

Interventional Management of Subcapsular Hepatic Hematoma with Hepatic Compartment Syndrome After Laparoscopic Adrenalectomy

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Abstract Hepatic compartment syndrome is an increase in intrahepatic pressure sufficient to cause compromised hepatic perfusion. Early recognition and prompt management are essential for preventing catastrophic consequences including ischemic liver failure and hypovolemic shock. This is the rare case of laparoscopic adrenalectomy resulting in subcapsular hepatic hematoma with hepatic compartment syndrome. Contrast-enhanced computed tomography demonstrated extensive subcapsular hepatic hematoma with collapse of portal vein and inferior vena cava suggesting impending compartment syndrome. Diagnostical catheter angiography of the liver revealed innumerable foci of petechial extravasation from disrupted isolated arteries. Emergent percutaneous catheter decompression and transarterial embolization were successful.

Keywords Subcapsular hepatic hematoma · Hepatic compartment syndrome · Laparoscopic adrenalectomy · Therapeutic embolization · Percutaneous catheter drainage

Abbreviations

CT Computed tomography
POD Postoperative day

IVC Inferior vena cava
DSA Digital subtraction angiography

Introduction

An expanding subcapsular hepatic hematoma can result in two endpoints: hemoperitoneum and hepatic compartment syndrome, according to the presence of capsular rupture. Hepatic compartment syndrome, defined by an increase in intrahepatic pressure sufficient to cause hepatic vascular compromise and ischemic hepatic necrosis, is reported in cases of intrahepatic and subcapsular hepatic hematomas [1–3]. It should be suspected when a patient with subcapsular hepatic hematoma experiences rapid deterioration of liver function. After identification of hepatic compartment syndrome, immediate decompressive management is required to prevent irreversible consequences. Herein, we report a rare case of subcapsular hepatic hematoma with hepatic compartment syndrome after laparoscopic adrenalectomy.

Case Report

A 25-year-old man diagnosed with multiple endocrine neoplasia type 2A was admitted to our institute for adrenalectomy. He had operation history due to left pheochromocytoma and medullary thyroid carcinoma. Laparoscopic adrenalectomy was scheduled due to enlarging right adrenal nodules at follow-up computed tomography (CT). The operation was performed under general anesthesia. The patient was positioned in lateral decubitus position. The abdominal cavity was explored

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using a 4-port laparoscopic approach, with a liver retraction holder. Adrenal dissection was performed without difficulty. The patient was transferred to the recovery room in stable condition after surgery. On postoperative day (POD) 2, he complained of aggravating pain at the right upper abdominal and right flank areas without definite peritoneal sign. Laboratory studies revealed markedly elevated liver enzymes with significant drop of hematocrit: alanine aminotransferase 2153 IU, aspartate aminotransferase 2953 IU, hemoglobin 8.6 g/dL and hematocrit 25.8%. Preoperative laboratory values of liver enzymes and hematocrit were within normal range. Vital signs were as follows: blood pressure 141/85, heart rate 99/min, respiratory rate 24/min and temperature 36.5 °C.

Abdomen CT scans before and during the portal venous phase of contrast enhancement were performed. A large hematoma was noted at right subhepatic and perihepatic spaces without active contrast extravasation (Fig. 1A). Parenchymal infarctions appeared as multiple low-attenuation parenchymal lesions aligned in a row (Fig. 1B). CT scan also identified complete collapse of intrahepatic inferior vena cava (IVC) and portal vein due to extrinsic compression, suggesting impending hepatic compartment syndrome (Fig. 1C).

Under ultrasound guidance, emergency percutaneous catheter decompression was performed with 10.2-F drainage catheter (Multipurpose Drainage Catheter Ultrathane;

Cook, Bloomington, IN, U.S.A.). For subsequent angiographic evaluation, arterial access was obtained via the right common femoral artery using 5-F vascular sheath (Radifocus Introducer II; Terumo, Tokyo, Japan). Abdominal aortography with a 5-F pigtail catheter (Beacon Tip Royal Flush; Cook, Bloomington, IN, U.S.A.) revealed no definite extravasation from branches of celiac axis, inferior phrenic artery and both renal arteries. Right renal arteriography, using 5-F catheter (Rosch Hepatic; Cook, Bloomington, IN, U.S.A.), demonstrated no abnormalities at right renal capsular and adrenal branches. Selective catheterization of right inferior phrenic artery failed due to narrowed orifice. Common hepatic angiography, using 5-F catheter, revealed scattered subtle staining dots, along the right hepatic margin (Fig. 2A). Review of digital subtraction angiography (DSA) image with magnification tool verified innumerable foci of petechial extravasation from disrupted isolated arteries (Fig. 2B). After selective catheterization of right hepatic artery with 1.9-F microcatheter (Tellus; Asahi INTECC Co., Ltd., Seto, Japan), embolization was performed with single vial Gelfoam particles (560–710 µm particle size, Cali-gel; Hangzhou Alicon. Pharm. Sci. & Tec. Co., Ltd.) mixed with diluted contrast agent. Successful pruning of peripheral branches was achieved on post-embolization angiography (Fig. 3). The patient received 2 units of packed red blood cells transfusion after the procedure. A total of 850 ml

Fig. 1 **A** Coronal reformatted contrast-enhanced CT scan shows extensive subcapsular hematoma and almost completely collapsed IVC (black arrows). **B, C** Axial contrast-enhanced CT scan demonstrates multiple low-attenuation parenchymal infarction aligned in a row. There is some free intraperitoneal and retroperitoneal air (white arrows) from previous surgery. Slit-like flattening of portal vein (black arrow) and IVC (black arrowhead), with intervening parenchyma of caudate lobe produce characteristic “sandwich appearance”



Fig. 2 **A** Common hepatic angiography demonstrates medial displacement of hepatic arterioles and innumerable foci of petechial staining (black arrows) along entire right hepatic margin. **B** Magnification image reveals multifocal minimal petechial extravasation from isolated arteries

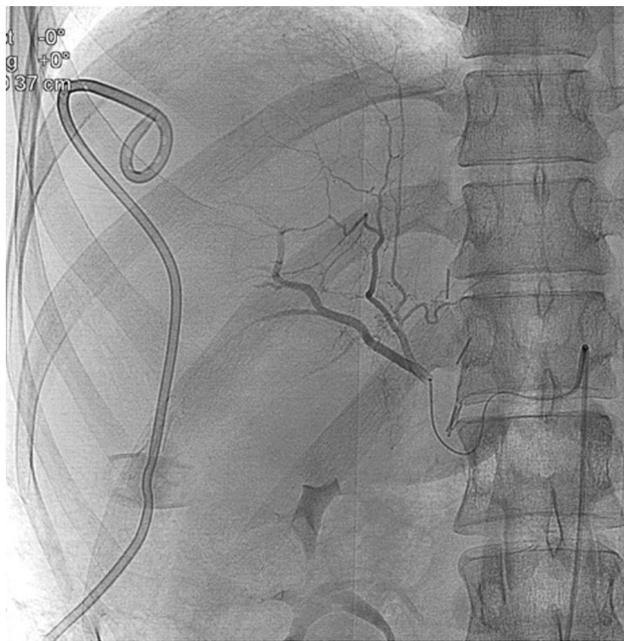
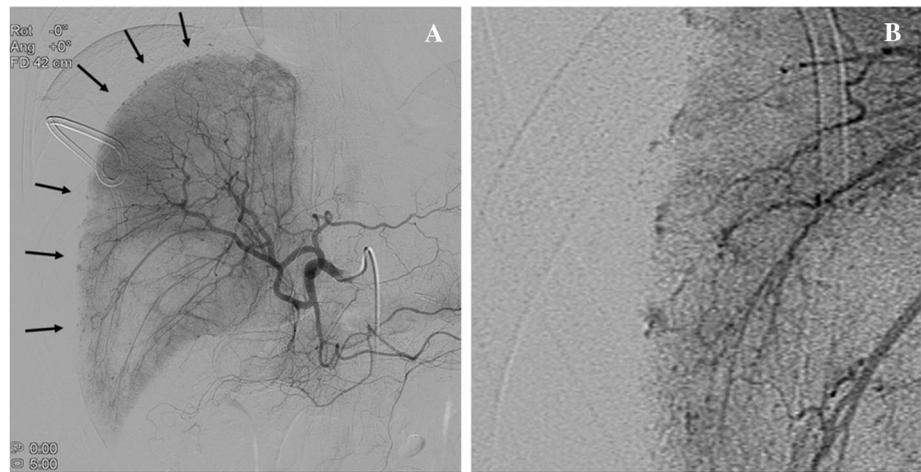


Fig. 3 Selective right hepatic angiography after embolization shows successful pruning of peripheral branches

hemorrhagic fluid was drained through pigtail catheter during the next 24 h. Catheter was left in place for 3 days, and catheter output was reduced to less than 10 ml/day and then removed. On POD 7, the patient was eventually discharged in stable condition with normalization of elevated liver enzymes. Follow-up abdomen CT scan obtained 2 months after embolization revealed interval decrease in the size of the subcapsular hepatic hematoma. Mild parenchymal atrophic change probably due to embolization was also noted at the periphery of right hepatic lobe.

Discussion

Loculated subcapsular hepatic hematoma usually demonstrates self-limiting course [4]. However, expanding hematoma may result in subsequent hepatic rupture or hepatic compartment syndrome and cause life-threatening problems. In the literature, iatrogenic subcapsular hepatic hematoma is usually related to laparoscopic cholecystectomy or endoscopic retrograde cholangiopancreatography [5, 6]. However, there has been only one case report following laparoscopic adrenalectomy, in which the patient demonstrated favorable clinical course with supportive care [7].

An isolated hepatic artery, a characteristic subgroup of the terminal hepatic arterioles, penetrates the liver parenchyma and connects to the hepatic capsular arterial plexus and hepatic venule without accompanying portal venule or bile duct [8]. The hepatic capsular arterial plexus usually communicates with extrahepatic arteries such as inferior phrenic, internal thoracic, intercostal and adrenal arteries. Dissection of the Glisson capsule by enlarging subcapsular hematoma may result in further disruption of isolated arteries. This self-aggravating process could be explained by angiographic findings of this case, which revealed active contrast extravasation from isolated arteries into subcapsular hematoma.

Hepatic compartment syndrome was first described with intrahepatic hematoma by Nissen et al. and defined as “The constellation of rapidly expanding intrahepatic hematoma with hepatic vascular compromise and hepatic necrosis” [1]. Because any rapid growing space-occupying lesion with intact hepatic capsule can lead to elevated intrahepatic pressure, the definition does not have to be limited to intrahepatic hematoma. Marcaire et al. reported that hepatic compartment syndrome could also be complicated by subcapsular hepatic hematoma [2]. Ando et al. emphasized that retrograde flow of portal vein on Doppler

ultrasonography and defined hepatic compartment syndrome as “intraparenchymal hypertension of the liver and reversal of portal flow resulting from large hepatic subcapsular hematoma” [3]. In our report, Doppler ultrasonography was not performed. If the flattening of portal vein is evident on CT scan, the diagnosis could be made based on clinical and CT findings [1, 2].

There is no established consensus for standard treatment of subcapsular hepatic hematoma with hepatic compartment syndrome. If there is no extravasation on dynamic CT, decompressive management may be sufficient to control complication [7]. However, extravasation of contrast may become evident after decompressive management as in this case. It was supposed that increased intrahepatic pressure obscured active bleeding focus until decompression was performed. When multiple extravasations are observed in subcapsular hematoma, Yoshida et al. recommend selective peripheral embolization of all active bleeding points to target isolated arteries [8]. Since the isolated hepatic artery is connected with hepatic capsular arterial plexus, additional embolization of extrahepatic collateral vessels is also often required [9]. In our report, limited embolization with pruning of distal hepatic vasculature might have been successful, because the active extravasation was not evident during angiography of extrahepatic collateral vessels [10].

In conclusion, we report the rare case of subcapsular hepatic hematoma with impending compartment syndrome after laparoscopic adrenalectomy. Hepatic compartment syndrome is a life-threatening condition, defined as increase in intrahepatic pressure sufficient to cause hepatic vascular compromise and ischemic hepatic necrosis. Immediate recognition and management are required to prevent catastrophic consequences including ischemic liver failure and hypovolemic shock. If the patient is hemodynamically stable, endovascular management may be considered as an alternative treatment option for hepatic compartment syndrome.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study. Consent for publication was obtained for every individual person's data included in the study.

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