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RESEARCH ARTICLE

SPONTANEOUS RUPTURE OF HEPATOCELLULAR CARCINOMA IN A HEALTHY LIVER: A CASE REPORT

Rebbani Mohammed, Njoumi Nourddine, Rahali Anwar, El Ktaibi Abderrahim, El Brahm Yasser, Najih Mohammed, El Hajjouji Abderrahman and Aitali Abdelmounaim

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Abstract

Spontaneous rupture of hepatocellular carcinoma (HCC) is a rare and potentially fatal complication with a poor prognosis. Its occurrence is increasing due to the high incidence of hepatocellular carcinoma. The mechanism of rupture is poorly understood. The clinical diagnosis is polymorphous and non-specific, but above all difficult to evoke it in subjects who are not known to be cirrhotic or to have hepatocellular carcinoma. The computed tomography scan remains the first choice for diagnosis. Initial stabilization of the patient remains the priority of emergency therapeutic management. The challenge of this complication is twofold: diagnosis and treatment. In this article, we report a case of spontaneous rupture of a hepatocellular carcinoma in a healthy liver.

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Introduction:-

Hepatocellular carcinoma is the 2nd most common cause of cancer-related death worldwide, occurring in most cases against a background of cirrhosis or chronic non-cirrhotic liver disease, and exceptionally in healthy livers. Spontaneous rupture of hepatocellular carcinoma is a rare complication, fatal in over 75% of cases [1]. we report the case of spontaneous HCC rupture in a healthy liver.

Case report:

Mr B.M, aged 63, non-insulin-dependent diabetic on oral antidiabetics, underwent surgery 20 years ago for perforated peptic ulcer. Admitted to emergency with acute episode of epigastralgia associated with nausea and vomiting, onset less than 04 hours previously. On initial admission, the patient was conscious and tachycardic at 120 beats per minute, with blood pressure measured at 90/60 mm hg. On physical examination, the abdomen was distended and sensitive. Laboratory investigations showed a hemoglobin of 11.7, a hematocrit of 35, a platelet count of 243,000 and a white blood cell count of 30,000, which 80% of was neutrophilic. Hepatic function was without abnormality. The blood prothrombin level was 65%. The patient was initially stable, and an injected abdominopelvic computed tomography scan revealed a medium-sized hemoperitoneum with no evidence of active bleeding or liver lesions (figure 1). At 03 hours after admission, the patient had developed hemorrhagic shock, which did not respond to medical reanimation measures, notably fluid repletion and transfusion of 03 red blood cells, which required emergency laparotomy. Surgical exploration has shown a medium-sized hemoperitoneum due to spontaneous rupture of a small mass in the caudate lobe of the liver (figure 2). Successful hemostasis was ensured by simple suturing of the fissured area with slow resorption filaments (figure 3). A biopsy was done and returned in favor of hepatocellular carcinoma (figures 4 and 5).

After 04 months and after exploration of the mass and discussion of the case in a multi-disciplinary consultation meeting, a segmentectomy of the caudate segment was decided, with simple post-operative follow-up. Follow-up was uneventful up to 6 months.

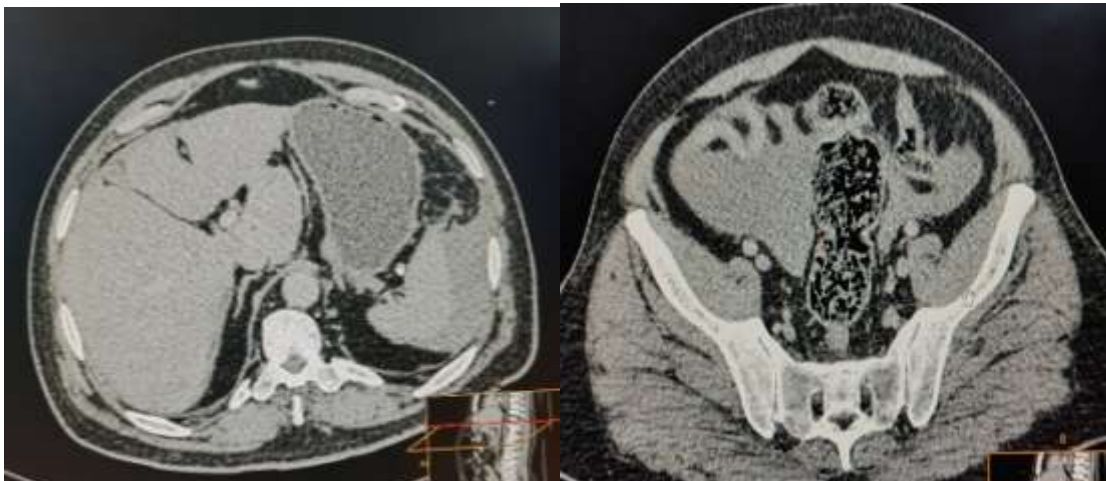


Figure 1:- Abdominopelvic computed tomography showing moderate hemoperitoneum.

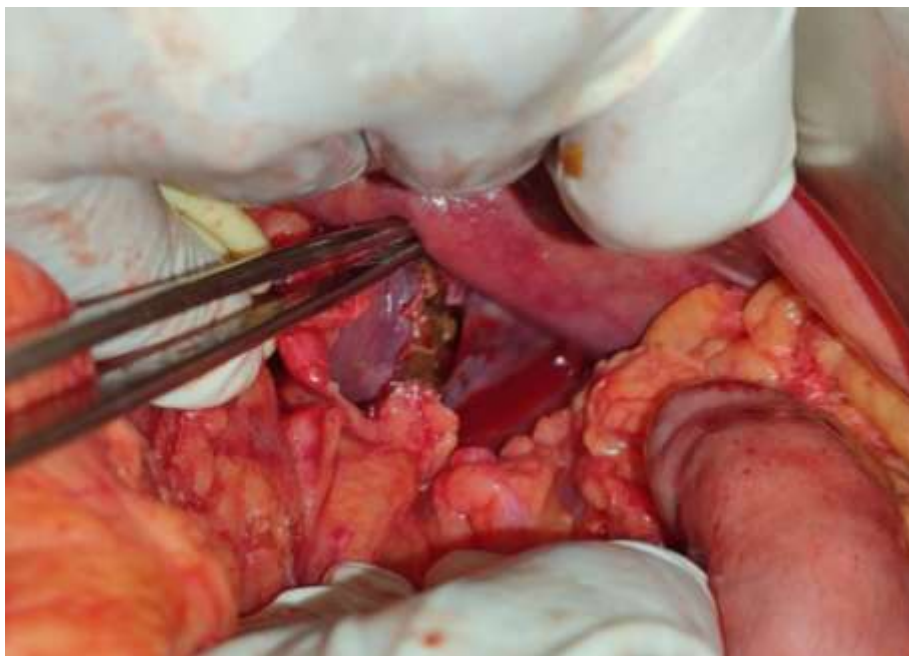


Figure 2:- Ruptured mass in the 1st liver segment.

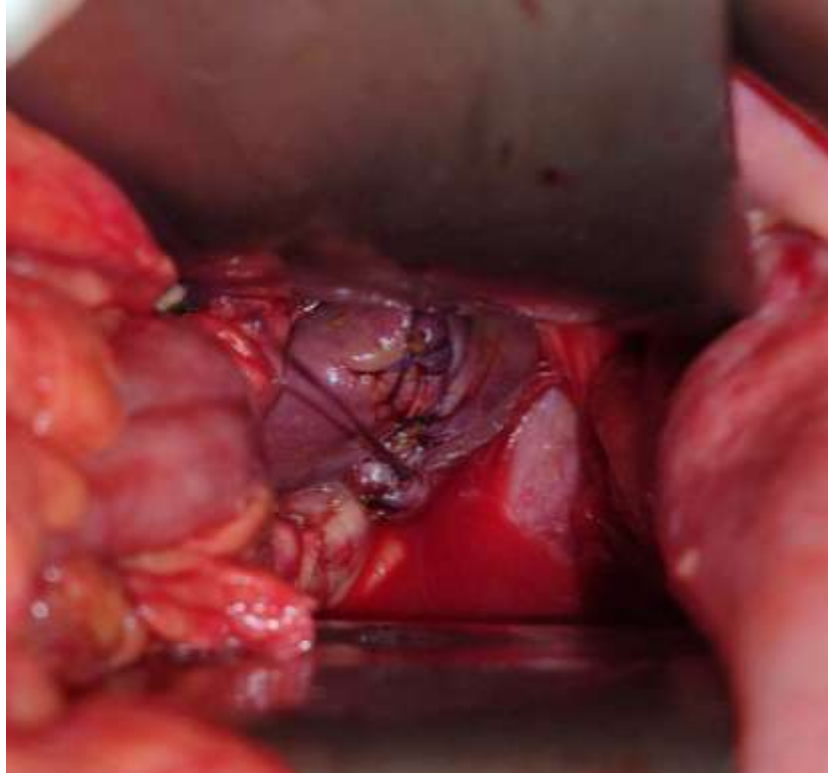


Figure 3:- Hemostasis using a slow resorption filament suture.

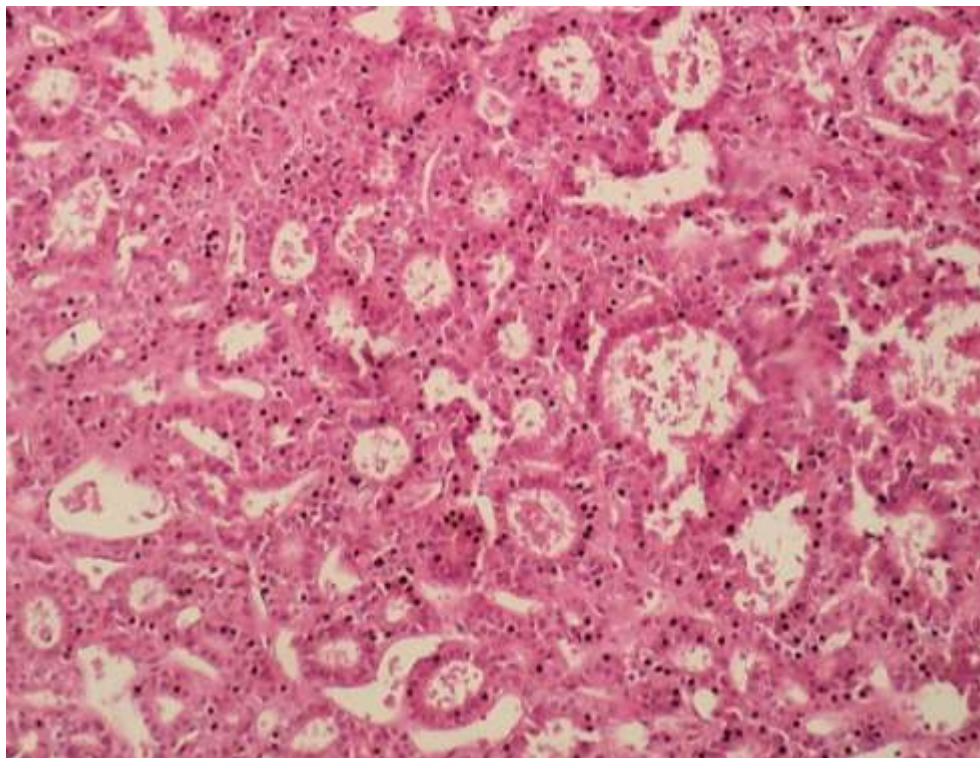


Figure 4:- Microphotography showing well differentiated hepatocellular carcinoma demonstrating pseudoacinar morphology (eosin hematoxylin staining power x200).

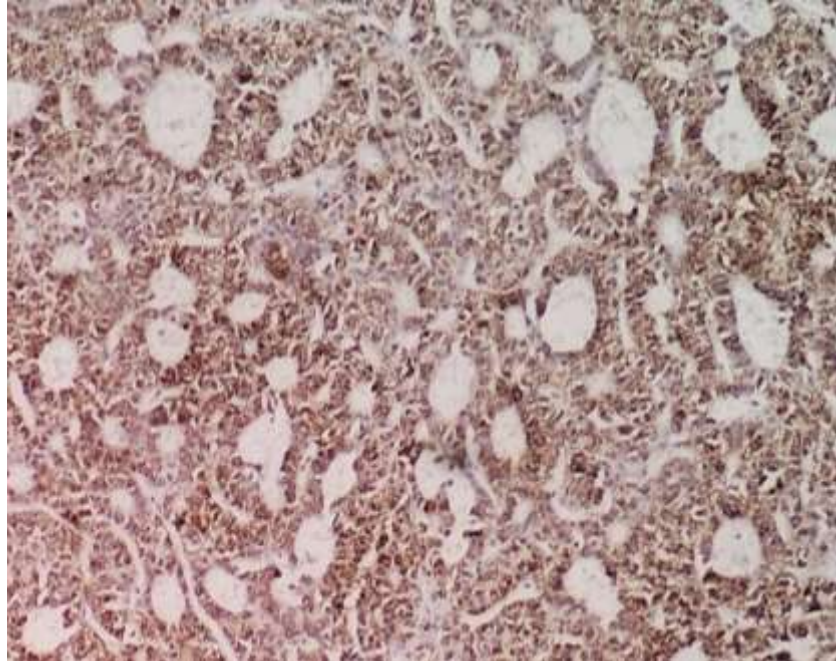


Figure 5:- Microphotography showing a positive immunohistochemistry for HepPar1 in HCC (200x).

Discussion:-

Hepatocellular carcinoma accounts for 85-90% of liver tumors. Worldwide, it is the sixth most common cancer in terms of incidence, but because of its poor prognosis, it has the second-highest cancer mortality rate [2]. An uncommon complication of hepatic neoplasms, mainly hepatocellular carcinoma, is spontaneous rupture causing a life-threatening haemoperitoneum. About 5–15% of patients with HCC develop this complication and the highest incidence has been observed in Far Eastern [3].

The mechanism of rupture is controversial. Factors such as central tumor necrosis, rapid tumor growth, venous congestion within the HCC due to obstruction of hepatic veins by tumor invasion, coagulopathy on pathological liver, mild trauma and respiratory movements cause intra-tumoral hemorrhage, which abruptly increases the size and consequently the intra-tumoral pressure, leading to rupture of the tumor itself or of the adjacent parenchyma [4]. Added to this is the risk of vascular rupture due to increased vascular rigidity and fragility resulting from degeneration of elastin and degradation of collagen IV under the conditions described above [1].

The clinical manifestations are different from compensated hemorrhage to massive peritoneal rupture leading to abundant life-threatening hemoperitoneum. In the acute form, the typical symptom is shock, which occurs after sudden onset of pain in the right hypochondrium or epigastrium. This may be preceded by nausea and vomiting with abdominal distension on clinical examination.

This picture is non-specific, as it most often occurs in patients with unknown HCC, making the diagnosis of rupture difficult to evoke.

Our patient initially presented with pain, nausea and vomiting, later complicated by hemorrhagic shock. As he was not a known HCC carrier and had no suggestive risk factors, the diagnosis of rupture could only be made by surgical exploration.

Imaging, particularly abdominal contrast ultrasonography highlighting the HCC and showing hemoperitoneum, peritumoral hematoma, or detecting bleeding and/or rupture.

Abdominal Computed Tomography scan with contrast injection also reveals the hemoperitoneum or hematoma that often accompanies HCC rupture. The absence of the latter may be due to resorption or dilution in pre-existing ascites. Hematomas are often close to the site of rupture, suggesting in such cases a hemorrhage originating from the

HCC. Extravasation of contrast medium into the HCC indicates active bleeding through rupture, requiring immediate management [5,6]. In our case, emergency imaging revealed hemoperitoneum. However, the segment 1 mass was not visible. Subsequent exploration revealed the mass and enabled the extension work-up to be established.

The aim of emergency care is to restore a stable hemodynamic state. This can be achieved through several therapeutic modalities.

Conservative medical treatment consists of resuscitation by conditioning, adequate filling and even blood transfusion. Trans-arterial embolization and chemoembolization is the least invasive treatment of choice for emergency bleeding control in frail patients with poor liver function and hemodynamic status unable to tolerate major surgery. Its success rate is encouraging, but its availability in emergencies is not always possible [7].

Surgical hemostasis is achieved by several methods. Direct packing with a second look after stabilization of the patient, suturing of the hemorrhagic zone, electrocoagulation and alcoholization. These methods are of mediocre efficacy, with a high recurrence and high mortality. Elective arterial ligation is the method of choice compared to other methods but exposes the patient to ischemic hepatocellular failure [8].

Atypical liver resection, segmentectomy and major hepatectomy are therapies with a dual objective: hemostatic and carcinologic. However, the hemodynamic state and condition of the underlying liver makes these procedures fraught with a high mortality rate. Delayed resections are therefore preferable to emergency resections and are associated with low morbidity and mortality in the short term [9]. In our patient, hemostasis was achieved by simple suturing of the site of rupture. A carcinological tumor resection was subsequently performed, with good post-operative outcome.

The TNM classification system classifies ruptured HCC as T4 and stage IV. This suggests that the prognosis and long-term survival of ruptured HCC is poorer than that of unruptured HCC. However, recent studies have shown that the long-term survival of ruptured HCC can be equivalent to that of unruptured HCC [10].

At last, the prognosis of patients with HCC cannot be correlated with rupture alone, but also depends on tumor size, differentiation or grade of tumor based on histopathology, severity of underlying liver disease, presence or absence of metastasis and tumor extension to adjacent structures [11].

Conclusion:-

Spontaneous rupture of HCC is a rare and fatal complication. The prognosis and therapeutic approach are conditioned by the hemodynamic state, liver function and tumor stage. However, there is currently no well-codified and standardized therapeutic consensus that can guide and therefore ensure optimal both vital and oncological outcomes of patients.

Conflict of interest:

The authors declare no conflict of interest.

Authors contribution:

All authors contributed to the conduct of this work. All authors also declare that they have read and approved the final version of the manuscript.

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