

Liver resection for hemoperitoneum caused by spontaneous rupture of unrecognized hepatocellular carcinoma

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SUMMARY: Liver resection for hemoperitoneum caused by spontaneous rupture of unrecognized hepatocellular carcinoma.

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Hepatocellular carcinoma (HCC) is an increasingly common form of cancer. Although its spontaneous rupture is rare in Western countries, it constitutes a surgical emergency and is associated with high mortality. There is a lack of consensus as to the best approach and what parameters to use in choosing it. The three main approaches are conservative, endovascular and resection - the treatment of choice for acute abdominal bleeding.

We report a case of hemoperitoneum following the spontaneous rupture of an unrecognized HCV-related HCC in a patient with no history of liver disease. The patient was successfully treated by emergency surgery, with resection of two segments of the left liver.

RIASSUNTO: Emoperitoneo da rottura spontanea di epatocarcinoma misconosciuto: trattamento resettivo.

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La rottura spontanea di un carcinoma epatocellulare (HCC) è un'evenienza rara nei Paesi Occidentali e costituisce un'urgenza/emergenza chirurgica, con alta mortalità. È dibattuto quale sia il migliore approccio a questa patologia e quali siano i parametri nella scelta. I tre orientamenti principali sono quello conservativo, quello endovascolare e quello chirurgico resettivo, che rimane la prima opzione per il trattamento dell'emorragia addominale acuta.

Riportiamo un caso di emoperitoneo da rottura spontanea di HCC misconosciuto HCV-correlato in paziente con anamnesi completamente negativa per epatopatia. Il paziente è stato trattato in urgenza, con successo, mediante resezione epatica bisegmentale del fegato sinistro.

KEY WORDS: Hepatocellular carcinoma - Hemoperitoneum - Liver resection - Emergency surgery.
Epatocarcinoma - Emoperitoneo - Resezione epatica - Urgenza.

Introduction

Hepatocellular carcinoma (HCC) is a primary liver cancer which is the fifth most common cause of cancer in men and the seventh in women worldwide (1). Its incidence is constantly rising, especially in the west, due to the increased spread of HBV and HCV infections (2-5). HCC is the third most common cause of death from cancer in the world (1). Early diagnosis is possible, but not easy if the first sign of HCC is its rupture, or if the-

re is no known history of cirrhosis, HCC or HBV/HCV infection (4, 5). Its rupture is a surgical emergency: in the west it occurs in just 3% of cases, while it seems more common in the east, affecting 12.4% of cases in Thailand and 14.5% in Hong Kong (5).

In countries with a high incidence of HCC, around 50% of ruptures prove fatal. Spontaneous rupture is in fact the third most common cause of HCC-related death, after disease progression and liver failure, and before rupture of esophageal varices (6). It can occur not only in large but also in small, aggressive carcinomas (5,6). The signs, symptoms and clinical presentation of HCC rupture depend mainly on its location with the parenchyma. Deep tumors can give rise to more indistinct signs and symptoms or to pain, while rupture of superficial tumors (below Glisson's capsule) can lead directly to hemoperitoneum, possibly associated with peritonitis and circulatory instability, and often accompanied by a sud-

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den abdominal pain (4,6-7). Larger and more superficial HCCs tend to be more subject to rupture (4,6).

We report a case of spontaneous HCC rupture in a patient with no history of liver disease, treated by resection of segments II and III of the left liver.

Case report

In December 2010, an 87-year-old man was referred to our attention from the Emergency Room with a diagnosis of "abdominal pain, constipation and anemia". Urgent blood tests revealed: RBC 3,210,000/uL; Hb 10.1 g/dl; Hct 30.6%; platelets 143,000/uL; AST/GOT 146 U/L; ALT/GPT 175 U/L; myoglobin 471 ng/ml. A direct abdominal x-ray revealed some air-fluid levels projecting into the left hypochondrium with no specific pathological significance, with no free air under the diaphragm.

There was nothing of note in the patient's medical history, except for an old prostatic hypertrophy and repair of a right inguinal hernia around 50 years earlier. He did not report any digestive or liver diseases. Recently, he had suffered colicky pain in the upper abdominal quadrants since the day before his admission. This was not accompanied by nausea, vomiting or temperature rise. His most recent bowel movement was the day before, with stools of a normal color and conformation. Rectal exploration confirmed the presence of stools of a normal color and conformation in the ampulla, with no signs of active or prior bleeding. Vital signs were monitored continuously during the first two days of admission, remaining stable. However, his Hb levels dropped steadily to 7.2 g/dL 18 hours after the first blood sample was taken, leading to authorization for the transfusion of concentrated erythrocytes (1 IU).

Emergency esophagogastrosocopy revealed LA grade A reflux esophagitis and congestive duodenitis. There were no signs of active or prior bleeding, or any conditions that could have been responsible for blood loss sufficient to cause anemia. A full abdominal ultrasound tomography revealed a moderate quantity of free liquid in the abdomen, non-homogenous liver structure and a growth with a mixed echo structure, probably in the left lobe, of around 8.6 x 7 cm. There was also sludge in the gallbladder.

A contrast-enhanced abdominal and pelvic CT revealed the liver had a normal size but non-homogenous density. There was a growth with non-homogenous enhancement and no clear cleavage plane from the adjacent gastric walls, which were thickened (Fig. 1). Routine preoperative blood tests were positive for anti-HCV antibodies (S/CO 156.03). An initial diagnosis of hepatitis C and HCV-related tumor was thus postulated, despite the negative tumor marker tests (AFP: 5.98 ng/ml; CEA: 2 ng/ml; CA 19.9: 21 U/mL).

As the Hb levels continued to drop, it was decided not to further delay surgery and this was carried out 48 hours after admission. The abdomen was opened through right subcostal median incision, revealing massive hemoperitoneum (around 2000 mL). The blood loss was replaced by transfusion with a total of 3 units of concentrated erythrocytes and 5 of plasma during the operation and immediate postoperative period. The source of the bleeding was found to be a 10.0 x 8.0 cm ulcerated nodular lesion at the back of segment III of the liver. As direct hemostasis was impossible, resection of segments II and III was carried out using Pringle's maneuver (Fig. 2). Bleeding of the section surface was controlled by the application of Floseal® high-viscosity gel (Fig. 3). A cholecystectomy was then performed and a subhepatic and pelvic drain were placed.

The postoperative course was normal. Two units of concentrated erythrocytes were transfused on post-operative days 3 and 4, and on day 4 both drains were removed and the patient began eating. He was discharged in a good general condition on day 10.

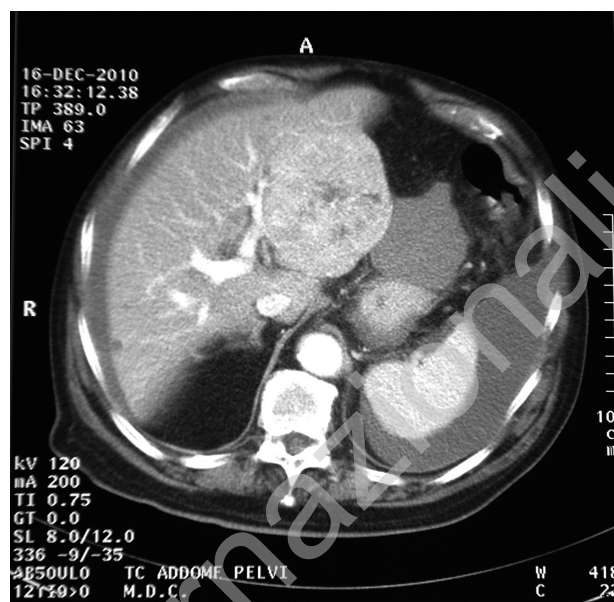


Fig. 1 - Contrast-enhanced CT revealing the formation in the left liver.



Fig. 2 - Dissection of the ruptured HCC. The harmonic scalpel indicates the breach from which the bleeding originated.

Examination of the specimen revealed another two 0.3 and 0.4 cm satellite nodules, in addition to the bleeding lesion (Fig. 4). Microscopic analysis led to the diagnosis of a poorly differentiated multicentric hepatocellular carcinoma (according to WHO 2000) ulcerating the Glisson's capsule, with underlying active chronic HCV-related hepatitis (pT3a pNx pMx, Stage IIIA).

Discussion

Full consensus has not yet been reached on the best approach to hemoperitoneum caused by HCC rupture. A conservative or more aggressive (endovascular or surgical) strategy, or a combination of the two, is cho-



Fig. 3 - Section surface after removal of segments II and III.

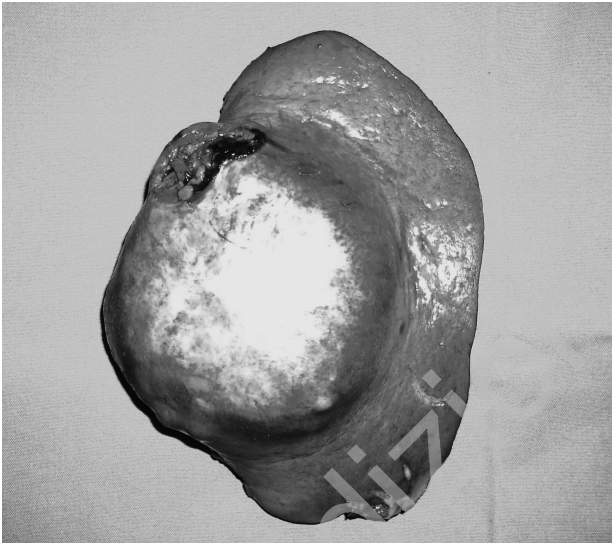


Fig. 4 - Specimen (segments II and III) with the HCC hemorrhagic point.

sen on the basis of liver function, the patient's general condition and the location of the source of the bleeding (4,6,8).

Historically, surgery was the first possible option in cases of massive hemoperitoneum caused by HCC rupture. However, the development of endovascular techniques, alone or in preparation for radical surgery, has expanded the range of possible treatments. The most commonly used of the invasive techniques are transarterial embolization (TAE), perihepatic packing and liver resection (4). Direct suturing of the bleeding lesion, ligation of the hepatic arteries, injection of alcohol into the bleeding vessels and radiofrequency ablation are less common (4).

The main objective is to stop the bleeding and pre-

serve the highest possible amount of functioning hepatic parenchyma (4). First, the patient's vital signs must be stabilized. The best palliative methods to achieve chemical hemostasis are transarterial embolization (TAE) and its variant, transarterial chemoembolization (TACE), although they are associated with high mortality (about 30%) (6). TAE is the treatment of choice in three particular conditions: when the tumor is in a position which is difficult to treat surgically, in the case of multifocal HCC and where liver function is already impaired (6). Its main risks are rebleeding, formation of an abscess and metastasis, while the high mortality seems to be due mainly to the underlying liver failure (4,6,9).

In recent years, there has been a growing conviction that radical surgery is destined to become a second choice treatment, indicated only when intravascular embolization is ineffective (4). However, some authors report its use after surgical resection (8).

Studies on the survival of patients with HCC have not clarified if there is a different life expectancy for patients with and without HCC rupture (10-12). Although it used to be thought that deferment of surgical resection increased the risks for patients and the likelihood of hematogenous metastasis, it is now considered that emergency resection should be carried out only in selected cases of small bleeding tumors which are surgically accessible and where there is no cirrhosis (4,9).

Conclusions

The choice of surgical strategy in patients with HCC rupture partly depends on the stability of vital signs. The presence of hypotension, acidosis or coagulopathy indicate the need for damage control surgery. This happened in another case of unrecognized spontaneous HCC rupture coming to our attention around three years ago. The critical instability of the patient's vital signs, concomitant cirrhosis and active coagulopathy, the extent of the bleeding and the location of the ruptured tumor (between segments VI and VIII) led us to perform emergency perihepatic packing followed by embolization of the right hepatic artery. This procedure, which was completed with depacking after 72 hours, resolved successfully.

In the case presented herein, the diagnosis of HCV-related HCC was reached completely by chance. Radical surgery was chosen above all due to the stability of the patient's vital signs and good liver function, as demonstrated by his blood tests and the absence of cirrhosis. CT images also demonstrated that the tumor was easily accessible. Three months after surgery the patient is in good general condition with normal liver function, has no anemia, and is negative for tumor markers.

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