Obesity and tendinopathies

Michele Abate
Department of Medicine and Sciences of Aging, University "G. d'Annunzio" Chieti, Pescara, Italy

Introduction and personal observations

Obesity is a world epidemic and an emerging risk factor for tendinopathies (1). Both load-bearing and non load-bearing tendons are frequently affected. Our group has recently added new data to the knowledge of this topic, with two complementary studies.
In the first study (2) we evaluated at which extent BMI can influence Achilles Tendon (AT) and Plantar Fascia (PF) thickness in type II diabetic patients.
Fifty-one participants with type II diabetes mellitus and eighteen controls were enrolled, and four groups were then considered: a) normoglycaemic-normal weight (BMI <25); b) diabetic subjects-normal weight (BMI <25); c) diabetic subjects-overweight (BMI 25 to 30); d) diabetic subjects-obese (BMI >30). By means of ultrasound evaluation, AT and PF thickness and their echotexture alterations were evaluated.
AT and PF thickness was increased in all diabetic subjects and a good correlation between BMI and PF was observed, while the relationship between BMI and AT was weaker.
In the second experiment (3) we assessed the prevalence of AT abnormalities in young normal weight and overweight amateur runners. Male runners and non-runners were recruited and, on the basis of BMI values, were divided in two subgroups: normal weight (BMI <25) and overweight (BMI >25).
AT thickness as well as tendon sonographic abnormalities and intratendinous microvessels were evaluated.
Overweight sedentary subjects showed AT thickness values significantly higher than normal weight sedentary subjects. Sonographic alterations and neovessels were more frequently observed in tendons of overweight participants and, among them, were significantly prevalent in runners.

Discussion

Our results, observed in two new experimental paradigms, confirm that overweight and obesity increase significantly the risk linked to some well known pathogenetic factors of tendinopathies, such as diabetes mellitus and overuse.
The mechanism by which obesity favours tendon degeneration is complex and can be ascribed both to systemic and local factors.
In obese subjects, bioactive peptides, released by adipose tissue, are provided of several activities on different mesenchimal cells, which may influence directly tendon structure. They can lead to a systemic state of chronic low-grade inflammation, influencing type II nitric oxide synthase, cytokines, prostanoids and metalloproteinases production (4). The failed tendon healing is furtherly "fed" by the migration of immune cells into adipose tissue with a decrease in their circulating levels and a reduced release of profibrotic factors.
To add complexity, obese subject frequently exhibit metabolic abnormalities, especially hyperglycemia, impaired glucose tolerance, and type II diabetes mellitus. In these pathological conditions, an increased cross-linking between collagen fibrils, mediated by Advanced Glycosilation End Products, and a state of low grade inflammation amplify tendon degeneration (5).
Besides the metabolic pattern, tendon overload/overuse must be taken into account. There is a threshold of loading frequency and magnitude that, once overcome, reverses tendon response from beneficial towards degenerative. An aberration in the proteoglycans metabolism, and an increased formation of degradation products and inflammatory molecules are implicated in the disease progression (6).
In the case of overweight subjects, modest increases in weight are amplified within the tendon, and especially weight-bearing tendons are exposed to higher load that leads to overuse tendinopathy.

Conclusions

Obesity is a strong risk factor for developing tendon disorders. Therefore, obese subjects, particularly those submitted to overuse, such as those practising sport activities, should be frequently monitored, being some sonographic abnormalities predictors of future symptomatic tendinopathy.

References

1. Gaida JE, Cook JL, Bass SL. Adiposity and tendinopathy. Disabil Rehabil 2008; 30(20-22):1555-1562.
2. Abate M, Schiavone C, Di Carlo L, Salini V. Achilles tendon and plantar fascia in recently diagnosed type II diabetes: role of body mass index. Clin Rheumatol 2012 Jul; 31(7):1109-1113.

3. Abate M, Oliva F, Schiavone C, Salini V. Achilles tendinopathy in amateur runners:role of adiposity. MLTJ 2012 Jan/Mar; 2(1):44-48.
4. Berry PA, Jones SW, Cicuttini FM, Wluka AE, Maciewicz RA. Temporal relationship between serum adipokines, biomarkers of bone and cartilage turnover, and cartilage volume loss in a population with clinical knee osteoarthritis. Arthritis Rheum 2011 Mar; 63(3):700-707.
5. DeGroot J. The AGE of the matrix: chemistry, consequence and cure. Curr Opin Pharmacol 2004; 4:301-305.
6. Abate M, Silbernagel KG, Siljeholm C et al. Pathogenesis of tendinopathies: inflammation or degeneration? Arthritis Res Ther 2009; 11(3):235.

Patellofemoral instability rehabilitation

Maria Grazia Benedetti, Elisa Brunelli
SC Physical and Rehabilitation Medicine, Rizzoli Orthopaedic Institute, Bologna, Italy

Patellar instability can be a difficult condition for clinicians to treat. The non operative treatment involves different steps (1).
Acute phase: a period of immobilization may be beneficial (2, 4). The goal of bracing is to restore proper alignment (3,5). If the patient has difficulty in activating the muscle because of pain, electrical stimulation can be effective in reducing swelling (1, 6).
Recurrent episodes: patella must be stabilized. Patella taping has been found to increase quadricipes muscle force (7), increase loading knee flexion response (8) and activate VMO earlier than VL during stairs (9, 10). For this joint, there is increasing evidence that closed chain training is more effective than open chain exercise (11, 12).
The patients need muscle training for VMO and the gluteal muscles. It is important to pay attention to the timing and intensity of the contraction of VMO relative to VL to improve distal control; in the same way it is important to enhance proximal control with gluteal training to improve stance phase of gait. The exercises should be done in front of a mirror to check the alignment and self-correcting (1). Some review investigates the early rehabilitation of patients after surgical intervention for patellofemoral instability (19-21). Some studies use postoperative knee braces to immobilize patients' knee, but there was a variation in the period of time (from 3 days to 4-6 weeks) (13-18). Moreover is very difficult to compare the bracing technique because the most common description is "knee immobilizer". It is not always possible to move joints immediately after surgery, but early motion is clearly desirable (22). An early goal of rehabilitation after surgery is to re-establish full knee extension. Return of passive flexion can be difficult for several reasons. The goal is to exceed 90° flexion within 6 week postoperatively (20). Concerning muscle strengthening, exercise should be started immediately after surgery. Studies have demonstrated that electrical stimulation alone and combined with voluntary exercise is superior to exercise alone in restoring strength and normal gait (23-26). Concerning optimal postoperative weight-bearing status, there were no obvious differences in outcome in the studies permitting immediate full weight bearing (16,18), compared to those of non weight-bearing (13-15,17), or partial weight-bearing patient (27). Fithian DC et al. (20) say that these patients can expect to return to unrestricted activities 6 months to 1 year postoperatively. Concerning outcome score, studies have shown that Lysholm, Fulkerso and Kujala scores are valid and reliable for patellar instability patients, (28). Several studies have compared surgical versus non-surgical treatment of patellofemoral instability, but in a recent review (29) it is concluded that there is insufficient evidence to confirm any significant difference in outcome between surgical or non-surgical initial management of people following primary patellar dislocation, and none examining this comparison in people with recurrent patellar dislocation.

References

1. McConnell J. Rehabilitation and nonoperative treatment of patellar instability. Sports Med Arthrosc 2007 Jun; 15(2):95-104.
2. Mäenpää H et al. Patellar dislocation. The long-term results of nonoperative management in 100 patients. Am J Sports Med. 1997;25:213-217.
3. Shellock FG. Effect of a patella-stabilizing brace on lateral subluxation of the patella: assessment using kinematic MRI. Am J Knee Surg 2000; 13(3):137-142.
4. Shellock FG, Mink JH, Deutsch AL, Fox J, Molnar T, Kvitne R, Ferkel R. Effect of a patellar realignment brace on patellofemoral relationships: evaluation with kinematic MR imaging. J Magn Reson Imaging. 1994; 4(4):590-594.
5. Shellock FG, Mink JH, Deutsch AL, Molnar T. Effect of a newly designed patellar realignment brace on patellofemoral relationships Med Sci Sports Exerc 1995 Apr; 27(4):469-72.
6. Malanga G, Craig C Young. Patellar Injury and Dislocation Treatment & Management (http://emedicine.medscape.com/article/90068-overview).
7. Conway A, Malone T, Conway P. Patellar alignment/tracking alteration: effect on force output and perceived pain. Isokinet exerc Sci 1992; 2:9-17.

8. Powers CM, Landel R, Sosnick T, Kirby J, Mengel K, Cheney A, Perry J. The effects of patellar taping on stride characteristics and joint motion in subjects with patellofemoral pain. J Orthop Sports Phys Ther 1997; 26(6):286-291.
9. Gilleard W, McConnell J, Parsons D. The effect of patellar taping on the onset of vastus medialis obliquus and vastus lateralis muscle activity in persons with patellofemoral pain. Phys Ther 1998; 78(1):25-32.
10. Cowan SM, Bennell KL, Hodges PW. Therapeutic patellar taping changes the timing of vasti muscle activation in people with patellofemoral pain syndrome. Clin J Sport Med 2002; 12(6):339-347.
11. Stensdotter AK, Hodges PW, Mellor R, Sundelin G, Häger-Ross C. Quadriceps activation in closed and in open kinetic chain exercise. Med Sci Sports Exerc 2003; 35(12):2043-2047.
12. Escamilla RF, Fleisig GS, Zheng N, Barrentine SW, Wilk KE, Andrews JR. Biomechanics of the knee during closed kinetic chain and open kinetic chain exercises. Med Sci Sports Exerc 1998; 30(4):556-569.
13 Nomura E, Horiuchi Y, Kihara M. A mid-term follow-up of medial patellofemoral ligament reconstruction using an artificial ligament for recurrent patellar dislocation. Knee 2000; 1; 7(4):211-215.
14. Drez D Jr, Edwards TB, Williams CS. Results of medial patellofemoral ligament reconstruction in the treatment of patellar dislocation. Arthroscopy 2001; 17(3):298-306.
15. Deie M, Ochi M, Sumen Y, Adachi N, Kobayashi K, Yasumoto M. A long-term follow-up study after medial patellofemoral ligament reconstruction using the transferred semitendinosus tendon for patellar dislocation Knee Surg Sports Traumatol Arthrosc 2005; 13(7):522-528.
16. Steiner TM, Torga-Spak R, Teitge RA. Medial patellofemoral ligament reconstruction in patients with lateral patellar instability and trochlear dysplasia. Am J Sports Med 2006 Aug; 34(8):1254-1261.
17. Nomura E, Inoue M. Hybrid medial patellofemoral ligament reconstruction using the semitendinous tendon for recurrent patellar dislocation: minimum 3 years' follow-up. Arthroscopy 2006; 22(7):787-793.
18. Cossey AJ, Paterson R. A new technique for reconstructing the medial patellofemoral ligament. Knee 2005; 12(2):93-98.
19. Smith TO, Russell N, Walker J. A systematic review investigating the early rehabilitation of patients following medial patellofemoral ligament reconstruction for patellar instability. Critical Reviews in Physical and Rehabilitation Medicine 2007; 19(2):79-95.
20. Fithian DC, Powers CM, Khan N. Rehabilitation of the knee after medial patellofemoral ligament reconstruction. Clin Sports Med 2010; 29(2):283-290.
21. Fisher B, Nyland J, Brand E, Curtin B. Medial patellofemoral ligament reconstruction for recurrent patellar dislocation: a systematic review including rehabilitation and return-to-sports efficacy. Arthroscopy 2010; 26(10):1384-1394.
22. Häggmark T, Eriksson E. Cylinder or mobile cast brace after knee ligament surgery. A clinical analysis and morphologic and enzymatic studies of changes in the quadriceps muscle. Am J Sports Med 1979; 7(1):48-56.
23. Anderson AF, Lipscomb AB. Analysis of rehabilitation techniques after anterior cruciate reconstruction. Am J Sports Med 1989; 17(2):154-160.
24. Snyder-Mackler L, Ladin Z, Schepsis AA, Young JC. Electrical stimulation of the thigh muscles after reconstruction of the anterior cruciate ligament. Effects of electrically elicited contraction of the quadriceps femoris and hamstring muscles on gait and on strength of the thigh muscles. J Bone Joint Surg Am 1991; 73(7):1025-1036.
25. Wigerstad-Lossing I, Grimby G, Jonsson T, Morelli B, Peterson L, Renström P. Effects of electrical muscle stimulation combined with voluntary contractions after knee ligament surgery. Med Sci Sports Exerc 1988; 20(1):93-98.
26. Beynnon BD, Johnson RJ, Abate JA, Fleming BC, Nichols CE. Treatment of anterior cruciate ligament injuries, part 2. Am J Sports Med 2005; 33(11):1751-1767.
27. Schöttle PB, Fucentese SF, Romero J. Clinical and radiological outcome of medial patellofemoral ligament reconstruction with a semitendinosus autograft for patella instability. Knee Surg Sports Traumatol Arthrosc 2005 Oct; 13(7):516-521.
28. Paxton EW, Fithian DC, Stone ML, Silva P The reliability and validity of knee-specific and general health instruments in assessing acute patellar dislocation outcomes. Am J Sports Med 2003 Jul-Aug; 31(4):487-492.
29. Hing CB, Smith TO, Donell S, Song F. Surgical versus non-surgical interventions for treating patellar dislocation. Cochrane Database Syst Rev 2011; 9,(11):CD008106.

The tendon niche

Anna C. Berardi

Laboratory of Stem Cells, Department of Transfusion Medicine, Pescara Spirito Santo Hospital, Pescara, Italy

The concept of the stem cell niche was firstly proposed theoretically by Schofield exactly 30 years ago in the context of hematopoietic stem cells (1).

The niche is defined as a specialized microenvironment surrounding stem cells that provides support and signals regulating cell number, self-renewal and cell fate of its resident stem cells. The cell outside of the niche differentiates, while the cell that maintains contact within the niche can go on to self-renew. Intrinsic fate determinants are unequally parti-

tioned during division and thereby regulate the choice to self-renew or to differentiate. Niches are restricted and specialized tissue microenvironments that integrate local and systemic signals for the regulation and maintenance for resident stem cells (2). The stem cell niche consists of cellular components and a cellular component, an extracellular matrix and molecular signals. Mis- or de-regulation of stem cell renewal and number via loss of the local niche microenvironment can lead to cancerous growths and premature ageing. Furthermore, current findings suggest that the age-imposed biochemical changes in the niches of tissue stem cells inhibit performance of this regenerative pool, which leads to the decline of tissue maintenance and repair. If true, slowing down stem cell and niche aging, thereby promoting tissue regeneration, could slow down the process of tissue and organismal aging (3, 4).
To date only Yanking Bi et al. showed, that human and mouse tendons harbor a unique cell population, termed tendon stem/progenitor cells (TSPCs), that has universal stem cell characteristics such as clonogenicity, multipotency and self-renewal capacity. The isolated TSPCs could regenerate tendon-like tissues after extended expansion *in vitro* and transplantation in vivo. They show that TSPCs reside within a unique niche predominantly comprised of an extracellular matrix (ECM), and identify biglycan (Bgn) and fibromodulin (Fmod) as two critical components that organize this niche (5). Previous studies have already demonstrated that ECM is an essential component of normal stem cell niche. ECM plays multiple roles in maintaining stem cell properties. ECM anchorage restricts stem cells in the niche and thus allows them to be exposed to paracrine and cell–cell contact signals that are essential for maintaining stem cell properties. Anchorage is also important for orienting the mitotic spindle and makes it possible for stem cells to undergo asymmetric cell division, which is essential for stem cell self-renewal and generation of daughter cells that are destined to undergo cell differentiation. The exact mechanism whereby ECM anchorage controls asymmetric cell division remains unclear, although one possibility is to allow cytoplasmic cell fate determinants to be differentially distributed between the daughter cells. In the presence of abnormal ECM or loss of ECM contact, stem cell properties fail to be maintained and undergo symmetric cell division instead, leading to an overexpansion of the stem cell pool. Abnormal changes of the ECM can also disrupt the cellular differentiation process, resulting in loss of differentiation and an increase of stem/progenitor cells (6).
The control and regulation of stem cells tendon and their niche are remaining challenges for cell therapy and regenerative medicine. These advances are important for both, the basic knowledge of stem cell tendon regulation and their practical translational applications into clinical medicine.

References

1. Schofield R. Blood Cells 1978.
2. Voog J, Jones DL. Cell Stem Cell 2010.
3. Choi J, Artandi S. Cell Stem Cell 2009.
4. Arai F, Suda T. StemBook (Internet). 2008.
5. Yanking Bi et al. Nature Medicine 2007.
6. Mecham RP. Curr Protoc Cell Biol 2012.

Pre participation screening in athletes with myopathies

Brancaccio Paola
Seconda Università di Napoli, Department of Experimental Medicine-Sport Medicine, Center of Excellence of Cardiovascular Disease, Napoli, Italy

Stiffness, muscle soreness and pain are normal features of physical training: often both athletes and coaches pay little attention to these symptoms. However, if they are resistant to rest and massage, or recur too frequently, they should prompt a diagnostic workup. Sometimes, these are the only signs of a silent myopathy. Kaar et al. diagnosed FSHD in a baseball player complaining of shoulder pain (1) even though long-term data were not available, authors studied the effect of exercise in FSHD (2). Strength training seems to be safe for these patients, even though the evidence for routine exercise prescription is still scanty (3, 4). In other athletes, biopsy-proven myopathies were the cause of unexplained exercise impairment (5). In these instances, evaluation of CK at rest and after exertion could be a simple and non invasive method to guide diagnosis. Creatine kinase (CK) is located in the sarcolemma and mithocondrial intermembrane space of healthy muscle cells. It catalyzes the movement of phosphate from phosphocreatine to adenosine diphosphate, forming adenosine triphosphate (ATP) and creatine (6). Elevated plasma levels of CK are observed in various neuromuscular conditions as a result of muscle damage and necrosis. A sustained increase in serum CK is often the results of myopathies, with levels depending on severity and course of the disease (7). Persistently high serum CK levels at rest may be encountered in individuals with idiopathic hyperCKemia (8), but it is also possible that subjects with abnormal increase in serum CK after exercise may have an unrecognised subclinical myopathy (9). Early diagnosis is important because it is difficult to evaluate the risks of sporting activities: it is possible that repeated intense prolonged exercise may produce negative effects (10), as it does not induce the physiological muscle adaptations to physical training given the continuous loss of muscles proteins. The assessment of the serum CK levels at rest is a simple and non invasive method to identify subjects with muscle disease without exercise intolerance in a preclinical stage of pathology. Often, the high serum levels of CK are considered normal in asymptomatic athletes because clinicians know that they are influenced by training, while they can be a sign of silent disease. Indeed, CK serum levels are almost always elvated even in the preclinical stages of frank myopathy (11).

Concluding remarks

It is probably safe to counsel athletes with suspected myopathy to continue to undertake physical activity at a lower intensity, so as to prevent muscle damage from high intensity exercise, and allow ample recovery to favour adequate recovery. Accurate history taking and clinical examination should help to clarify whether more invasive investigations, including muscle biopsies, should be considered. The pre participation screening aimed at the moment to mainly exclude cardiovascular and respiratory impairment, should include clinical or laboratory examinations to determine the heath's state of skeletal-muscle apparatus.

References

1. Kaar S, Hazard D, Miller BS. Facioscapulohumeral muscular dystrophy presenting as shoulder pain in a baseball player. Arthroscopy. 2005 Sep;21(9):1145.
2. Lue YJ, Chen SS. The strength and functional performance in patients with facioscapulohumeral muscular dystrophy. Kaohsiung J Med Sci 2000;16:248-54.
3. van der Kool EL, Vogels OJ, van Asseldonk RJ, Lindeman E, Hendriks JC, Wohigemuth M, van der Maarel SM, Padberg GW. Strength training and albuterol in facioscapulohumeral muscular dystrophy. Neurology 2004 24;63:702-8.
4. van der Kool EL, Lindeman E, Riphagen I. Strength training and aerobic exercise training for muscle disease. Cochrane Database Syst Rev 2005 25;CD003907.
5. Cavanagh NP, Preece MA. Calf hypertrophy and asimmetry in female carriers of Xlinked Duchenne muscular dystrophy: an over-diagnosed clinical manifestation. Clin Genet 1981; 20(2): 168-72.
6. Wyss M, Kaddurah-Daouk R. "Creatine and creatine metabolism" Physiol Rev 2000; 80:1107-1213.
7. Engel A.G., Franzini-Armstrong C. "Myology" Mc Graw Hill third edition vol II p. 978 2004.
8. Capasso M, De Angelis MV, Di Muzio A, Scarciolla O, Pace M, Stuppia L, Comi GP, Uncini A. Familial idiopathic hyperkemia: an unrecognised condition Muscle Nerve 2006; 33:760-5ù.
9. P.Brancaccio, F.M. Limongelli, N. Maffulli. Monitoring of serum enzymes in sport Br J Sports Med 2006; 40:96-97.
10. L. Vercelli, T. Mongini, R. Tupler, N. Oliviero, E. Vittonatto, R. Mutani, L. Palmucci. "Selective hypotrophy of pectoralis muscle reveals an atypical case of FSHD" Basic and Applied Myology 2006; 16: 62.
11. Miyoshi K, Kawai H, Iwasa M, Kusaka K, Nishino H. Autosomal recessive distal muscular dystrophy as a new type of progressive muscular dystrophy. Seventeen cases in eight families including an autopsied case. Brain 1986; 109:31-54.

Physics and technology behind ESW

Domenico Caputo
Department of Information Engineering, Electronics and Telecommunications, University of Rome "La Sapienza", Rome, Italy

The first observation of the effect of shock waves (SW) on human body have been observed during World War II in the crews of tanks hit by bombs and in castaways whose lung tissue were injured due to the explosion of water bombs even though visible damage were not detected (1). Since then the interaction between shock waves and biological tissue has been extensively investigated leading to SW application in urology (2) and more recently in musculoskeletal disorders using extracorporeal SW (ESW) (3).
The aim of this talk is to briefly review the physical principles and technology behind the SW and ESW. Shock waves are acoustic waves characterized by a sudden variation of pressure that propagates in the space with a speed greater than the speed sound in the same medium. In a fixed point of the space, the SW is described by a pressure rise up to 200 MPa in less than 10 ns, followed by a decrease to atmospheric pressure in about 1µs and to a negative pressure around -10 MPa in about 5 µs. Therefore, each space point experiences both a compressive and a tensile stress when the SW propagates. As a consequence of the SW time evolution, the SW frequency spectrum covers the 100 kHz-1 MHz range, with a peak around 300 kHz. At fixed time, the SW has a Gaussian-like spatial distribution and is charac-

terized by an ellipsoidal focal zone where the pressure perturbation is concentrated. Diameter and length of this region depend on the type and focusing mechanism of each specific SW generator. The time evolution determines also the energy carried by the SW and together with the medium acoustic characteristics (i.e. the acoustic impedance given by the product of the medium density and the speed of propagation) the propagation properties of the SW that in turn determine the effect of SW on human body.
From a technological point of view, even though different kinds of generators are available all of them rely on the following physical principles:
1. transformation of electrical energy into mechanical energy;
2. generation of an abrupt pressure variation;
3. focusing of the energy into the therapeutic focal volume at a precise focal depth.

In order to achieve a good acoustic coupling shock waves are generated depositing energy in a propagation medium whose acoustic impedance is similar to the soft tissue in the body. In this way the waves enter the human body without significant energy loss. During propagation, shock waves experiment reflection, diffraction, transmission and attenuation at the interfaces between the different tissues.
The various SW generators are based on the following technologies (4):
a) electrohydraulic: a high voltage is applied between two electrodes, deepen in a medium, to induce an electrical discharge that generates a shock wave that is focused by an ellipsoidal reflector. Therefore, the electrodes are positioned in the first focus while the focal zone will be located in the second focus of the ellipse;
b) piezoelectric: a voltage is applied to an array of piezoelectric crystals placed in an half spherical container. The crystals transform the electrical energy in crystal vibrations generating an acoustic wave for each piezoelectric element. All the acoustic waves are focused in the center of the sphere to achieve a shock wave;
c) electromagnetic: an electric coil is excited by an electric pulse, which generates an electromagnetic field which in turn induces vibrations in a metal plate positioned closed to the coil. Depending on the relative position between the coil and the metal plate, the acoustic waves are focused, to form a shock wave, either by a parabolic reflector or an acoustic lens.

A comparison of the different generator performances in terms of focal sizes and energy density will be provided during the talk.

References

1. Thiel M. Application of shock waves in medicine. Clin Orthop 2001; 387:18-21.
2. Cleveland RO, McAteer JA. Smith's Textbook of Endourology. Wiley-Blackwell 2012; 38:316-332.
3. Chang G. An Overview of Shock Wave Therapy in Musculoskeletal Disorders. Med J 2003; 220-231.
4. Ogden JA. Tóth-Kischkat A, Schultheiss R. Principles of Shock Wave Therapy. Clin Orthop and Related Research 2001; 387:8-17.

The LHB pathology associated or not with RCTS. Choosing the most appropriate treatment

Fabio Cautiero, Raffaele Russo
UOC of Orthopaedics and Traumatology, Pellegrini Hospital, Naples, Italy

Tendinopathy of the long head of the biceps is a common cause of shoulder pain.
We can found the articular portion of Long Head Biceps Tendon (LHBT) in the Interval Rotator Cuff, between the anterior border of supraspinatus tendon and the superior third of subscapularis.
It's stabilized into bicipital groove by a capsulo-ligamentous structure called "pulley system", created by coracohumeral ligament, superior glenohumeral ligament and fibers of supraspinatus and subscapularis tendon.
The role of the LHBT has yet to be defined. Recent biomechanical and electromyogram studies suggested that decrease the anterior-posterior and superior-inferior translations, limit internal and external rotations and plays an active compensatory role in the unstable shoulder.
Lesions of the LHBT are frequent causes of shoulder pain and disability and can be related to a different etiologic pathologic process, traumatic, congenital and degenerative.
The lesion of the pulley system leads to instability of the articular portion of LHBT, with pain and discomfort in some positions of the shoulder, and gives rise to various disorders of tendon from fraying, tendinitis until the complete rupture.
The injury of the pulley may be caused by trauma, repeated micro-trauma (as in the antero-superior impingement) or degenerative factors.
Bennet and later Habermeyer have proposed a classification of pulley lesion related to location and severity of the lesion.

The instability of LHBT can be associated or not with cuff tears and may lead over time to damages of cartilage, cuff tear or extension of existing lesions.
The LHB tendon can be also the location of tendonopathy primary due to trauma, congenital abnormalities or a degenerative lesion as the “hourglass hypertrophy”, commonly found in tears cuff degenerative.
Diagnosis of pulley lesion can be very difficult, we can use some clinical test and instrumental investigations as the MRI but often the definitive diagnosis is made by arthroscopy. Greater tear with dislocation of the tendon can be easier to identify.
The surgical treatment according to the recent Literature may range from simple debridment of the lesion (if involving less than 25%), to repair of the pulley until the tenotomy followed or not by tenodesis.
Although reconstruction of the pulley is a anatomical procedure with the aim of leaving a mobile tendon, some authors have reported frequent complications: persistent pain in 12-30%, failure 10% and a static tendon into the groove in 50% of patients.
Tenotomy is a easy and quick surgical procedure, can be performed in arthroscopy and the patients can return to normal activities very soon. Some author reported a more frequent Popey sign (3% to 70%) while conflicting data are reported about the effect on muscle strength. In addition, although tenotomy of LHB is discouraged in pseudo-paralitic shoulder, is not demonstrated a statistically significant difference with tenodesis in the proximal migration of the humeral head.
Tenodesis (TD) can be performed in arthroscopy or in open surgery, the tendon can be locked at the bicipital groove (proximal TD) or at sub-pectoralis zone (distal TD), and finally we can use anchors or screws to fix tendon at the bone (bony TD) or suturing the tendon to the interval, conjoined tendons or cuff (soft tissues TD).
In our department we perform the TT if ther is a lesion great then 25%, presence of tendinopathy, patient older than 50 y, obese, women. In the other case we prefer the tenodesis at sulcus with a absorbable anchor.
In isolated pulley lesion and in young patients with high demand and sport practice, we repair the pulley.

References

Bennett WF. Arthroscopic bicipital sheath repair: two-year follow-up with pulley lesions. Arthroscopy 2004; 20(9):964-973. PubMed PMID: 15525930.
Boileau P, Baqué F, Valerio L, Ahrens P, Chuinard C, Trojani C. Isolated arthroscopic biceps tenotomy or tenodesis improves symptoms in patients with massive irreparable rotator cuff tears. J Bone Joint Surg Am. 2007; 89(4):747-757. PubMed PMID: 17403796.
Chen CH, Hsu KY, Chen WJ, Shih CH. Incidence and severity of biceps long head tendon lesion in patients with complete rotator cuff tears. J Trauma. 2005; 58(6):1189-1193. PubMed PMID: 15995469.
Franceschi F, Longo UG, Ruzzini L, Papalia R, Rizzello G, Denaro V. To detach the long head of the biceps tendon after tenodesis or not: outcome analysis at the 4-year follow-up of two different techniques. Int Orthop 2007; 31(4):537-545. Epub 2006 Sep 1. PubMed PMID: 16947053; PubMed Central PMCID: PMC2267623.
Frost A, Zafar MS, Maffulli N. Tenotomy versus tenodesis in the management of pathologic lesions of the tendon of the long head of the biceps brachii. Am J Sports Med 2009; 37(4):828-833. Epub 2008 Sep 1. Review. PubMed PMID: 18762669.
Gerber C, Sebesta A. Impingement of the deep surface of the subscapularis tendon and the reflection pulley on the anterosuperior glenoid rim: a preliminary report. J Shoulder Elbow Surg 2000; 9(6):483-490. PubMed PMID: 11155300.
Habermeyer P, Magosch P, Pritsch M, Scheibel MT, Lichtenberg S. Anterosuperior impingement of the shoulder as a result of pulley lesions: a prospective arthroscopic study. J Shoulder Elbow Surg 2004; 13(1):5-12. Pub Med PMID: 14735066.
Itoi E, Newman SR, Kuechle DK, Morrey BF, An KN. Dynamic anterior stabilisers of the shoulder with the arm in abduction. J Bone Joint Surg Br 1994; 76(5):834-6. PubMed PMID: 8083280.
Kim SH, Ha KI, Kim HS, Kim SW. Electromyographic activity of the biceps brachii muscle in shoulders with anterior instability. Arthroscopy 2001; 17(8):864-868. PubMed PMID: 11600985.
Koh KH, Ahn JH, Kim SM, Yoo JC. Treatment of biceps tendon lesions in the setting of rotator cuff tears: prospective cohort study of tenotomy versus tenodesis. Am J Sports Med 2010; 38(8):1584-1590. Epub 2010 Jun 15. PubMed PMID: 20551285.
Lafosse L, Reiland Y, Baier GP, Toussaint B, Jost B. Anterior and posterior instability of the long head of the biceps tendon in rotator cuff tears: a new classification based on arthroscopic observations. Arthroscopy 2007; 23(1):73-80. PubMed PMID: 17210430.
Le Huec JC, Schaeverbeke T, Moinard M, Kind M, Diard F, Dehais J, Le Rebeller A. Traumatic tear of the rotator interval. J Shoulder Elbow Surg 1996; 5(1):41-46. PubMed PMID: 8919441.
Maier D, Jaeger M, Suedkamp NP, Koestler W. Stabilization of the long head of the biceps tendon in the context of early repair of traumatic subscapularis tendon tears. J Bone Joint Surg Am 2007; 89(8):1763176-9. PubMed PMID: 17671016.
McClelland D, Bell SN, O’Leary S. Relocation of a dislocated long head of biceps tendon is no better than biceps tenodesis. Acta Orthop Belg 2009; 75(5):595-598. PubMed PMID: 19999869.

Russo R, Cautiero F, Giudice G, Ciccarelli M, Visconti V. Arthroscopic repair of rotator cuff tears using absorbable anchors with a single-row technique. J Orthop Surg (Hong Kong) 2010; 18(3):332-337. PubMed PMID: 21187546.
Shank JR, Singleton SB, Braun S, Kissenberth MJ, Ramappa A, Ellis H, Decker MJ, Hawkins RJ, Torry MR. A comparison of forearm supination and elbow flexion strength in patients with long head of the biceps tenotomy or tenodesis.
Arthroscopy 2011; 27(1):9-16. Epub 2010 Oct 29. PubMed PMID: 21035992.
Walch G, Nové-Josserand L, Boileau P, Levigne C. Subluxations and dislocations of the tendon of the long head of the biceps. J Shoulder Elbow Surg 1998; 7(2):100-108. PubMed PMID: 9593086.
Walch G, Edwards TB, Boulahia A, Nové-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elbow Surg 2005; 14(3):238-246. PubMed PMID: 15889020.
Werner A, Mueller T, Boehm D, Gohlke F. The stabilizing sling for the long head of the biceps tendon in the rotator cuff interval. A histoanatomic study. Am J Sports Med 2000; 28(1):28-31. PubMed PMID: 10653540.
Youm T, El Attrache NS, Tibone JE, McGarry MH, Lee TQ. The effect of the long head of the biceps on glenohumeral kinematics. J Shoulder Elbow Surg 2009; 18(1):122-129. Epub 2008 Sep 16. PubMed PMID: 18799325.

Pelvic floor impairment associated with pregnancy and childbirth

Nicola Comi
Gynecologic Unit, BMI The London Independent Hospital, London U.K.

Pregnancy and vaginal delivery can negatively affect the pelvic floor function.
Pelvic floor disorders (PFDs) include urinary symptoms (urgency, urge and stress incontinence), fecal incontinence and pelvic organ prolapse.
These disorders affect one-third of adult women in the United States, with substantial impact on their quality of life. The pelvic viscera are supported by the pelvic floor, which is composed of muscle, fascia and ligamentous support.

Urogenital symptoms occur in almost all the women during pregnancy:

- Frequency and urgency symptoms can start from 12 weeks and remain stable during the pregnancy;
- Urinary incontinence symptoms increase with gestational age;
- SUI is significantly prevalent after vaginal delivery, mainly after instrumental delivery; but the presence of SUI in early pregnancy give an increased risk of SUI one year postpartum both in women after vaginal delivery and women who underwent a caesarean section.

Fecal incontinence is mainly related to anal sphincter lesions.

Pelvic organ prolapse is a protrusion of a pelvic organ beyond its normal boundaries. The pelvic floor includes the levator ani, internal obturator and piriform muscles and superficial and deep perineal muscles. The levator ani (which is in two parts - pubococcygeal and ileococcygeal) is covered by pelvic fascia and arises from the pelvic surface of the pubic bone and posteriorly from the ischial spine.
These muscles act and support for the pelvic viscera and as sphincter for the rectum and the vagina. Contraction of the pubococcygeal muscles will also arrest the urinary stream. The pelvic floor fascia is divided into that covering the pelvic floor muscles and the endopelvic fascia which are connections between muscles and pelvic viscera.
The endopelvic fascia is divided in the following: pubocervical ligament, pubourethral ligament, cardinal ligament and uterosacral ligament.
The pubourethral ligament maintains elevation of the bladder neck and prevents excess posterior displacement of the urethra. It may facilitate micturition and is important in maintaining continence. A pelvic organ or structure prolapse may include: bladder (cystocele), urethra (urethrocele), uterus (uterine descent), bowels (enterocele - rectocele).
The aetiology of a prolapse may be congenital or acquired and childbirth with trauma and denervation is the most important acquired factor.
Both constitutional and obstetric factors are involved in impairment of pelvic floor muscle function (PFM) postpartum. Reduced PFM strength is associated with prolapse in the postpartum period.

Management

- Conservative: Behavioural therapy - Pelvic Floor Exercises - Physiotherapy- Ring pessary insertion.
- Surgical: TVT - sacrohysteropexy - vaginal hysterectomy - anterior and posterior vaginal repair.

- Medical: Antimuscarinics (e.g. Oxybutinin, Tolterodine, Solifenacin) - Antidepressant (Imipramine hydrochloride) - PTNS (Percutaneous tibial nerve stimulation).

Case report

Multipara with grade 2 uterine prolapse treated with ring pessary in pregnancy and laparoscopic sacrohysteropexy after delivery.

References

Agur W et al. Does antenatal pelvic floor muscle training affect the outcome of labour? A randomised controlled trial. International Urogynecology Journal and Pelvic Floor Dysfunction 2008; 19:1,85-88.
Allen RE et al. Pelvic floor damage and childbirth: a neurophysiological study. British Journal of Obstetrics and Gynaecology 1990; 97:9,770-779.
Bump RC et al. Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction. American Journal of Obstetrics and Gynaecology 1991; 165:2, 322-327.
Carroli G, Belizan J. Episiotomy for vaginal birth.Cochrane Database Systematic Reviews 2000; (2):CD000081.
Chiarelli P, Campbell E. Incontinence during pregnancy. Prevalence and opportunities for continence promotion. Australian and New Zealand Journal of Obstetrics and Gynaecology 1997; 37:1,66-73.
Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. Obstetrics and Gynecology 2005; 106: 4,707-712.
Geneeskunde Proefschriften; 2006.
Groutz A et al. (2004) Caesarean section: does it really prevent the development of postpartum stress urinary incontinence? A prospective study of 363 women one year after their first delivery. Neurourology and Urodynamics 2004; 23:1, 2-6.

Talalgia

Maria Conforti
Physiatrist and Sports Medicine in Bergamo, Directorate General INAIL Milan, Italy

Italian Sport Medicine can claim 35 years of experience and therefore can today express an authoritative opinion about granting sport eligibility to patients with rheumatic ilnesses like spondyloarthritis, choosing the right sport in this state of non perfect health and aids to play sport. The symptoms for most patients with spondyloarthritis (AS) begin in the second to fourth decades of life, a typical age for competitive sports activity. There is a mean delay of five to eight years between onset of symptoms and diagnosis of AS. This is due to the fact that radiographic sacroiliitis is delayed and that the first symptom is often peripheral enthesitis with talalgia and not pain in the axial skeleton. Talalgia is defined as spontaneous or evoked pain at the posterior part of the heel (along the Achilles tendon and at its insertion, or in the area of the sub-Achilles bursa) or at the insertion the plantar fascia on the calcaneus.
Talalgia in AS caused on weight bearing, decreased slowly weightless, and it is evoked by minimal local pressure.
In each patient, the presence of valgus or varus deformities of the hindfoot or in forefoot and soft tissue swelling (Achilles tendon, sub-Achilles bursa, or the plantar fascia) and the articular mobility of the foot joints are recorded.
Most of the session should be dedicated to collecting history and to cataloging the aching joints.
Higher coefficients of correlation have been found with BASDAI rather than DFI Questionnaires regarding the signs of illness activity. BASDAI have to be filled in during the SPORT medical visit.
Conservative medical treatment and not vigorous physical activity involving strenuous running and jumping or contact are considered as an important part of treatment for AS in order to counteract musculoskeletal system atrophy, joint dysfunction, loss of coordination and to avoid the secondary damage from immobility. Heel surgery is contraindicated during the inflammation phase, since it may cause local aggravation and risk of ankylosis of the talocalcaneal articulation.

References

de Jong Z, Munneke M, Zwindermann AH, Kroon HM, Ronday KH, Lems WF,et al. Long term hight intensity exercise and damage of small joints in rheumatoid arthritis.
Ann Rheum Diseases 2004; 63:1399-1405.
Harper BE, Reveille JD. Spondyloarthritis: Clinical Suspicion, Diagnosis, and Sports.

Published in final edited form as: Curr Sports Med Rep. 2009; 8(1): 29-34. doi: 10.1249/JSR.0b013e3181967ac6 Curr Sports Med Rep. Author manuscript; available in PMC 2010.
Kraag G, Stokes B, Groh J, Helewa A, Goldsmith CH. The effects of comprehensive home physiotherapy and supervision on patients with ankylosing spondylitis-an 8-month followup.
J Rheumatol. 1994; 21(2):261-263. Department of Medicine (Rheumatology), Ottawa Civic Hospital, University of Ottawa, ON, Canada.
Sweeney S, Taylor G, Calin A. The effect of a home based exercise intervention package on outcome in ankylosing spondylitis: a randomized controlled trial. J Rheumatol 2002; 29(4):763-766.
Royal National Hospital for Rheumatic Diseases, Bath, United Kingdom.

Ultrastructural modifications of myotendinous junction in hind-limb suspended rats

Davide Curzi[1], Davide Lattanzi[1], Stefano Ciuffoli[1], Sabrina Burattini[1], James G. Tidball[2], Elisabetta Falcieri[1,3]
[1]*Department of Earth, Life and Environmental Sciences, University of Urbino "Carlo Bo", Urbino, Italy*
[2]*Department of Integrative Biology and Physiology, University of California, Los Angeles, CA, USA*
[3]*Institute of Molecular Genetics and Rizzoli Orthopaedic Institute, Bologna, Italy*

At ultrastructural level, myotendinous junction (MTJ) reveals several tendon interdigitations penetrating in muscle mass (1). We recently demonstrated that MTJ changes occur as an adaptation to exercise-induced tension increase. In particular, branching of finger-like processes increases, so enlarging the whole tendon-muscle surface area and, consequently, allowing a better tension resistance (2). The aim of this study is to analyze MTJ behavior in the atrophic condition, generated by disuse, and after particular rehabilitation protocols. In this work we used twenty male albino Sprague-Dawley rats assigned randomly and equally to one of five groups: 1) control, saline injected (CTRL); 2) hindlimb suspended, saline injected (HS); 3) hindlimb suspended and exercised (HS+Ex); 4) hindlimb suspended, GH injected (HS+GH); 5) hindlimb suspended, GH injected and exercised (HS+GH+Ex). The exercise protocol consisted of three daily bouts of 10 repetitions to ladder climbing (85° incline) with an additional 50% animal body weight attached to their tails. The GH, equivalent to a replacement dose in hypophysectomized rats, was supplied in two daily intraperitoneal injections. After sacrifice, MTJs of plantaris muscle were withdrawn, fixed in 1.4% glutaraldehyde in 0.2M sodium cacodylate and processed as previously described (3, 4). The contact between muscle and tendon was measured using "Image J" software that calculated the base (B) and "perimeter" (P) of tendon finger-like processes at MTJ level (5). After 5 days of suspension, skeletal muscle highlights signs of atrophy in response to decreased mechanical loading (6). The P/B ratio decreases from 6.39 in CTRL to 3.92 in HS. After suspension, the three different rehabilitation protocols increase this P/B ratio to 4.18 in HS+GH, 5.25 in HS+Ex and 7.3 in HS+GH+Ex, respectively. In conclusion, along with muscle atrophy features, evident ultrastructural changes occur at MTJ organization level, as an adaptation to muscle unloading. The present investigation indicates that a combination of daily resistance exercise and GH treatment was capable of maintaining muscle-tendon interface. For investigating the causes of the decrease in P/B ratio in HS, we measured the finger-like process extension. HS rats showed a significant marked decrease -from 1.84μm to 0.97μm-in the mean of interdigitation extension respect to CTRL. So it is possible to explain the reduction of MTJ interface area in HS rats with a decrease in the finger-like process length. Therefore, disuse, as well as exercise, not only affect muscle, but MTJ too.

References

1. Ciena AP et al. Microsc Res Tech 2012; 75(9):1292-1296.
2. Curzi D et al. Eur J Histochem 2012; 56(2):e19.
3. Tidball JG, Quan DM. J ApplPhysiol 1992; 73(1):59-64.
4. D'Emilio A et al. Histol Histopathol 2010; 25(1):21-32.
5. De Palma L et al. Rom J Morphol Embryol 2011; 52(1):105-109.
6. Linderman JK et al. Am J Physiol 1994; 267(2 Pt2):R365-371.

Other fields of clinical applications of extracorporeal shock waves

Cristina D'Agostino
Extracorporeal Shock Wave Unit, Istituto Clinico Humanitas, Rozzano, Italy

ESW have known in last decade a wide diffusion in the field of musculoskeletal disorders, both for soft tissuesd iseases and bone healing disturbances.
Experimental studies and clinical trials have been clarified, in recent years, that the clinical results (through mechanotrasduction) are related, other than their anti - inflammatory and analgesic effects, to a general "throphic" effect. This effect can be considered effectively as a regenerative potential, mainly due to anangiogenicaction and stimulation of mesenchimal stem cells to mitosis and tissue - specific differentiation.
From the clinical point of view, these recent "discoveries" have widened the field of application of ESW therapy, towards new, interesting and promising fields of clinical application, that can be so summarized:

1) Bone Healing Disordersand Stress Fractures;
2) Vascular Bone Diseases;
3) Wounds and Scar Pathologies;
4) Spasticity;
5) Cellulitis.

Some other ones are still under studies, but they represents the near future:

6) Implantology;
7) Tissue Engineering;
8) Cardiology.

In the field of Bone and Healing Disorders, ESW therapy can be considered, nowadays, as the first choice tool for improving reparative osteogenesis, both in pseudoarthrosis and in delay healing disturbances. In these pathologies it has been demonstrated a direct stimulatory effect both on mature osteoblasts and undifferentiated bony precursor cells. More recently, it has been described also the positive influence of ESW on subperiosteal cells, that are well known to participate to bone healing processes.
On the topic of stress fractures, it is recognized that the pathogenetic mechanism is due to an imbalance in osteoblasts and osteoclasts coupling, with a prevalent bony resorption. In this case, the reparative and trophic effect of ESW therapy has tobe related, other than to the osteogenic activity, to the inhibition of osteoclastic function as well.
In all bone healing disturbances for which it is indicated (mechanical stability has to be guaranteed), ESW therapy represents a valid not invasive therapeutical option, in alternative to surgical intervention: it is repeatable, does not imply collateral negative effects and allow, in any case, the possibility of secondary surgery.
The field of Vascular Bone Diseases includes a wide spectrum of osseuspathologies, characterized by locally altered bone turnover, due to a vascular impairment of different origins. From the anatomo - pathological point of view, it ranges from simple Bone Marrow Edema Syndromes to aseptic osteonecrosis. According to some of the authors, osteochondropaties may be included in this chapter as well, as a particular form of osteonecrosis, localized to a more restricted bony osteoarticular area.
While in osteonecrosis the vascular impairment is a real anatomical alteration (vascular occlusion and lack of perfusion), in all those syndromes characterized only by subchondral bone marrow edema, the vascular impairment can be considered a functional disorder, that, if persistent, could be probably cause osteonecrosis. The functional vascular impairment seems to be able to induce an altered bone turnover, with prevailing osteoclastic activity. This condition, described as "Regional Acceleratory Phenomenon" or "RAP", is characterized by a local up - regulation of bone remodeling. Tipically, it can be observed in the subchondral bone of osteoarthritic painful joints (other than in active osteonecrotic areas), and could be responsible for progressive degenerative changes and osteophytes formation as well. ESW therapy has been described to be effective in reversing the early stages of osteonecrosis; it would be able to stop the progression of osteonecrotic area in some cases, and it has been postulated to "reset" also an altered bone remodeling, when up - regulated. From a general point of view, the mechanisms of action of ESW therapy in Vascular Bone Diseases have been described to be related to an angiogenic and locally trophic effect (in osteonecrosis), other than a "reset" of an altered bone turnover, with a prostacyclin - mimetic effect, perhaps due to eNOS activation (bone marrow edema syndromes).
The angiogenetic effect, coupled to the stimulus on undifferentiated mesenchimal stem cells, is the mechanism that makes it possible to currently apply ESW therapy for improving the healing of wounds and ulcers, other than to treat all "scar pathologies" (included the painful ones). After the original observation, during the treatment of a tibia non - union, associated with a skin ulcer, new ESW devices, producing unfocused shock waves, allowed the treatments of many different skin healing problems. Originally employed only for chronic conditions, recent studies seem to indicate that the more precociously we apply them, the faster we will obtain skin healing. Also in this interesting field of application, ESW

therapy can be considered a valid tool, to be used in combination with some other current procedures, recommended in wound care management.

Closely related to these pathological conditions, it is the application of ESW in cellulitic conditions, an aestethical one, but based on the same tissutal effects (to improve the trophism of the collagen septa in subcutaneous tissue, and circulation as well, while reducing lymphedema at the same time). For optimizing clinical results, ESW therapy should be used in combination with some nutritional reccomandations and proper physical activity.

Clinical applications of ESW therapy in spasticity have been introduced in clinical practice some years ago. Its efficacy is nowadays widely recognized, both in childhood and in adult life, for spastic disorders of different origins, but the mechanism of action is still under studies.

The results can be observed soon after application, although in most of the cases, during the following weeks, there seems to be a return to the original condition, thus requiring further applications. In any case, due to its safety, loss of invasivity and the absence of collateral effects, it can be considered a valid therapy, especially if applied in combination with a rehabilitation program. Moreover, a part of the clinical results, if properly associated to stretching and physiotherapy, can be mainteined in the following time, allowing to the patients to improve week by week (especially if precociously applied after an acute events).

Closely related to clinical applications in the field of musculo - skeletal system, we can describe those of dental implantology and tissue engineering (both still under studies). In the first case, by ESW therapy we will have the opportunity to improve and accelerate bone healing, in all those cases characterized by bone loss and/or implant loosening (for example in peri implantitis), that requirebone integration and augmentation.

Moreover, while applying shock waves for stimulating cell migration and replication, we will have the possibility to do, in some ways, a “cell therapy” and to optimize the use of “loaded scaffolds” in the field of Tissue Engineering in Orthobiological Treatment.

One of the most representative applications of ESW therapy, in the field of regenerative medicine is surely that one on ischemic myocardium. After originary animal studies, further confirmed by cells experiments, in some centers of the world (still few), ESW therapy is applied in clinical practice for ischemic heart diseases, in order to obtain tissue regeneration (from stem cells) and functional recovery, while avoiding fibrosis and reducing the risk of heart fealure and dilated cardiomyopathies.

In summary, ESW therapy, originally introduced in daily clinical practice for urolithiasis, that is only for a pure mechanical effect (to break renal stones), has revealed, some unexpected biological effects, when applied on leaving tissues. They are mainly described as a regenerative potential, in part under studies, but already confirmed by animal and clinical studies. This allow us to include ESW therapy in the field of “Orthobiological Treatment”, toward the new frontieers of Regenerative Medicine.

References

d’Agostino MC, Amelio A, Frairia R. L’Evoluzione delle Onde d’Urto e l’Apporto Scientifico dell’Italia, i Meccanismi d’Azione (Capitolo 2) In: Le Onde D’urto Nella Pratica Medica, WIP Edizioni, Bari, 1° edizione settembre 2010.

d’Agostino MC, Boniforti B et al. ESWT In “Bone Vascular Diseases: The Rationale For Treatment And The Reset Theory”. ISMST Newsletter, May 2007; Vol, 3, Issue 3.

d’Agostino MC, Romeo P et al. Effectiveness of ESWT In The Treatment of Kienbock’s Disease. Ultrasound in Med & Biol, 2011; Vol. -, No. -, pp. 1-5.

d’Agostino MC, Romeo P et al. Onde D’urto Extracorporee: Dalla Litotripsia Alla Rigenerazione Tissutale (considerazioni cliniche ed esperienza sperimentale). Eur Med Phys 2009; 45(suppl. 1 to no. 3).

d’Agostino MC, Romeo P et al. Osteogenesis& Bone Turnover. ISMST Newsletter May 2010; Vol, 6, Issue 1.

Di Meglio F, NurzynskaD et al. Cardiac Shock Wave Therapy: Assessment Of Safety And New Insights Into Mechanisms Of Tissue Regeneration. J. Cell. Mol. Med. Vol XX, No X, 2011 pp. 1-7.

Kearney CJ, Hsu HP et al. The Use Of Extracorporeal Shock Wave-Stimulated Periosteal Cells For OrthotopicBone Generation. Tissue Eng Part A. 2012 Jul;18(13-14):1500-8.

Li X, Chen M et al. Extracorporeal Shock Wave Therapy: A Potential Adjuvant Treatment ForPeri-Implantitis. Medical Hypotheses 74 2010; 120-122.

Manganotti P, Amelio E. Long-Term Effect of Shock Wave Therapy on Upper Limb Hypertonia in Patients Affected by Stroke. Stroke September 2005; DOI: 10.1161/01.STR.0000177880.06663.5c http://www.strokeaha.org

Mariotto S, Cavalieri E et al. Extracorporeal Shock Waves: From Lithotripsy To Anti-Inflammatory Action by NO Production Nitric Oxide 12 2005; 89-96.

Mariotto S, Carcereri de Prati A et al. Extracorporeal Shock Wave Therapy in Inflammatory Diseases: Molecular Mechanism that Triggers Anti-Inflammatory Action Current Medicinal Chemistry, 2009; 16:2366-2372.

Mittermayr R, Hartinger J et al. Extracorporeal Shock Wave Therapy (ESWT) Minimizes Ischemic Tissue Necrosis Irrespective of Application Time and Promotes Tissue Revascularization by Stimulating Angiogenesis AnnSurg 2011; 00:1-9. www.annalsofsurgery.com

Moretti B, Notarnicola A et al. Shock Waves In The Treatment Of Stress Fractures Ultrasound in Med & Biol 2008; Vol. -, No. -, pp. 1-8.

Moretti B, Notarnicola A et al. The Management Of Neuropathic Ulcers Of The Foot In Diabetes By Shock Wave Therapy. BMC Musculoskeletal Disorders 2009; 10:54 doi:10. 1186/1471-2474-10-54.
Muzio G, Verne` A et al. Shock Waves Induce Activity of Human Osteoblast-Like Cells in Bioactive Scaffolds The Journal of TRAUMA® Injury, Infection, and Critical Care • Volume XX, Number XX, XXX 2010.
Nishida T, Shimokawa H et al. Extracorporeal Cardiac Shock Wave Therapy Markedly Ameliorates Ischemia-Induced Myocardial Dysfunction in Pigs *in Vivo* Circulation 2004; 110; 3055-3061.
Olav P. van der Jagt et al. Unfocused Extracorporeal Shock Wave Therapy as PotentialTreatment for Osteoporosis Published online 13 May 2009 in Wiley Inter Science (www.interscience.wiley.com). DOI 10.1002/jor.20910.
Romeo P, d'Agostino MC et al. Extracorporeal Shock Wave Therapy In Pillar Pain After Carpal Tunnel Release: A Preliminary Study Ultrasound. Med &Biol 2011; Vol. 37, No. 10, pp. 1603-1608.
Sansone V, d'Agostino MC et al. Early Angiogenic Response To Shock Waves In A Three Dimensional Model Of Human Microvascular Endothelial Cell Culture (HMEC – 1).
Journal of Biological Regulators & Homeostatic Agents 2012; Vol 6, n° 1: 29-37. Sansone V, d'Agostino MC. Onde d'Urto nelle Patologie Muscolo-Scheletriche Medicina e Doctor - Anno XVI numero 14-22 aprile 2009.
Sathishkumar S, Meka A et al. Extracorporeal Shock Wave Therapy Induces Alveolar Bone Regeneration J Dent Res. 2008; 87(7):687-691.
Siems W, Gruneb T et al. Anti-Fibrosclerotic Effects Of Shock Wave Therapy In Lipedema And Cellulite BioFactors 24 2005; 275-282.
Schaden W, Thiele R et al. Shock Wave Therapy For Acute And Chronic Soft Tissue Wounds: A Feasibility Study. J Surg Res 2007; 143(1):1-12.
Stojadinovic A, Elster EA et al. Angiogenic Response To Extracorporeal Shock Wave Treatment In Murine Skin Isografts Angiogenesis 2008; 11:369-380.
Vulpiani MC, VetranoM et al. Extracorporeal Shock Wave Therapy In Early Osteonecrosis Of The Femoral Head: Prospective Clinical Study With Long-Term Follow-Up. Arch Orthop Trauma Surg, 2012; DOI 10.1007/s00402-011-1444-9.
Wang CJ, Wang FS et al. Treatment For Osteonecrosis Of The Femoral Head: Comparison Of Extracorporeal Shock Waves With Core Decompression And Bone-Grafting. J Bone Joint Surg Am. 2005; 87:2380-2387.
Wang CJ. An Overview of Shock Wave Therapy in Musculoskeletal Disorders Chang Gung Med J 2003; Vol. 26 No. 4.
Wang CJ, Wang FS et al. Extracorporeal Shockwave Therapy Shows Regeneration In Hip Necrosis Rheumatology 2008; 47:542-546.
Wang CJ, Kunder D et al. The Effects Of Shockwave On Bone Healing And Systemic Concentrations Of Nitric Oxide (NO), TGF-B1, VEGF And BMP-2 In Long Bone Non-UnionsNitric Oxide 2009; 20:298-303.

Orthokine - ACS in the post-surgical treatment of joints

Nikica Darabos
Department for Sports trauma and Orthopaedics University Clinic for Traumatology, CMC Sisters of Mercy Medical School, University of Zagreb, Croatia

New biologically based therapeutic agents that modify the action of cytokines and growth factors have a high therapeutic potential. They promise to improve the management of a variety of orthopaedic conditions as a part of a post-surgical treatment, also. Results from a different veterinary and human studies are published a years ago. Combination of biologic Autologous Conditioned Serum – Orthokine therapy and operative treatment could influence a function recovery and clinical result after operation.
For example, the bone tunnel widening appear after knee anterior cruciate ligament (ACL) single bundle technique (SB) reconstruction and could result in a ligament laxity or lead to increased failure rates. It could be affected on the biomechanical and biomolecular base. We carried out a research to establish whether a double bundle technique (DB) in a combination with biologic regenerative treatment of intra-articular application of Autologous Conditioned Serum (ACS) containing endogenous anti-inflammatory cytokines including IL-1Ra and several growth factors, could enhance a result of ACL surgery. In a prospective, randomized, double-blind trial with four parallel groups, 124 patients were treated. We compared a tibial bone tunnel width measured by CT-scans in three different post-operative periods. The clinical efficacy was assessed by patient administered outcome instruments (IKDC 2000, LYSHOLM, TEGNER) up to two years following the ACL-reconstruction in 4 groups of patients regarding a different combination of treatment: A. SB + Placebo, B. SB + ACS, C. DB + Placebo, D. DB + ACS.
Results show that postoperative tibial tunnel diameters in all groups were significantly larger than they were directly after surgery. Bone tunnel enlargement was significantly less in Group D than in the other groups ($p<0.005$). Clinical outcomes were consistently better in patients treated with double boundle technique, especially in a group D, at all data

points and for all outcome parameters, but there were no statistically significant differences, even in the IKDC 2000 Ligament examination subscale after 1 year.
Also, biologic Orthokine therapy could be successfully used in a combination with the operative treatment of ligament, meniscus, tendon, muscle and chondral tissue injury of different joints.
Combination of biologic Autologous Conditioned Serum – Orthokine therapy and operative treatment give have a positive influence on a function recovery and clinical result after operation. In a combination with operative DB ACL reconstruction it gives the best postoperative knee stability and clinical result by affecting tunnel widening.

The treatment of the instability caused by bony defect. A treatment algoritm

Francesco Franceschi
Department of Orthopaedic and Trauma Surgery, Campus Biomedico University of Rome, Italy

The glenohumeral joint is one of great mobility facilitated through the complex interplay of soft tissue and osseous anatomy. Bone loss of the glenoid and/or humerus is a common consequence of traumatic anterior shoulder instability and can be a cause of recurrent instability after a Bankart repair. Accurate characterization of the size and location of osseous defects associated with traumatic instability is important when planning treatment.
Clinically significant glenoid and/or humeral head defects are large enough to cause or exacerbate shoulder instability. Bone loss can lead to very severe instability, including dislocations without raising the arm, during routine activities of daily living or while asleep. If significant bone loss is suspected, a CT scan is needed. MRI cannot be relied upon for to evaluate bone loss, as MRI often underestimates the degree of bone loss. If less than 20-25% of the glenoid is eroded, the shoulder may still be treated arthroscopically with good success. However, if >20-25% of the socket has been lost, open surgery will often be required to replace the lost bone of the socket with bone from another part of the shoulder. This open surgery (Modified Laterjet Procedure) is still an outpatient procedure and leads to good or excellent results in 83-95%.
When the humeral head dislocates, part of its ball shape can be smashed in by the corner of the glenoid socket that it rests against in the dislocated position. If the humeral head is damaged, unless the bone defect is very large, this can often be treated arthroscopically by repairing part of the rotator cuff into the bony defect (Arthroscopic Reimplissage).
Arthroscopic shoulder stabilization has become the standard of care in the surgical management of glenohumeral instability. However, the management of the unstable shoulder associated with a bony defect (glenoid, humeral or combined) can be challenging and preclude arthroscopic treatment.
Biomechanical data from Itoi et al. has shown that the force required to translate the humeral head in relation to the glenoid with the arm in abduction and external rotation was significantly smaller in the glenoid with a defect of equal to or greater than 21% of its length. Adequate diagnosis of bony defects is paramount to successful treatment and entails a careful history, clinical exam, and specific radiographic imaging. In general, higher energy shoulder trauma leads to more significant glenoid and/or humeral head defects. In addition, the severity of these defects corresponds with the number and frequency of instability episodes. Non-operative methods of treatment are not sufficient for treating these cases. Although successful arthroscopic management of instability associated with osseous defects has been described, open reconstruction is often indicated.
There are different indication and different technique.
The management of the unstable shoulder with bony defects is challenging and differs depending on the individual case. Diagnosis relies on a thorough clinical and radiographic evaluation. Of significant importance is the size and location of the defect encountered. Treatment strategies are emerging, and our ability to create successful outcomes is improving.

References

Boileau P, O'Shea K, Vargas P, Pinedo M, Old J, Zumstein M. Anatomical and functional results after arthroscopic Hill-Sachs remplissage. J Bone Joint Surg Am 2012; 94(7):618-626.
Hantes ME, Venouziou A, Bargiotas KA, Metafratzi Z, Karantanas A, Malizos KN. Repair of an anteroinferior glenoid defect by the latarjet procedure: quantitative assessment of the repair by computed tomography.
Arthroscopy 2010; 26(8):1021-1226.
Lynch JR, Clinton JM, Dewing CB, Warme WJ, Matsen FA 3rd. Treatment of osseous defects associated with anterior shoulder instability. J Shoulder Elbow Surg 2009; 18(2):317-328. Review.
Milano G, Grasso A, Russo A, Magarelli N, Santagada DA, Deriu L, Baudi P, Bonomo L, Fabbriciani C. Analysis of risk factors for glenoid bone defect in anterior shoulder instability. Am J Sports Med 2011; 39(9):1870-1876. E pub 2011 Jun 27.

Park MJ, Tjoumakaris FP, Garcia G, Patel A, Kelly JD 4th. Arthroscopic remplissage with Bankart repair for the treatment of glenohumeral instability with Hill-Sachs defects. Arthroscopy 2011; 27(9):1187-1194. E pub 2011 Aug 6.

Relation between tendon biomechanic and water

Marco Franchi
Science Department for the Quality of Life, University of Bologna, Rimini, Italy

In tendon the extracellular matrix including collagen, proteoglycans, glycoproteins and elastin in a small amount, and water support the functional mechanical roles of transmitting forces of muscle contraction and recoiling of collagen fibres when muscle relaxes. Insoluble fibrillar collagen mainly consists of type I collagen which represents the 80-90% of tissue dry weight. However the major tissue component is water representing the 55-70% of the total wet weight. Fibrillar collagen shows a regular arrangement in a repeated, alternated and hierarchical handedness, from a molecular to a supra molecular level. This array increases the resistance of fibrils and fibres to tensional loads and tendon elongation. In particular three polypeptide left-handed chains coil around each other to form a unique triple right handed tropocollagen helix. Five of these tropocollagen molecules stag side-by-side and twist in a left-handed helix forming a single microfibril. Many microfibrils array into rightward helices to form a single fibril. Then fibrils form fibres running mostly parallel and densely packed following an almost straight course, interrupted only by periodic crimps where fibrils always twist leftward. In tendon small proteoglycans like decorin and a small amount of biglycan fill inter fibrillar spaces, whereas large proteoglycans like aggrecan and versican seem to be located in inter fibre spaces. Collagen fibers seems to retain the main part of interstitial bound water whereas proteogly can aggregates are supposed to retain large amount of free water. Water is a major constituent of connective tissues and altering the water content may affect the tissue elastic and viscous behavior. Connective tissues are very sensitive to dehydration as they tend to get dry when exposed to air: dehydration of the Achilles tendon due to air exposure during open surgical repair causes a low quality healing both at the cellular and biomechanical level. Fluids move within tendon when it is stretched since it has been shown that in humans, *in vivo*, there is a marked negative tissue pressure in the peritendinous space around the Achilles tendon during exercise. In this study we investigated the morphological aspects and biomechanical behavior of collagen fibrils/fibres under physiological stretching in Achilles tendon of rat. Relaxed and physiologically stretched tendons were analyzed at the polarized light microscope, TEM and SEM. Under stretching, fibre crimps straightened and disappeared. Morphometric analysis showed that the diameter of physiologically stretched fibrils reduced. We believe that an increased molecular packing with a fluid displacement occurs in fibrils under tension. We suggest that both small (decorin and biglycan) and large proteoglycans (aggrecan and versican) binding large amount of water could play a role in favoring and regulating a fluid shift or a water flow during stretching and recoil of tendon. This hydrodynamic mechanical model is supported by a mechanical role of muscle and tendon sheets during physical activity.

References

Kjaer M. Physiol Rev 2004; 84: 649-698.
Franchi M et al. J Anat 2007; 210:1-7.
Franchi M et al. J Anat 2010; 216:301-309.
Magnusson SP et al. J Physiol 2008; 586:71-81.
Yoon JH, Halper J. Neuronal Interact 2005; 5:22-34.

Eccentric exercises in tendinopathies

Antonio Frizziero, Michele Barazzuol, Patrizia Poli
School of Specialization in Physical and Rehabilitation Medicine, University of Padua, Italy

The widespread practice of sports, especially those at high-level, has led clinicians to an increasing interest for tendinopathies and to realize that the lack of recognition of the disease, as well'as the mistreatment of the acute event, can affect the evolution towards chronic conditions with risk of disability and significant lengthening of recovery time. Starting from researches published by Stanish, in order for the healing tendon to be adequately rehabilitated, the treatment program must include specific eccentric strength rebuilding exercises.
A number of consequences on the tendon have been demonstrated after an eccentric exercise program: in fact the eccentric exercise determines active lengthening of the muscle-tendon unit and causes an increase of the tendon mass by favouring the deposition of collagen type I fibers, thus it's now widely accepted that the eccentric exercise is more effective on pain than a general exercise program.

Alfredson was the first to propose a protocol of intensive exercises as opposed to concentric activities, but unfortunately maybe the only one to demonstrate such good results with eccentric exercise only. However, some Authors recently have confirmed long-term success of this protocol in patients with chronic Achilles tendinopathy treated with Alfredson'sheel-drop exercise programme, showing a constant improvement in strength and resistance to pain, up to 5 years after completion of the programme.
Stanish et al. recommend that patients should perform the eccentric exercises with no pain, while Scandinavian authors recommend pushing through pain; though effective in Scandinavian population, the results of eccentric exercises observed from other study groups are less convincing than those reported from Scandinavia, with only around 60% of good outcome.
On the other hand, the literature is lacking in studies comparing the eccentric exercise protocol with other rehabilitation programs, while many authors evaluated the significance of the year in combination with other therapies, such as shockwave therapy or the use of platelet-rich plasma (PRP).
The encouraging results obtained by comparative studies show that the combined approach of painful eccentric loading plus repetitive low-energy shockwave therapy produced significantly better results than eccentric muscle training alone.
In conclusion, many aspects of the relation between eccentric exercise and tendinopathy remain uncleared, and a future goal will be to identify new specific protocols for musculo-tendinous pathologies. The clinician, and the patient as well, must recall that the beginning of the rehabilitation treatment should be gradual, to avoid the onset of more severe forms of tendinopathy.

References

Alfredson H. The chronic painful Achilles and patellar tendon: research on basic biology and treatment. Scand J Med Sci Sports 2005; 15:252-259.
Cacchio A, Rompe JD, Furia JP, Susi P, Santilli V, De Paulis F. Shockwave. Therapy for the Treatment of Chronic Proximal Hamstring Tendinopathy in Professional Athletes. Am J Sports Med 2011; 39(1):146-153.
Jonsson P, Alfredson H. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. Br J Sports Med 2005; 39(11):847-850.
Lindstedt SL, La Stayo PC, Reich TE. When active muscles lengthen: Properties and consequences of eccentric contractions. News Physiological Science 2001; 16:256-261.
Maffulli N, Longo UG. How do eccentric exercises work in tendinopathy? Rheumatology 2008; 47(10):1444-1445.
Maffulli N, Longo UG, Denaro V. Novel approaches for the management of tendinopathy J Bone Joint Surg Am 2010; 92(15):2604-2613.
Rompe JD, Furia JP and Maffulli N. Eccentric Loading Versus Eccentric Loading Plus Shock-Wave Treatment for Midportion Achilles Tendinopathy Am J Sports Med 2009; 37(3):463-470.
Stanish WD, Rubinovich RM, Curwin S. Eccentric exercise in chronic tendinitis. Clin Orthop Relat Res 1986; (208): 65-68.
Van der Plas A et al. A 5-year follow-up study of Alfredson's heel-drop exercise programme in chronic midportion Achilles Tendinopathy. Br J Sports Med 2012; 46(3):214-218.
Visnes H, Bahr R. The evolution of eccentric training as treatment for patellar tendinopathy (jumper's knee): a critical review of exercise programmes. Br J Sports Med 2007; 41:217-223.

Arthroscopic treatment of basal joint arthritis

John Furia
P. SUN Orthopedics and Sports Medicine, Lewisburg, USA

Basal joint arthritis is the second most common type of arthritis seen in females and the most common location for osteoarthritis in the hand. The condition afflicts approximately 44% of adult females and 28% of adult males. The condition is diagnosed clinically based on pain, tenderness and crepitance with decreased range of motion of the basal joint and confirmed radiographically, often utilizing the Eaton classification system.
Traditional nonoperative management includes relative rest, activity modification, splinting, anti-inflammatory medicines and injections. Non operative management may be helpful in the early stages of the condition but is much less helpful as the condition progresses.
There are numerous surgical options to treat basal joint arthritis. Depending on the stage, metacarpal osteotomy, anterior oblique ligament reconstruction, suspensionplasty, hematoma arthroplasty, ligament reconstruction tendon into position, total joint replacement, as well as fusion may all be helpful. There is data to support each of these procedures, however, much of this data is level IV and the optimal procedure remains unclear.

Over the past decade, arthroscopic measures to treat vasal joint arthrosis have become more popular. There are now several reports that support the use of this minimally invasive technology. The purpose of this presentation is to review the prevalence, nonoperative, traditional operative and newer arthroscopic methods for the treatment of basal joint arthrosis. Various “tips and tricks” can be utilized to simplify the arthroscopic approach to basal joint arthrosis.

Calcific process in tendon pathology

Francesco Oliva[1], Alessio Giai Via[1], Nicola Maffulli[2]
[1] Department of Orthopaedics and Traumatology, University of Rome “Tor Vergata” School of Medicine, Rome, Italy
[2] Centre for Sports and Exercise Medicine Queen Mary University of London, Barts and The London School of Medicine and Dentistry, Mile End Hospital, London, U.K.

Abstract

In calcific tendinopathy (CT), calcium deposits in the substance of the tendon, with chronic activity-related pain, tenderness, localized edema and various degrees of decreased range of motion. CT is particularly common in the rotator cuff tendons (RCTs), Achilles and patellar tendons. The presence of calcific deposits may worsen the clinical manifestations of tendinopathy with an increase in rupture rate, slower recovery times and a higher frequency of post-operative complications. CT of the rotator cuff shows a tendency toward spontaneous resorption of the deposits and symptoms often resolve spontaneously, although some authors described persistent pain and reduction of ROM at long time follow-up. Osteolys is of the greater tuberosity is an uncommon and distinctive form of CT of the shoulder. The clinical course of insertional CT has been poorly investigated, although experience suggests that pain seems to improve in older patients even if insertional calcification persists. The nomenclature of CT is confusing, perhaps in part because of the many terms classically used to describe localized deposits of calcium in tendons, including calcifying tendinitis, calcific tendinitis, calcified tendinitis, calcareous tendinitis, tendinosis calcarea, calcific periarthritis, periarticular apatite deposit.
The specimens of RCTs obtained during surgery consist of a gritty mass of sandy material or a toothpaste-like fluid. X-ray diffraction and infrared spectrometry identified the material of calcific deposits as calcium carbonate apatite. Computed tomography studies of patellar tendon revealed instead a three-dimensional structure of calcific deposits, which have a porous structure throughout the tendon. But the macroscopic differences of calcific deposits seems not to reflect a change neither chemical composition nor in the mineralogical structure.
The etiopathogenesis of CT is largely unknown and still controversial. Many different theories have been developed. “Reactive calcification” is the first theory which has been proposed for CT of rotator cuff. Favorable environment permits an active process of cell-mediated calcification, usually followed by spontaneous phagocytic resorption. The process has been divided into four stages (precalcific phase, calcific phase, resorptive phase, and repair phase) which may all occur concomitantly in the same tendon. A process resembling endochondral ossification has been proposed as a possible mechanism to explain the physiopathology Achilles and patellar tendon calcifications. Others authors confirmed this theory suggesting that insertional CT can develop by endochondral ossification of fibrocartilage at the enthesis of the Achilles tendon. The calcific process begins in the enthesis and grows into the tendon. Fibrocartilage cells appear by metaplasia from tenocytes. Other authors thought that ectopic bone derives from metaplasia of tendon cells into osteogenic cells. Mesenchymal stem cells are present in tendon tissues and Tendon-Derived Stem Cells (TDSCs) have been isolated from the flexor tendon and patellar tendon in animals models.
Some predisposing factors could also influence the development of CT. Emerging data seem to indicate an association between tendinopathies and endocrine disorders such as diabetes, hypercholesterolemia, hypertriglyceridemia, thyroid disorders, estrogen levels alterations. A familial predisposition and inherited genetic components have also been postulated in some circumstances. Unfortunately the association with CT is unclear, and no physiopathological investigations have been performed. Rather than formed by precipitation of inorganic ions, CT results from an active cell-mediated process in which resident progenitor cells with multidifferentiation potential may play a determinant role. Many different factors such as acute injury, repetitive micro-trauma, and chemical-induced injury may cause damage to the tendon and start the natural healing process. Tendon healing includes many sequential processes. It is possible that the erroneous differentiation of tendon progenitor cells into chondrocytes or osteoblasts instead of tenocytes may contribute to the pathogenesis of CT. The mechanism leading to the erroneous differentiation of TDSCs is not completely understood. Probably, the expression of BMPs, biglycan, fibromodulin and an unfavorable micro-environment induced by overuse modify the natural healing process of the tendon.
In conclusion the aetiopathogenesis of CT is still controversial and many questions remain unanswered. We advocate to use the terms “calcific tendinopathy” and “insertional calcific tendinopathy” to differentiate conditions that seem to be caused by two different pathogenetic mechanisms. Better understanding of the pathogenesis is essential for development of effective treatment modalities and for the improvement of clinical outcomes.

Classification and treatment of ankle antero-lateral instability

Marco Guelfi, Alessandro Civani, Matteo Guelfi
Head of Department of Osteo-Articular Diseases Clinic Montallegro, Genoa, Italy

Introduction

The instability of the ankle has been well framed by many authors including Freeman (1964) and subsequently Pisani (1982). We have to distinguishthe term "laxity than the instability"; laxity is defined as a failure of the capsule-ligament structures normally appointed to maintain stable articular heads, instability is a subjective sensation which no longer allows the ankle a stable and securely to the ground. Instability and laxity can the propose both as a stand-alone or in combination framework. The treatment of the instability resulting in chronic lesion laxity for the ligamentous apparatus external ankle counts sever al surgical techniques (over 50 described in the literature).

Among the most used:
Methods with the use of the tendon of the peroneus brevis:
1) Watson-Jones
2) Evans
3) Larsen
4) Chrisman-Snook
5) Pisani (with associated stabilization of the subtalar joint).

Methods with the use of a periosteal flap fibular drawbridge:
1) Chiappara-Rettagliata
2) Priano (with arthroscopic assistance) (1994).

Methods with direct repair of ligaments:
1) Brostrom (1966)
2) Brostrom amended by Gould (1980).

Necessarily recall some concepts of anatomy and joint kinematics underlying the treatment of ankle instability. The stability of the ankle joint is given by three groups of ligaments: the lateral ligaments, the tibiofibular syndesmosis and medial ligaments. The lateral ligaments are: the anterior talofibular ligament, calcaneo fibular, posterior talofibular. The anterior talofibular ligament is a thickening of the intra-articular capsule anterolateral which extends from the anterior edge of the fibula distal to the distal anterolateral neck of the talus. It is about 2 mm of thickness, 10 mm wide and 20 mm long. Calcaneal fibular ligament is a cord-extra-capsular, which runs from the lower surface of the lateral malleolus in a distal-to-back on a small tubercle on the lateral aspect of the calcaneus. It forms the medial wall of the sheath of the peroneal and stabilizes the ankle and the talo-calcaneal size 3 mm thick, 5 mm wide and 20 of length. Posterior talofibular ligament is the most robust of the lateral ligaments. Runs horizontally from the distal fossa of the fibula to the lateral tubercle of the posterior process of the talus. Deep on average is 8 mm long and 30 mm. A cadaveric study shows that the higher antero-lateral section of the joint capsule is responsible for about 18% of the laxity remaining after a ligament injury grade II and 30% after a ligament injury grade III (Boardman, Liu 1997). A facility that is not in itself a mechanical stabilizer of the ankle but plays an important role for execution of the surgical technique in question is the extensor retinaculum. Marconetto and Parino have accurately described the anatomy and function: it is composed of 2 parts top and bottom. The lower part has a configuration Y formed by three bands or less frequently to X composed of four bands, stretched between heel and medial edges of the tibia and foot. The formation of main interest for the execution of the technique in question is the stem the Y (frondiforme ligament) and is the restraint system III peroneal tendon and the extensor digitorum communis, and originates from the heel taking up and medially to extend the supero-medial band. The origin calcaneal is composed of three roots: lateral from the lateral margin of the floor of the sinus tarsi, intermediate from the floor of the sinus tarsi and medial origin with endosenotarsica in turn composed of three beams, which run on the lateral side of the talar neck until to the lower surface of the ligament frondiforme consituing the package deep. Medial tendons of the extensor digitorum ligament frondiforme continued in the supero-medial oblique band that goes on the tendon below the EPA and the tendon of the TA to terminate the medial malleolus. Microscopically can detect a rich innervation with free terminations and particles Pacini, who appear to function more proprioceptive and mechanical. In fact, according to the authors, taking up some considerations Viladot in 1984, the retention of the retinaculum has a positive effect on the stability of the ankle and subtalar also their mechanism of alerting reflex muscle pronator.

Surgical technique

Since with a certain frequency the lateral instability of the ankle is associated with intra-articular pathologies, in accordance with other authors we usually use the technique of ligament reconstruction external preceded by a time arthroscopic intra-articular. In our experience, intra-articular pathology that often we find that we treat in the same session and during the time associated with arthroscopic ligament reconstruction of antero-lateral ankle impingement fibrous. According to the literature it is not uncommon to find a detachment of osteochondral talar dome, a lesion of the distal syndesmosis tibio fibular, a osteophyte tibialis anterior, intra-articular loose bodies. As regards the part arthroscopic use a manual traction sling that turns out to be sufficient both for the inspection for the treatment of articular lesions. Use a toolbox with standard optical, 2.8 mm, 30° inclined, with access anteromedial and antero-lateral after distension of the joint with 20 cc of saline sol. It examines the chondral surfaces, the malleolus, the distal syndesmosis tibio fibular and soft tissues including the joint capsule side. Synoviectomy is performed to remove scar tissue anterolateral painful intra-articular reactive. If this is removed the osteophyte tibialis anterior. Prefer treat chondral lesions using chondroplasty with the technique of micro-fractures. Once the start time arthroscopic ligament reconstruction according to the technique of Brostrom amended by Gould. We practice a linear incision along the anterior margin of the distal fibula starting 5 cm proximal to the tip of the fibula and ending, after having bent downward and posteriorly, a few cm before the sheath of the peroneal, then with an incision shorter than the classical technique, since they do not deal with this procedure patients with lesion also of the peroneal-calcaneal ligament. We are careful to observe the superficial peroneal nerve before while the sural nerve is in an area far enough away from our incision being rear peroneal tendons. Continue dissection up to the joint capsule and simultaneously identify the inferior extensor retinaculum. Let inserted to the fibula a sleeve capsular of 3 mm. Interior of the articular capsule is identified the anterior astragalar fibular ligament as a thickening of the capsule. We suture the remaining distal fibular ligament anterior talar directly anchored on the fibula and sutured to the periosteum over the previously prepared with capsular sleeve points to coat. It is then isolated the extensor retinaculum, which is above the suture and pulled, and anchored to the fibula. We complete the repair with suture and the subcutaneous tissue of the skin and protect the joint with a bandage soft. The next day you apply a guardian bivalve protection and allows the load touched with two sticks. From day 15 you can ambulation with partial load is gradually increasing and started the re-education program with exercises of flexion-extension of the ankle and knee continues with specific strengthening exercises of the peroneal and proprioception exercises. The race will be granted from the third month and the sport of jumping and contact recovered gradually from the fourth to the sixth month post-operatively.

Case study

From January 2000 to January 2005 were treated with this method 18 patients, 11 males and 7 females, aged between 17 and 35 years (mean 27.5 y), the affected side was the right in 12 patients and left in 6 patients. All were suffering from instability of the ankle. The instability persisted for at least six months to a maximum of four years. The sport was football in 9 cases, the court in one case, a gym with bodyweight exercises in two cases while the other did not practice sports. All of them correspond to the following criteria:
1) Failure of previous conservative treatment, including use of braces, physical therapy, pharmacological and proprioceptive exercises. Ten of these were treated with plaster cast, while the others had not received any treatment in acute.
2) Drawer front clinically evident compared to the healthy side at 20° of plantar flexion to evaluate the PAA and the reversal maneuver forced to evaluate the PC. May be documented with RX stress by Telos device. While knowing the limits of reproducibility of this examination, we consider, in agreement with the majority of the authors, a limit of normality of 9 mm for anterior translation and 15° for the talar tilt (Karlsson J.).
3) Complete rupture of the ligament PAA and integrity of the ligament PC and PAP MRI examination performed routinely in all cases.
4) Subjective feeling of instability often associated with pain in normal walking, especially on uneven ground or inability to sports activities as well as recreational-amateur. All patients had one foot with the heel in line footprint and indifferent, except for one patient with bilateral pes cavus, a patient with mild varus heel ipsilateral to the lesion and one patient with a small footprint with calcaneal valgus flat I can ipsilateral to lesion. In all cases we performed the first time arthroscopic, highlighting always aimpigement by scar synovial antero-lateral, which has been treated by synovectomy anterolateral selective. Were excluded from the study patients who had an osteochondral disease.

Results

For the evaluation of the results we refer to the scale AOFAS minimum follow-up was 1 year and up to 5 years (average F-UP = 3.2 years)
- pain:

absent in 17 patients
occasional in 1 patient
- walking without limitation all subjects
- difficulty walking on uneven ground one patient (occasional)
- abnormalities of gait in any patient
- dysmotility in flexion-extension of the Ankle in any patient
- dysmotility in inversion-eversion in two patients (moderate limitation pronosupination)

- anteroposterior stability: the presence of a moderate anterior drawer in a patient
- varus-valgus stability: good in all patients
- changes in the axis of the support and heel: heel valgus and flat-footedness in one case, heel varus in one case, existing intervention.

The results have been so good in 94.5% of cases and good in the remaining 5.5%. We had unsatisfactory results in this study. All patients who were doing sports have returned to practice and two patients who previously held the trauma sedentary life began after the procedure to practice sports at a recreational level.

Discussion and Conclusions

In agreement with the literature we have found satisfactory results with the use of this surgical technique, which has proved simple and quick, without the sacrifice of other structures and with a post-operative course well accepted by patients. In the light of the results and findings during surgery we believe that the combination of a time arthroscopic helpful to identify comorbid conditions sometimes misunderstood by the methods of preoperative imaging and clinical examination to treat them simultaneously instability with a wave of technical simple to perform and less aggressive for the patient, in the context of the intervention of base. Excellent stability obtained by the method of Brostrom-Gould in the cases treated, the rapid recovery of joint function and return to sports comfort us in continuing this surgical technique and encourage us to use it in calcaneal-fibular ligament injuries, as indeed suggested by the inventor of the technique. Bell SJ et al. reported the long-term results obtained with this method also repairing the ligament PC in a group of patients used high-level military sports. For an F-UP of 26.3 years, the results were excellent and good in 91% of cases studies performed on cadaver showed a greater mechanical stability with the intervention of Brostrom modified compared to the techniques of Watson-Jones and Chrisman-Snook especially in subjects top sportsmen commitment and encourage us in the use of this method, especially in this category of sports.

References

Bell JL, Walthour CS, Provencher MT, Sitler D. Chronic lateral ankle instability: the brostromprocedure. Operative Techniques in sports medicine 2005; 13:(3):176-182.
Bordmann DL, Liu SH. Contribution of the lateral Joint capsule to the mechanical stability of the ankle. Clin. Ort. Relat. Res. 1997; (341):224-232.
Cannon LB, Slater HK. The role of ankle arthroscopy and and surgical approach in lateral ankle ligament repair. Foot and ankle surgery 2005; vol. 11, n°1, 1-4.
Giannini S, Ceccarelli F, De Benedictis M, Perna L, Faldini C. Lesioni capsulo legamentose croniche della tibiotarsica; progressi in chirurgia del piede. Il piede nello sport 1997; 127-139.
Gould J.S. Chirurgia del piede. Verduci editore 1996; vol II; capitolo 46.
Karlsson Lasinger. Lateral instability of the ankle joint. Clin. Orthop. and Rel. Research 1992; 253-261.
Liu SH, Baker CL. Comparison of lateral ankle ligamentous reconstruction procedures, Am J Sports Med 1994; 22(3):313-317.
Marconetto M, Parino E. Il retinacolo inferiore degli estensori, anatomia funzionale, utilizzo nelle plastiche legamentose. Chirurgia del piede 2003; vol. 27.
Messer TM, Cummins CA, Ahn J, Kelichian AS. Outcome of the modified Brostrom procedure for chronic lateral ankle instability using suture anchors. Foot Ankle Int 2000; 21 (12):996-1003.
Salvi AE, Metelli GP, Pazzaglia UE. Artroscopia di caviglia nello sportivo. Minerva ortopedica traumatologica; 2004, vol.55; 257-263.

The effect of femoral tunnel position and graft tension on patellar contact mechanics and patellar kinematics following MPFL reconstruction

Deiary F Kader
Orthopaedic & Trauma Department, Newcastle upon Tyne, U.K.

Background: non-anatomic femoral tunnel position or graft over tensioning during Medial Patellofemoral Ligament (MPFL) reconstruction may cause altered patellofemoral joint kinematics and contact mechanics, potentially resulting in pain and joint degeneration.

Hypothesis: non-Anatomic positioning of the tunnel or graft over tensioning during MPFL reconstruction will have an adverse effect on patellar tracking and patellofemoral joint contact mechanics.

Study Design: Controlled Laboratory Study.

Methods: Eight fresh frozen cadaveric knees were placed on a customised testing rig, with a fixed femur and tibia mobile through 90° of flexion. Individual quadriceps heads and the Iliotibial Band were separated and physiologically loaded with 205N using a weighted pulley system. Patellofemoral contact pressures and patellar tracking were measured through flexion range at 10° intervals, using Tekscan pressure sensitive film inserted between the patella and trochlea and an Optical Tracking System. The MPFL was transected and then reconstructed using a double strand gracilis tendon graft. Tekscan and tracking were recorded for reconstruction with the graft positioned in anatomic, proximal and distal tunnel positions. Measurements were then repeated with an anatomic tunnel and graft tension of 2N, 10N and 30N, fixed at 3 different flexion angles of 0°, 30° and 60°. Statistical analysis was undertaken using repeated-measures ANOVA, Bonferroni post hoc analysis and paired t-tests.

Results: Anatomically placed MPFL reconstruction tensioned with 2N resulted in restoration of intact medial and lateral joint contact pressures and patellar tracking ($P > 0.05$), but femoral tunnels positioned proximal or distal to the anatomic origin, resulted in significant increases in peak and mean medial pressures and medial patellar tilt during knee flexion or extension, respectively ($P < 0.05$). Grafts tensioned with 10N or 30N also caused significant increases in medial pressure and tilt. Fixation of the graft at 30° and 60° restored all measures to intact state ($P > 0.05$), but fixed at 0° caused significant increases in medial joint contact pressures compared to intact ($P < 0.05$) knees.

Conclusions: Anatomically positioned reconstruction with 2N tension fixed at 30° or 60° restored joint contact pressures and tracking. However, graft over tensioning, or femoral tunnels positioned too proximal or distal caused significantly elevated medial joint contact pressures and increased medial patellar tilting. The importance of correct femoral tunnel position and graft tensioning in restoring normal patellofemoral joint kinematics and articular cartilage contact stresses is therefore evident.

Propioception in knee rehabilitation after ACL reconstruction

Gianfranco Lisitano
Medical Center Physical Rehabilitation Kinecenter, Messina, Italy

The proprioceptive rehabilitation of a patient who has suffered a knee injury can be started right from the earliest stages proposed repositioning joint exercises performed in discharge and under the control of the therapist. With the progress of rehabilitation are proposed closed chain exercises with the use of different tools: tablets mono and multi-axial, elastic carpet and balloons Physioball. The “Romberg test” on both feet and monopodalic, with eyes open and closed can give important information and is itself an exercise to be included in the rehabilitation protocol. This exercise can be complicated with a spongy pillow of medium density. And then goes to exercises of perturbation of the balance, it’s done with the therapist behind the patient, making unpredictable the time and the type of perturbation. Typically the exercises of proprioceptive are specific for the joint because they must stimulate the whole propriocetive system. But it’s, possible to propose specific exercises for the knee joint, for example varying the angle of flexion of the joint or using technical equipment, such as, ski boots for a skier. When you plan the exercise program, for the proprioceptive rehabilitation it’s necessary to consider the muscular strenght of the extensor and flexor knee muscles, including the strenght of the muscles of the upstream and downstream joints. To recover the muscle strength is basic for the correct execution of proprioceptive exercises in load. Finally, we must not forget the strength of the stabilizing muscles of the trunk (Core Stability), which is also a prerequisite for the correct execution of many exercises.

References

Cressey EM et al. “The effect of ten weeks of lower-body unstable surface training on markers of athletic performance”. J Sport Cond Res 2007.
Heitkamp HC et al. “Gain in strength and muscular balance after balance training”. Int J Sports Med 2001.

Kvist J et al. “Fear of re-injury: a hindrance for returning to sports after ACL reconstruction”. Knee Surg Sports Traumatol Arthrosc 2005.

Endoscopic assisted release for CECS of the lower leg

Heinz Lohrer, Tanja Nauck
Institute of Sports Medicine Frankfurt/Main, Germany

Introduction

Endoscopic treatment of intractable CECS of the lower leg in athletes is reported rarely and anecdotically.

Methods

We developed a minimally invasive, endoscopically assisted technique for release of CECS of the lower leg. Follow-up 1 was performed by telephone interview 4.0±1.8 (0-7) years; [n=17] after surgery. Follow-up 2 was performed by telephone interview 9.3±1.6 (5-9) years; [n=8] after surgery.

Results

38 compartments were released in 17 athletes (19 deep posterior, 16 anterior, and 3 lateral compartments). All these patients were available for follow-up 1analyses. No complications were seen following anterior and lateral compartment decompression. Contrasting to this, two patients operated under tourniquet for deep posterior compartment syndrome suffered intraoperative hemorrhage and open revision surgery was required. No complications due to vascular injuries were seen in all further patients when the tourniquet was omitted. Ten patients returned to previous sport activity. At follow-up, results were good or excellent in 10 out of 17 patients. Visual analogue pain scale improved from 7.4 (range 5 to 9) before surgery to 2.4 (range 1 to 8) at follow up ($p = 0.0005$). At follow-up 2 the results were not deteriorated.

Discussion

Our data indicate that endoscopic release for CECS of the lower leg is feasible und successful even in the long term. The procedure is at risk for vascular complications during deep posterior compartment fasciotomy. Consequently, it is emphasized to perform the procedure without tourniquet.

Single minimal incision fasciotomy for chronic exertional compartment syndrome of the lower leg

Nicola Maffulli[1], Filippo Spiezia[2], Mattia Loppini[2], Umile Giuseppe Longo[2], Gayle D. Maffulli[1], Vincenzo Denaro[2]
[1] Centre for Sports and Exercise Medicine, Barts and The London School of Medicine and Dentistry, Mile End Hospital, England
[2] Department of Orthopaedic and Trauma Surgery, Campus Biomedico University, Trigoria, Rome, Italy

Chronic exertional compartment syndrome (CECS) is a pathologic condition characterized by exercise-induced pain, swelling, and impaired muscle function (1, 2). It has been related to repetitive movements, typical of military training, or running or endurance sports, and may impair physical performances (3, 4). Any muscle harboring an enveloping fascia can be affected by CECS. In the leg, it can arise in all four compartments, including anterior, lateral, superficial posterior, and deep posterior compartment. The anterior and deep posterior compartments are most frequently affected, accounting for 50% of cases (25% each) (5). Physiopathologically, CECS results from an abnormal increase in muscle compartment pressure secondary to insufficient compliance of the surrounding fascia or exercise-induced muscle hypertrophy, which leads to an increase of the muscle volume within the confined nonelastic space of the compartment. The high intracompartmental pressure (ICP) affects the tissue circulation causing ischemia and, at times, temporary neurological deficits (6, 7). In physiological conditions, the skeletal muscle is perfused only during muscle relaxation, between two muscle contractions during dynamic exercise. When CECS occurs, the high intramuscular pressure prevents the perfusion of the muscles during exercise, resulting in ischemic pain and impaired muscle function.

The intracompartmental pressure measurement is currently considered as the gold standard diagnostic exam for CECS. Although the measurements are obtained before, during, and after exercise (8), the post-exercise values of ICP are the best documented parameter to confirm this condition. Some authors suggest to measure this parameter at 1 minute after exercise, indicating values higher than 27.5 mm Hg as strongly suggestive of CECS in patients with a history of leg pain during physical exercise (9). The ICP measurement is performed introducing a needle or catheter through the skin and fascia into the muscular compartment. For this reason, it is invasive and painful, with risks of bleeding, neuropraxia, and infection. Among noninvasive diagnostic methods, magnetic resonance imaging (MRI) (10) seems to be a promising alternative. Near-infrared spectroscopy (NIRS) aims to measure the hemoglobin saturation of tissues. Patients with typical complaints and elevated ICPs show a larger decrease in StO2 after exercise than those without pressure elevation or healthy controls (11). However, the role of both MRI and NIRS in clinical diagnosis has not been clarified.

The treatment of choice for CECS is considered the faciotomy consisting in surgical decompression of the involved muscular compartments to decrease the pressure. Over time, different surgical procedures have been proposed to manage chronic exertional compartment syndrome of the leg performed in open, endoscopically assisted and minimally invasive fashion (4, 12-20). Recurrence secondary to incomplete release, and complications such as infections, postoperative haematoma, nerve injury, cosmetically unacceptable scarring, muscle fascia adhesions, swelling, lymphocele or haemorrhage of the leg are reported after fasciotomy for CECS (13, 14, 17, 21).

We assessed the effectiveness and safety of the minimally invasive fasciotomy in 18 consecutive athletes (12 males and 6 females, median age: 27 years) with unilateral or bilateral CECS. Clinical evaluation was performed with questionnaires, including SF-36 and European Quality of Life-5 Dimensions scale (EQ-5D). The ability to participate in sport before and after surgery, and the time to return to training (RTT) and to sport (RTS) were also recorded. The median follow up after surgery was 36 months. Both questionnaires showed a statistically significant improvement ($P<0.0001$) after surgery. At the time of latest follow up, 17 of 18 patients (94%) returned to pre-injury or higher levels of sport. Only one patient (6%) returned to sport at lower levels than those of pre-injury status. The median time to RTT and RTS was 8 and 13 weeks respectively. No severe complications or recurrence of the symptoms were recorded.

Our results suggest that the minimally invasive fasciotomy allows to release the anterior and lateral compartments through a single incision in patients suffering from unilateral or bilateral CECS of the leg. This procedure is safe, despite it can be technically challenging. Moreover, the complete recovery and return to pre-injury sport levels are likely in the most patients, and satisfaction and physical function remain high in the mid term. Given these findings, the minimally invasive fasciotomy can be considered as alternative approach to traditional open surgery for the management of CECS.

References

1. Styf J, Korner L, Suurkula M. Intramuscular pressure and muscle blood flow during exercise in chronic compartment syndrome. J Bone Joint Surg Br 1987; 69:301-305.
2. Reneman RS. The anterior and the lateral compartmental syndrome of the leg due to intensive use of muscles. Clin Orthop Relat Res 1975:69-80.
3. Allen MJ, Barnes MR. Exercise pain in the lower leg. Chronic compartment syndrome and medial tibial syndrome. J Bone Joint Surg Br 1986; 68:818-823.
4. Balduini FC, Shenton DW, O'Connor KH, Heppenstall RB. Chronic exertional compartment syndrome: correlation of compartment pressure and muscle ischemia utilizing 31P-NMR spectroscopy. Clin Sports Med 1993; 12: 151-165.
5. Raikin SM, Rapuri VR, Vitanzo P. Bilateral simultaneous fasciotomy for chronic exertional compartment syndrome. Foot Ankle Int 2005; 26:1007-1011.
6. Edmundsson D, Toolanen G, Sojka P. Chronic compartment syndrome also affects nonathletic subjects: a prospective study of 63 cases with exercise-induced lower leg pain. Acta Orthop 2007; 78:136-142.
7. Mohler LR, Styf JR, Pedowitz RA, Hargens AR, Gershuni DH. Intramuscular deoxygenation during exercise in patients who have chronic anterior compartment syndrome of the leg. J Bone Joint Surg Am 1997; 79:844-849.
8. Styf JR, Korner LM. Diagnosis of chronic anterior compartment syndrome in the lower leg. Acta Orthop Scand. 1987; 58:139-144.
9. Aweid O, Del Buono A, Malliaras P, Iqbal H, Morrissey D, Maffulli N et al. Systematic Review and Recommendations for Intracompartmental Pressure Monitoring in Diagnosing Chronic Exertional Compartment Syndrome of the Leg. Clin J Sport Med 2012.

10. van den Brand JG, Nelson T, Verleisdonk EJ, van der Werken C. The diagnostic value of intracompartmental pressure measurement, magnetic resonance imaging, and near-infrared spectroscopy in chronic exertional compartment syndrome: a prospective study in 50 patients. Am J Sports Med 2005; 33:699-704.
11. van den Brand JG, Verleisdonk EJ, van der Werken C. Near infrared spectroscopy in the diagnosis of chronic exertional compartment syndrome. Am J Sports Med 2004; 32:452-456.
12. Blackman PG. A review of chronic exertional compartment syndrome in the lower leg. Med Sci Sports Exerc 2000; 32:S4-10.
13. Cook S, Bruce G. Fasciotomy for chronic compartment syndrome in the lower limb. ANZ J Surg 2002; 72:720-723.
14. Howard JL, Mohtadi NG, Wiley JP. Evaluation of outcomes in patients following surgical treatment of chronic exertional compartment syndrome in the leg. Clin J Sport Med 2000; 10:176-184.
15. Martens MA, Backaert M, Vermaut G, Mulier JC. Chronic leg pain in athletes due to a recurrent compartment syndrome. Am J Sports Med 1984; 12:148-151.
16. Rorabeck CH. A practical approach to compartmental syndrome. Part III. Management. Instr Course Lect 1983; 32:102-113.
17. Rorabeck CH, Bourne RB, Fowler PJ. The surgical treatment of exertional compartment syndrome in athletes. J Bone Joint Surg Am 1983; 65:1245-1251.
18. Slimmon D, Bennell K, Brukner P, Crossley K, Bell SN. Long-term outcome of fasciotomy with partial fasciectomy for chronic exertional compartment syndrome of the lower leg. Am J Sports Med 2002; 30:581-588.
19. Bramante C, Gandolfo L, Bosco V. Minimally invasive fasciotomy in the treatment of chronic exertional anterior compartment syndrome of the leg: personal technique. Chir Ital 2008; 60:711-715.
20. de Fijter WM, Scheltinga MR, Luiting MG. Minimally invasive fasciotomy in chronic exertional compartment syndrome and fascial hernias of the anterior lower leg: short- and long-term results. Mil Med 2006; 171:399-403.
21. Detmer DE, Sharpe K, Sufit RL, Girdley FM. Chronic compartment syndrome: diagnosis, management, and outcomes. Am J Sports Med 1985; 13:162-170.

Hamstring injuries classification. Anatomical, clinical or imaging orientated?

Nikolaos G. Malliaropoulos
National Track & Field Centre, Sports Injury Clinic, Sports Medicine Clinic of S.E.G.A.S., Thessaloniki, Greece

Little information is available in the international literature about non contact acute (NCA) muscle injury classification systems (1).
Various systems have been used to classify the severity of the non contact acute (NCA) muscle injury. Acute muscle injuries are commonly classified as strains (Grade I), partial tears (Grade II) and complete tears (Grade III) (2, 3).
Particularly in elite athletes, where decisions regarding return to play and player availability have significant financial or strategic consequences for the player and the team, there is an enormous interest in optimising the diagnostic, therapeutic and rehabilitation process after muscle injuries, to minimise the absence from training and to reduce recurrence rates (1, 11).

Anatomical

Recently describe an imaging (magnetic resonance or ultrasound) nomenclature, which considers the anatomical site, pattern and severity of the lesion in the acute stage. By site of injury, they define muscular injuries as proximal, middle and distal. Anatomically, based on the various muscular structures involved, muscle injuries distinguish intramuscular, myofascial, myofascial/perifascial and musculotendinous injuries (4).
A general rule of thumb is, “the closer to the ischial tuberosity, the longer rehabilitation period”. Intramuscular or intramuscular haematoma should be also differentiated.

Imaging

Muscle injuries has been classified according to their imaging findings, and Ultrasound and MRI are used to image the muscle lesion.
Previous grading systems based up imaging: Takebayashi et al. published in 1995 an ultrasound-based three-grade classification system ranging from a grade 1 injury with less than 5% of the muscle involved, grade 2 presenting a partial

tear with more than 5% of the muscle involved and up to grade 3 with a complete tear (5). Peetrons has recommended a similar grading (6).
An MRI study described findings with poor prognosis of muscle injury when the muscle rupture and retraction, haemorrhage, ganglion-like fluid collections, are greater than 50% cross-sectional involvement were associated with convalescent periods of more than 6 weeks.
In general, MRI findings, particularly the length and cross-sectional area of injury, may be used as an estimate of time for rehabilitation and can sometimes be predictive of the time high-performance athletes will be away from play.
The currently most widely used classification is an MRI-based graduation defining four grades: grade 0 with no pathological findings, grade 1 with a muscle oedema only but without tissue damage, grade 2 as partial muscle tear and grade 3 with a complete muscle tear (7).
In patients with an equivocal or remote history of trauma, imaging is advised, as it may help to better define a soft tissue mass if a neoplastic mass is clinically suspected (4).

Clinical

In terms of Clinical Classification time to walk pain-free, has been used to define the severity of the injury and predict the time to recovery, 4 weeks to RTP for the time to walk (8).
Also in muscle-tendon complex of the long head of biceps femoris, a clinical assessment of the point of highest pain on palpation, within 3 weeks from the injury, is predictive of recovery time (9).
During my lecture I will present Our Clinical classification for non contact Hamstring injuries, which is based on estimating the knee active range of motion deficit between the injured and the healthy side (10).
We also correlate our fining with the time needed to full return to play. Following the clinical classification we are able to define the severity of the injury decide for the treatment, to design the rehabilitation protocol and follow them during that period, to predict the time to full rehabilitation and to assess the reinjure rate (11).

Conclusion

Careful combination of a Clinical Classification including medical history, mechanism of the injury, injury location, inspection, clinical examination, active range of motion deficits and choosing the appropriate imaging will most likely lead to an accurate diagnosis on NCA Hamstring injuries.

References

1. Mueller-Wohlfahrt HW et al. Terminology and classification of muscle injuries in sport: a consensus statement. Br J Sports Med 2012; 0:1-9.
2. O'Donoghue DO. Treatment of injuries to athletes. Philadelphia: WB Saunders 1962.
3. Brandser EA, el-Khoury GY, Kathol MH, Callaghan JJ, Tearse DS. Hamstring injuries: radiographic, conventional tomographic, CT, and MR imaging characteristics. Radiology 1995; 197:257-262 24, 43.
4. Chan O, Del Buono A, Best TM, Maffulli N. Acute muscle strain injuries: a proposed new classification system. Knee Surg Sports Traumatol Arthrosc 2012; DOI 10.1007/s00167-012-2118-z.
5. Takebayashi S, Takasawa H, Banzai Y et al. Sonographic findings in muscle strain injury: clinical and MR imaging correlation. J Ultrasound Med 1995; 14:899-905.
6. Peetrons P. Ultrasound of muscles. Eur Radiol 2002; 12:35-43.
7. Stoller DW. MRI in orthopaedics and sports medicine. 3rd edn. Philadelphia: Wolters Kluwer/Lippincott 2007.
8. Warren P, Gabbe BJ, Schneider-Kolsky M et al. Clinical predictors of time to return competition and of recurrence following hamstring strain in elite Australian footballers. Br J Sports Med 2010; 44:415-419.
9. Askling CM, Tengvar M, Saartok T, Thorstensson A. Acute first-time hamstring strains during high-speed running: a longitudinal study including clinical and magnetic resonance imaging findings. Am J Sports Med 2007; 35:197-206.
10 Malliaropoulos N, Papacostas E, Kiritsi O, Papalada A, Gougoulias N, Maffulli N. Posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med 2010; 38:1813-1819.
11. Malliaropoulos N, Isinkaye T, Tsitas K, Maffulli N. Reinjury after acute posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med 2011; 39:304-310.

Pectoralis major tendon rupture. Acute vs chronic repair

Giovanni Merolla, Giuseppe Porcellini
Unit of Shoulder and Elbow Surgery, D. Cervesi Hospital, Cattolica, Italy

Introduction: pectoralis major (PM) muscle is the powerful dynamic stabiliser of the shoulder that acts as a flexor, adductor and internal rotator. The rupture of the PM tendon is a relatively rare injury and to date, over 200 cases have been published. PM injuries are male dominant and occur in patients during their second to fourth decade of life; SP is the most commonly involved, CP being hit so rarely to make these lesions sometimes misdiagnosed as a muscle sprain.

Classification and examination: PM injuries are classified on the degree as muscle sprain (type I), partial tear (type II), complete tear (type III); complete tear are further subdivided according to location in: type IIIA (muscle origin), type IIIB (muscle belly), type IIIC (myotendinous junction) and type IIID (tendinous). A recent subclassification introduced a bony avulsion from the insertion (type IIIE) and a muscle tendon substance rupture (IIIF). Type IIID is the most frequent with a rate of 65% folllowed by the type IIIC that occurs with a frequency of 27%. PM ruptures have been described almost exclusively in young men weight lifters and high-performance athletes (boxing, football, water skiing, wrestling, rugby) as results of eccentric contraction of the musculotendinous unit. Rare cases occurred when resisted forces are applied to the extended and abducted arm. Physical examination shows loss of the anterior axillary fold and pectoralis contour and performing the test in “prayer position” asking the patients to press the hands together with the arm adducted, the chest wall reveals to be asymmetryic, particularly evident when the chest wall moves medially. Loss of strength is notable to internal rotation of the arm when tested at neutral. Conventional X-ray is limited as diagnostic tool while ultrasound is effective and less expensive to identify and locate PM rupture, however, MRI remain the method of choice to identify partial and complete tears and to assess the amount of muscle retraction.

Surgical procedures: conservative management is indicated in elderly patients, partial tendon tears and muscle belly ruptures; furthermore, lower demands subjects could be cadidated for nonoperative treatment. Surgical approach is required in young active patients with acute ruptures, despite the chronicity does not represent a limit for tendon reattachment. The deltopectoral is the most common method used for shoulder exposure, with the proximal extent of the incision slightly medial to make easier access to the retracted tendon, while the distal incision should be more lateral for a better exposure of the pectoralis insertion.

Acute ruptures: the clavicular fibes are usually preserved and often a connection (“cord”) between the medial brachial and antebrachial septum is found simulating an intact tendon. In acute ruptures (< 6 weeks) the terminal end of the tendons are identified and fixed with non absorbable sutures (# 5) that are used to pull on the muscle belly. The sutures used to reattack the tendon include size of both types, absorbable and nonabsorbable. Mason-Allen and Krachow stitches have been described to guarantee adequate strength to the knot, while a modified Kessler stitches in multiples layers should be preferred to repair intramuscular tears. PM can be fixed to the humerus with suture anchors, bone tunnels or bone trough.

Chronic ruptures: in cases of chronic ruptures in which direct tendon attachment is not feasible, PM can be reconstruct using tendon autograft or allograft. Zafra et al. described the use of a bone patellar autograft procured from the knee, sutured to the musculotendinous junction with the bone fragment inserted into the humerus by means of a 4.5 mm cortical screw with a washer. Schachter et al. reported the use of autologus gracilis and semitendinous tendon harvested from the knee in a standard fashion and looped to form a standard 4-strand graft. Achilles tendon allograft has been described in delayed repair of PM rupture including both, sternal and clavicular portion, fixed using metal suture anchors. In our study which is still in progress we found encouraging results using the fascia lata allograft for tendon augmentation.

Discussion: acute repair of tendon tears should be performed within 6 weeks to achieve an optimum mobilization of the muscle belly and reattach the tendon to its anatomic origin on the humeral shaft. The mobilization of the musculotendinous junction is more difficult in case of chronic ruptures due to the surface and deep adhesions of the chest wall. Several authors reported that there were significantly more cases with an excellent outcome when surgery was performed within 8 weeks of injury than with delayed surgery. Similarly, additional research findings showed how early surgical approach was associated with better outcome than delayed treatment. Conversely, Schepsis et al. found no significant subjective or objective differences in the outcome between the patients treated operatively for acute or chronic injuries, but all these patients achieved better results than patients treated nonoperatively. In conclusion, PM ruptures are rare injuries requiring immediate diagnosis to set the most appropriate treatment which is identified with surgical reattachment in case of complete tendon tears, both acute and chronic. Although some authors described similar outcomes in acute vs chronic repair, we suggest an early repair to obtain the best clinical and functional results.

References

Aärimaa V, Rantanen J, Heikkilä J, Helttula I, Orava S. Rupture of the pectoralis major muscle. Am J Sports Med 2004; 32:1256-1262.
Bak D, Cameron EA, Henderson IJ. Rupture of the pectoralis major: a meta-analysis of 112 cases. Knee Surg Sports Traumatol Arthrosc 2000; 8:113-119.
de Castro Pochini A, Ejnisman B, Andreoli CV, Monteiro GC, Silva AC, Cohen M, Albertoni WM. Pectoralis major muscle rupture in athletes: a prospective study. Am J Sports Med 2010; 38:92-98.
Merolla G, Paladini P, Campi F, Porcellini G. Surgical approach to acute pectoralis major tendon ruptures. G Chir 2009; 30:53-57.
Schepsis AA, Grafe MW, Jones HP, Lemos MJ. Rupture of the pectoralis major muscle: outcome after repair of acute and chronic injuries. Am J Sports Med 2000; 28:9-15.

Orthokine derived autologous conditioned serum in the treatment of back pain and joints

Carsten Moser
Groenemeyer Institute for Microtherapy, Bochum, Germany

Introduction

As the purely mechanical view of orthopaedic diseases gradually yields to a more biological perspective, novel, biologically based therapeutic agents that modify the action of such cytokines have a high therapeutic potential in a variety of orthopaedic conditions, including osteoarthritis, tendon and muscles injuries and radiculopathies. A new method based on exposure of blood leukocytes to glass beads elicits an accumulation of anti-inflammatory cytokines, including IL-1Ra, and several growth factors, e.g. insulin like growth factor-1 (IGF-1), platelet derived growth factor (PDGF) and transforming growth factor beta (TGF-ß1). This article summarizes details about the production procedure, techniques of clinical application and the status of randomized controlled trials (RCT) using this new method.

Methods

For the ACS production, venous blood is drawn into syringes containing glass beads (Orthogen, Duesseldorf, Germany). When the blood is incubated for six to nine hours, peripheral blood leukocytes reproducibly produce elevated amounts of endogenous anti-inflammatory cytokines. After incubation, the blood is centrifuged and the serum is extracted and injected locally in a series of injections.

Results

ACS has shown to improve signs and symptoms in recently published randomized controlled trials (RCT). In patients with knee OA ACS was superior to Hyaluronan and Saline in terms of pain reduction, patients´ satisfaction, and functional outcome. This marked improvement was still present in a follow-up evaluation after 2 years. Authors from a second RCT concluded that ACS clearly induces a biologic answer and improves function and symptoms in patients with knee osteoarthritis. Further results of a controlled study in a horse-model (experimentally induced OA) indicated that there were significant clinical and histological improvements in osteoarthritis-affected joints of horses following treatment with ACS, compared with placebo-treatments. In view of the significant concentrations of growth factors that have been shown to favorably affect tendon healing, an animal model evaluated the effect of ACS on the healing of the transacted rat Achilles tendon and showed favorable results in contrast to placebo.
In a RCT in patients after anterior cruciate ligament reconstruction, the therapeutic use of intraarticular ACS injections was proven in comparison to saline injections, showing statistically less bone tunnel widening as well as consistently superior clinical outcomes in a follow up period of 12 months.
In a RCT to study ACS for the treatment of sciatic back pain in patient suffering from lumbar radiculopathy three injections of Orthokine once a week for three consecutive weeks showed a beneficial effect on pain decrease and was superior when compared to steroids.

Discussion

Injection of ACS into affected joints, muscles or spinal tissue(s) has shown clinical effectiveness and safety in animal models and human clinical studies for treatment of OA, lumbar radiculopathies and muscle injuries. Because of the autologous nature of the treatment and the physiological levels of cytokines and growth factors that are achieved, the risk/benefit ratio is excellent.

The feasibility and effectiveness of such injections with ACS opens a promising new therapeutic perspective and will be examined further.

References

Baltzer AW, Moser C et al. Osteoarthritis Cartilage 2008 Jul 30.
Becker C et al. Spine 2007 Aug 1; 32(17):1803-1808.
Darabos N, Moser C et al. KSSTA 2011 Dec; 19 Suppl 1:S36-46.
Frisbie DD et al. Am J Vet Res 2007 Mar; 68(3):290-6.
Meijer H, Reinecke J et al. Inflamm Res 2003; 52(10):404-7.
Wehling P, Moser C et al. Biodrugs 2007; 21(5):223-32.
Yang KG et al. Osteoarthritis Cartilage 2008 Apr; 16(4):498-505.

Placental stem cells in the treatment of tendon disorders: experimental preclinical and clinical settings

Aurelio Muttini[1], Luca Valbonetti[1], Michele Abate[2]

[1] *Department of Comparative Biomedical Sciences, University of Teramo, Italy*

[2] *Department of Medicine and Sciences of Aging, University "G. d 'Annunzio" Chieti-Pescara, Chieti Scalo (CH), Italy*

Tendon injuries are very common both in humans and in horses. The Achilles and patellar tendons are most commonly involved in humans and superficial digital flexor tendon is most often affected in horses. In both species very serious economic impact is caused by tendinopathies. It is well known that a true regeneration of tendon tissue is only possible in fetus (Favata, 2006) and that healing of tendinopathies in adult occurs through a scar formation. The scar, in turn, has mechanical properties inferior than healthy tissue (Woo et al. 1999), and affected subjects are predisposed to re-injury. Several different strategies has been suggested for the treatment of tendon injuries and this large range of solutions reflect the absence of a validated effective protocol enabling to induce a true regenerative process. There is consensus agreement, that amnion derived cells may be a possible reserve of cells usefull for clinical application (Muttini et al. 2012) as they are available in nearly unlimited supply, ethically problem-free and easily procured (Evangelista et al. 2008). The cells of the fetal component of the placenta named Amniotic Epithelial Cells (AECs) and Amniotic Mesenchimal Stromal Cells (AMSC) are formed by the epiblast and they appear before gastrulation. Since gastrulation has primary importance in the differentiation and specification of cell fate (cell commitment) amnion cells may retain the pluripotent properties of early epiblast cells (Miki et al. 2005). It has been demostrated by *in vitro* studies that human and ovine AECs display unique properties that can support tendon regeneration. In particular AECs display the self renewal and pluripotency characteristics of stem cells. Furthermore these cells show a high level of plasticity, are able to *in vitro* differentiate into all three germ layers (Miki et al. 2005) and are characterized by a high immunomodulatory activity (Parolini et al. 2009). Recent studies demostrate that AECs isolated from pregnant sheep maintain a stable expression profile during culture and are able to differentiate toward the tenogenic lineage when co-coltured with tenocytes (Muttini et al. 2010, Barboni et al. 2012). Our preclinical studies clearly showed that, when injected into experimental tendon defects af sheep Achilles tendon, PKH 26 marked AECs can survive for at least 28 days. In a recently published clinical study our group also demonstrated that the xenotransplantation of ovine AEC into spontaneous teninopathy of horses survive in the tissue for long period and are able to produce collagen tipe I protein. These data confirm the low immunogenicity and immunomodulatory properties demonstrated in the elegant studies by Magatti et al. 2008.

References

Barboni B, Russo V, Mauro A, Martelli A, Muttini A, Bernabò N, Valbonetti L, Marchisio M, Di Giacinto O, Berardinelli P, Mattioli M. Achille's tendon regeneration can be improved by amniotic epithelial cells allotransplantation. Cell Transplantation. In press (2012).
Evangelista M, Soncini M, Parolini O. Placenta-derived stem cells: new hope for cell therapy? Cytotechnology 2008; 58:33-42.
Favata M, Beredjiklian PK, Miltiadis HZ, Beason DP, Crombleholme TM, Jawad AF, SoslowskyLJ. Regenerative prop-

erties of fetal sheep tendon are not adversely affected by transplantation into adult environment. J Orthop Res 2006; 24:2124-2132.
Magatti M, De Munari S, Vertua E, Gibelli L, Wengler GS, Parolini O. Human amnion mesenchyme harbors cells with allogeneic. T-cell suppression and stimulation capabilities. Stem Cells 2008; 26(1)182-192.
Miki T, Lehmann T, Cai H, Stolz DB, Strom SC. Stem cell characteristics of amniotic epithelial cells. Stem Cells 2005; 23(10):1549-1559.
Muttini A, Valbonetti L, Abate M, Colosimo A, Curini V, Mauro A, Berardinelli P, Russo V, Cocciolone D, Marchisio M, Mattioli M, Podaliri Vulpiani M, Barboni B. Ovine amniotic epithelial cells: in vitro characterization and transplantation into equine superficial digital flexor tendon spontaneous defects. Research in veterinari Sciences (in press) 2012.
Parolini O, Alviano F, Bagnara GP et al. Concise review: isolation and characterization of cells from human term placenta: outcome of the first international workshop of placenta derived stem cells. Stem Cell 2008; 26:300-311.
Woo SL, Hilebrand K, Watanabe N, Fenwick JA, Papageorgiou CD, Wang JH. Tissue engineering of ligament and tendon healing. Clinical orthopaedics and related research. 367, S312-S323 (1999).

Muscles injuries recidives. How i treat?

Gianni Nanni
Education Research Department, Isokinetic Medical Group, Bologna, Italy

A recidive is defined as a new lesion that can occur at the same point of the first lesion, or in different areas, but at the same muscle.
Some studies consider recidive a lesion that occurs in the same muscle until two years after the primary lesion (1).
In our study we instead consider recidives that occur in a shorter period of interval between the two lesions, precisely six months.
We believe that it is difficult to think that an athlete may compete for a period of two years from the primary lesion, without complaints, if the primary lesion is not completely healed.
The early recidives, which occur after only a few days from the primary lesion, usually affecting the same area and the scar tissue that it is forming, are caused by diagnostic errors (2) and/or by eccessive loads when the scar tissue cannot withstand such loads.
Late recidives instead, most often affect the healthy muscle tissue, closed to the scar tissue of the primary lesion or in a more distant areas upstream or downstream the previous injury, but in the same muscle belly. They are usually caused by thick, irregular and inelastic fibrous scar tissue from the previous injury or, in this case too, from excessive loads for a not completely recovered muscle.
In recent years PRP's (Platelet Rich plasma) infiltrations have improved the prognosis of muscle lesions and of recidives too, although the correct rehabilitation protocol, with an adequate number of rehabilitation sessions on the field, has proved to be of fundamental importance in muscle injuries recidives and therefore also in the prevention of further recidives. Exercise seems to affect and influence the quality of the tissue being formed to fill the gap that the injury caused (2, 3).
Our study of 1343 muscle lesions from indirect trauma, in athletes, has done a lot to make us reflect on the importance of rehabilitation on the field after muscle injury and made us understand how the complete recovery of extensibility, especially eccentric of muscle strength, of the primary athletic parameters, characteristic of the sport practiced by the injured, the specific technical movements for that sport discipline and finally the reliability to perform the specific act that caused the injury, are key elements in order to consider an athlete suitable to return to sport activities as before the muscle injury happened (2, 4-7).
In fact, this first period of full return to sport activity we call it "conditioned healing" and we still consider as still taken over by the coach, the athletic trainer and the medical staff, in order to adopt a careful monitoring of the athlete and in particular of the muscle involved by the lesion, after trainings and competitions.
These concepts and principles that guide rehabilitation after muscle injuries are even more important when we talk about the treatment of recidives (2).
We must also be aware of the predisposing factors to recidives, some of which can be detected and removed before starting the work with loads at high intensity, such as ipoestensibility of the antagonistic muscle groups and biomechanical compensations that have been established as a consequence of the first injury (2, 5, 6).

References

1. Malliaropoulos N, Isinkaye T, Tsitas K, Maffulli N. Reinjury after acute posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med 2011; 39(2):304-310.

2. Orchard J, Thomas M. The management of muscle strain injuties: An early return versus the risk of recurrence. Clin J Sport Med 2002;12(1):3-5.
3. Kjaer M. Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. Physiol Rev 2004; 84:649-698.
4. Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Med 2012; 42(3):209-226.
5. Schmitt B, Tyler T, McHugh M. Hamstring injury rehabilitation and prevention of reinjury using lengthened state eccentric training: a new concept. Int J Sports Phys Therapy 2012; 7(3):333-341.
6. Heiderscheit B, Sherry M, Slider A, Chumanov E, Thelen D. Hamstring strain injuries: recommendation for diagnosis, rehabilitation and injury prevention. J Orthop Sports Phys Ther 2010; 40(2):67-81.
7. Croiser J, Forthomme B, Namurois M, Vanderthommen M, Crielaard J. Hamstring muscle strain recurrence and strength performance disorders. Am J Sports Med 2002; 30(2):199-203.

Acromion-clavicular ligament reconstruction with allograft

Simone Nicoletti
Orthopaedic and Traumatology Unit Pistoia Hospital, Italy

Introduction

The acromioclavicular (AC) joint is one of the most commonly injured joints of the body, with injuries to the AC Joint accounting for roughly 9% of upper limb injuries seen in clinical practice (1). They are almost ten times more likely to occur in males than females and most frequently occur within the first three decades of life (1). These injuries typically occur in contact sports athletes (American Football, Rugby, Australian rules and Soccer) (2, 3). It has also been noted that one-fifth of shoulder injuries suffered by recreational skiers involves the AC joint (4). The treatment of AC joint injuries remains a controversial topic. There are many different techniques used but as of yet no technique has been shown to have the significantly best results (5-9). A few studies address the topic and seem to confirm the biomechanical and clinical advantages of using free tendon graft reconstruction for Coracoclavicular (CC) ligaments (10-15). The purpose of this study was to examine the clinical outcomes for CC ligaments reconstruction with tendon allografts.

Methods

Over a two years period, eleven patients underwent CC ligament reconstruction at our institution using cadaveric tendon allografts having suffered AC Joint dislocation. Indications forearly surgery (< 3 months) included symptomatic Rockwood Type IV and V injuries (16) that prevented early return to activities and sports. Indications for delayed surgery (> 3 months) included symptomatic Type III, IV and V injuries. There were nine men and two women. The operations were performed as an open procedure in 8 patients and arthroscopically assisted in three. Preparation for clavicle and coracoid tunnels placement was performed by drilling a 3 mm hole and enlarge by increasing drill bit diameters in increments of 1 mm. A semitendinosus tendon allograft was anchored to the coracoid with an endobutton. An interference 6 mm screw was used to fix the tendon into the clavicle tunnel and a cerclage of PDS band was made as an additional support. In five patients were used a Graftrope instead of fixation with endobutton and PDS cerclage.
The age of the patients ranged from twenty to sixty years old, with a mean age at the time of evaluation of 39 years old. The dominant shoulder was involved in eight patients.
Outcome measures included pre- and post-operative Constant and Oxford shoulder scores, patient satisfaction, return to sports and activities and standardize antero-posterior and Axillary radiographs. The follow-up in this series was between 6 and 16 months post operatively.
Statistical analysis was performed using the Stats Direct 2006 software. The preoperative and post-operative scores for the patients as a single group were compared. This was done separately for both the Oxford and Constant Scores. The statistical significance of the changes was assessed using the Wilcoxon signed-rank test.

Results

The mean follow-up was 12.7 months with a range between six to seventeen months. One of the eleven patients was chronic type III dislocation with unresolved pain having previously been treated conservatively for at least six months. The remaining ten were all type IV (n =6) or V (n = 4) dislocations. Five patients were involved in collisions whilst skiing or snowboarding, four fell whilst cycling and one were rugby injuries. Each patient reported landing heavily on the point of the shoulder.

Over all the patients showed a significant improvement following intervention. The mean pre-operative Oxford score was 42.14 (range 22-59) versus 18 (range 11-38) postoperatively ($p = <0.05$). The mean pre-operative Constant score was 21.71 (range 8-61) versus 85.45 (range 61-92) post operatively ($p = <0.05$). The mean pre-operative pain was 10.43 (5-15) and the mean post-op pain was 1.27 (range 0-6), where 15 is maximal pain and 0 is no pain. All patients were very satisfied (n=7) or moderately satisfied (n = 4). The mean satisfaction score was 85% (range 50-100%). Six patients returned to their pre-injury level of sports activity, three returned to a reduced leveland one returned to a significantly reduced level.

The patients were divided into two groups depending from the time interval between the injury date and operation date. Group A contained patients having had surgery less than three months since injury. Group B included patients operated on more than three months from the date of injury. There was no significant difference in any of the outcome measures between the post-operative outcomes between the two groups.

There were two complications. One patient was affected by post-operative frozen shoulder. This had completely resolved by four months with rehabilitation only. The other patient complained of periscapular pain. This was a revision case.

Conclusion

Coracoclavicular ligament reconstruction using cadaveric tendon allografts is a viable technique for the treatment of AC Joint dislocation both acutely and chronically. It offers good medium term results, with early return to previous levels of sports and activities. There were no significant differences in between the group of patients that underwent the operation within three months of the injury and those that had the surgery more than three months from the injury date.

References

1. Mazzocca AD, Arciero RA, Bicos J. Evaluation and treatment of Acromioclavicular Joint injuries. Am J Sports Med 2007; 35:316.
2. Petron DJ, Hanson RW Jr. Acromioclavicular joint disorders. American College Sport Med 2007; 300-306.
3. Webb J, Bannister G. Acromioclavicular disruption in first class rugby players. Br J Sports Med 1992; 26:247-248.
4. Kocker MS, Feagin JA Jr. Shoulder injuries during alpine skiing. Am J Sports Med 1996; 24:665-669.
5. Fraser-Moodie JA, Shortt NL, Robinson CM. Aspects of Current Management; Injuries to the acromioclavicular joint. J Bone Joint Surgery (Br) 2008; 90-B:697-707.
6. Cooper ES. New method for treating longstanding dislocations of the scapuloclavicular articulation. Am J Med Sci 1861; 41:389-392.
7. Weaver JK, Dunn HK. Treatment of acromioclavicular injuries, especially complete acromioclavicular separation. J Bone Joint Surg Am 1972; 54:1187-1194
8. Weinstein DM, McCann PD, Mcllveen SJ, Flatow EL, Bigliani LU. Surgical treatment of complete acromioclavicular dislocations. Am J Sports Med 1995; 23:324-331.
9. Hessmann M, Gotzen L, Gehling H, Ritcher A. Reconstruction of complete acromioclavicular separations (Tossy III) using PDS-banding as augmentation: experience in 64 cases. ActaChisrBelg 1995; 95(3):147-151.
10. Jones HP, Lemos MJ, Schepsis AA. Salvage of failed acromioclavicular joint reconstruction using autogenous semitendinosus tendon from the knee: surgical technique and case report. Am J Sports Med 2001; 29:234-237.
11. LaPrade RF, Hilger B. Coracoclavicular ligament reconstruction using a semitendinosus graft for failed acromioclavicular separation surgery. Arthroscopy 2005; 21:1277.
12. Mazzocca AD, Santangelo SA, Johnson ST, Rios CG, Dumonski ML, Arciero RA. A biomechanical evaluation of an anatomical coracoclavicular Ligament reconstruction. Am J Sports Med 2006; 34:236-246.
13. Nicholas SJ, Lee SJ, Mullaney MJ, Tyler TF, McHugh MP. Clinical outcomes of coracoclavicular ligament reconstructions using tendon grafts. Am J Sports Med 2007; 35:1912-1917.
14. Lee SJ, Nicholas SJ, Akizuki KH, McHugh MP, Kremenic IJ, Ben-Avi S. Reconstruction of the coracoclavicular ligaments with tendon grafts: a comparative biomechanical study. Am J Sports Med 2003; 31:648-654.
15. Jari R, Costic RS, Rodosky MW, Debski RE. Biomechanical function of surgical procedures for acromioclavicular joint dislocations. Athroscopy 2004; 20:237-245.
16. Rockwood CA Jr, Williams GR Jr, Young DC. Disorders of the acromioclavicular joint. In: Rockwood CJ, Matsen FA III, eds. The Shoulder, 2nd ed. Philadelphia, PA: WB Saunders 1963; 56:486-495.

Role of magnetic resonance imaging in the evaluation of heart muscle in athletes

Francesco Palmieri
UOC of Radiology and Diagnostic Imaging P.O. Santa Maria delle Grazie ASL Na 3, Pozzuoli, Naples

Advanced cardiac imaging, using cardiac magnetic resonance imaging (MRI), is increasingly used in the work-up of athletes with suspected abnormalities on screening. This imaging modality produce highly accurate and reproducible structural and functional cardiac information. Cardiac MRI has the advantage of imaging without radiation exposure or the use of iodine-containing contrast agents, but is sometimes not possible due to claustrophobia or other contraindications. For patients less than 35 years of age, cardiac MRI is the first option after initial echocardiography for further assessment of cardiomyopathies, myocarditis, aortic stenosis and diseases of the aorta and coronary anomalies, which are major causes of sudden cardiac death in young athletes. In healthy individuals and endurance athletes, cardiac MRI systematically shows larger values of ventricular and atrial dimensions and volumes compared to echocardiography, while wall-thickness and wall-mass are smaller.
To evaluate left ventricular myocardial mass and function as well as ostial coronary artery cross-sectional area in endurance athletes, an athlete group of 12 highly trained rowers and a control group of 12 sedentary healthy subjects underwent MR examination. An ECG-gated breath-hold cine gradient-echo sequence was used to calculate myocardial mass, end-diastolic and end-systolic volumes, stroke volume, and cardiac output, all related to body surface area, as well as ejection fraction. A 3D fat-saturated ECG and respiratory-triggered navigator echo sequence was used to evaluate coronary arteries: left main (LM), left anterior descending (LAD), left circumflex (LCx), and right coronary artery (RCA). Magnetic resonance imaging is a useful tool in evaluating the myocardial hypertrophy and function of athlete's heart. Magnetic resonance angiography is a valuable noninvasive method to visualize the correlated cross-sectional area increase of the left coronary artery system.

References

Baggish AL, Yared K, Weiner RB, Wang F, Demes R, Picard MH, Hagerman F, Wood MJ. Differences in cardiac parameters among elite rowers and subelite rowers. Med Sci Sports Exerc 2010; 42:1215-1220.
Biffi A, Maron BJ, Di Giacinto B, Porcacchia P, Verdile L, Fernando F, Spataro A, Culasso F, Casasco M, Pelliccia A. Relation between training-induced left ventricular hypertrophy and risk for ventricular tachyarrhythmias in elite athletes. Am J Cardiol 2008; 101:1792-1795.
Corrado D, Pelliccia A, Bjornstad HH, Vanhees L, Biffi A, Borjesson M, Panhuyzen-Goedkoop N, Deligiannis A, Solberg E, Dugmore D, Mellwig KP, Assanelli D, Delise P, van-Buuren F, Anastasakis A, Heidbuchel H, Hoffmann E, Fagard R, Priori SG, Basso C, Arbustini E, Blomstrom-Lundqvist C, McKenna WJ, Thiene G. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol: consensus statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Eur Heart J 2005; 26:516-524.
Darling EA. The effects of training: a study of the Harvard University crews. Boston Med Surg J 1899; 161:229-233.
Fisman EZ, Pelliccia A, Motro M, Auerbach I, Frank AG, Tenenbaum A. Effect of intensive resistance training on isotonic exercise Doppler indexes of left ventricular systolic function. Am J Cardiol 2002; 89:887-891.
Gott PH, Roselle HA, Crampton RS. The athletic heart syndrome: five-year cardiac evaluation of a champion athlete. Arch Intern Med 1968; 122:340-344.
Harper RW, Mottram PM. Exercise-induced right ventricular dysplasia/cardiomyopathy: an emerging condition distinct from arrhythmogenic right ventricular dysplasia/cardiomyopathy. Heart Lung Circ 2009; 18:233-235. CrossRef Medline.
Jose AD, Collison D. The normal range and determinants of the intrinsic heart rate in man. Cardiovasc Res 1970; 4:160-167.
Levine BD. Exercise and Sports Cardiology. New York, NY: McGraw Hill 2001.
Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. Ann Intern Med 1975; 82:521-524. Abstract/FREE Full Text.
Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. Ann Intern Med 1999; 130:23-31. Abstract/FREE Full Text.
Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991; 324:295-301.
Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete's heart: a meta-analysis of cardiac structure and function. Circulation 2000; 101:336-344.
Reindell H, Roskamm H, Steim H. The heart and blood circulation in athletes. Med Welt 1960; 31:1557-1563.

Roeske WR, O'Rourke RA, Klein A, Leopold G, Karliner JS. Noninvasive evaluation of ventricular hypertrophy in professional athletes. Circulation 1976; 53:286-291.
Roskamm H, Reindell H, Musshoff K, Koenig K. Relations between heart size and physical efficiency in male and female athletes in comparison with normal male and female subjects. Arch Kreislaufforsch 1961; 35:67-102.
Roy A, Doyon M, Dumesnil JG, Jobin J, Landry F. Endurance vs. strength training: comparison of cardiac structures using normal predicted values. J Appl Physiol 1988; 64:2552-2557.
Sharma S, Maron BJ, Whyte G, Firoozi S, Elliott PM, McKenna WJ. Physiologic limits of left ventricular hypertrophy in elite junior athletes: relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. J Am Coll Cardiol 2002; 40:1431-1436.

Imaging of muscle lesions managment

Gianluigi Pasta
Traumatology Department and Angelo Bianchi Bonomi Hemophilia Center, IRCCS, Maggiore Hospital Foundation, Milan, Italy

The muscle-tendon pathology is a very common problem in the general population and even more in the sport population; a muscle-tendon injury may be very debilitating for an athlete and also if treated improperly can become chronic and risking the season and even athlete's career.
It is therefore essential the correct diagnosis and timely at the same time so you can start as early as possible a correct and appropriate therapeutic procedure and thus enabling the athlete to return to sport activity in the best possible way and as quickly as possible.
The imaging takes a direct role, it helps detect the presence of any abnormalities or muscle-tendon injury, is very important to characterize these changes and also for follow-up distance.
In this sense the imaging mainly relies on the ultrasound and magnetic resonance imaging and the development of these imaging techniques in recent years has allowed us to make great strides in the study of muscle-tendon pathology.
These methods allow the meantime, in fact, not only to highlight even minor alterations in the muscle-tendon and to characterize them in a precise manner by giving the clinician with all the information necessary to establish an appropriate treatment protocol, but also to provide the clinician with all the information necessary for an adequate follow-up at a distance.
In this report, then give way to a series of images that will allow us to understand such as ultrasound and magnetic resonance imaging, clearly linked to the clinic, have become so important not only in the study of muscle-tendon pathology but also in the follow-up distance alterations of muscle-tendon itself.

Resorbable biopolymeric scaffolds seeded with stem cells: evolution from bone application to tendon regeneration

Ernesto Reverchon, Stefano Cardea, Lucia Baldino, Iolanda De Marco
Department of Industrial Engineering, University of Salerno, Fisciano (SA), Italy

Tissue Engineering (TE) is the ensemble of techniques that have been developed to repair diseased or damaged tissue or to replace and regenerate part of organs of the human body; many of them are still at the stage of research proposal or have moved the first step of development. TE originates from reconstructive surgery used for the direct transplantation of a donor tissue to repair damaged tissues and organs. Many difficulties arise with direct transplantation, due to insufficient organ donors, rejection of the donor organ and pathogens transmission. An autogenic tissue engineering transplant (using patient own cells) would address most limitations of the direct transplantation and avoid difficulties concerning rejection and pathogens transmission. Therefore, constructing a tissue-engineered replacement *in vitro* can be an excellent alternative to the direct transplantation of donor organs (1, 2). A crucial step of the *in vitro* replacement is the scaffold fabrication. One opportunity is to use decellularized tissues and has been largely used to create heart valves, epithelial tissue and blood vessels; however, these "natural" scaffolds suffer of several limitations, among all infections transmission, and rejection. It is also possible to propose scaffolds obtained as 3-D synthetic constructs which serve as temporary support to allow isolated cells to form a new tissue, before being transplanted to the host. A resorbable biopolymeric matrix with sufficient mechanical strength, optimized architecture and suitable degradation rate, could perform this aim.
The temporary substitution of different biological materials (bones, cartilages, nerves, tendons, vessels, cardiac valves,

skin and tissues for the gastrointestinal system) requires different structural characteristics; but, all tissues share a series of common characteristics that have to be simultaneously obtained such as a highly regular and reproducible 3-D structure, a very high porosity, a suitable pore size and nanostructured surface, suitable mechanical properties, etc. All these specifications would be simultaneously required to allow the colonization of the scaffold by stem or specialized cells and all the requirements have to be fulfilled to avoid apoptosis or necrosis of the delicate biological guests. The techniques proposed to generated scaffolds for TE, at present, are not able to assure the obtainment of the described characteristics. In particular, the presence of solvent residue, low mechanical resistance and difficulty in 3-D scaffolds generations are the most common limitations of these techniques.
For these reasons, supercritical carbon dioxide (SC-CO_2) assisted processes have been proposed in this field; the general aim of SC-CO_2 assisted technique is to improve the traditional TE processes, using the characteristic properties of SC-CO_2 to control scaffolds morphologies thanks to the modulability of mass transfer properties and zero surface tension.
We successful applied supercritical drying procedures for bone TE applications: PLLA scaffolds were produced, mimicking the bone-like structure from macroscopic (3-D), microscopic (cellular structure) and nanometric (mimicking the extracellular matrix) point of view, with adequate mechanical resistance (3, 4). Human mesenchymal stem cells (hMSC) adhered, grown, migrated and differentiated during the cultivation in a perfusion reactor. They uniformly colonized all the structure, differentiated to osteoblasts, and some early bone formation proteins were consistently expressed (5).
Recently, an hardest challenge has been proposed by our group, that consists of the applications of SC-CO_2 processes to the formation of scaffolds suitable for tendon TE applications (6). Indeed, this target is particularly difficult to be obtained, due to the special characteristics of tendon structure; it is mainly formed by long collagen fibrils that are uniaxially oriented, organized in fiber bundles, according to a hierarchical structure. We are performing experiments on natural polymers, such as chitosan, elastin and collagen, with the aim of generating hierarchically organized fibrillar structures.

References

1. Langer R, Vacanti JP. Tissue engineering. Science 1993; 260:920-926.
2. Fuchs JR, Nasseri BA, Vacanti JP. Tissue engineering: a 21st century solution to surgical reconstruction. Annu Thorac Surg 2001; 72:577-591.
3. Reverchon E, Cardea S, Rapuano C. A new supercritical fluid-based process to produce scaffolds for tissue replacement. J Supercritical Fluids 2008; 45:365-373.
4. Cardea S, Pisanti P, Reverchon E. Generation of chitosan nanoporous structures for tissue engineering applications using a supercritical fluid assisted process. J Supercritical Fluids 2010; 54: 290-295.
5. Pisanti P, Yeatts AB, Cardea S, Fisher JP, Reverchon E. Tubular perfusion system culture of human mesenchymal stem cells on poly-L-lactic acid scaffolds produced using a supercritical carbon dioxide-assisted process. J Biomed Mater Res Part A 100A 2012; 2563-2572.
6. Reverchon E, Baldino L, Cardea S, De Marco I. Biodegradable synthetic scaffolds for tendon regeneration, Muscles, Ligaments and Tendons Journal 2012; 2:181-186.

ACL deficiency and cartilage lesion

Donato Rosa
Department of Orthopaedics, Faculty of Medicine and Surgery, University of Naples Federico II, Naples, Italy

The anterior cruciate ligament (ACL) is commonly injured, with a reported injury rate of 0.38 per 100 individuals (1). ACL injuries often cause knee joint laxity and instability, leading to pain and varying levels of disability. An ACL tear is often associated with meniscal tears, chondral lesions and the onset of post-traumatic osteoarthritis.
Maffulli et al. (2) reported a prevalence of chondral defects in patients with ACL tear of 43% (163 of 378 knees). Time from injury was significantly greater in these patients compared with patients with normal articular surface. The weight-bearing portion of the medial femoral condyle showed the highest frequency of articular cartilage lesions (77 of 163) knees.
Porter et al. (3) asserted that all patients with acute, traumatic ACL distruption sustained a chondral injury at the time of initial impact with sub-sequent longitudinal chondral degradation in compartments unaffected by the initial "bone bruise", a process that is accelerated at 5 to 7 years' follow-up.
In a recent systematic review (4) the incidence of severe articular cartilage injury in acute ACL tears was between 16% and 46%. Although surgical repair of damaged articular cartilage remains a significant challenge, a growing number of

options that have shown at least some efficacy in restoring hyaline-like material to the articular surface are available, such as microfracture, mosaicoplasty, ACI and osteochondral allograft transplantation. However results of combined ACL reconstruction and articular cartilage repair have only been reported for microfracture, osteochondral autograft transplantation and Autologous Chondrocyte Implantation showing good results at short-term follow-up.
Many studies have evaluated the long-term onset of osteoarthritis after anterior cruciate ligament injury. Neumann et al. (5) reported 15 per cent of radiographic tibio-femoral osteoarthritis 15 years after non-operative treatment of ACL injuries.
There is a great range of values in several studies for the prevalence of osteoarthritis after ACL reconstruction. In 2009 a systematic review published on the AJSM (6) reported an overall prevalence of tibio-femoral joint that varied between 1 and 100%. Differences in the prevalence of radiographic OA between individual studies can be attributed to different lengths of follow-up, different characteristics within the patient population, and different methods used to evaluate radiographic OA. Of 31 studies included in the review only two reported a differences of prevalence between surgically treated and non-surgically treated subjects.
Li et al. (7) described a prevalence of 39% of radiographic OA an average of 7.8 years after ACL reconstruction. For authors the strongest predictors of OA were obesity, increased length of follow-up, grade 2 or greater medial chondrosis, and prior or concurrent medial meniscectomy. Significant predictors were also male sex and grade 2 or greater chondrosis in patella-femoral joint while the use of hamstring graft was found to be a significant protective factor.

Conclusion

ACL injuries are often associated with chondral lesions and meniscal tears. Development of post-traumatic OA is a multifactorial event. An instable knee lead to a quickly progression of the cartilage lesion until the onset of a post-traumatic OA. Meniscectomy or meniscal tear accelerate this process. Alterated load characteristics, hematoma, subchondral bone bruises, inflammation, synovitis, capsular damage, surgical trauma also contribute to cartilage degeneration. ACL reconstruction restores knee stability and allows the patient to resume his o her preinjury activities but does not prevent the development of post-traumatic knee OA. More research needs to be done to determine factors that explain the development of OA and to describe the management of chondral lesions associated with ACL tears.

References

1. Myasaka KC, Daniel DM, Stone ML, Hirshman P. The incidence of knee ligament injuries in the general population. Am J Knee Surg 2001; 4:3-8.
2. Maffulli N, Binfield PM, King JB. Articular cartilage lesions in the symptomatic anterior cruciate ligament-deficient knee. Arthroscopy 2003; 19:685-690.
3. Potter HG, Jain SK, Ma Y, Black BR, Fung S, Lyman S. Cartilage Injury After Acute, Isolated Anterior Cruciate Ligament Tear: Immediate and Longitudinal Effect With Clinical/MRI Follow-up. Am J Sports Med 2012; 40:276.
4. Brophy RH, Zeltser D, Wright RW, Flanigan D. Anterior cruciate ligament reconstruction and concomitant articular cartilage injury: incidence and treatment. Arthroscopy 2010 Jan; 26(1):112-120.
5. Neuman P, Englund M, Kostogiannis I, Friden T, Roos H, Dahlberg LE. Prevalence of tibiofemoral osteoarthritis 15 years after non operative treatment of anterior cruciate ligament injury: a prospective cohort study. Am J Sports Med 2008; 36(9):1717-1725.
6. Oiestad BE, Engebresten L, Storheim K, Risberg MA. Knee Osteoarthritis after anterior cruciate ligament injury. A systematic review. Am J Sports Med 2009; 37(7):1434-1443.
7. Li RT, Lorenz S, Xu Y, Harner CD, Fu FH, Irrgang JJ. Predictors of radiographic knee osteoarthritis after anterior cruciate ligament reconstruction. Am J Sports Med 2011; 39(12):2595-2603.

Biological mechanisms of ESWT

Sergio Russo
Department of Orthopaedics, University of Naples "Federico II", Naples, Italy

The mechanisms of action of s.w. are twofold: mechanical and biological.
The first depends on a number of factors such as: number of shots, energy, focal size, gradient acoustic-impedance and cavitation.
The direct mechanisms are based on the idea that the crystalline aggregates, present in nonunion area, undergoes direct physical influence which leads to microlesions of heads of pseudoarthrosis and then formation of hematoma evolving in formation of callus.

Cavitation would play in this case positive effect but represents an important factor of tissue damage in other therapy on soft tissues.
The biological mechanisms instead assume an increasingly important role in tissue regeneration. In recent years, research has been directed to identify the way in which the energy of shockwave is converted into biological response.
The answer is in “Mechanic biology“, science that studies how cells subjected to load adapt them selves to biophysical stimuli by an active response. It represents the meeting point between classical mechanic and molecular biology. Its critical point is to identify those molecules that transmit to the cell-nucleus the mechanic signal which is subjected the cell-membrane. (Mechanotransduction-Meccanoresponses). The study therefore focused on mechanism by which cells convert mechanical signals into biochemical responses, and on the identification of molecules and cellular components mechanosensitive.
It is shown that ion channels, integrins, growth factors, filaments of the cytoskeleton, extracellular matrix and other molecules contribute to the response of the cell (mechanotransduction).
In recent years, numerous studies draw attention to the increase of VEGF and nitricoxide, substances produced in response to sw therapy that increase blood flow in the treated area for vasodilatation and new angiogenesis. Other molecules whose increase is highlighted are TGF b1, PCNA, BMP2, and ostocalcina. All these factors, through the increase of vascularization, stimulation of bone and tendon metabolism leading to tissue regeneration.
A new experimental studies on the effects of sw on the heart of rats shows new interesting data on stem cells.
C-kit (also known as CD117) positive cardiac cells represent a population of cardiac stem cells and progenitors/precursors of cardiac cell lineages in the developing and adult heart in contrast to control hearts, immunohistochemical analysis of the rat myocardium at 3 months after SW treatment revealed the presence of c-kit–positive cell clusters.
So cardiac SW treatment increased the number of c-kit–positive cells 2.65-fold compared to control hearts.

References

Di Meglio F, Russo S, Montagnani S et al. Cardiac shock wave therapy: assessment of safety and a new insights into mechanisms of tissue regeneration. Journal of cellular and molecular medicine Vol XX, No X, 2011; 1-7.
Gotte G, Russo S, Marlinghaus E, Musci G, Suzuki H. Short-time non-enzymatic nitric oxide synthesis from L-Arginine and hydrogen peroxide induced by shock waves treatment. FEBS Letters 520 2002; 153-155.
Mariotto S, Russo S, Suzuki H et al. Extracorporeal shock waves: from lithotripsy to anti-inflammatory action by NO production. Nitric Oxide 12 2005; 89-96.
Nurzynska D, Russo S, Montagnani S et al. Shock waves activate in vitro cultured progenitors and precursors of cardiac cell lineages from the human heart.
Ultrasound in Med. & Biol, Vol. 34, No 2, 2008; 334-342.

Return of sport after ligament/tendon surgery of foot/ankle

Amol Saxena
Department of Sports Medicine, Palo Alto Medical Foundation, Palo Alto, CA, USA

Key words: athlete, Achilles, Peroneal, Lisfranc’s, plantar fasciitis, anti-gravity treadmill, ESWT

Background

Patients who sustain injuries or undergo surgery wish to know their period of “down-time” and convalescence. Return to activity (RTA) is often not cited in most musculoskeletal publications. A review of current literature that does document RTA is reviewed in order to better help guide the medical professional and patient. Speeding up the RTA may be possible with certain processes.

Methods

A retrospective review of several studies pertaining to common foot and ankle surgeries is presented. This includes surgeries for Achilles rupture and tendinopathy, Peroneal tendinopathy, plantar fasciitis and Lisfranc’s ligament repair. Classification of activity level is also presented and analyzed. Return to activity as defined and reported in studies on the above topics is presented.

Results

RTA from Achilles tendon surgery ranges from 1.5 months to seven months with Peritenolysis having the fastest RTA and delayed rupture repair being the slowest. Many studies with Achilles surgery RTA show women and those with a higher BMI have slower RTA while athletes have a faster RTA as compared to the rest of the population. Peroneal tendinopathy patients have an RTA ranging from 2.5 months for retinaculum repair to more than three months for tendon repair without osteotomy. Isolated Lisfranc ligament repair with a bioresorbable, metallic or syndesmotic suture device has an RTA of four or more months. Plantar Fasciotomy has been shown to have a RTA of 2.8 months when performed endoscopically. One study on an anti-gravity treadmill utilized post-operatively for Achilles surgery patients showed a faster RTA by two weeks. Lab studies on the utilization of ESWT post-surgery and tendinopathy also have shown faster "healing".

Clinical significance

When study design includes RTA parameters and activity level, definite time frames can be helpful to the clinician and patient. More studies should include this. The information presented will help set realistic expectations for recovery and return to sports with foot and ankle surgery. New rehabilitation items such as an anti-gravity treadmill and ESWT may help speed up RTA, but further study is needed.

References

Curtis M, Myerson M, Szura B. Tarsometatarsal joint injuries in the athlete. Am J Sports Med 1993; 21(4):497-502.
DeOrio M, Erickson M, Usuelli F, Easley M. Lisfranc injuries in sport/ Foot Ankle Clin 2009; 14(2):169-186.
Ferran NA, Oliva F, Maffulli N. Recurrent subluxation of the peroneal tendons. Sports Med 2006; 36:839-846.
Han SH, Lee JW, Guyton GP, Parks BG, Courneya JP, Schon LC. J. Leonard Goldner Award 2008. Effect of extracorporeal shock wave therapy on cultured tenocytes. Foot Ankle Int. 2009; 30(2):93-98.
Harwood M, Raikin S. A Lisfranc Fracture Dislocaton in a Football Player. J Am Board Fam Pract 2003; 16(1): 69-72.
Leach RE, Schepsis AA, Takai H. Long-term results of surgical management of Achilles tendinitis in runners. Clin Orthop Relat Res 1992:208-212.
Maffulli N, Longo UG, Testa V, Oliva F, Capasso G, Denaro V. Italian translation of the VISA-A score for tendinopathy of the main body of the Achilles tendon. Disabil Rehabil 2008; 30:1635-1639.
Maffulli N, Testa V, Capasso G, Bifulco G, Binfield PM. Results of percutaneous longitudinal tenotomy for Achilles tendinopathy in middle- and long-distance runners. Am J Sports Med 1997; 25:835-840.
Maffulli N, Testa V, Capasso G, Oliva F, Sullo A, Benazzo F, Regine R, King JB. Surgery for chronic Achilles tendinopathy yields worse results in nonathletic patients. Clin J Sport Med 2006; 16:123-128.
Maffulli N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc 2001; 9:42-47.
Maffulli N, Tallon C, Wong J, Lim K, Bleakney R. Early Weightbearing and Ankle Mobilization after Open Repair of Acute Midsubstance Tears of the Achilles Tendon. Am J Sports Med 2003; 31:692-700.
Maffulli N, Longo U, Maffulli G et al. Achilles tendon ruptures in elite athletes. Foot Ankle Int 2011; 32(1):9-14.
Maffulli N, Ferran NA, Oliva F, Testa V. Recurrent subluxation of the peroneal tendons. Am J Sports Med 2006; 34:986-992.
Nunley J, Vertullo C. Classification, Investigation and Management of Midfoot Sprains: Lisfranc Injuries in the Athlete. Am J Sports Med 2002; 30(6):871-878.
Ogawa BK, Thordarson DB. Current concepts review: peroneal tendon subluxation and dislocation. Foot Ankle Int 2007; 28:1034-1040.
Oliva F, Ferran N, Maffulli N. Peroneal retinaculoplasty with anchors for peroneal tendon subluxation. Bull Hosp Jt Dis 2006; 63:113-116.
Oliva F, Saxena A, Ferran N, Maffulli N. Peroneal Tendinopathy In International Advances in Foot & Ankle Surgery 2011 Springer (Saxena A ed.) pp 193-210.
Orhan Z, Ozturan K, Guven A, Cam K. The effect of extracorporeal shock waves on a rat model of injury to tendo Achillis. A histological and biomechanical study. J Bone Joint Surg Br 2004; 86(4):613-618.
Parekh SG, Wray WH 3rd, Brimmo O, Sennett BJ, Wapner KL. Epidemiology and outcomes of Achilles tendon ruptures in the National Football League. Foot Ankle Spec. 2009; 2(6):283-286.

Porter D, McCarroll J, Knapp E, Torma J. Peroneal tendon subluxation in athletes: fibular groove deepening and retinacular reconstruction. Foot Ankle Int 2005; 26:436-441.
Robinson JM, Cook JL, Purdam C, Visentini PJ, Ross J, Maffulli N, Taunton JE, Khan KM. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. Br J Sports Med 2001; 35: 335-341.
Roles NC, Maudsley RH. Radial tunnel syndrome: resistant tennis elbow as nerve entrapment. J Bone Joint Surg Br 1972; 54:499-508.
Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion Achilles tendinopathy: a randomized controlled trial. Am J Sports Med 2009; 37:463-470.
Saxena A. Guest Editorial. Stand up and start counting. J Foot Ankle Surg 2012; 51(1):
Saxena, A. Return to athletic activity after foot and ankle surgery: a preliminary report on select procedures. J Foot Ankle Surg 2000; 39:114-119.
Saxena A, Ramdath S, O'Halloran P, Gerdesmeyer L, Gollwitzer H. Extra-corporeal pulsed-activated therapy (low-energy sound-wave) for Achilles Tendinopathy: a prospective study. J Foot Ankle Surg 2011; 50(3):315-319.
Saxena A. Results of chronic Achilles tendinopathy surgery on elite and nonelite track athletes. Foot Ankle Int 2003; 24:712-720.
Saxena A. Return to athletic activity after foot and ankle surgery: a preliminary report on select procedures. J Foot Ankle Surg 2000; 39:114-119.
Saxena A, Cheung S. Surgery for chronic Achilles tendinopathy. Review of 91 procedures over 10 years. J Am Podiatr Med Assoc 2003; 93:283-291.
Saxena A, Ewen B, Maffulli N. Rehabilitation of the operated Achilles tendon. J Foot Ankle Surg 2011.
Saxena A, Granot A. Use of an anti-gravity treadmill in the rehabilitation of the operated Achilles tendon: a pilot study. J Foot Ankle Surg 2011.
Saxena A, Fullem B. Plantar fascia ruptures in athletes. Am J Sports Med. 2004; 32(3):662-665.
Saxena A. Uniportal endoscopic plantar fasciotomy: a prospective study on athletic patients. Foot Ankle Int 2004; 25(12):882-889.
Saxena A. Bioabsorbable screws for reduction of Lisfranc's diastasis in athletes. J Foot Ankle Surg 2005; 44(6):445-449.
Saxena A, Wolf SK. Peroneal tendon abnormalities. A review of 40 surgical cases. J Am Podiatr Med Assoc 2003; 93:272-282.
Saxena A, Cassidy A. Peroneal tendon injuries:an evaluation of 49 tears in 41 patients. J Foot Ankle Surg 2003; 42(4):215-220.
Saxena A, Ewen B. Peroneal subluxation: surgical results in 31 athletic patients. J Foot Ankle Surg 2010; 49(3):238-241.
Saxena A, Granot A. Post-operative physical therapy for foot and ankle surgery. In International Advances in Foot & Ankle Surgery 2011 Springer (Saxena A ed.)
Saxena A, Longo U, Denaro V, Maffulli N. Insertional and midsubstance Achilles tendinopathy International Advances in Foot & Ankle Surgery 2011 Springer (Saxena A ed.) pp179-192.
Silbernagel KG, Thomee R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med 2007; 35:897-906.
Silbernagel KG, Thomee R, Karlsson J. Cross-cultural adaptation of the VISA-A questionnaire, an index of clinical severity for patients with Achilles tendinopathy, with reliability, validity and structure evaluations. BMC Musculoskelet Disord 2005; 6:12.
Tallon C, Coleman BD, Khan KM, Maffulli N. Outcome of surgery for chronic Achilles tendinopathy. A critical review. Am J Sports Med 2001; 29:315-320.
Willits K, Amendola A, Bryant D, et al. Operative versus Non-operative Treatment of Acute Achilles Tendon Ruptures: A Multicenter Randomized Trial Using Accelerated Functional Rehabilitation. J. Bone Joint Surg. Am 2010; 92(17): 2767-2775.

Clinical practice guide for muscle injuries. FC Barcelona experience (1)

Luís Til
Sports Medicine. Futbol Club Barcelona

Muscular injuries are very frequent in sports, especially in football. The most recent epidemiological studies show that muscular injuries represent more than 30% of all injuries (1.8-2.2/1,000 hours of exposure), which means that a professional football team suffers an average of 12 muscular injuries per season, equivalent to more than 300 lost sporting days (2). In other professional sports like basketball and handball the incidence is also high, although not reaching the figures shown in football.

Despite their high frequency and the interest in finding solutions, there is little scientific evidence on aspects as important as prevention and treatment. We will outline some weak points below.
The diagnosis of muscular injuries is based on clinical medicine, fundamentally on symptomatology and especially on the anamnesis of the injury mechanism and physical examination. Imaging studies through the musculoskeletal echography and magnetic resonance are complementary studies, despite the fact that they could be increasingly more useful when confirming a diagnosis or especially when living a prognosis. There is no sufficiently specific biochemical marker available that could help with the diagnosis of the seriousness and the definitive prognosis of each of the different muscular injuries.
Treatment guidelines for muscular injuries do not follow a unique model, despite the fact that the different alternatives have been modified very little. Finally, new expectations have been raised thanks to research within the field of biological repair and regeneration.
Certain primary and secondary prevention programmes can reduce the incidence of suffering muscular injuries, but scientific evidence is still limited and it has only been possible to verify them in certain groups of sportsman.
The objective of this document (1) is to record the diagnostic, therapeutic and preventative approaches that should be taken when faced with the various muscular injuries suffered by the football players of Barcelona FC. This is not intended as an exhaustive review of muscular pathology in sport, but rather a working document that is clear, practical and comprehensive. The protocols are based on current knowledge from recent years in the daily work of dealing with these types of injuries. The guide also explain the sequential steps model applied in the muscle injuries treatment. Finally the paper presents the criteria for return to play after the injury, where the decision is taken based on experience, strength test, and ultrasound/MRI. The decisión for return to play is modulated through the factors proposed by Orchard as high and low risk (3).
The guide is becoming obsolete and it is necessary a new version. In 2012 are appearing new proposals for the classification of muscle injuries based on the nature of the injury (4), the severity of the injury and the affected tissue (5).

Reference

1. Clinical Practice Guide for muscular injuries. Apunts Med Sport 2009; 164:179-203.
2. Ekstrand J, Hägglund M, Waldén M. Epidemiology of Muscle Injuries in Professional Football (Soccer). Am J Sports Med 2011; Vol. XX, No. X, XXXX: 1-7.
3. Orchard J, Best TM, Verrall GM. Return to play following muscle strains. Clin J Sport Med 2005; 15:436-441.
4. Mueller-Wohlfahrt HW, Haensel L, Mithoefer K et al. Terminology and classification of muscle injuries in sport: a consensus statement. Br J Sports Med 2012; 0:1-9. doi:10.1136/bjsports-2012-091448.
5. Chan O, Del Buono A, Best TM, Maffulli N. Acute muscle strain injuries: a proposed new classification system. Knee Surg Sports Traumatol Arthrosc published online on July 7th 2012; DOI 10.1007/s00167-012-2118-z.

Posterolateral corner reconstruction. A La Prade modification

Gian Felice Trinchese, Antonio Toro
Orthopaedic and Traumatology Unit, "Villa Malta" Hospital, Sarno, Salerno, Italy

Introduction

Injuries to the posterolateral corner (PLC) of the knee produce a spectrum of pathologic states of ligamentous laxity resulting in various amounts of varus, external rotation, and posterior translation of the knee. Consequently, they may result in significant functional disability. For years, posterolateral corner injuries have been a challenging diagnostic and therapeutic problem for the orthopaedic surgeon. Various surgical techniques to treat posterolateral knee instability have been described. The goal of this study is to determine the clinical outcome of PLC reconstruction following the La Prade technique modified by the authors.

Materials and methods

We retrospectively studied 5 patients who underwent a PLC reconstruction with the La Prade technique modified by the authors from september 2007 to november 2009. Three patient had a concomitant ACL rupture, one an isolated PLC injury and one a residual posterolateral instability after a previous ACL reconstruction. In the modified technique the reconstruction of PLC was performed with a single femoral tunnel, using tibialis anterior allograft for the popliteus reconstruction and peroneus longus allograft for the collateral ligament and popliteofibular reconstruction. In the cases of concomitant ACL lesion the reconstruction was performed using a tibialis posterior allograft. Mean age was 30.2 years (range 16-40). Mean follow-up was 21.4 months (range 9-35). All patients underwent the same postoperative protocol.

Results

We evaluated varus stress, recurvatum, dial, manual Lachman and pivot shift tests, maximum anterior tibial translation by Rolimeter, Lysholm and IKDC forms. All patients showed negative dial and recurvatum tests; one showed 1+ on varus stress. Mean Lysholm score was 93 points (range 82-100). On IKDC evaluation 1 patient was grade A and four were grade B.

Conclusions

Although clear limits of the study due to small number of cases, the modified La Prade technique showed good results in terms of clinical outcome, restoring varus and rotation stability of knees in treatment of chronic PLC injury.

References

Apsingi S, Nguyen T, Bull AM, Unwin A, Deehan DJ, Amis AA. A comparison of modified Larson and 'anatomic' posterolateral corner reconstructions in knees with combined PCL and posterolateral corner deficiency. Knee Surg Sports Traumatol Arthrosc 2009; 17:305-312.
Clancy WG Jr, Sutherland TB. Combined posterior cruciate ligament injuries. Clin Sports Med 1994; 13:629-647.
Fanelli GC, Larson RV. Practical management of posterolateral instability of the knee. Arthroscopy 2002; 18(2 Suppl 1):1-8.
Harner CD, Ho¨ her J, Vogrin TM, Carlin GJ, Woo SL. The effects of a popliteus muscle load on in situ forces in the posterior cruciate ligament and on knee kinematics: a human cadaveric study. Am J Sports Med 1998; 26:669-673.
Hughston JC, Jacobson KE. Chronic posterolateral instability of the knee. J Bone Joint Surg Am 1985; 67:351-359.
La Prade RF, Johansen S, Agel J, Risberg MA, Moksnes H, Engebretsen L. Outcomes of an anatomic posterolateral knee reconstruction. J Bone Joint Surg Am 2010; 92:16-22.
La Prade RF, Resig S, Wentorf F, Lewis JL. The effects of grade III posterolateral knee complex injuries on anterior cruciate ligament graft force: a biomechanical analysis. Am J Sports Med 1999; 27:469-475.
La Prade RF, Tso A, Wentorf FA. Force measurements on the fibular collateral ligament, popliteofibular ligament, and popliteus tendon to applied loads. Am J Sports Med 2004; 32:1695-1701.
La Prade RF, Wozniczka JK, Stellmaker MP, Wijdicks CA. Analysis of the static function of the popliteus tendon and evaluation of an anatomic reconstruction: the "fifth ligament" of the knee. Am J Sports Med 2010; 38:543-549.
McCarthy M, Camarda L, Wijdicks CA, Johansen S, Engebretsen L, La Prade RF. Anatomic posterolateral knee reconstructions require a popliteofibular ligament reconstruction through a tibial tunnel. Am J Sports Med 2010; 38:1674-1681.
Nau T, Chevalier Y, Hagemeister N, Deguise JA, Duval N. Comparison of 2 surgical techniques of posterolateral corner reconstruction of the knee. Am J Sports Med 2005; 33:1838-1845.
Noyes FR, Barber-Westin SD, Albright JC. An analysis of the causes of failure in 57 consecutive posterolateral operative procedures. Am J Sports Med 2006; 34:1419-1430.
Noyes FR, Barber-Westin SD. Surgical restoration to treat chronic deficiency of the posterolateral complex and cruciate ligaments of the knee joint. Am J Sports Med 1996; 24:415-426.
Pasque C, Noyes FR, Gibbons M, Levy M, Grood E. The role of the popliteofibular ligament and the tendon of popliteus in providing stability in the human knee. J Bone Joint Surg Br 2003; 85:292-298.
Schinhan M, Bijak M, Unger E, Nau T. Electromyographic study of the popliteus muscle in the dynamic stabilization of the posterolateral corner structures of the knee. Am J Sports Med 2011; 39:173-179.
Sigward SM, Markolf KL, Graves BR, Chacko JM, Jackson SR, McAllister DR. Femoral fixation sites for optimum isometry of posterolateral reconstruction. J Bone Joint Surg Am 2007; 89:2359-2368.
Veltri DM, Deng XH, Torzilli PA, Maynard MJ, Warren RF. The role of the popliteofibular ligament in stability of the human knee: a biomechanical study. Am J Sports Med 1996; 24:19-27.
Yoon KH, Bae DK, Ha JH, Park SW. Anatomic reconstructive surgery for posterolateral instability of the knee. Arthroscopy 2006; 22:159-165.

Chronic exertional compartment syndrome in lowerleg

Antonio Turmo
Department of Sports Physiology and Medical Services CAR FC Barcelona

Chronic Exertional Compartment Syndrome (CECS) of thelegis well documented in the literature since it was first described by Mavor in 1956.
CECS usually refers to myoneural ischaemia due to a reversible increase in tissue pressure with in a my of as cial compartment. Exercise causes anabnormal in crease in intramuscular pressure (IMP), leading to vascular occlusion and ischemic pain in the involved muscles.
The CECS is the leading cause of chronic pain in the leg in athletes and the second cause if it is located in the front.
Diagnosis of CECS require: 1) history of exercise-induced pain/symptoms, 2) appropriater e production of symptoms and pain during the exercise test, 3) intramuscular pressure (ICP) at rest of > 15 mm Hg and/or intramuscular pressure of > 30 mm Hg 1–2 min after the end of the exercise test, and/or intramuscular pressure of > 20 mm Hg 5 min after the end of the exercise test.
We present a series of 31 patients with CECS in 69 compartments. Its distribution is bilateral in 49% of cases and is the most frequent compartment to above (46%). The sport with the high est incidence is soccer (n = 11) and track and field (n = 8).
Mean ICP registered in our casesis 24 ± 9,7 in rest, 39±20 after exercise test and 29±13 at 5 min.
Some of the cases were considered CECS despite not having the classic diagnostic criteria. Fur ther treatment by fasciotomy confirmed the diagnosis.

Conclusions

The indication of the diagnostic test should be made from the clinical process. There is a delay in diagnosis CECS from the time of on set of symptoms.
The traditional criteria for the diagnosis should be reviewed for a better and earlier diagnosis.

References

1. Boody A. Accuracy in the Measurement of Compartment Pressures: A Comparison of Three Commonly Used Devices. J BoneJoint Surg 2005; 87-A:2415-2422.
2. Edmundsson D. Chronic compartment syndrome also affects non athletic subjects: A prospective study of 63 cases with exercise-induced lower leg pain Acta Orthopaedica 2007; 78(1):136-142.
3. Padhiar N.Chronic exertional compartment syndrome of the foot. Br J SportsMed 2011; 45:e1.
4. Pedowitz RA. Modified Criteria for the Objective Diagnosis of Chronic Compartment Syndrome of theleg. Am J SportsMed 1990; 18:35-40.
5. Roberts A.The validity of the diagnostic criteria used in chronic exertional compartment syndrome: A systematic review. Scand J MedSciSports 2012; 585-595.
6. Styf J. Intramuscular pressure measurement during exercise. OperativeTechniques in Sports Medicine 1995; 3(4):243-249.

Evidence based physical therapy protocols for the treatment of muscles injuries

Alessandro Valent[1], Mattia Marazzi[2]
[1] *Specialist in Physical and Rehabilitation Medicine, Modena Football Club 1912, Italy*
[2] *Medical Student, University of Padova, Italy*

Muscular injuries are very frequent during sport activities. For example, in soccer they represent the first cause of injury (about 30 percent of all injuries) and time loss (1, 3). Muscular injuries could be classified in direct traumas (contusion and laceration) and indirect traumas (cramp, contracture, delayed onset muscle soreness, elongation and tear) (2, 3). The most serious event is muscle tear that is a rupture of a variable number of muscular fibers, always accompanied by edema and local bleeding. Muscle tear is generally determined by indirect trauma and can be classified in three degrees, depending on the severity of the injury.

The approach to this type of injury should be based on a correct diagnosis and on a correct therapeutic strategy. Ultrasonography and Magnetic Resonance are essential for diagnosis (4), together with the objective examination.
Muscle repair is a complex process that includes three phases, regulated by many molecules and cellular events (5):

1) Degeneration phase,
2) Regeneration phase,
3) Remodeling phase.

The current treatment principles of muscle injuries are mainly based on experimental findings, with limited scientific evidence (2, 3, 5, 22).
Physical Therapies such as Therapeutic Ultrasound, Laser Therapy, Diathermy, Electrotherapy and Hyperthermia are frequently used in clinical practice for muscle injuries, although there is low scientific evidence of their use.
However, the lack of scientific evidence does not mean that the Physical Therapies are ineffective, but it only means that no randomized-controlled trials have yet been made (these kinds of trials are in fact very difficult to perform in physiotherapy).
In the last years technology has actually developed new and really effective Physical Therapies.
Therapeutic Ultrasound reduces pain and swelling following muscle injury, but it does not have proven therapeutic effects on muscle regeneration (6, 7, 20, 23, 24).
Low Level Laser Therapy (LLLT) has many therapeutic effects on injured muscle: anti-inflammatory, anti-oxidative, analgesic and bio-stimulating (increasing the production of ATP and the synthesis of DNA, RNA and proteins). These therapeutic effects improve the quality of the healing process and reduce the time of recovery (8-11).
A Brazilian study (11) revealed that both LLLT and Ultrasound therapies have positive effects on muscle metabolism after muscular injury in rats, but LLLT seems to produce a better response.
Moreover, Valent in his clinical trial (12) has highlighted that High Intensity Laser Therapy (Pulsed wave Nd-Yag laser) is more effective than traditional therapy with CO_2 Laser and Ultrasound in the treatment of grade I muscular tears.
Electrotherapy has an analgesic effect, in addition it promotes muscle relaxation. This second effect restores muscular function, which is very useful in tears and D.O.M.S. (13, 26).
Temperature increase (14-17) (heat therapy), induced by Capacitive-Resistive Diathermy or 434 MHz Hyperthermia, stimulates the repair process, decreases pain and induces an active hyperemia that promotes the drainage of toxic wastes. Moreover, hyperthermia reduces muscle stiffness.
In case of grade I muscular tear, according to our experience, we divide the rehabilitation protocol into three different phases:

1) Acute phase (1st - 3rd day),
2) Sub acute phase (4th – 10th day),
3) Healing phase (after the 10th day).

We use the Physical Therapies in all the clinical phases as a support and as a complement to the rehabilitation techniques (2, 3, 5, 18-22).
In the acute phase, in agreement with international literature, we suggest “cold” therapies according to the RICE protocol (18, 20-22). Indeed if heat is introduced too early, it can determine problematic complications such as fibrosis and calcifications (2-4, 23). In this phase we recommend Cryoultrasound therapy, analgesic Electrotherapy and Low Intensity Laser Therapy.
In the sub acute phase (4-8 days after trauma) it’s possible to introduce heat therapies to determine hyperemia and deep bio-stimulation. According to our experience the most recommended heat therapies are the Endogenous Thermotherapy (Capacitive-Resistive Diathermy and 434 MHz Hyperthermia) and High Intensity Laser Therapy.
In the healing phase, that mostly takes place on the field, Physical Therapies are principally used to relax muscles and relieve muscle fatigue. The most used Physical Therapies in clinical practice are Electrotherapy and Capacitive-Resistive Diathermy.
Some possible complications of muscular tears and contusions are fibrosis, myositis ossificans, calcifications, encysted hematoma, thrombophlebitis and re-injuries (4).
For the treatment of fibrosis, calcification and myositis ossificans we propose Ultrasound (6, 7, 23, 24), Extracorporeal Shock Wave Therapy (ESWT) (25) and 434 MHz Hyperthermia. Instead for encysted hematoma we prefer to use Ultrasound and 434 MHz Hyperthermia.
Finally, in case of functional muscle alterations (DOMS, elongation or contracture) we use especially Electrotherapy (26), High Intensity Laser therapy and Endogenous Thermotherapy.

References

1. Ekstrand J, Hägglund M, Waldén M. Epidemiology of Muscle Injuries in Professional Football (Soccer). Am J Sports Med, Vol. 39, No. 6, 2011.
2. Nanni G, Vincentelli F. Le lesioni Muscolari. In Roi G.S., Della Villa S. (eds): Health, prevention and rehabilitation in soccer. Calzetti Mariucci editore (Torgiano PG), 2007; 10.
3. Roi GS. Gli infortuni muscolari dello sportivo. Medicina Riabilitativa 2008,22:185-195.

4. Valent A. Infortuni muscolari: diagnosi attraverso le immagini. Sport&Medicina, 4, 2011.
5. Jarvinen TAH et Al. Muscle injuries. Biology and treatment. Am J Sports Med, vol. 33, No. 5, 2005.
6. Markert CD, Merrick MA, Kirby TE, Devor ST. Nonthermal ultrasound and exercise in skeletal muscle regeneration. Arch Phys Med Rehabil 2005; 86:1304-1310.
7. Wilkin LD, Merrick MA, Kirby TE, Devor ST. Influence of therapeutic ultrasound on skeletal muscle regeneration following blunt contusion. Int J Sports Med. 2004; 25:73-77.
8. Pertille A, Macedo AB, Oliveira CPV. Evaluation of muscle regeneration in aged animals after treatment with low-level laser therapy. Rev Bras Fisioter 2012.
9. De Souza TO, Mesquita DA, Ferrari RA, Dos Santos Pinto D Jr, Correa L, Bussadori SK, Fernandes KP, Martins MD. Phototherapy with low-level laser affects the remodeling of types I and III collagen in skeletal muscle repair. Lasers Med Sci. 2011; 26(6):803-814.
10. Dos Santos DR, Liebano RE, Schiavinato Baldan C, Bordello Masson I, Paranhos Soares R, Esteves Junior I. The low-level laser therapy on muscle injury recovery: literature review. J Health Sci Inst 2010; 28(3):286-288.
11. Renno AC, Toma RL, Feitosa SM, Fernandes K, Bossini PS, De Oliveira P, Parizotto N, Ribeiro DA. Comparative effects of low-intensity pulsed ultrasound and low level laser therapy on injured skeletal muscle. Photomed Laser Surg 2011; 29:5-10.
12. Valent A. Muscular lesions in athlete: case comparison between Hilterapia and traditional Therapy. Energy for Health vol.3, n°3, 2009; 22-25.
13. McLoughlin TJ, Snyder AR, Brolinson PG, Pizza FX. Sensory level electrical muscle stimulation: effect on markers of muscle injury. Br J Sports Med 2004; 38:725-729.
14. Giombini A, Giovannini V, Di Cesare A, Pacetti P, Ichinoseki-Sekine N, Shiraishi M, Naito H, Maffulli N. Hyperthermia induced by microwave diathermy in the management of muscle and tendon injuries. Br Med Bull 2007; 83:379-396.
15. Ganzit GP, Stefanini L, Stesina G. Nuove metodiche nel trattamento della patologia muscolo articolare dell'atleta: la TECAR terapia. Medicina dello Sport, 2000; 53(4):361-367.
16. Ichinoseki-Sekine N, Naito H, Norio S, Yuji O et al. Changes in Muscle Temperature induced by 434 MHz Microwave Hyperthermia. Br J Sport Med 2007; 41:425-429.
17. Suros Romagos J, Suros Frigola J, Parrilla Forcada I. Profound diathermy uhf in sport traumatological pathology. Br J Sports Med 2011; 45:2.
18. Bleakley CM, Glasgow P, Webb MJ. Cooling an acute muscle injury: can basic scientific theory translate into the clinical setting? Br J Sports Med 2012; 46(4):296-298.
19. Nakano J, Yamabayashi C, Scott A, Reid WD. The effect of heat applied with stretch to increase range of motion: a systematic review. Phys Ther Sport 2012; 13(3):180-188.
20. Järvinen TA, Järvinen TL, Kääriäinen M, Äärimaa V, Vaittinen S, Kalimo H et al. Muscle injuries: optimizing recovery. Best Pract Res Clin Rheumatol 2007; 21:317-331.
21. Valle X. Clinical practice guide for muscular injuries: epidemiology, diagnosis, treatment and prevention. Br J Sports Med 2011; 45:2.
22. Orchard JW, Best TM, Mueller-Wohlfahrt HW, Hunter G, Hamilton BH, Webborn N et al. The early management of muscle strains in the elite athlete: best practice in a world with a limited evidence basis. Br J Sports Med 2008; 42:158-159.
23. Miller DL, Smith NB, Bailey MR, Czarnota GJ, Hynynen K, Makin IR. Overview of therapeutic ultrasound applications and safety considerations. J Ultrasound Med 2012; 31(4):623-634.
24. ter Haar G. Therapeutic applications of ultrasound. Prog Biophys Mol Biol 2007; 93(1-3):111-129.
25. Buselli P, Coco V, Notarnicola A, Messina S, Saggini R, Tafuri S, Moretti L, Moretti B. Shock waves in the treatment of post-traumatic myositis ossificans. Ultrasound Med Biol 2010; 36(3):397-409.
26. Curtis D, Fallows S, Morris M, McMakin C. The efficacy of frequency specific microcurrent therapy on delayed onset muscle soreness. J Bodyw Mov Ther 2010; 14(3):272-279.

Effects of ESW on tenocytes and ECM synthesis

Vincenzo Visco[1], Mario Vetrano[2]
[1] Department of Clinical and Molecular Medicine, Sant'Andrea Hospital, La Sapienza University of Rome, Italy
[2] Physical Medicine and Rehabilitation Unit, Sant'Andrea Hospital, La Sapienza University of Rome, Italy

In the last two decades, focused extracorporeal shock-wave therapy (ESWT) has been used in the treatment of tendinopathies. Despite its extensive clinical application, the biological mechanisms underlying the clinical effectiveness of ESWT in musculoskeletal disorders have not yet been defined so far and their elucidation might hold important clues for ameliorating tendon repair strategies.
Several authors suggest that ESWT effect can be ascribed to the transduction of the acoustic shock wave signal into biochemical signals, and this process has been defined as "mechanotransduction" (1).
Experimental studies performed through the evaluation of histological and biomechanical parameters showed that ESWT

induces tissue regeneration and facilitates tendon healing. Previous reports have demonstrated that ESWT can increase the number of neovessels at the normal tendon-bone junction (2), through the release of growth factors and some other active substances (3). More recent studies on fibroblasts *in vitro* and *in vivo* showed that shockwaves activate fibroblasts proliferation rate, collagen synthesis and gene expression for growth factors and/or cytokines (4-8).
To better elucidate the therapeutical effects of ESWT on chronic tendinopathies, we analysed for the first time the ESWT-mediated effects on primary cultured human tenocytes (9). Because several authors emphasized the fundamental role of cell proliferation and collagen synthesis for the healing process during tendinopathies, we have measured those variables in relation to the effects of ESWT on cultured tenocytes.
We obtained primary cultures of human tenocytes from explants of semitendinosus tendons, harvested during anterior cruciate ligament (ACL) reconstructions, because we assumed that the biological activities of such cells could be representative to any other tendon derived cells. An energy flux density of 0.14 mJ/mm^2 was applied to the cultures, primarily considering that medium energies showed a clinical efficacy *in vivo*, and we investigated their behavior over a 12 days period following the exposure.
We observed that ESWT significantly interferes with the overall cell morphology, by impairing de-differentiation of the cells. Furthermore, shock wave-induced growth promoting effects were analyzed by the MTT (tetrazolium) colorimetric assay and by expression of the proliferation marker Ki67. Lastly we found a significant increase in collagen (mainly type I) synthesis by ESWT-stimulated tenocytes compared to control cells.
Our findings seem to indicate that human cultured tenocytes are metabolically “activated” by ESWT and significantly induced to proliferate. They synthesize *in vitro* increasing amounts of total collagen (mainly type I) compared to untreated cells, which in contrast enter in a dedifferentiation program, characterized by heterogeneous cell morphology and lower secretive activity.
In summary, we demonstrated that a single dose of ESWT applied on primary cultured human tenocytes induces -over a 12 days period- a clear enhancement of cell growth and collagen synthesis. The principal findings of this work provide a possible explication of ESW-mediated therapeutical benefits in patients affected by chronic tendinopathies.

References

1. Frairia R, Berta L. Biological effects of extracorporeal shock waves on fibroblasts. A Review. MLTJ 2011; 1:137-146.
2. Wang CJ, Huang HY, Pai CH. Shock wave enhances neovascularization at the tendon–bone junction. J Foot Ankle Surg 2002; 41:16-22.
3. Wang CJ, Wang FS, Yang KD, Huang CS, Hsu CC, Yang LC. Shock wave therapy induced neovascularization at the tendon–bone junction. A study in rabbits. J Orthop Res 2003; 21:984-989.
4. Berta L, Fazzari A, Ficco AM et al. Extracorporeal shock waves enhance normal fibroblast proliferation in vitro and activate mRNA expression for TGF beta1 and for collagen types I and III. Acta Orthop 2009; 80:612-617.
5. Wang FS, Wang CJ, Huang HJ et al. Physical shock wave mediates membrane hyperpolarization and ras activation for osteogenesis in human bone marrow stromal cells. Biochem Biophys Res Comm 2001; 287:648-655.
6. Wang FS, Yang KD, Chen RF et al. Extracorporeal shock wave promotes growth and differentiation of bone-marrow stromal cells towards osteoprogenitors associated with induction of TGF-β1. J Bone Joint Surgery (Br) 2002; 84:457-461.
7. Martini L, Fini M, Giavaresi G et al. Primary osteoblasts response to shock wave therapy using different parameters. Artif Cells Blood Substit Immobil Biotechnol 2003; 31:449-466.
8. Chen YJ, Wang CJ, Yang KD et al. Extracorporeal shock waves promote healing of collagenase-induced Achilles tendinitis and increase TGF-beta1 and IGF-I expression. J Orthop Res 2004; 22:854-861.
9. Vetrano M, d’Alessandro F, Torrisi MR, Ferretti A, Vulpiani MC, Visco V. Extracorporeal shock wave therapy promotes cell proliferation and collagen synthesis of primary cultured human tenocytes. Knee Surg Sports Traumatol Arthrosc 2011; 19(12):2159-2168.

Clinical experience of ESWT in orthopaedics and traumatology

Maria Chiara Vulpiani
Physical Medicine and Rehabilitation Unit, Sant’Andrea Hospital, La Sapienza University of Rome, Italy

In the last decade, focused ESWT has been used in the treatment of tendinopathies and orthopaedic pathologies. Despite its success in clinical application in the treatment of soft tissues and orthopaedic pathologies, the exact mechanism of ESWT is not yet fully understood. Several authors suggest that its effect can be ascribed to the transduction of the acoustic shock wave signal into a biological signal. Studies performed through the evaluation of histological and biomechanical parameters on animals showed that ESWT induces tissue regeneration and facilitates tendon healing after trauma. These experimental studies have pointed out significant increase in neovascularization, reduced adhesion formation and increase in hydroxyproline levels on tendon tissues treated with ESWT. In addition, in the tendons treated with ESWT, a greater force is required to damage the tendon when compared with the control group (1, 2). Biopsies performed on canine Achilles tendons 4 and 8 weeks after treatment with ESWT showed neovascularisation growth and blood supply improvement, a result that was not found in the control group (3). In a later study, neovascularization in the bone-tendon junction was confirmed by the presence of such angiogenic markers as vessel endothelial growth factor, endothelial nitric oxide synthase and by endothelial cell proliferation indicated by proliferating cell nuclear antigen (PCNA). The results showed earlier release of angiogenic growth factors and increase in neo-vessels and tissue proliferation about four weeks after treatment, that persisted for up to 12 weeks (4). Recently, it has been shown that the shock waves promoted cell growth and collagen synthesis of primary cultured human tenocytes (5, 6). The efficacy of ESWT is strictly related to energy level and number of impulses used during treatment (7). Effects of ESWT appear, in fact, to be dose-related. In our experience we used a medium-low energy flux density and different energy levels for insertional tendinopathies and tendon belly pathologies (8, 9). Higher levels of energy (0.12-0.40 mJ/mm2) were used in insertional tendinopathy, since the focal zone is the tendon-bone junction and related calcifications, if any, while in tendon belly pathologies a lower energy level was used (0.08-0.33 mJ/mm2). While several studies are available in literature on the effects of ESWT on animal models, fewer are the clinical studies.

Basic scientific studies have shown that application of shock waves produces a biological effect on treated bone. Using animal models, SWT has been shown to induce healing of fracture defects, usually by creating micro-fractures and bleeding in the treated bone (10-21). It has been hypothesized that the treated tissues release local growth factors such as bone morphogenic proteins which ultimately recruit stem cells and mesenchymal progenitor cells and initiate the healing cascade (22-25). That said, the precise biological mechanism of action remains only partially understood. Shock wave application clearly has an osteogenic effect on treated bones. Maier et al. used a rabbit model to demonstrate that application of shock waves with an energy flux density of 0.5 mJ/mm2 resulted in new periosteal bone formation in treated femurs (26). In another series of trials, selective destruction of osteocyctes, microfractures of trabeculae, and minor bleeding in the medullary space were observed in rabbits treated with SWT. Approximately 3 weeks after treatment, histological and biochemical analysis revealed thickening of the cortex, increase in the number of bony trabeculae, and a significant increase in the number and activity of treated osteoblasts (14). In regards to fracture nonunions, Johannes et al. used a canine nonunion model to study the effects of high energy SWT on cortical bone (12). All of the treated subjects reached radiographically observable bony union 12 weeks after the shock wave treatment whereas untreated control subjects had radiographically persistent nonunions at termination of the study.

Uncontrolled clinical trials using SWT as a method to treat fracture nonunions have been promising. Schaden et al. reported on 115 patients with nonunions or delayed unions of various fractures treated with high energy SWT and immobilization. Follow-up ranged from 3 months to 4 years. Overall, 87 patients (75.7%) were reported to have healed fractures (27).

Rompe et al. reported their experience using high energy shock wave therapy to treat 43 patients with either a tibial or femoral diaphyseal nonunion. They noted bony consolidation in 31 of 43 cases (72%) after an average of four months post-treatment (28). Wang et al. used high-energy SWT as a treatment for 72 nonunions of long bone fractures. Twelve month follow-up was available for 55 patients. For the entire cohort, they noted an overall healing rate of 80% (44 of 55 patients) (29).

The results from our experience suggest that SWT can be a safe and effective treatment for stable fracture nonunions. Like other studies, our data shows that trophic nonunions respond better to SWT than atrophic nonunions (30). Additional randomized, prospective clinical trials are needed to substantiate these conclusions.

High energy ESWT can be effective in the management of orthopaedic pathologies. Early diagnosis and staging are, therefore, of extreme importance to obtain benefits from ESWT (31). Additionally, ESWT is non-invasive, has a low complication rate, does not require hospitalization and has a relatively low cost compared to other types of conservative and surgical approaches, with a relatively short time of application. In literature, various protocols have been used as well as number of sessions, impulses and energy levels. In the mentioned studies, the parameters used were different, in particular concerning the energy levels provided. In our clinical practice, we apply shock waves according to the guidelines of the Italian Society of Shock Wave Therapy (Società Italiana Terapia con Onde d'Urto, SITOD) and the variability of energy flux density can be referred to some individual parameters of a single patient that we have to consider: degree of tendinopathy, presence of calcifications, and tolerance to pain. In this case, it is necessary that focalized ESWT be applied by expert physicians.

Further studies are needed with larger cohorts of patients using the homogenous classification system, and standardized treatment protocols to further assess the effectiveness of ESWT in the management of orthopaedic disease.

Extracorporeal Shock Wave Treatment (ESWT) improves *in vitro* functional activities of ruptured human tendon derived-tenocytes.

References

1. Orhan Z, Alper M, Akman Y, Yavuz O, Yalciner A. An experimental study on the application of extracorporeal shock waves in the treatment of tendon injuries: preliminary report. J Orthop Sci 2001; 6:566-570.
2. Orhan Z, Ozturan K, Guven A, Cam K. The effect of extracorporeal shock waves on a rat model of injury to tendo Achillis. A histological and biomechanical study. J Bone Joint Surg Br 2004; 86:613-618.

3. Wang CJ, Huang HY, Pai CH. Shock wave-enhanced neovascularization at the tendon-bone junction: an experiment in dogs. J Foot Ankle Surg 2002; 41:16-22.
4. Wang CJ, Wang FS, Yang KD, Weng LH, Hsu CC, Huang CS et al. Shock wave therapy induces neovascularization at the tendon-bone junction. A study in rabbits. J Orthop Res 2003; 21:984-989.
5. Vetrano M, d'Alessandro F, Torrisi MR, Ferretti A, Vulpiani MC, Visco V. Extracorporeal shock wave therapy promotes cell proliferation and collagen synthesis of primary cultured human tenocytes. Knee Surg Sports Traumatol Arthrosc 2011; 19(12):2159-2168.
6. Leone L, Vetrano M, Ranieri D, Raffa S, Vulpiani MC, Ferretti A, Torrisi MR, Visco V. Extracorporeal Shock Wave Treatment (ESWT) improves in vitro functional activities of ruptured human tendon-derived tenocytes. PLoS One. In Press. http://dx.doi.org/ 10.1371/journal.pone.0049759.
7. Orhan Z, Cam K, Alper M, Ozturan K. The effects of extracorporeal shock waves on the rat Achilles tendon: is there a critical dose for tissue injury? Arch Orthop Trauma Surg 2004; 124:631-635.
8. Vulpiani MC, Vetrano M, Savoia V, Di Pangrazio E, Trischitta D, Ferretti A. Jumper's knee treatment with extracorporeal shock wave therapy: a long-term follow-up observational study. J Sports Med Phys Fitness 2007; 47(3):323-328.
9. Vulpiani MC, Trischitta D, Trovato P, Vetrano M, Ferretti A. Extracorporeal shockwave therapy (ESWT) in Achilles tendinopathy. A long-term follow-up observational study. J Sports Med Phys Fitness 2009; 49(2):171-176.
10. Haupt G, Haupt A, Ekkernkamp A et al. Influence of shock waves on fracture healing. Urology 1992; 39(6):529-532.
11. Kaulesar Sukul DM, Johannes EJ, Pierik EG et al. The effect of high energy shock waves focused on cortical bone: an in vitro study. J Surg Res 1993; 54(1):46-51.
12. Johannes EJ, Kaulesar Sukul DM, Matura E. High-energy shock waves for the treatment of nonunions: an experiment on dogs. J Surg Res 1994; 57(2):246-252.
13. Forriol F, Solchaga L, Moreno JL et al. The effect of shockwaves on mature and healing cortical bone. Int Orthop 1994; 18(5):325-329.
14. Delius M, Draenert K, Al Diek Y et al. Biological effects of shock waves: in vivo effect of high energy pulses on rabbit bone. Ultrasound Med Biol 1995; 21(9):1219-1225.
15. Augat P, Claes L, Suger G. In vivo effect of shock-waves on the healing of fractured bone. Clin Biomech (Bristol, Avon) 1995; 10(7):374-378.
16. Haist J. Die Osteorestauration via Stoßwellenanwendung. Eine neue Möglichkeit zur Therapie der gestörten knöchernen Konsolidierung. In: Chaussy C, Eisenberger F, Jochum D, Wilbert D. eds. Die Stoßwelle – Forschung und Klinik. Attempo, Tuebingen 1995; 157-161.
17. Rompe JD, Kullmer K, Vogel J et al. Extracorporeal shock-wave therapy. Experimental basis, clinical application. Orthopade 1997; 26(3):215-228.
18. Vogel J, Rompe JD, Hopf C et al. High-energy extracorporeal shock-wave therapy (ESWT) in the treatment of pseudarthrosis. Z Orthop Ihre Grenzgeb 1997; 135(2):145-149.
19. Graff J, Pastor J, Richter KD. Effect of high energy shock waves on bony tissue. Urol Res 1988; 16(3):252-258.
20. Ikeda K, Tomita K, Takayama K. Application of extracorporeal shock wave on bone: preliminary report. J Trauma 1999; 47(5):946-950.
21. Uslu MM, Bozdogan O, Guney S et al. The effect of extracorporeal shock wave treatment (ESWT) on bone defects. An experimental study. Bull Hosp Jt Dis 1999; 58(2):114-118.
22. Wang CJ, Huang HY, Chen HH et al. Effect of shock wave therapy on acute fractures of the tibia: a study in a dog model. Clin Orthop Relat Res 2001; (387):112-118.
23. Maier M, Milz S, Tischer T et al. Influence of extracorporeal shock-wave application on normal bone in an animal model in vivo. Scintigraphy, MRI and histopathology. J Bone Joint Surg Br 2002; 84(4):592-599.
24. Wang FS, Yang KD, Kuo YR et al. Temporal and spatial expression of bone morphogenetic proteins in extracorporeal shock wave-promoted healing of segmental defect. Bone 2003; 32(4):387-396.
25. Maier M, Averbeck B, Milz S et al. Substance P and prostaglandin E2 release after shock wave application to the rabbit femur. Clin Orthop Relat Res 2003; (406):237-245.
26. Wang FS, Yang KD, Chen RF et al. Extracorporeal shock wave promotes growth and differentiation of bone-marrow stromal cells towards osteoprogenitors associated with induction of TGF-beta1. J Bone Joint Surg Br 2002; 84(3):457-461.
27. Schaden W, Fischer A, Sailler A. Extracorporeal shock wave therapy of nonunion or delayed osseous union. Clin Orthop Relat Res 2001; (387):90-94.
28. Rompe JD, Rosendahl T, Schollner C et al. High-energy extracorporeal shock wave treatment of nonunions. Clin Orthop Relat Res 2001; (387):102-111.
29. Wang CJ, Chen HS, Chen CE et al. Treatment of nonunions of long bone fractures with shock waves. Clin Orthop Relat Res 2001; (387):95-101.
30. Vulpiani MC, Vetrano M, Conforti F, Minutolo L, Trischitta D, Furia JP, Ferretti A. Effects of Extracorporeal Shock Wave Therapy on Fracture Nonunions. Am J Orthop 2012; 41(9):E122-E127.
31. Vulpiani MC, Vetrano M, Trischitta D, Scarcello L, Chizzi F, Argento G, Saraceni VM, Maffulli N, Ferretti A. Extracorporeal shock wave therapy in early osteonecrosis of the femoral head: prospective clinical study with long-term follow-up. Arch Orthop Trauma Surg 2012; 132(4):499-508.