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Endovascular Management of a Case of Large Intraparenchymal and Subcapsular Hematoma of the Liver with Hepatic Arterioportal Shunt

Case Report

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Abstract

Hepatic arterioportal shunts are abnormal communications between hepatic artery and portal vein. They are seen in a variety of conditions like Hepatocellular carcinoma, penetrating trauma to the liver, Cirrhosis, portal vein thrombosis, marked extrinsic hepatic parenchymal compression by tumour or hematoma. They can also arise spontaneously without any cause and rarely can be congenital. We here report a case endovascular management of a large sub capsular hematoma compressing the liver parenchyma and causing secondary hepatic contusions with an arterioportal shunt.

Case Report

A 19 year old male came with complaints of severe right hypochondriac pain since 2 days. There was no history of trauma. He had past history of two attacks of acute pancreatitis for which he underwent conservative management in outside hospital. The last attack of pancreatitis was 4 months back. On examination at admission, there was mild tenderness in the hypochondriac region. His vitals were stable.

Ultrasound abdomen revealed a large intraparenchymal hematoma measuring 8 x 7 x 6 cm in segment VI and VII of liver which has ruptured with resultant massive sub capsular hematoma compressing the right lobe of liver parenchyma, which measured 14 x 12 x 11 cm. CT angiogram was advised for further evaluation which confirmed the ultrasound findings. There was marked extrinsic compression of right lobe of liver. There were, in addition, multiple small bleeders into the intraparenchymal hematoma as well as extensive surface bleeders on the raw surface of right lobe of liver, most of which appear to arise from the peripheral portal vein radicles. The right anterior branch of the portal vein was enhancing in the arterial phase with wedge shaped transient hepatic parenchymal

enhancement during the arterial phase, suggesting the presence of arterioportal shunt (Figure 1).

His Liver function tests and other blood parameters were normal. As the patient was clinically stable except for pain, it was decided to manage conservatively. However, during the hospital stay, there was a rapid and significant drop in the haemoglobin from 13 gm/dl on the day of admission to 6gm/dl in 36 hours. His pain worsened and there was tachycardia. So, it was decided to evaluate him with hepatic angiogram to look for abnormal bleeders. Selective angiogram of segment VI branch of hepatic artery revealed abnormal vascularity which was embolized by 75 - 150 micron size PVA (Polyvinyl alcohol) particles. Segment VIII branch angiogram revealed an arterioportal shunt. Rest of the segmental arteries were unremarkable. Since the surface bleeders were from the peripheral portal vein radicles, it was decided to selectively embolize the arterioportal shunt to reduce the pressure in the portal vein system (Figure 2).

Two 018 inch coils, 3 mm x 14 cm and 3 mm x 7 cm (Micronester coils, Cook)was placed in the segment VIII branch of hepatic artery and the arterioportal shunt was embolized. Post procedure, there was significant resolution in the right hypochondriac pain within 6 hours. There was no further drop in haemoglobin. His liver enzymes, especially the transaminases were transiently elevated (AST - 1200 U/L and ALT - 1300 U/L) on the second day post procedure, but became normal in next 48 hours. Patient was discharged in stable condition on the 5th day post procedure. His haemoglobin at the time of discharge was 6.7 gm/dl. Follow up USG after 10 days revealed organising sub-capsular hematoma which was mildly regressed in size and extent (Figure 3).

Discussion

Hepatic arterioportal shunts are a well-known entity. These can be broadly classified into tumorous and non-tumorous arterio portal shunts. These can be usually seen in hepatocellular carcinoma. Small peripheral arterioportal shunts are well known in cirrhosis. Large direct fistulas can be seen in penetrating injuries of liver. Some of the arterio portal shunts can be seen in portal vein and hepatic vein thrombosis and also in marked extrinsic compression of liver as in our case. The proposed mechanism for this is increased sinusoidal pressure leading to shunting of

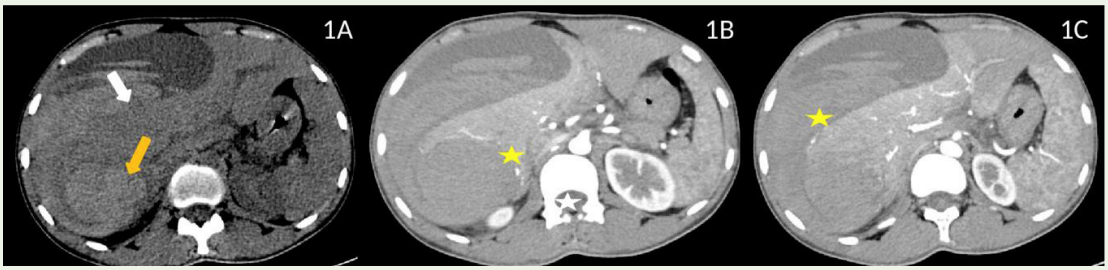

Figure 1: 1A: Plain CT scan abdomen axial section reveals a large hyperdense intraparenchymal hematoma in segment VI and VII of liver (yellow arrow) and a large sub capsular hematoma compressing the right lobe of liver (white arrows). **1B:** Contrast enhanced CT scan arterial phase Images reveal active bleeders into the intraparenchymal hematoma (asterisk). **1C:** Similar active surface bleeders are also noted bleeding into the sub capsular hematoma.

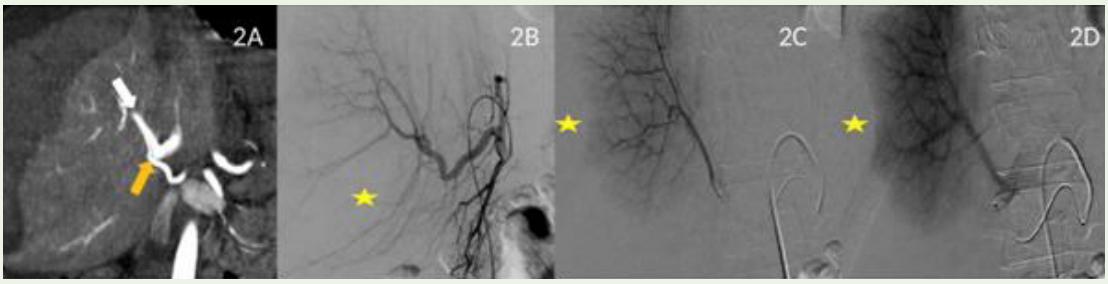

Figure 2: 2A: Coronal reformatted image arterial phase demonstrating opacification of hepatic artery segment VIII branch (yellow arrow) and early opaciflcation of portal vein (white arrow). **2B:** Hepatic anglogram demonstrating marked splaying of right lobe segmental branches (asterisk) due to intraparenchymal hematoma. **2C, 2D:** On selective angiogram of segment VIII branch of the hepatic artery, there is early filling of the portal venous system, suggesting arterio portal shunt. Note the dense hepatic parenchymal blush due to extrinsic compression by sub capsular hematoma (asterisk).

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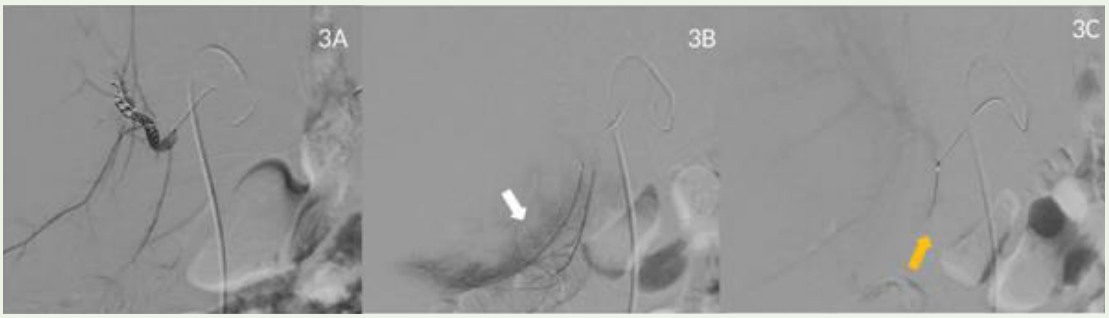

Figure 3: 3A: Hepatic angiogram post embolization of the arterio portal fistula demonstrating complete occlusion of segment VIII branch of hepatic artery. **3B:** Superselective angiogram of segment VI branch demonstrating abnormal vascularity splayed by the intraparenchymal hematoma (white arrow). **3C:** Anglogram post embollsatlon by PVA particles demonstrating complete occlusion of the segment VI branch (yellow arrow).

blood from the hepatic arterioles to portal vein radicles in the liver sinusoids, leading to opacification of the portal vein [1]. Large direct arterioportal fistulas can be seen in congenital vascular malformations and can also be iatrogenic post liver biopsy [2]. Large arterioportal shunts can be symptomatic and can result in portal hypertension and gastric variceal haemorrhage or ascites [2,3]. In our case, there was also a large intraparenchymal hematoma apart from sub capsular hematoma, which can be either due to concealed history of trauma or secondary to bleed within primary hepatic tumour like hepatic adenoma or hemangioma. Such cases are reported in literature, especially in HELLP (Hemolysis, elevated liver enzymes and low platelets) syndrome in patients with preeclampsia .Most of these cases are usually conservatively managed, if the clinical and lab parameters are within normal range. Sometimes, patients present with intra peritoneal rupture and shock [4]. We had to intervene in this case as there was significant drop in Haemoglobin in 36 hours of admission with tachycardia. Such hematomas are also reported in post liver transplant cases were hepatic angiogram was done in necessary cases to look for source of bleed. Hepatic arterioportal shunts can be seen in such conditions, which were embolised [5].

Due to shearing away of the liver capsule from raw surface of liver, extensive surface bleeders are usually seen in such rapidly expanding sub-capsular hematomas. In our case, most of these surface bleeders appear to arise from the peripheral portal vein radicles in CT angiogram and the presence of arterioportal shunt in hepatic angiogram is an indirect indicator of active haemorrhage [5]. The shunts could have made these surface bleeders as high pressure bleeders as the portal vein pressures would have increased due to shunting. These surface bleeders cannot be embolized. As there was Haemoglobin drop and early signs of hypotension, embolization of arterioportal fistula was considered to stop arterial haemorrhage and also reduce the pressure of surface bleeders from portal radicles which could help in spontaneous haemostasis.

Various embolization agents can be used to embolize the hepatic arterioportal shunts like detachable balloons, coils and microspheres [6]. The transient increase in the liver enzymes can be explained by marked parenchymal compression. These abnormal parameters, however, were normalised in 48 hours post procedure. Still conservative management has to be initially tried in these cases if the patient is clinically stable. If there is continuing drop in haemoglobin and signs of hypotension/shock, the causative factors have to be evaluated and an attempt for embolization should be done.

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