

Spontaneous Rupture of a Hepatocellular Carcinoma: Is a Liver Transplant Indicated?☆



Rotura espontánea de hepatocarcinoma ¿está indicado el trasplante hepático?

Spontaneous rupture of a hepatocellular carcinoma (HCC) is potentially life-threatening. Urgent diagnosis and treatment are necessary, but there is great controversy about which therapeutic management is best. Embolization followed by scheduled surgery is considered an effective treatment as it reduces the mortality rate; on the other hand, emergency hepatectomy has very high mortality rates.¹

HCC are the fifth most frequent tumours in the world and usually occur in patients with alcoholic or viral cirrhosis.²⁻⁷ In western countries, the incidence of HCC rupture is 3%, with a mortality rate of between 32 and 100%.²⁻⁹ It is the third cause of death in HCC cases after tumour progression and liver failure.²

The patient is a 46-year-old male with alcoholic liver cirrhosis (Child stage B8 and MELD) who, in December 2004, had an episode of haemorrhagic shock due to haemoperitoneum secondary to rupture of a space-occupying lesion of the liver measuring 7 cm in the left lobe, which was previously undetected. Emergency embolization of the left liver artery was performed. Extension studies were negative, so the patient was added to the waiting list for liver transplantation, which took place in July 2006. Currently, the patient is alive and has shown no recurrence of the disease.

The TNM classification qualifies tumoral proliferation towards the peritoneum as T4, which is the most advanced stage, even if the tumour is small and solitary.¹ HCC rupture can cause peritoneal dissemination and recurrence, although intraperitoneal metastases are very rare.¹⁰ Even palliative surgical treatment in peritoneal dissemination can improve survival and the quality of life in selected patients.¹

The physiopathology is not completely clear, although there are several hypotheses that could explain it: subcapsular location, size, portal hypertension, rapid growth with tumour necrosis, vessel erosion, venous thrombosis, local increase in venous pressure, and previous vascular lesion.^{1,2,4,6,8-10}

The typical form of presentation includes abrupt epigastric pain in the right hypochondrium, abdominal distension, associated signs of hypovolemic shock and peritoneal irritation.^{1-6,9}

Preoperative diagnosis without previous history of HCC or cirrhosis is very difficult. CT scan and ultrasound are very useful to demonstrate the presence of haemoperitoneum and liver tumours,^{4-6,8,9} together with the presence of bloody ascitic fluid in a paracentesis.^{1,4,5,9}

In the literature, there is much debate about which therapeutic approach is best in cases of HCC rupture.^{5,8} The most effective therapeutic strategy in HCC rupture is arterial

embolization followed by scheduled hepatectomy.¹ Initial treatment using embolization improves the mortality rate and is able to make time in order to decide on the definitive treatment.^{2,3,6,8,9} When there is hypovolemic shock, immediate management involves haemodynamic resuscitation and haemostasis before considering the definitive treatment.^{4,5,10} If there is liver failure (Child C), cirrhosis, multifocal or bilobar HCC, arterial embolization is an effective, well tolerated emergency treatment.⁸ Liver resection is the best therapeutic option, but it is technically difficult in cirrhotic livers and its success depends on the degree of cirrhosis severity,³ while also presenting a high rate of mortality.³⁻⁵ Embolization is effective for controlling haemorrhage (70%-100%), with a mortality rate from 0 to 30%. The mortality rate of emergency hepatectomy is 28%-55%, compared to 0% when scheduled.¹

In the year 2000, Chen et al. published a case in which an HCC was treated with liver transplantation. They suggest that transplantation can increase recurrence-free survival in cases with extrahepatic metastatic disease.⁷

Several factors have been reported to influence short-term mortality, such as hepatic encephalopathy, Child-Pugh, bilirubin, AST, and albumin. Moreover, the number of tumours, their size and the presence of portal thrombosis have not been shown to be factors that influence short-term mortality.³

Short-term survival benefits can be obtained with surgical treatment, which may or may not be associated with interventionist radiology. Nonetheless, long-term survival seems to correlate with the disease stage, local dissemination after the rupture and residual liver functionality.⁸ In ruptured HCC, the survival rate is higher compared to non-ruptured stage IV HCC, and the disease-free survival rate is similar to non-ruptured HCC in stages I-II-III.¹

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- Tatiana Prieto-Puga Arjona*, Laura Romacho López, Miguel Ángel Suarez Muñoz, Belinda Sánchez Pérez, Julio Santoyo Santoyo
- Unidad de Cirugía Hepatobiliopancreática y Trasplantes, HRU Carlos Haya, Spain
- *Corresponding author.
E-mail address: prietopugaarjona.tatiana@gmail.com (T. Prieto-Puga Arjona).
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Pancreatic Adenocarcinoma During Pregnancy[☆]

Adenocarcinoma pancreático durante el embarazo



Malignant tumours occur in 0.1% of pregnant women. The most common types are cervical, breast, melanoma, lymphoma and leukaemia.¹ Pancreatic cancer is unusual in women of child-bearing age.² The average age for the diagnosis of pancreatic ductal adenocarcinoma is 72, and less than 3% of diagnosed patients are under the age of 45.³ We present a case of pancreatic adenocarcinoma during pregnancy.

The patient is a 35-year-old woman who had had 2 pregnancies, 1 normal delivery, and was at 16 weeks gestation. She presented with vomiting, pain in the left epigastrium and hypochondrium, general weakness and weight loss. Physical examination revealed a painful abdomen with positive Murphy sign and normal vitals. Lab results were: haemoglobin 8.6 g/dL, total bilirubin 2.49 mg/dL, direct bilirubin 1.4 mg/dL, aspartate aminotransferase 120 IU/L, alanine aminotransferase 125 IU/L, phosphatase alkaline 313 IU/L, serum amylase 136 IU/L and urine amylase 426 IU/L.

Abdominal ultrasound reported the absence of cholelithiasis or choledocholithiasis and multiple hypodense liver masses. Fine-needle biopsy demonstrated the presence of cells compatible with adenocarcinoma of the pancreas.

Tumour marker levels were: CA15-3, 36 U/mL; carcinoembryonic antigen, 860 ng/mL; CA19-9, 2750 U/mL; and CA125, 2221 U/mL. Endoscopic ultrasound demonstrated metastatic liver disease, several lymphadenopathies and a neoplastic mass in the tail of the pancreas measuring 40 mm in diameter with central necrosis. Magnetic resonance confirmed the existence of an area of necrosis measuring 2 cm×3 cm in the pancreas, lesions in the liver and several lymphadenopathies in the hepatic-pancreatic hilum (Fig. 1).

Six days later, obstetric ultrasound confirmed the death of the foetus. Uterine evacuation obtained a foetus weighing 120 g, followed by curettage.

The patient was discharged on the third day to later initiate outpatient chemotherapy, but she died 26 days later. The pathology study demonstrated the presence of a poorly differentiated adenocarcinoma of the pancreas. The tumour was larger than 6 cm in diameter, with extension beyond the pancreas towards the lymph nodes and perineural invasion.

Although the exact cause is unknown, pancreatic cancer has several risk factors that have been identified (older age, male sex, diabetes, obesity, chronic pancreatitis and smo-

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