

# Disseminated necrotic mediastinitis spread from odontogenic abscess: our experience

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## Summary

**Aims.** Deep neck infections are rare but potentially fatal complication of pulp abscess of the teeth. If an infection can progress rapidly from a toothache to a life threatening infection, then it is critical that dentists be able to recognize the danger signs and identify the patients who are at risk. Mediastinitis is a severe inflammatory process involving the connective tissues that fills the intracellular spaces and surrounds the organs in the middle of the chest. This pathology has both an acute and a chronic form and, in most cases, it has an infectious etiology. This study want to expose the experience acquired in the Oral and Maxillo-facial Sciences Department, Policlinico Umberto I, "Sapienza" University of Rome, regarding two clinical cases of disseminated necrotizing mediastinitis starting from an odontogenic abscess.

**Methods.** We report two clinical cases of disseminated necrotic mediastinitis with two different medical and surgical approaches. The radiographic and photographic documentation of the patients was collected in the pre-and post-operatively. All patients underwent a CT scan and MRI.

**Results.** Mediastinitis can result from a serious odontogenic abscess, and the extent of its inflammation process must be never underestimated. Dental surgeons play a key role as a correct diag-

nosis can prevent further increasing of the inflammation process.

**Conclusions.** A late diagnosis and an inadequate draining represent the major causes of the elevated mortality rate of disseminated necrotizing mediastinitis.

**Key words:** odontogenic abscess, mediastinitis, pulpal abscess.

## Introduction

Mediastinitis is a severe inflammatory process involving the connective tissues that fills the intracellular spaces and surrounds the organs in the middle of the chest. This pathology has both an acute and a chronic form and, in most cases, it has an infectious etiology. Major sources of mediastinal infections can be: direct contamination, haematic or lymphatic dissemination, spreading from the neck or retroperitoneal space, from the lung or the pleura (1).

The disseminated necrotic mediastinitis (DNM) is, on the other hand, a suppurative acute infection of the mediastinal tissue always coming from infective pathologies of the neck or odontogenic abscesses (2). This rare pathology includes a high mortality rate (between 25 and 40%) which is made worse by delayed diagnosis and therapy (2). Whenever an odontogenic or cervical abscess fails to respond to the therapy, it spreads into the mediastinum and the pleura, causing a severe and generalized sepsis.

Patients suffering from this pathology are admitted to hospitals in very critical conditions and in many cases the therapy is not effective even if started on time. In fact, DNM mortality rate is still very high (about 40%) and comes with many complications, such as septic shock, occlusion of respiratory airways, jugular thrombosis and erosion of major vascular structures with consequent blood overflowing (3).

With this research we want to show the experience acquired at the Maxillo-facial Department of Policlinico Umberto I, "Sapienza" University of Rome, regarding two patients with disseminated necrotizing mediastinitis starting from an odontogenic abscess. Their clinical character, diagnosis and treatment were analyzed. A protocol is recommended for managing suspected cases.

## Patients and methods

A retrospective review was conducted of patients who

were diagnosed with odontogenic abscess and disseminated necrotizing mediastinitis from December 2009 to December 2011. Two patients were included in this study. Their etiology, associated systemic diseases, bacteriology, radiology, treatments, duration of hospitalization, complications and outcomes were reviewed. The diagnosis of disseminated necrotizing mediastinitis was confirmed through clinical examinations and thoracic CT scans.

**Patient 1.** A 26-year-old patient admitted to the Emergency Room with fever (39° C), a tense-elastic bruised area in the right buccal region and spread side-cervical bilateral, with massive pain, hyperemic skin and bilateral trismus. CT scan results showed a voluminous abscess mass involving the right parotid and laterocervical area up to the carotid bifurcation, and the bilateral under-chin and sub-mandibular area that was displacing the laryngeal lamp to the left up to the plan over the tongue. There was also a thickening and edema of right genial area soft tissues, but there was no remarkable fluid collection in the mediastinum, or presence of lymphadenopathy. Later on, the patient underwent an incision in the under chin area, with drainage of purulent materials; the pull-out of the third molar with drainage of purulent materials on the level of the alveolus and finally the positioning of a drainage. Cultures exams found gram positive cocci and gram negative bacilli; in addition, streptococcus anginosus and ralsstonia picketti, both sensitive to ampicillin (MIC<2), were isolated.

There was an improvement of the patient's general condition, together with lower body temperature and an enhancement of ABG values. The mediastinal extension with the fluid collection in the right para-tracheal area, was 30x35 mm and there was a spreading for 15 cm in pre- and retro-tracheal spaces. Afterwards, he was treated with beta-lactam antibiotic 2 g every 8 hours and aminoglycoside 1,5 g every 24 hours, while therapy was started with enoxaparin sodium 6000 U every 24 hours. Due to the persistence of breathing difficulties, the patient underwent a right videothoracoscopy that showed the lung completely adherent to the mediastinum.

A right thoracotomy was then performed, which revealed parietal pleura with intense inflammatory reaction. On the pleural dome there was a purulent abscess, that was drained washing the pleural cavity with iodine solution and with the resection of the lung apex, hemostasis and drainage catheters positioning. So the patient started improving his condition. 7 days after surgery another CT of neck and thorax was performed that showed a big reduction of the many superior abscesses with normal and symmetric airways. The right pre-tracheal and the small localized fluid collection at the mediastinum disappeared. There was just a fluid collection behind the right bronchus and pulmonary vein (2 x 2 cm).

After three weeks of antibiotic therapy, the patient was discharged in good clinical conditions, after the removal of draining wires.

**Patient 2** A 50-year-old patient was admitted to the maxillofacial Department with a phlegmon in the bilateral latero-cervical area and in the anterior-superior mediastinal area. The patient suffered from a slight toothache at the left mandibular mid-arcade, and was therefore under antibiotic therapy with clindamycin and amoxicillin.

Afterwards, he had fever and on clinical examination he presented swelling in the left mandibular and sub-mandibular area, lasting for about a month, loss of function of ATM, fever with shivers and breathing difficulties. At CT of the neck was performed. This showed a voluminous swelling (6 cm approximately) with visible gas-filled bubbles in the left sub-mandibular and under chin area, with impregnated tissues in the deep peripheral area.

The patient underwent a tracheotomy and the phlegmon drainage through a percutaneous laterocervical and sub-mandibular incision.

He was then administered an antibiotic therapy with clindamycin and amoxicillin; due to the persistence of his critical conditions, after 7 days he underwent a neck and chest CT that showed many abscesses emerging in the left latero-cervical sub-mandibular area with gas-filled bubbles. These bubbles were spreading upwards, involving the masseter muscle, and downwards involving the parotid up to the biking area, involving also the para-pharyngeal space. The collection was extending medially to the pre-vertebral area and right latero-cervical space, where a fluid collection with air bubbles was detected, involving left supra-clavear area and anterior-superior mediastinum, where fluid collections, delimited by vascular walls, occupied the right para-tracheal region (10 cm). For these reasons the patient underwent a toilette and phlegmon drainage through laterocervical access.

Later on, a new angio-CT of the neck and the chest was performed: it showed a slight improvement of abscess lesions, that fairly change at the latero-cervical level, and the presence of increasing abscess lesions at the anterior-superior mediastinum, localized mostly to the right side. Because of these complications the patient had a right cervicotomy and a right thoracotomy 10 days after hospitalization in order to evacuate those fluid collections. There was an improvement of general conditions and no fever. Microbiological culture found the presence of Streptococcus and Candida. Thorax X-ray didn't show any important alteration. CT of neck and thorax showed parenchymatous inflammatory tissue (about 5 cm) in the left sub-mandibular area spreading to parapharyngeal and vascular space, which caused a light compression of the left lateral wall. There was also a little abscess (3 cm) in the laterocervical area which obliterated the left pyriform sinus. In the mediastinum there was inflammatory tissue (4 cm) that spread in the right para-tracheal area, but was reduced compared to previous control (8 cm). The post-surgery course showed a slow improvement of the patient's clinical conditions. He underwent regular washings with disinfected and peroxide while daily medications were carried on with the neck exposed and plugged with

iodoform gauze. Finally, after 2 weeks, the patient was discharged in good general condition.

## Discussion

Descending mediastinitis is an acute infection secondary to severe cervical infection, and odontogenic abscess is the most common primary infection. Infections in the head and neck can spread down into the retropharyngeal space (71% of cases) or carotid sheath (21% of cases), gravity-facilitated, breathing and negative intrathoracic pressure (4, 5).

According to the International Literature, the clinical cases above described, show most of the potential emergencies that might arise from an odontogenic infection. Their complications can cause systemic onsets that might compromise to the death the patient's general conditions. It was reported that 49% of patients died during their treatment in the first know article published about mediastinitis in 1938 (6).

An example of most critical clinical evolution of odontogenic infections, that usually spread directly, is Ludwig's Angina, a necrotic fasciitis of head and neck that finally reaches the mediastinum causing mediastinitis (7).

Odontogenic infections can be divided into: den-toalveolar, periodontal, and facial plans. They can spread through contiguity causing jaws sinus, mandibular osteomyelitis, cavernous sinus thrombophlebitis, internal jugular vein thrombophlebitis, erosion of the internal carotid artery, Ludwig's Angina and descending necrotic mediastinitis (7). They can also spread in an hematogenic way causing an infective endocarditis, infections of articular prothesis, spondylodiscitis, streptococcus sepsis (in patients affected by leukemia) (7).

Delayed diagnosis and inadequate drainage processes were primary underlying factors contributing to this high mortality (8). The earlier detection and treatment as a result of contrast-enhanced CT imaging contributed to decrease in mortality rate.

**Microbiology:** intravenous antibiotic therapy is the initial treatment, and empirical medication is often used. The bacteriological results of these patients show that pus culture and sensitivity tests are essential. The most common organism isolated seem to be streptococcus sanguis, mutans and actinomyces viscosus, that live mostly on the tooth area (9), also streptococcus salivarius and veillonella, that are easily detected on the tongue and in the mucous membrane, together with fusobacterium, bacteriodes pigmentati and spirochete anaerobie. They establish compounds within the gum recess (6). Dental cavities, gum diseases and several forms of periodontitis are associated to different patterns of bacterial compounds, such as mutans streptococcus, usually detected on teeth with cavities (9, 10).

**Anatomic considerations:** diagnosis of disseminated necrotic mediastinitis mandates that the relation-

ship between mediastinitis and oropharyngeal infection is clearly established. Primary sites of infection are periodontal abscess, retropharyngeal abscesses and peritonsillar abscess. According to Wheatley et al. (11) most common primary oropharyngeal infection is odontogenic with mandibular second or third molar abscess.

Soft tissues infections with odontogenic origins tend to spread through the tissue surrounding the teeth, through *locus minoris resistentiae*, up to all closest areas (12).

**Clinical symptoms:** periodontal infections include gum disease, periodontitis, fascial plans infections can cause trismus, pain of the body and the corner of the mandible, swelling and disfagia. It can spread: through inferior hole of the orbit or through the orbit itself, causing a cellulitis with proptosis, optic neuritis and abducent nerve palsy. The infection can also spread in extra-oral position and present like an evident swelling, a low pain, trismus, and just few general symptoms. Potentially it could spread to periorbital tissues and paranasal sinuses. Moreover it could be an intraoral extension without swelling and drainage. We could find a swelling in parotid space with pain and clinical signs. Potentially it could spread to posterior pharyngeal space (so called "danger space"). Finally the infection can affect sublingual space, causing sub-chin edema with rash and tense skin bilaterally.

**Ludwig's angina:** it's a neck and mouth floor soft tissue gangrenous cellulitis (4). Both patients, at the moment of hospitalization, were suffering from Ludwig's Angina.

Diagnostic criteria are:

- 1) bilateral involvement
- 2) submandibular and sublingual areas involvement
- 3) rapidly progressive cellulitis with no abscess or lymphatic involvement
- 4) the infection must be begin from the mouth floor, and in 80% of cases, from the II and III molar (4, 13).

Gram positive cocci and anaerobics are the etiologic agents involved with the infection. Leaning factors are: cavities, recent dental treatments, diabetes mellitus, malnutrition, alcoholism, immunodepression and trauma (4).

On a clinical examination, it can be identified as a severe pathology with fever, septic status, pain wooden-hard swelling, uplist and protrusion of the tongue, trismus (50%), disfagia, disartria, disфонia, neck stiffness (13). Clinical complications are due to airways obstruction.

According to the International Literature, therapy includes the constant monitoring of airways, difficult intubation and a preventive tracheotomy (4).

The following medications must be administered:

- Desametasone (10 mg bolus followed by 4 mg every 6 h to 48 h)
- Nebulized epinephrine (1 ml 1:1000 in 5 ml of physiological solution 0.9%)

- High doses of antibiotics intravenously: G penicillin + metronidazole or clyndamicine; amoxicillin/clavulanic acid
- Patient must be remained seated
- Surgical draining and/or tissue decompression from on side (13).

Clinical symptoms of Ludwig's angiooma infections of the facial plans can affect the lateropharyngeal area, that is divided through the stiloideus process into front compartment (soft and connective tissues) and back compartment (vascular-nervous wreck). This partition is fundamental to detect the different syndrome that may arise (4, 13).

Front compartment syndrome, is characterized by fever with shiver, high pain, trysmus, swelling of the mandibular corner, dysphagia and medial displacement of the pharyngeal lateral wall (4, 7).

Back compartment syndrome, is characterized by septic status, low pain, cranial nerves involvement (IX-XII), airways obstruction due to laryngeal edema, internal jugular thrombosis, internal carotid erosion (7).

Retro-pharyngeal infections present in back visceral compartment (esophagus, trachea, thyroid), that goes all the way down to the superior mediastinum. It also compromises the "danger space" that goes from the back of the mediastinum to the diaphragm. Clinical symptoms include high fever, dysphagia, dispnea, neck stiffness, esophagus regurgitation, spontaneous draining of airways with suction, laryngeal spasm, bronchial erosion and jugular vein thrombosis (4).

According to the Literature and with regard to our experience, although we analyzed two patients only, where serious inflammatory processes are already ongoing, we usually adopt the diagnostic and therapeutic pattern as indicated in the charts below.

## Complications

We can divide them in:

- 1) Hematogenic complications: transient bacteremia, infective endocarditis, infections of articular prosthesis (7).
- 2) Contiguity complications: jaws sinus, mandibular osteomyelitis, cavernous sinus thrombophlebitis, internal jugular vein thrombophlebitis (Lemierre Syndrome), erosion of the internal carotid artery and descending necrotic mediastinitis (7).

## Lemierre Syndrome

It's a serious oro-pharynx infection with bacteremia, internal jugular vein pyogenic thrombophlebitis with septic embolism. It's unusual (<1/1.000.000), but it has been increasing during last year because changes in antibiotic therapy for high airways infections. It arises in young people, after EBV infection or pharyngitis caused by streptococco. The main etiologic agent is fusobacterium necrophorum. Main clinical findings are: high fever, cervical pain, septic shock, thrombocitopeny (14).

## Mediastinitis

It's an acute or chronic inflammatory disease of mediastinic connective tissue. Acute mediastinitis are due to Gram positive cocci which cause a mediastinic suppuration; they can be primitive or often follow esophageal lesions for trauma, foreign body or tumors; they are characterized by fever, retrosternal pain, leukocytosis; they can affect mediastinic organs (lung, heart, large vessels) with really serious complications.

Therapy is based on antibiotics administration, checking general conditions and removing the cause if possible.

Chronic mediastinitis are characterized by fibrotic sclerosis of mediastinic space, with serious functional complications for circulation and breathing; they often follow granulomatous lesions (TBC, syphilis) in the mediastinic connective tissue or autoimmunitary disease. We must also consider that a mediastinitis could cause a mediastinic syndrome (Tabs. 1-3) (15).

Table 1. Diagnostic examination.

### Microbiology:

- Collection, through an aspiration needle, of deep samples
- Immediate ground transfer for anaerobics and aerobics
- Bacteriological examination

### Imaging:

- Scintigraphy with marked leukocytes: useful to mark eventual osteomielitis
- Ultrasound: useful to analyzed collections from the surface
- Lateral neck X-Ray: it allows to estimate tracheal deviation or compression, and the presence of gas within necrotic tissues
- CT: useful for evaluation of head, neck and facial areas (must be extended to the chest in any cases)

Table 2. Surgical approach.

- Sick toot extraction
- Surgical drainage and necrotic tissues removal
- Drainage optima Timing
- Systematic monitoring of fascial areas

Table 3. Medical Therapy.

- Antibiotic therapy (antibiotics retain local diffusion and prevent the hematogen diffusion):
  - A. Clinical findings:
    - a. Localised infection, no sepsis
      - ✓ Clyndamicine
      - ✓ Amoxicillin/clavulanic acid or ampicillin/sulbactam
      - ✓ Metronidazole (no in monotherapy)
    - b. Life-threatening infection
      - ✓ Piperacillin/tazobactam or ticarcillin/clavulanic acid or carbapenemics
- Broad spectrum empirical therapy for immunodepressed people

## Conclusions

From data collected at the Oral and Maxillo-facial Sciences Department, Policlinico Umberto I "Sapienza" University of Rome and according to the International Literature, we reached the following conclusions: mediastinitis remains a potential lethal infection that can result from a serious odontogenic abscess, and the extent of its inflammation process must be never underestimated. Dental surgeons play a key role as a correct diagnosis can prevent further increasing of the inflammation process. They should identify as soon as possible the infection symptoms, especially when it's reaching deeper tissues. These symptoms are: fever, mouth floor swelling, inferior mandibular swelling, asymmetric pharyngeal walls swelling, trismus.

A late diagnosis and an inadequate draining represent the major cause of the elevated mortality rate of DNM. The use of CT scan is highly recommendable in cases with deep cervical inflammation in order to identify still showing no sign. To effectively contrast the elevated mortality rate, an aggressive surgical drainage, the removal of residues from neck area and mediastine drainage through a postero-lateral toracotomy performed by a multi-function medical team are necessary.

Moreover, it is extremely important to research and register all symptoms of an imminent collapse of airways, that include muffled voice, tongue or mouth floor uplift, beside the inability to tolerate or even swallow salivary secretions.

## References

1. Kinzer S, Pfeiffer J, Becker S, Ridder GJ. Severe deep neck space infections and mediastinitis of odontogenic origin: clin-

- ical relevance and implications for diagnosis and treatment. *Acta Otolaryngol.* 2009;129:62-70.
2. Estrera AS, Landay MJ, Grisham JM, Sinn DP, Platt MR. Descending necrotizing mediastinitis. *Surg Gynecol Obstet.* 1983;157:545-52.
3. Kiernan PD, Hernandez A, Byrne WD, Bloom R, Dicicco B, Hetrick V, Graling P, Vaughan B. Descending cervical mediastinitis. *Ann Thorac Surg.* 1998;65:1483-88.
4. Furst IM, Ersil P, Caminiti M. A rare complication of tooth abscess-Ludwig's angina and mediastinitis. *J Can Dent Assoc.* 2001;67:324-27.
5. Mihos P, Potaris K, Gakidis I, Papadakis D, Rallis G. Management of descending necrotizing mediastinitis. *J Oral Maxillofac Surg.* 2004;62:966-972.
6. Pearse HE. Mediastinitis following cervical suppuration. *Ann Surg.* 1938;108:588-611.
7. Furst IM, Ersil P, Caminiti M. A rare complication of tooth abscess-Ludwig's angina and mediastinitis. *J Can Dent Assoc.* 2001;67:324-27.
8. Mihos P, Potaris K, Gakidis I, Papadakis D, Rallis G. Management of descending necrotizing mediastinitis. *J Oral Maxillofac Surg.* 2004;62:966-972.
9. Lautermann J, Lehnerdt G, Beiderlinden M, Sudhoff H. Infections of the deep neck spaces with accompanying mediastinitis. *Laryngorhinootologie.* 2005;84:171-75.
10. Hardie JM, Bowden GH. The normal microbial flora of the mouth. *Soc Appl Bacteriol Symp Ser.* 1974;3:47-83.
11. Wheatley MJ, Stirling MC, Kirsh MM, Gago O, Orringer MB. Descending necrotizing mediastinitis: transcervical drainage is not enough. *Ann Thorac Surg.* 1990;49:780-84.
12. Kang SK, Lee S, Oh HK, Kang MW, Na MH, Yu JH, Koo BS, Lim SP. Clinical features of deep neck infections and predisposing factors for mediastinal extension. *Korean J Thorac Cardiovasc Surg.* 2012;45:171-76.
13. Saifelddeen K, Evans R. Ludwig's angina. *Emerg Med J.* 2004;21:242-43.
14. Chirinos JA, Lichtstein DM, Garcia J, Tamariz LJ. The evolution of Lemierre syndrome: report of 2 cases and review of the literature. *Medicine (Baltimore).* 2002;81:458-65.
15. Bulut M, Balci V, Akköse S, Armağan E. Fatal descending necrotising mediastinitis. *Emerg Med J.* 2004;21:122-23.