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Traumatic common hepatic artery injury causing isolated right hepatic ischemia due to a left accessory artery. A case report☆

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ABSTRACT

INTRODUCTION: Hepatic arterial liver flow is renowned for its redundancy. Previous studies have demonstrated that the common hepatic artery is not essential for liver survival. We present a case of a 31year-old involved in a high-speed motor vehicle accident whose liver survived thanks to the presence of an accessory hepatic artery.

PRESENTATON OF THE CASE: We present the case of a 31year-old male who sustained a traumatic injury of the proper hepatic artery following a motor vehicle accident. The patient suffered temporary right liver lobe ischemia due to the presence of an accessory left hepatic artery. This resulted in the selective formation of 'biliary lakes' distinctively within the territory of the right hepatic artery supply.

Simultaneously the patient developed a pseudo-aneurysm of the proper hepatic artery which required radiology intervention. At the time of pseudo-aneurysm embolisation, a rich network of arterial collaterals had formed between the accessory left hepatic and the inferior phrenic artery. On follow up the biliary lakes to the right lobe had resolved, but a small area at the periphery of the right lobe had encountered atrophy.

DISCUSSION: This case report is an '*in vivo*' demonstration of liver resilience to arterial flow re-distribution and demonstrates the ability of the biliary epithelium to recover from and ischemic injury.

CONCLUSION: Parenchymal liver survival is mostly independent from flow within the common hepatic artery. Acute and chronic liver parenchyma changes following interruption of hepatic artery flow can still occur.

1. Introduction

The main blood supply to the liver is through the common hepatic artery; however, there are several other possible sources of liver arterial flow. These can arise from the right or the left accessory hepatic artery (from the superior mesenteric artery and the left gastric artery respectively), and also from the inferior phrenic artery. Previous studies have demonstrated that the common hepatic artery is not essential for liver parenchymal survival [2].

We present the case of a 31 year-old male who was admitted to our emergency department following a motor vehicle collision. The injury had caused a traumatic interruption of flow within the common hepatic artery. As a result, the liver suffered temporary, but limited ischemic changes due to its rich collateral vascularization through an accessory left hepatic artery.

Forty-eight hours after the injury, the gall bladder became necrotic and was surgically removed. Three months later, multiple bilomas exclusively affecting the right liver lobe occurred. Due to the development of a hepatic artery pseudoaneurysm requiring embolization, we were able to angiographically demonstrate the collateral arterial flow within the liver. This consisted of a rich anastomotic network between the left accessory hepatic artery and the inferior phrenic artery. Interestingly, eight months following the initial injury, a '*restitutio ad integrum*' of 90% of the liver parenchyma had occurred.

2. Presentation of case

A 31 year-old male presented to the emergency room of our hospital following a high-speed motor vehicle accident. He was intubated on arrival due to a Glasgow Coma Scale (GCS) of 6 and, following the primary survey and initial resuscitation, he underwent a computed tomography (CT) scan of the chest, abdomen and pelvis. The CT scan showed a large traumatic left diaphragmatic hernia warranting surgical repair and a pancreatic head hematoma with interruption of the common hepatic artery arterial flow (Fig. 1). In addition, the presence of an accessory left hepatic artery was noted on the arterial phase of the CT (Fig. 2).

☆ This case report is reported in line with the SCARE criteria [1].

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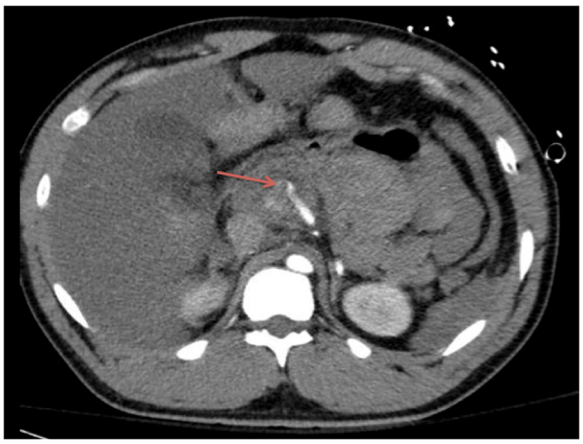

Fig. 1. Arterial phase of contrast enhanced CT. The arrow indicates the point at which flow in the hepatic artery abruptly stops.

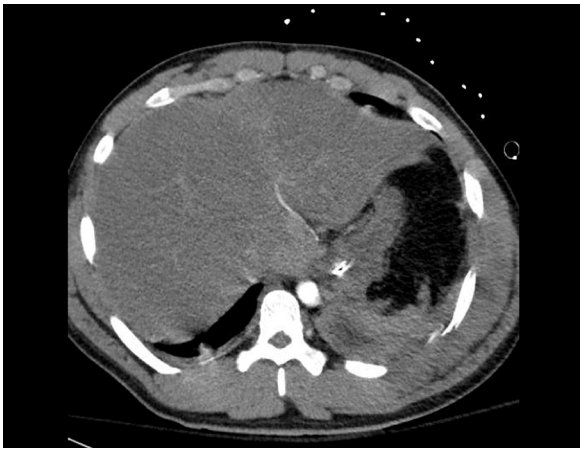

Fig. 2. Left accessory hepatic artery is visible on the same CT scan.

The patient was taken to the operating room for an exploratory laparotomy, repair of diaphragmatic defect. During the procedure, exploration of the lesser sac was carried out, which revealed a grade 1 pancreatic laceration with a small stable hematoma of the superior aspect of the pancreatic head. The liver parenchyma was viable with mild stigmata of blunt injury on segment 4 and no signs of ischemia.

On post-operative day 2 the patient became more acidotic, had peritonitis and an increasing requirement of vasopressors to maintain adequate perfusion. The patient was taken back to the operating room for re-exploration. The gall bladder was found to be necrotic, and a cholecystectomy was performed. The liver appeared mottled and was attributed to the high doses of vasopressors. The abdomen was left open with a temporary vacuum dressing. During the following 2 days, aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels increased (1480 and 1520 respectively) while the alkaline phosphatase (Alk Phos) level remained normal. Following the initial rise, liver enzymes started to downtrend 24 h following the injury, and returned to baseline by post-operative day 14. The patient was taken for an abdominal washout every 48 h, and full abdominal wall closure was eventually achieved on post-admission day 18. The patient made a slow but steady recovery and was discharged to a rehabilitation facility on post-admission day 46.

One month following discharge the patient presented again to the emergency room of our institution with abdominal pain, acute anemia and shortness of breath. Biochemistry tests showed a rise of the AST, ALT and alk phos (152, 154 and 458). Bilirubin level was 1.3 mg/dl. A CT scan was performed and showed a pseudoaneurysm (Fig. 3A) of the proper hepatic artery and multiple cystic biliary dilatations, which selectively affected the right lobe of the liver (Fig. 3B). The left lobe of the liver appeared normal. Further magnetic resonance cholangio-pancreatograhy examination showed the presence of the pseudoaneurysm exerting some compression on the porta hepatis as well as a non-dilated (2 mm) common bile duct.

CT and MRI both showed that the caudate lobe of the liver was atrophic. Furthermore, the accessory left hepatic artery had increased in size and appeared to the have become the dominant arterial flow within the liver.

Due to the presence of the pseudoaneurysm, the patient was taken for celiac axis angiography and embolization of the common hepatic artery pseudoaneurysm.

Angiography showed enlargement of the accessory left hepatic artery with several interlobar communications between the accessory left hepatic artery and the inferior phrenic artery (Fig. 4). No arterial flow was seen within the caudate lobe.

Three months after the hepatic artery embolization, liver function tests had normalized. A follow-up CT scan 9 months later showed complete resolution of the intrahepatic bilomas and restitutio ad integrum of the liver (Fig. 5).

3. Discussion

This case is an exceptional '*in vivo*' example of the liver's resilience to arterial ischemia.

Our patient presented with a blunt traumatic injury to the epigastrium, which caused a grade 1 injury to the head of the pancreas and interruption of flow within the common hepatic artery.

In the acute phase, this did not cause any major signs of acute liver ischemia such as large enzymatic derangement or lactic acidosis. The only structure that suffered from the acute interruption of the hepatic artery flow was, expectedly, the gall bladder. In fact, it became necrotic 48 h after the initial injury and had to be promptly

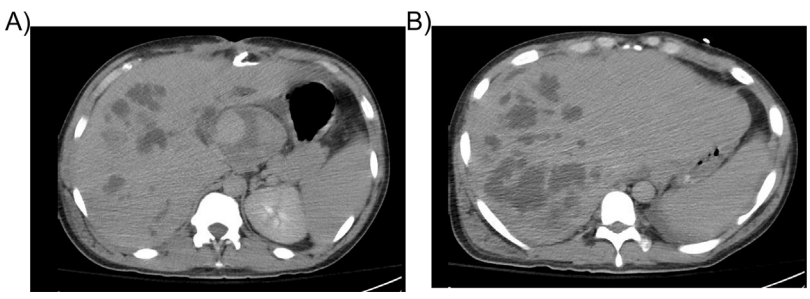

Fig. 3. A) Hepatic artery pseudoaneurysm lying adjacent to the porta hepatis. Caudate lobe is atrophic. B) Multiple *'bile lakes'* affecting exclusively the right lobe of the liver are visible 3 months following initial injury.

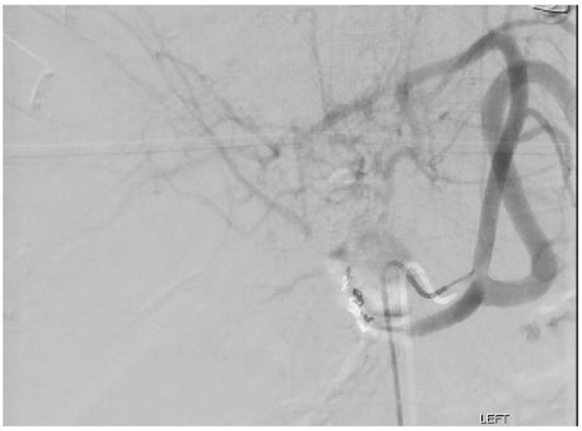

Fig. 4. Hepatic artery pseudoaneurysm has been embolized. Collaterals to the right hemiliver via the left accessory hepatic artery are visible.

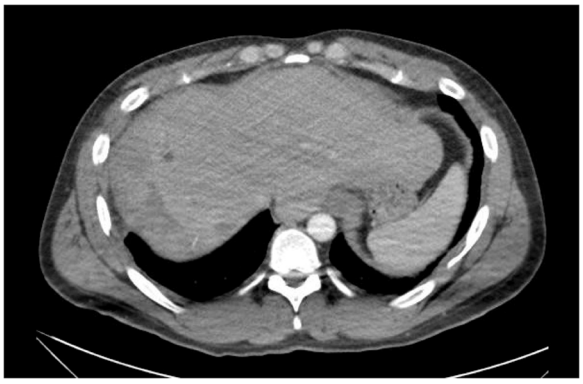

Fig. 5. Twelve months from the injury, biliary abnormalities have resolved. Minor abnormality of the vascularization of the periphery of the right liver lobe persists.

removed. Previous studies have determined the redundancy of the hepatic artery flow. In 1974, Mays et al., [2] demonstrated collateral arterial flow following ligation of the hepatic artery through inferior phrenic artery, interlobar arteries, intercalary '*de novo*' collaterals and gastric and pancreato-duodenal arteries forming the peri-biliary plexus. Collaterals would appear as soon as 10 h following hepatic artery ligation. It is reasonable to think that during that period of time a degree of liver ischemia does occur. In our case, this might have been reflected by the transient increase of the AST and ALT levels. Interestingly, 1 year after the injury, our patient developed marked atrophy of the caudate lobe. It is possible that the caudate lobe artery lacks an adequate collateral network of vessels due to its anatomical connection to the rest of the liver [3].

The fact that other authors [4] have described right liver necrosis following transection of the right hepatic artery during cholecystectomies does not assign the lobar hepatic arteries an 'end-vessel' connotation. In their series out of 86 patients with right hepatic artery injury only 2 developed liver necrosis. Those patients also had a transection injury of the bile duct and therefore damage to the peri-biliary plexus, a very important source of collateral arterial flow.

Another interesting finding in our case is the selective, right-sided intrahepatic biliary dilatation/extravasation observed 3 months from the initial injury.

One possible explanation for this finding may be the selective right hepatic duct compression caused by the pseudoaneurysm.

Obstructive jaundice secondary to hepatic or gastro-duodenal artery pseudoanuerysm has been previously described [5–8]. However, there are no previous reports of selective right or left hepatic duct obstruction. The pseudoaneurysm shown in Fig. 1 is undoubtedly lying at the porta hepatis. The common bile duct is non-dilated and it is difficult to envisage the compression of the right hepatic duct, which anatomically has a very short extra hepatic course. More likely, the large cystic biliary dilatations represent bilomas secondary to temporary right-sided liver ischemia.

Previous reports from different transplant groups have reported similar findings of multifocal biliary disruption with resultant '*bile lakes*' secondary to hepatic artery thrombosis [9,10]. Similar findings have been reported following hepatic artery thrombosis in pediatric liver transplants [11,12]. Interestingly, in transplant patients, the natural history of this condition is favourable as long as arterial flow is promptly restored. Our patient likely underwent a few hours of right hemi-liver ischemia resulting in the onset of '*bile lakes*' exclusively to the right lobe. The accessory left hepatic artery would have spared the left lobe from such a disease process. Eventually, the collateralization of flow between the accessory left hepatic and the inferior phrenic artery has allowed adequate supply to the biliary duct epithelium. To this respect, the superior mesenteric artery (SMA) angiogram done at the time of the hepatic artery embolization does not show an accessory right hepatic artery arising from the SMA. The full revascularization through the left accessory artery is proven by the fact that on the follow-up CT scan obtained 1 year following the injury, the liver appears essentially normal. The only abnormalities observed at that time include the atrophy of the caudate lobe and a semilunar area at the periphery of the right lobe (segments 5–8) with irregular contrast uptake (Fig. 5). Possibly, such area has suffered chronic ischemia and has undergone atrophy or secondary biliary cirrhosis changes. Nonetheless, overall liver function was entirely maintained.

This case is a clear demonstration of the resilience of the liver to ischemia.

Thanks to a series of fortuitous events such as the presence of an accessory left hepatic artery and also of the common hepatic artery pseudoaneurysm requiring embolization, we were able to unveil the natural history of a rare event such as the temporary ischemia of the right liver lobe, its effect on the biliary tree and the collateralization of flow through the left accessory hepatic artery.

4. Conclusions

The learning point of this case report is that although the arterial blood flow to the liver is redundant, common hepatic artery injuries can cause acute and chronic ischemic changes to the liver parenchyma.

This is a unique case of an '*in vivo*' demonstration of liver collateral re-vascularization through interlobar artery and '*de novo*' intercalary artery.

Conflicts of interest

The authors have NO conflicts of interest.

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Ethical approval

The Institutional Review Board (IRB) of our institution was consulted and apporpriate ethical approval was granted.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request".

Author contribution

Eduardo Fernandes : wrote the paper.
Corrado Pedrazzani: reviewed paper.
Marielia Gerena: Provided radiology advice.
Ellen Omi: Study design, paper writing.

Registration of research studies

researchregistry2551.

Guarantor

Eduardo Fernandes.

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