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Liver and Gallbladder Infarction as a Complication of Acute Aortic Dissection in the Splenomesenteric Trunk

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Abstract

Hepatic ischemia is a rare complication of acute aortic dissection. There are few reports about liver and gallbladder infarction occurring from aortic dissection; their main cause was hypotension due to cardiac tamponade. Here we report a case of type B aortic dissection with isolated liver and gallbladder infarction from visceral artery malperfusion of a splenomesenteric trunk. Interventional radiology effectively detected the cause of ischemia and enabled treatment of the visceral artery malperfusion.

Keywords: Acute aortic dissection; Visceral artery malperfusion; Liver infarction; Splenomesenteric trunk; Interventional radiology

Introduction

Case Presentation

Mesenteric malperfusion complicated by acute aortic dissection is a rare (3% to 7%) but ominous complication carrying a high risk of hospital mortality [1]. Although hepatic infarction is very rare because of the double blood supply from the hepatic artery and portal vein, abrupt hypoperfusion of the hepatic artery may be the underlying mechanism of hepatic infarction [2]. The splenomesenteric trunk is a very rare variation with an occurrence rate of less than 1% [3]. The collateral network in this variation is yet to be confirmed because of its rareness.

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A 57-year-old man was transferred to our institute with sudden-onset severe chest and back pain. He had not been treated for hypertension for 10 years despite being diagnosed. The patient's hemodynamics were stable on admission and emergency enhanced Computed Tomography (CT) showed an acute type B aortic dissection with occlusion of the celiac artery and stenosis of the superior mesenteric artery due to aortic dissection (Figure 1A,1B). The celiac artery was occluded because of compression of a false lumen (Figure 1A) and the superior mesenteric artery was stenosed owing to the involvement of the dissection (Figure 1B). Because he had no abdominal pain or abdominal symptoms and the laboratory data associated with visceral malperfusion were almost normal (Figure 2A), he was admitted to our hospital for conservative treatment. However, next-day laboratory tests showed significant elevation of the serum liver enzyme levels (Figure 2A) for which urgent angiography was indicated. Abdominal angiography showed a rare variation of the visceral branches: splenomesenteric artery (Figure 2B). Angiography also showed a poor collateral network between the superior mesenteric artery and the celiac artery (Figure 2B). After catheter intervention consisting of ballooning to the celiac artery and stent placement in the superior mesenteric artery, aortography demonstrated improved flow within the visceral arteries. Nine days later, because follow-up CT showed rapid enlargement of the distal arch, he underwent a total arch replacement. Although the serum liver enzyme level was decreased almost to the normal range (Figure 2A), the follow-up CT scan also revealed infarction of the broad right lobe of the liver and the wall of the gallbladder (Figure 3A,3B). The postoperative course was uneventful and a postoperative CT scan showed no significant changes. He was discharged 22 days postoperatively and is currently doing well without abdominal symptoms at 9 months of follow-up. The patient provided consent to publish the details and results of his case. The patient's identity has been protected.

Discussion

The liver has a dual blood supply. The portal vein, which is rich in nutrients and relatively high in oxygen, provides two-thirds of the liver's blood flow, while the hepatic artery, which is oxygenrich, supplies the rest. When the portal vein blood flow increases, the hepatic artery flow decreases

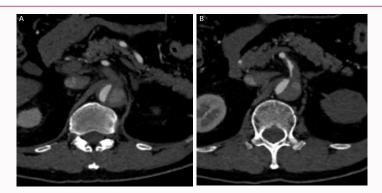


Figure 1: A: Initial computed tomography scan showing occlusion of the celiac artery. B: Initial computed tomography scan showing severe stenosis of the superior mesenteric artery.

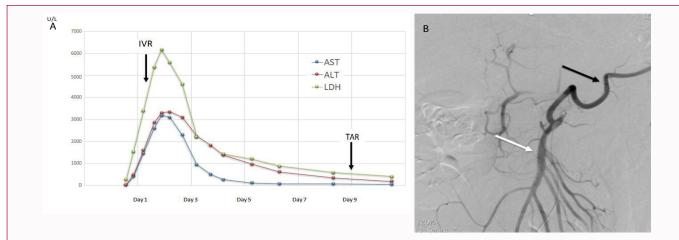


Figure 2: A: Laboratory data showed increased serum liver enzyme levels before interventional radiology that decreased after revascularization IVR, interventional radiology; AST, aspartate transaminase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; TAR, total arch replacement. B: Visceral angiography showing the splenomesenteric trunk: the superior mesenteric artery (white arrow) and splenic artery (black arrow) originate from a common trunk, and a sudden decrease in collateral flow is seen from the superior mesenteric artery to the celiac artery.

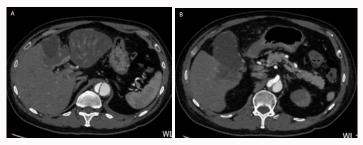


Figure 3: A and B: Follow-up computed tomography scans showing infarction of the right liver lobe and the gallbladder.

and vice versa because of the hepatic arterial buffer response. This dual reciprocal compensatory blood supply provides some protection against hepatic ischemia in healthy people. Despite its dual blood supply, the liver, a metabolically active organ, can be injured by acute ischemia, insufficient venous drainage, and specific vascular lesion. To our knowledge, cases of type B aortic dissection with celiac artery malperfusion resulting in liver and gallbladder infarction are extremely rare [4]. Gallbladder infarction is also rare, with a reported incidence of 1.1% [5]. Abdominal pain is not a reliable symptom of mesenteric ischemia due to the aortic dissection itself or may be masked by other prominent symptoms such as chest pain or an altered level of consciousness. Elevated blood enzyme levels may not be apparent before necrotic intestinal changes [6]. This situation carries a high mortality rate in mesenteric malperfusion of aortic dissection [7]. In the present case, he had no specific abdominal symptoms even after infarction occurred in the entire right lobe of the liver and the gallbladder. Multiple studies have been published regarding the safety of celiac artery coverage in thoracic endovascular aortic repair, celiac trunk resection in gastric and pancreatic surgery, and revascularization of the superior mesenteric artery alone for the treatment of acute intestinal ischemia [8]. This case demonstrates that single vessel revascularization may be inadequate in some cases. Aortic dissection causing branch vessel occlusion is traditionally treated by graft repair with concomitant fenestration and visceral reconstruction when necessary [7]. With recent advances in endovascular technology, preceded percutaneous stenting of the true lumen in malperfusion branch vessels when necessary has become an increasingly favored management option. Interventional radiology is

favorable in cases of type B aortic dissection with visceral malperfusion. In the present case, the splenic artery and superior mesenteric artery originated from a common trunk, while the common hepatic artery and left gastric artery originated from a common trunk [3]. This variation, termed the "splenomesenteric trunk," is quite rare. Based on angiography findings, it has a reported frequency of 0.3% to 0.8% [9]. Although its detailed mechanism has not yet been clarified, the collateral network between the celiac artery and the superior mesenteric artery might not have developed sufficiently because the splenic artery and superior mesenteric artery are already connected in this variation. Further studies regarding collateral network in this variation are needed to establish the appropriate treatment.

Conclusion

Visceral malperfusion in acute aortic dissection can cause liver infarction, and interventional radiology can enable its effective treatment. Further studies on the splenomesenteric trunk are needed.

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