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Case Report of Spontaneous Rupture of Hepatocellular Carcinoma: Clinical Presentation, Management, and Outcome

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ABSTRACT

Spontaneous rupture of hepatocellular carcinoma (HCC) is a rare but life-threatening complication, presenting with acute abdominal pain and hemodynamic instability. This case report describes the clinical manifestations, diagnostic challenges, and management strategies employed in a patient with spontaneous rupture of HCC. The patient, with underlying cirrhosis, presented with signs of hemorrhagic shock. Immediate resuscitation, imaging, and subsequent embolization were performed to stabilize the patient, followed by definitive treatment. The rarity of this complication, combined with the precarious nature of the underlying liver disease, creates significant therapeutic challenges. This case underscores the need for rapid diagnosis and a multidisciplinary approach to optimize patient outcomes.

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INTRODUCTION

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy, often arising in the background of chronic liver disease and cirrhosis. The natural history of HCC is highly variable, ranging from indolent tumors to aggressive cancers with early metastasis and vascular invasion. Among its myriad complications, spontaneous rupture is a rare but dire event, occurring in 3% to 15% of cases depending on the geographic region and patient cohort. Spontaneous rupture is frequently associated with significant intra-abdominal hemorrhage, resulting in profound hemodynamic instability and high mortality rates, often exceeding 25%. The mechanism behind the rupture remains unclear, though hypotheses include tumor necrosis, rapid growth, increased intra-tumoral pressure, and vascular fragility due to underlying cirrhosis.1,2

The clinical presentation of ruptured HCC typically involves sudden onset of severe abdominal pain, hypotension, and signs of hemorrhagic shock. These symptoms may mimic other causes of acute abdomen, making early recognition and diagnosis challenging. Imaging modalities such as contrast-enhanced computed tomography (CT) or ultrasound are vital for confirming the diagnosis and assessing the extent of bleeding. Management strategies primarily focus on hemodynamic stabilization through fluid resuscitation, blood transfusions, and embolization of the bleeding vessels. In select cases, surgical intervention may be warranted, though it carries a high risk in patients with cirrhosis due to impaired liver function.3,4,5

This case report highlights the diagnostic and therapeutic complexities of managing a spontaneous rupture of HCC in a patient with underlying cirrhosis. We present the clinical course, including initial stabilization, definitive treatment, and outcome, emphasizing the importance of rapid intervention and a multidisciplinary approach in such critical situations.

CASE PRESENTATION

Patient: 76-year-old female with a history of suffering from diabetes mellitus 35 years ago diagnosed on insulin treatment, diagnosed with arterial hypertension 35 years ago on telmisartan treatment. Surgical history: Hysterectomy for uterine myomatosis. Traumatologic: Right radius fracture

treated by reduction and internal fixation 28 years ago. Other history denied.

She began her illness 10 days prior to her admission with abdominal pain in the right hypochondrium of intensity 7/10 on the analogue pain scale, colic type, without irradiation, treated with a private physician with butylhioscine with partial improvement, later she presented again with abdominal pain of the same characteristics, She was treated with Sylibum marianum and alpha lipoic acid, however the patient presented exacerbation of pain and symptoms with mild dyspnea, so she went to the emergency room for evaluation.

On arrival he has the following vital signs: Blood pressure: 80/55 mmHg, Heart rate: 88 beats per minute, Respiratory rate: 19 breaths per minute, Temperature: 36 °C, Oxygen saturation: 90%.

It was decided to place a central venous catheter with the start of amine infusion and supplemental oxygen through nasal prongs. Physical examination: the patient was awake, alert, oriented, with mild jaundice of skin and integuments +/+++, vesicular murmur present, without integrating pleuropulmonary syndrome, rhythmic heart sounds with adequate tone and intensity, synchronous pulses, hemodynamically unstable with vasopressor support of norepinephrine type at 0.09 mcg/kg, abdomen globose at the expense of soft adipose panniculus, with slight pain on deep palpation of the mesogastrium, normal peristalsis, without data of peritoneal irritation, the rest of the examination without alterations. After initial resuscitation, vital signs improved, oxygen saturation increased to 98%, maintaining a mean arterial pressure (MAP) of 65 mm/Hg.

Laboratory tests: Leukocytes 11.9 x 10^3/µL, neutrophils 46.0% , Hb 10.1g/dL, Hto 31.9, Platelets 301 000/µ, PT 12.3 s, TTP 25.3 s, INR 1.12, LDH 186.7, Glucose 306.0 mg/dL, Urea 47.4 mg/dL, BUN 22.1 mg/dL, Creatinine 0.99 , Na:140.4 mEq/L, K: 4.36 mEq/L, Cl: 108.7 mEq/L, Ca: 9.0 mEq/L, arterial blood gases reported metabolic acidosis with lactate of 13.1 mmol/L.

An abdominopelvic CT scan was performed showing a lobulated liver in relation to probable chronic liver disease and a neoplastic lesion of 104x107 mm and free perihepatic fluid in its thickness.(Figure 1)

Due to the clinical deterioration and the decrease of 2 g of hemoglobin at the beginning and data of hypovolemic shock secondary to probable ruptured hepatocarcinoma, the need for angioembolization of the hepatic artery was considered; however, the resource was not available in the hospital, However, the patient presented deterioration and data of peritoneal irritation, so surgical treatment was decided after transfusion of 2 erythrocyte concentrates and availability of blood products, and exploratory laparotomy was performed, found hemoperitoneum of approximately 1300 cc with organized clots in the right slider, liver tumor of approximately 10x10cm depending on segment V and IV with active bleeding of liver tumor apparently in segment V near the gallbladder, The rest of the liver with macronodular characteristics, during surgery the hemoperitoneum is drained, hepatorraphy of the tumor is performed with chromic 0 with U and transfixed and X points, placing gelfoam to control the bleeding, liver biopsy is taken and placement of penrose drainage.

The patient evolves adequately after surgery with stabilization of vital signs without the need for vasoactive amines, being however the drainage output persists with hematic characteristics and quantifying approximately 200 cc in 24 hours with gradual decrease of hemoglobin until reaching 8g/dL without presenting hemodynamic deterioration, The relatives decide to perform hepatic chemoembolization by private means, the procedure is performed without complications, after which he is readmitted again for surveillance, the evolution is adequate, drains are removed on the 6th day without complications; The pathology report is obtained with a report of well differentiated hepatocellular carcinoma, she is evaluated with tomography by surgical oncology cataloging it as a Barcelona C hepatocellular carcinoma, not a candidate for surgical treatment and suggests evaluation by medical oncology, who comments that she is not a candidate for systemic oncologic treatment and a candidate for palliative treatment at home due to the advanced stage of the disease, so it is decided to discharge her for maximum benefit.

Abdominopelvic CT on 03-08-24: The liver is lobulated in relation to probable chronic liver disease and in its thickness there is a neoplastic lesion of 104x107 mm. In the peritoneal cavity free liquid of minimal quantity is observed. (Figure 1)(Figure 2)

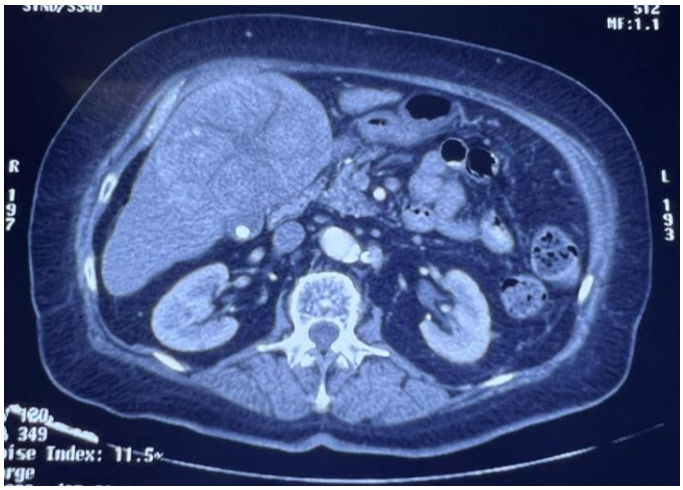

Figure 1. Abdominal tomography showing a liver tumor measuring 104x107 mm with a lobulated appearance.

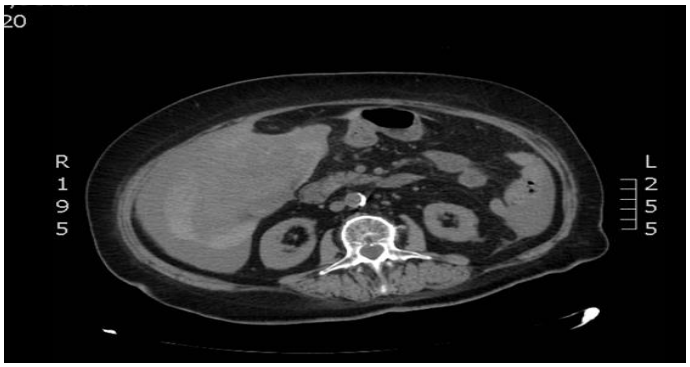

Figure 2. Abdominal tomography showing perihepatic free fluid.

Due to the clinical deterioration and the decrease of 2 g of hemoglobin at the beginning and data of hypovolemic shock secondary to probable ruptured hepatocarcinoma, the need for angioembolization of the hepatic artery was considered; however, the resource was not available in the hospital, However, the patient presented deterioration and data of peritoneal irritation, so surgical treatment was decided after transfusion of 2 erythrocyte concentrates and availability of hemoderivasos, exploratory laparotomy + drainage of hemoperitoneum + hepatorraphy of liver tumor + liver biopsy + placement of drainage penrose 16 was performed.02.23.

During the surgery a biopsy of bleeding tumor tissue was taken, hepatorraphy was performed with chromic 0 with U and transfixed and X points, placing gelfoam to control bleeding, hemostasis was corroborated and reviewed again by quadrants without finding bleeding data.

Trans-surgical findings; hemoperitoneum of approximately 1300 cc with organized clots in the right side, liver tumor of approximately 10x10cm dependent on segment V and IV with active bleeding of liver tumor apparently in segment V near the gallbladder, rest of liver with macronodular characteristics.(Figure 3)

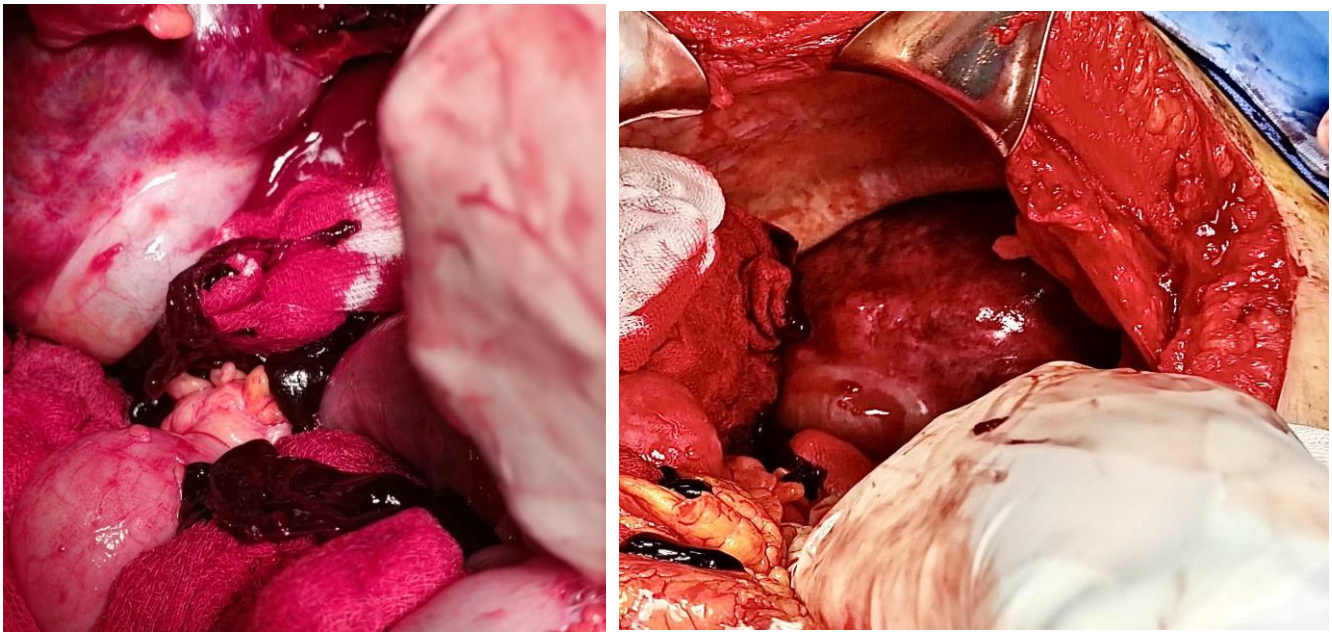

Figure 3.

Transoperative photograph showing organized clots and hepatic tumor that almost completely envelops the gallbladder.

The patient evolves adequately after surgery with stabilization of vital signs without the need for vasoactive amines, being however the drainage output persists with hematic characteristics and quantifying approximately 200 cc in 24 hours with gradual decrease of hemoglobin until reaching 8g/dL without presenting hemodynamic deterioration, The need for angioembolization of the hepatic artery with referral to the 3rd level was suggested, the family decided to perform hepatic chemoembolization by private means, which was performed without complications, after which he was readmitted for follow-up, the course is adequate, with adequate tolerance to the oral route, the drains with scarce serohematic expense are removed after 6 days and the pathology report is obtained with a report of well-differentiated hepatocellular carcinoma, She is evaluated with tomography by surgical oncology cataloging it as a Barcelona C hepatocellular carcinoma, not a candidate for surgical treatment and followed by evaluation by medical oncology, who commented that she was not a candidate for systemic oncological treatment and a candidate for palliative treatment at home due to the advanced stage of the disease, so it was decided to discharge her for maximum benefit.

CONCLUSION

Spontaneous rupture of hepatocellular carcinoma (HCC) represents a rare but catastrophic complication of this malignancy, often leading to acute and severe hemodynamic compromise due to massive intra-abdominal hemorrhage. This case underscores the complexity and urgency of managing such events, particularly in patients with underlying cirrhosis, who are already burdened by diminished hepatic reserve, coagulopathy, and other systemic complications. The initial presentation of spontaneous rupture often mimics other acute abdominal conditions, necessitating a high index of suspicion and rapid diagnostic workup using imaging techniques such as contrast-enhanced computed tomography (CT) or ultrasound. Early recognition and prompt intervention are paramount to improving survival outcomes.

The treatment of ruptured HCC is multifaceted and demands a coordinated, multidisciplinary approach involving interventional radiologists, hepatologists, critical care specialists, and surgeons. In this case, successful stabilization was achieved through immediate resuscitative efforts and subsequent transarterial embolization (TAE), which remains the cornerstone of initial management due to its minimally invasive nature and effectiveness in controlling hemorrhage. For select patients, definitive treatment of the underlying tumor may require surgical resection or liver transplantation, though these options are limited by the patient's liver function, tumor burden, and overall clinical status.

Despite advances in both diagnostic imaging and therapeutic modalities, the prognosis of ruptured HCC remains guarded. The overall mortality associated with this complication remains high, reflecting not only the severity of hemorrhage but also the advanced stage of liver disease and malignancy in many affected patients. This case reinforces the importance of vigilant follow-up in patients with known HCC, particularly those with large or vascular tumors, which may carry a higher risk of rupture.

Moreover, emerging therapies, including molecularly targeted agents and immunotherapies, may play a role in the future management of patients with ruptured HCC, particularly in the context of stabilizing the tumor biology post-hemorrhage. However, further research is required to

delineate the optimal therapeutic pathways and improve long-term outcomes in this challenging patient population.

In conclusion, while the rupture of hepatocellular carcinoma presents a formidable clinical challenge with high associated morbidity and mortality, timely intervention—particularly via transarterial embolization—can provide a critical window of opportunity for stabilization. As highlighted by this case, early diagnosis, aggressive management, and a tailored, multidisciplinary approach are the keys to optimizing survival and improving outcomes in patients with this life-threatening complication.

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