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Complete title

Two cases of suspected liver compartment syndrome
treated with transarterial embolization

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Running head

Liver Compartment Syndrome

ABSTRACT We herein present two cases suspected of having liver compartment syndrome that were successfully managed with transarterial embolization (TAE). The first patient was a 40-year-old female involved in a car accident. Contrast-enhanced computed tomography (CT) showed a large intraparenchymal hematoma and active hemorrhaging in the hematoma. Transarterial embolization was performed. A hepatofugal portal flow was only detected in the right lobe of the liver, and a normal antegrade flow was observed in the left lobe. The second patient was a 73-year-old man who had fallen down a flight of stairs. Contrast-enhanced CT showed a large intraparenchymal hematoma. On angiography, a small hemorrhage was observed and TAE was performed. A hepatofugal portal flow was detected in the right lobe of the liver. Liver compartment syndrome is defined as intraparenchymal hypertension induced by a large subcapsular hematoma in a patient with blunt hepatic injury. Liver compartment syndrome causes a disruption in the normal liver circulation and may result in either hepatic ischemia or Budd-Chiari syndrome. It is important to prevent an enlargement of the hematoma by applying TAE.

Key words: angiography, blunt hepatic injury, compartment syndrome, arterial portal venous shunting, non-operative management

INTRODUCTION

Compartment syndrome is well known to be a complication of blunt injury to an extremity. This syndrome is defined as a condition in which increased pressure in a confined anatomical space adversely affects the circulation and threatens the function and viability of the tissues therein. Blunt hepatic injury has traditionally been operatively managed. Recently, however, some blunt hepatic injuries such as intrahepatic hematoma have been successfully managed non-operatively ¹⁾. Compartment syndrome of the liver was reported in 1999 by Pearl and Trunkey ²⁾. They presented a case in which a large subcapsular hematoma in the liver causing an elevated intraparenchymal pressure led to hepatic ischemia ²⁾. The ischemic appearance of the liver thereafter rapidly improved after surgical decompression ²⁾. We herein present two cases with the suspicion of liver compartment syndrome successfully managed non-operatively with arterial embolization.

CASE REPORTS

Case 1

A 40-year-old female operating a motor vehicle while under the influence of alcohol was involved in a car accident in which she fell a few meters into a ditch. Consequently, she was admitted to a nearby hospital. Although a hepatic injury was detected by computed tomogram (CT), she was conservatively observed with some fluid infusion. Six hours afterward, she was transferred to our emergency

department because of hypotension and progressive anemia.

Upon arrival, her consciousness was clear and she was not in distress. Her vital signs were as follows: blood pressure, 100/60 mmHg; pulse, 130/min; respiratory rate, 15/min and oxygen saturation, 99% on room air. Contrast-enhanced CT showed a moderate pooling of an intraperitoneal hemorrhage and a large intraparenchymal and subcapsular hematoma (**Fig. 1**). A contrast medium extravasation was detected in the hematoma. An active hemorrhage from a branch (A8) of the right hepatic artery was detected on angiography, and embolization was performed with micro coils and gelatin sponges. On hepatic arteriography, a hepatofugal portal flow was only detected in the right lobe of the liver (with a right hepatic arterial inflow and right portal venous drainage) (**Fig. 2**). In the left lobe, the left hepatic arterial inflow and the left hepatic venous drainage were observed. The day after undergoing transarterial embolization, her lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) levels were transiently elevated (**Table 1**). However, these laboratory findings gradually improved. No ascites was observed. She was eventually discharged in good condition.

Six months after the accident, follow-up angiography was performed and the previous segmental hepatofugal portal flow was found to have completely disappeared.

Case 2

A 73-year-old man fell down a flight of stairs in his house. He rested at home for an hour, and then sought admission to a nearby hospital because of increasing back pain. Eight hours after admission, he was transferred to our

emergency department because of liver injury and hypotension.

Upon arrival, his consciousness was clear. His vital signs were as follows: blood pressure, 127/72 mmHg; pulse, 114/min; respiratory rate, 18/min, and oxygen saturation, 99% on O₂ 5L/min administration. Contrast-enhanced CT showed a large intraparenchymal and subcapsular hematoma without any obvious contrast medium extravasation (**Fig. 3**). No intraperitoneal hemorrhaging was observed. On selective angiography, a small hemorrhage was observed from a branch (A6) of the right hepatic artery. Embolization was performed using gelatin sponges. A hepatofugal portal flow was detected in the right lobe of the liver (the right hepatic arterial inflow and the right portal venous drainage). A normal hepatic blood flow was observed in the left lobe (with a left hepatic arterial inflow and left hepatic venous drainage) (**Fig. 4**). Following transarterial embolization, he gradually improved without any elevation in the LDH, AST, and ALT levels (**Table 1**). In addition, no Ischemic damage of the liver or ascites were observed. He was discharged from our hospital in good condition.

Six months after the accident, his liver function was normal and no ascites were observed. Follow-up angiography was not performed because of his advanced age and due to his good liver function.

DISCUSSION

The concept of liver compartment syndrome was previously reported in 1999 by Pearl and Trunkey ²⁾. Liver compartment syndrome is the result of intraparenchymal hypertension induced by a large subcapsular hematoma ²⁾. In the

compensated period, the elevated intraparenchymal pressure changes the hepatic blood flow. Hepatic angiography shows the hepatic arterial inflow and the portal venous drainage in the whole area of the affected lobe, and the hepatic arterial inflow and the hepatic venous drainage in the unaffected lobe. When the intraparenchymal pressure further increases to a decompensated period, the either hepatic ischemia or Budd-Chiari syndrome (hepatic venous outflow obstruction) may occur ^{2,3)}.

Markert et al ³⁾ reported three cases of traumatic Budd-Chiari syndrome resulting from compression of the intrahepatic inferior vena cava (IVC) or confluence of the main hepatic veins due to a large intraparenchymal hematoma. In other previous reports ^{4,5)}, however, traumatic Budd-Chiari syndrome has often been regarded as traumatic thrombosis of the hepatic veins or IVC following IVC injury. No intraparenchymal hematoma was detected in the previous reports ^{4,5)}. Based on the findings of the previous reports ^{4,5)}, we suspect that the three cases reported by Markert et al. all represented a decompensated period of liver compartment syndrome.

A definitive diagnosis of liver compartment syndrome is based on the measurement of intraparenchymal pressure ²⁾. However, the measurement of pressure without a celiotomy is dangerous for trauma patients, such for our cases. Therefore, we could not measure the intraparenchymal pressure in our two patients. Notably, a large subcapsular or intraparenchymal hematoma was observed. The hepatic blood flow was also observed to have changed in the lobe affected by the hematoma. A hepatic arterial inflow and portal venous drainage were observed. However, no hepatic venous obstruction was detected. These observations may result from the intraparenchymal hypertension induced large hematoma, and such

patients are suspected of having liver compartment syndrome.

The operative management to decompress the intraparenchymal pressure for liver compartment syndrome has previously been reported ^{2,3}). Surgical decompression promptly improved the hepatic ischemia or Budd-Chiari syndrome that had resulted from liver compartment syndrome ^{2,3}). We were able to manage our cases non-operatively with arterial embolization. Even though a large subcapsular or intraparenchymal hematoma was detected in the liver by abdominal CT, the enlargement of the hematoma was successfully stopped in both cases by arterial embolization. Despite the altered hepatic blood flow in the affected lobe, the occurrence of both hepatic ischemia and Budd-Chiari syndrome could be prevented. These results suggest the importance of arterial embolization in order to prevent an increase in the intraparenchymal pressure leading to hepatic ischemia or Budd-Chiari syndrome.

A hepatofugal portal flow is also observed in trauma patients with arterial portal venous fistulae ⁶). However, the arterial portal venous flow is more focal than is that of liver compartment syndrome. In liver compartment syndrome, the hepatic arterial blood spreads throughout the peripheral area of the liver and then conversely flows to the portal vein. This blood flow is considered to be a hepatofugal portal flow. In arterial portal venous fistulae, the hepatic arterial blood flows into the portal vein via the fistulae. The hepatofugal portal flow in our two cases could thus be clearly distinguished from the arterial portal venous fistulae by hepatic angiography.

The optimal management of liver compartment syndrome remains to be clarified. Pearl and Trunkey ²) recommended operative decompression of the liver capsule to prevent a progression to permanent Budd-Chiari syndrome. We,

however, instead performed arterial embolization and carefully observed the liver function and other laboratory findings. In addition, follow-up angiography conducted six months after discharge showed that the first patient had progressed to permanent Budd-Chiari syndrome. In contrast, the second patient did not progress to permanent Budd-Chiari syndrome based on the clinical symptoms. We successfully managed these two cases non-operatively. We consider that the optimal management regimen for liver compartment syndrome to be as follows: 1) to prevent an enlargement of the hematoma in the liver by applying arterial embolization; 2) to observe and follow-up the liver function and other laboratory findings; and 3) to promptly decompress the intraparenchymal pressure when any signs of hepatic ischemia or Budd-Chiari syndrome are observed.

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FIGURE LEGENDS

- Fig. 1.** A contrast-enhanced abdominal CT of case 1.
Contrast-enhanced CT showed an intraperitoneal hemorrhage and a large intraparenchymal and subcapsular hematoma. Contrast medium extravasation was detected in the hematoma (arrow).
- Fig. 2.** Angiography findings of case 1.
Left, right hepatic arteriography after embolization is shown.
Right, a hepatofugal portal flow was identified.
- Fig. 3.** A contrast-enhanced abdominal CT of case 2.
Contrast-enhanced CT showed a large intraparenchymal and subcapsular hematoma. No active hemorrhage was detected.
- Fig. 4.** Angiography findings of case 2.
Left, right hepatic arteriography is shown.
Right, a hepatofugal portal flow was identified.

Table 1. Laboratory data of cases 1 and 2.

			Day 0	Day 1	Day 2	Day 3	Day 4	Day 5
Case 1	AST	(IU/l)	801	1264	2971	1478	574	93
	ALT	(IU/l)	474	955	1930	1703	1142	500
	LDH	(IU/l)	2067	2572	3896	1999	888	680
Case 2	AST	(IU/l)	623	2217	1614	612	236	99
	ALT	(IU/l)	717	1876	1723	1084	669	424
	LDH	(IU/l)	1116	4648	2141	658	384	327

Day 0; on admission to the former hospital, Day 1; on admission to our emergency department, AST; aspartate aminotransferase, ALT; alanine aminotransferase, LDH; lactate dehydrogenase

和文タイトル

Liver compartment syndrome を疑われ経動脈塞栓術にて治療した 2 症例

キーワード

動脈造影
鈍的肝外傷
コンパートメント症候群
動門脈シャント
非手術療法

要旨 肝コンパートメント症候群を併発した鈍的肝損傷に対し，経動脈的塞栓術 (transarterial embolization, TAE) と慎重な経過観察により保存的に治療可能であった 2 症例を経験したので，文献的考察を加え報告する．【症例 1】交通事故にて受傷した 40 歳の女性．造影 computed tomogram (CT) にて，肝右葉実質内の巨大血腫と，その血腫内への造影剤の血管外漏出を認めため TAE を施行した．血腫のある肝右葉にのみ遠肝性の門脈血流を認めた．左葉は左肝動脈から肝静脈に流れる正常血流であった．その後，順調な経過で退院となった．6 ヶ月後の血管造影では，肝血流は正常化していた．【症例 2】階段から転落し受傷した 73 歳の男性．造影 CT にて，肝右葉実質内に巨大血腫を認め，TAE を施行した．肝右葉にのみ遠肝性の門脈血流を認めた．左葉は正常血流であった．その後順調な経過で退院となった．高齢のため，フォローアップの血管造影は施行していないが，肝機

能に異常はなく，腹水も認めていない．【考察】

肝コンパートメント症候群とは，血腫の増大により肝皮膜下の内圧が高まり，肝血流に異常を来たした状態である．初期の段階では，皮膜下の血腫により内圧が高まり，肝静脈への血流が阻害され，遠肝性の肝動脈－門脈血流を認める．血腫の影響を受けていない他の部位では，正常の肝血流を示す．血腫が更に増大すると，肝虚血や，肝静脈の圧迫による **Budd-Chiari** 症候群へ進展する可能性もある．

このため，肝コンパートメント症候群の治療では，初期に **TAE** により血腫増大を防止することが重要である．また，その後も，肝逸脱酵素や肝機能などを慎重に経過観察し，肝虚血や **Budd-Chiari** 症候群の兆候が出現した場合は，外科的な血腫除去等による減圧を行う必要があると考える．