A Rare Case of Spontaneous Rupture of Hepatocellular Carcinoma in Renal Transplant Patient Under Chronic Immunosuppressive Therapy

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ABSTRACT

Spontaneous rupture of hepatocellular carcinoma (HCC) is a potentially life-threatening condition. Diagnosis may be difficult, particularly in the absence of known liver cirrhosis or tumor. A 53-year-old African male came to the emergency unit presented with severe abdominal pain and shock. Past medical history of immunosuppressive medication after renal transplantation, 20 years ago. CT scan of the abdomen revealed large hepatic mass with cortical discontinuity and hemoperitoneum, also sentinel clot sign is demonstrated due to active bleeding, the findings suggestive of spontaneous HCC rupture. In this case transarterial embolization was preferable as a bridging therapy prior to further diagnostics and therapy, to lower the morbidity and mortality. In this case report, we present the computed tomography finding of a rare case of spontaneous rupture of HCC in a non-cirrhotic liver in a patient with chronic immunosuppressive treatment after renal transplantation. The immunosuppressive drugs are associated with an increased risk for cancer following transplantation. The diagnosis is proven by surgical specimen.

Keywords: Hepatocellular carcinoma, Spontaneous rupture, Transarterial embolization

1. INTRODUCTION

HCC has the fifth most common incidence of all cancers. The rupture incidence in HCC is reduced by early diagnosis [1]. Spontaneous rupture has been reported in patients with HCC between 3% and 26% and the mortality rate due to ruptured HCC is between 32% and 66% [2]. The lack of initial diagnosis such as cirrhosis and HCC lead to difficulty in the diagnosis of ruptured HCC in emergency conditions. The computed tomography (CT) is the useful imaging tool in primary diagnosis. In kidney transplant recipients, the incidence of cancer is generally increased 2- to 3-fold compared with the general population [3].

Here we report a case of renal transplant patient with spontaneous ruptured multifocal HCC, which was detected for the first time in the emergency room and required a special approach.
2. CASE PRESENTATION

A 53-year-old male patient arrived in the emergency department with acute right upper quadrant abdominal pain and signs of hypovolemia. On examination, he was pale, dehydrated, with a tense and distended abdomen but no signs of external bleeding. The past history of the patient was renal transplantation, 20 years ago with maintenance dose of prednisolone and cyclosporine. His blood pressure was 110/60 mmHg and his heart rate was 96 bpm. Laboratory parameters were as follows: hemoglobin: 8.5 g/dL (normal range: 12.2–18.1), alanine aminotransferase: 245 U/L (16-63), aspartate aminotransferase: 119 U/L (15-37) and direct bilirubin: 7.6 mg/dL (0.0–3). The other parameters were in the normal range. In an interval of one hour, the patient presented by shock. He was shifted to the emergency radiology department for abdominal dynamic CT.

CT abdomen(figure1,2) coronal and axial images show right hepatic lobe large mass measures around 8 x 10.7 x 10.2 cm in maximum dimensions occupying segment V and segment VIII with typical mosaic architecture (i.e. compartments of variable enhancement in random distribution). Cortical discontinuity of the mass and high density peritoneal fluid suggest tumor rupture with hemoperitoneum, also sentinel clot sign is demonstrated due to active bleeding. We diagnosed the case as massive hemoperitoneum related to primary liver tumor rupture most likely HCC.

Figure 1: coronal CT image shows large right hepatic mass with bleeding into adjacent peritoneal cavity with typical sentinel clot sign. Also, the transplanted kidney is visualized.
An emergent interdisciplinary discussion was initiated. To achieve hemostasis, we initiated a transarterial embolization (TAE). By a super selective angiography of the liver arteries with embolization of right hepatic artery by using the VA particles 500-700 microns followed by Gelfoam. Although the tumor was highly vascularized, an adequate hemostasis was achieved, and the control showed a successful embolization (figure3, 4).

**Figure 2:** axial image shows capsular discontinuity of the mass with hemoperitoneum

**Figure 3:** Highly vascular liver lesion
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After clinical recovery of the patient, further radiological examinations were made. One month after diagnosis, the liver magnetic resonance imaging (MRI) showed non-cirrhotic liver with a tumor centered on segment VIII and segment V with a large hemorrhagic center and peripheral soft tissue components. The peripheral soft tissue components demonstrate arterial hyper enhancement followed by washout, but no signs of further bleeding or ascites. Additional 3 small lesions with maximum diameter of 7mm were detected in segments V, VII and VIII with early atrial enhancement and early washout. Metastatic disease was excluded by thoracic and abdominal CT scan.

The patient underwent surgery with right hepatectomy. The pathological result was moderate differentiated multifocal HCC. The serology findings were negative for hepatitis B and C viruses. Postoperative MRI abdomen showed collection at the surgical bed suggestive of seroma which was resolved after interventional aspiration.

3. DISCUSSION

One of the life threatening complication of HCC is spontaneous rupture of the tumor. Ruptured HCC has a mortality rate of between 32% and 66% [5]. The incidence of spontaneous rupture in HCC has been reported as being between 3% and 26% [5].

The cause of spontaneous rupture remains unclear. The main causes of spontaneous rupture are thought to be minor trauma of surface localized tumors, a fragile feeder artery, tumor size, superficial location, increased intra-tumoral pressure secondary to hepatic tumor invasion, portal hypertension, invasion, and tumor necrosis [6]. Our patient had no trauma history, but the tumor size was more than 5cm which is risk factor for rupture.

The following CT finding are useful for diagnosing a ruptured HCC: hemoperitoneum, HCC with surrounding perihepatic hematoma ,active extravasation of contrast material ,tumor protrusion from the hepatic surface, focal discontinuity of the hepatic surface and an enucleation sign [7]. Our case had most of these signs including( sentinel clot sign ) which is a very important CT sign pointing to the site of bleeding where the clotting occurs at the site of tissue rupture (higher HU of 45 - 60 with heterogeneous appearance) whereas blood that leaks away from the liver remains liquid in the peritoneal cavity ( layered effect with HU of 40).

Maintenance immunosuppression is essential after kidney transplantation to prevent allograft rejection. Although it is accepted that overall immunosuppression dose is associated with an increased cancer risk following transplantation, the contributive effect of different immunosuppressive agents is not established. The mechanisms linking

Figure 4: successful embolization
immunosuppression dose to the increased incidence of cancer are numerous and include decreased immune surveillance of tumors, decreased antiviral responses resulting in a specific increase of virus-induced tumors and possibly the direct carcinogenic effect of immunosuppressive drugs such as cyclosporine and azathioprine [8]. Our patient was known case of renal transplant, 20 years ago on maintenance dose of cyclosporine and prednisolone. So, the history of renal transplantation and immunosuppressive drugs help our diagnosis that the liver lesion was HCC.

4. CONCLUSION

Spontaneous rupture in HCC patients without initial diagnosis leads to difficulties in emergency conditions. The treatment of ruptured HCC needs to be a consensus of emergency doctors, radiologists, and surgeons as well. HCC should be considered as a primary differential diagnosis in spontaneous hemoperitoneum in patients with liver lesion especially in post renal transplant patients with long immunosuppressive treatment involving cyclosporine due to possible direct carcinogenic effect with high incidence of malignancy.

REFERENCES