Open surgical repair of a giant common hepatic artery pseudoaneurysm that perforated into the duodenum and common bile duct

Ken Tsuchida, MD, PhD,[a] Kyosuke Kokaguchi, MD, PhD,[a] Daijirou Akamatsu, MD, PhD,[b] *and* Kenji Namiki, MD, PhD,[c] *Osaki and Sendai, Japan*

ABSTRACT

This is a case of 60-year-old male patient with a history of heavy alcohol consumption and liver dysfunction who presented with a giant hepatic aneurysm. The incidence of giant hepatic aneurysms exceeding 10 cm in diameter is rare, particularly in the context of pseudoaneurysms. Furthermore, simultaneous perforation into the bile duct and duodenum is highly unusual. This case report elucidates the successful surgical management of a large pseudoaneurysm of the common hepatic artery that concurrently perforated the bile duct and duodenum, without any complications or deterioration of liver function. (J Vasc Surg Cases Innov Tech 2023;9:101226.)

Keywords: Common hepatic artery; Perforation into the bile duct; Perforation into the duodenum; Pseudoaneurysm; Surgical repair

According to Society for Vascular Surgery guidelines, treatment is indicated for common hepatic artery (CHA) aneurysms that rupture, manifest symptomatically, or present asymptomatically when they measure more than 2 cm or are associated with polyarteritis nodosa.[1] Pseudoaneurysms exceeding 10 cm in diameter are extremely rare. The common bile duct and duodenum were simultaneously perforated during open surgery in our case. It is crucial to assess collateral blood vessels in patients with impaired liver function. This article and photographs were published with the consent of the patient.

CASE REPORT

The patient was a 60-year-old man with a history of heavy alcohol consumption and liver dysfunction. He complained of diarrhea lasting 6 months, weight loss of 15 kg, and icterus in both eyes. A suspected hepatobiliary disorder brought the patient to our hospital. Epigastric examination revealed a pulsatile mass, but with no tenderness or pain. Contrast-enhanced computed tomography (CT) revealed dissection from the celiac artery (CA) to the CHA and a 10 cm × 15 cm large pseudoaneurysm protruding downward from the distal end of the false cavity (Fig 1). The common bile duct and duodenum were stenosed secondary to compression.

He was admitted to our hospital for further assessment and management despite a stable hemodynamic status. Laboratory investigations revealed white blood cell of 8.8 × 103/mm^3, hemoglobin of 7.9 g/dL, total bilirubin of 8.35 mg/dL, direct bilirubin of 5.35 mg/dL, albumin of 2.9 g/dL, prothrombin time-international normalized ratio of 1.27, and C-reactive protein of 2.3 mg/dL. According to the Child-Pugh classification, the patient was grade B with a score of 8. It was difficult to determine whether the liver damage was caused by obstructive jaundice or alcohol-induced liver damage.

Given the high degree of liver damage caused by billary obstruction and the associated perioperative risk, reduction of jaundice was initially attempted. Endoscopic retrograde cholangiopancreatography was performed and revealed compression of the duodenum with no evidence for the presence of a fistula. A stent was placed in the bile duct to promote effective bile excretion. Hemodynamics remained stable, and total bilirubin gradually decreased to 3.25 mg/dL.

Angiography was conducted for further assessment and potential ad hoc intervention.

A large pseudoaneurysm originating from the distal side of the CHA, 8 mm proximal to the branch point, was found (Fig 2). As endovascular treatment was not pursued, concerns regarding hepatic ischemia secondary to branch embolization or insufficient stent graft patency persist.

The patient experienced fever and abdominal pain 2 days after angiography. The biochemical profile remained unchanged, and there were no signs that would indicate gastrointestinal bleeding. Due to symptomatic changes, emergency surgery was deemed necessary despite stable vital signs. We utilized a hybrid operating room to allow balloon blockade of the aorta or CA in cases where central blockade was impractical. We evaluated the great saphenous vein for possible use, but it was

From the Division of Vascular Surgery, Osaki Citizen Hospital, Osaki[a]; the Division of Vascular Surgery, Department of Surgery, Tohoku University Hospital, Sendai[b]; and the Osaki Citizen Hospital, Osaki.[c]

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Correspondence: Ken Tsuchida, MD, PhD, Division of Vascular Surgery, Osaki Citizen Hospital, 3-8-1 Furukawahonami, Osaki 989-6183, Japan (e-mail: kent@surg.med.tohoku.ac.jp).

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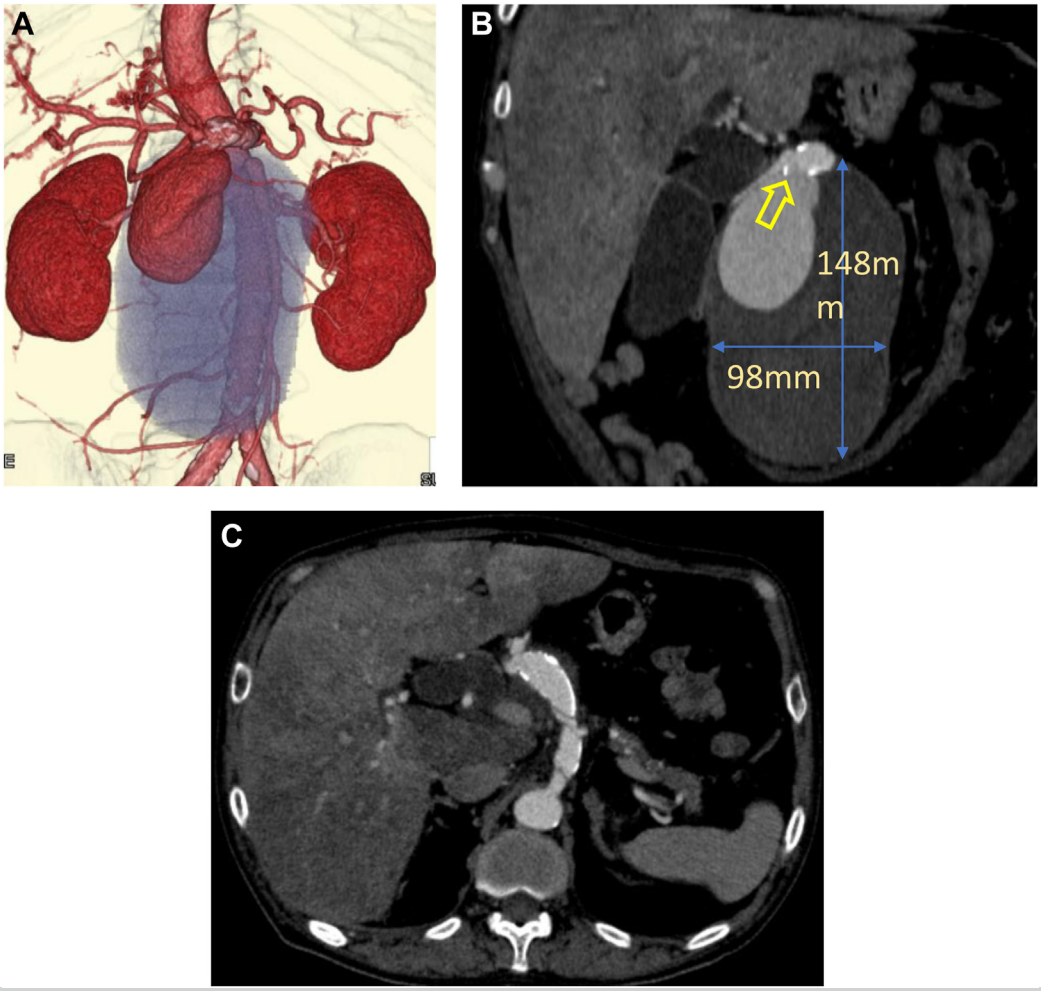

Fig 1. Preoperative computed tomography (CT) angiography (CTA). **A,** Three-dimensional reconstructed CT image: Preoperative CTA images showed dissection from the celiac artery (CA) to the common hepatic artery (CHA), as well as a large pseudoaneurysm downward from the distal end of the false cavity. **B,** Sagittal view of the pseudoaneurysm measuring 98 × 148 mm. The *arrow* denotes the orifice of the pseudoaneurysm. **C,** Dissection from the CA to the CHA.

extremely thin. The abdomen was opened via a median skin incision. The lesser omentum was then incised, revealing thickening of the wall and dense adhesions with surrounding tissue. (Fig 3, *A*). Therefore, no attempt was made to expose the celiac trunk.

The proximal CHA, as verified by ultrasound guidance, was carefully controlled, which was similarly applied to left hepatic artery (LHA), middle hepatic artery (MHA), right gastric artery (RGA), and gastroduodenal artery (GDA). Because RGA and GDA arose from a pseudoaneurysm, ligation was performed at their origin to preserve the superior mesenteric artery (SMA)-posterior inferior pancreaticoduodenal artery (PIPDA)/anterior inferior pancreaticoduodenal artery (AIPDA)-GDA-right hepatic artery (RHA) route.

The aneurysm was incised followed by excision of the thrombus. After careful removal of the mural thrombus, a biliary leak was discovered within the pseudoaneurysm. Moreover, 1-cm fistulas were found in the descending portion of the duodenum and the distal bile duct (Fig 3, *B*). The presence of the biliary duct stent was noted; however, the fistula was simply sutured without extraction of the biliary stent to prevent stenosis. The duodenal fistula was also simply sutured. Absence of bile leakage was confirmed.

Following meticulous trimming and verification of the pseudoaneurysm origin's structural integrity, we performed primary closure without incurring CHA stenosis. The dissection flap, which extended into the CHA, was excised. Each hepatic artery branch was audible via Doppler ultrasound. Ultrasonography revealed a type I waveform for LHA, MHA, and RHA, and liver blood flow was determined to be acceptable. The greater omentum was used to cover the repair sites of the CHA, duodenum, and common hepatic duct.

Postoperative CT revealed a preserved blood flow in the CA-CHA-LHA/MHA and also in the SMA-PIPDA/AIPDA-RHA (Fig 4). Gastrointestinal fluoroscopy revealed no duodenal leakage. The patient's postoperative course was uneventful, and he was

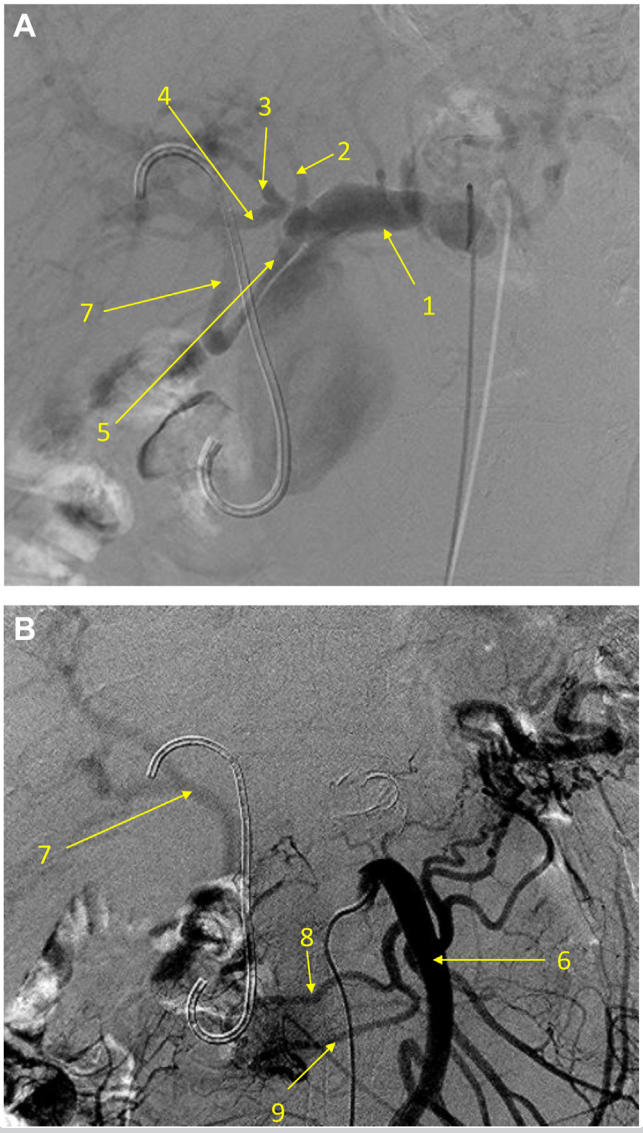

Fig 2. Preoperative angiography. **A,** Selective angiography of the common hepatic artery (CHA) showed that the left hepatic artery (LHA) and middle hepatic artery (MHA) formed a common trunk. The common trunk, gastroduodenal artery (GDA), and right gastric artery (RGA) originated from the pseudoaneurysm. The right hepatic artery (RHA) bifurcated from the GDA. **B,** Selective angiography of the superior mesenteric artery (SMA) showed that the RHA was contrasted via the collateral vessels from the posterior inferior pancreaticoduodenal artery (PIPDA) and the anterior inferior pancreaticoduodenal artery (AIPDA). The arrow numbers indicate: (1) CHA; (2) RGA; (3) MHA; (4) LHA; (5) GDA; (6) SMA; (7) RHA; (8) PIPDA; and (9) AIPDA.

discharged. The patient attended the outpatient clinic 8 months after surgery without liver dysfunction. Hepatic ultrasound revealed good hemodynamics and no pseudoaneurysms. He was in good condition and had gained more than 10 kilograms.

The pathological diagnosis was a CHA pseudoaneurysm (Fig 5).

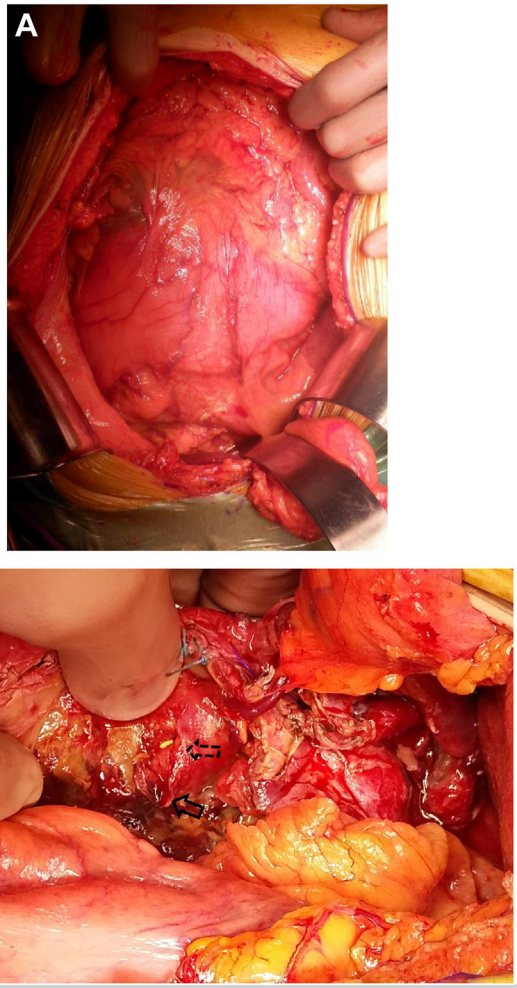

Fig 3. Intraoperative findings. **A,** Laparotomy findings. An approximately 20-cm median skin incision was made to the abdomen. The stomach was ventrally compressed by a massive pseudoaneurysm. **B,** Findings of an open pseudoaneurysm. The *dotted arrow* denotes perforation of the bile duct and presence of the bile duct stent. The arrow denotes perforation into the duodenum.

DISCUSSION

Extrahepatic hepatic artery aneurysms (HAAs) primarily occur in the CHA and are classified as true aneurysms.[1-3] True HAAs exceeding 10 cm have been reported 11 times.[4-13] However, this is the first case documenting a giant, chronic pseudoaneurysm exceeding 10 cm in the CHA. Although most CHA pseudoaneurysms stem from traumatic or iatrogenic origins, no previous history of endovascular or open surgery, trauma, or infection was noted in this case. The development of this pseudoaneurysm may have arisen from dissection of the CA branching into the CHA.

Pseudoaneurysms necessitate urgent surgical intervention. However, Child-Pugh class B liver cirrhosis increases the perioperative risk.[14] Despite hepatic dysfunction, the

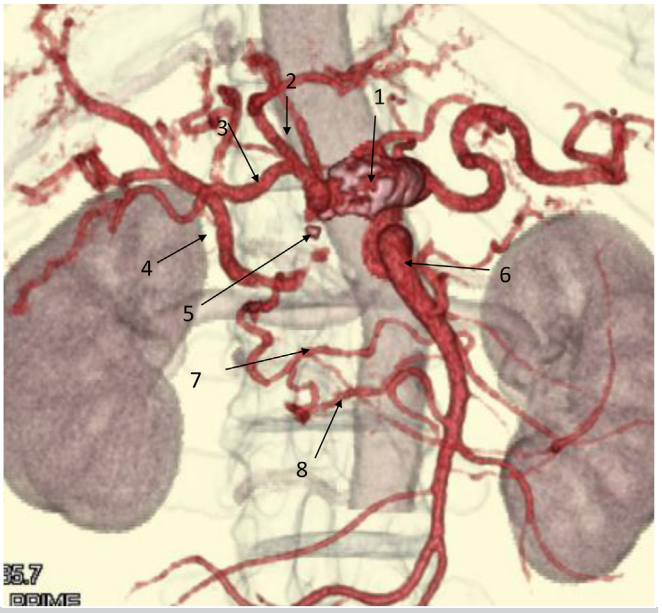

Fig 4. Postoperative computed tomography angiography (CTA). Postoperative CTA revealed preserved blood flow in the celiac artery (CA)-common hepatic artery (CHA)-lower hepatic artery (LHA)/middle hepatic artery (MHA) and also in the superior mesenteric artery (SMA)-posterior inferior pancreaticoduodenal artery (PIPDA)/anterior inferior pancreaticoduodenal artery (AIPDA)-right hepatic artery (RHA). The arrow numbers indicate: (1) CHA; (2) LHA; (3) MHA; (4) RHA; (5) gastroduodenal artery (GDA); (6) SMA; (7) PIPDA; and (8) AIPDA.

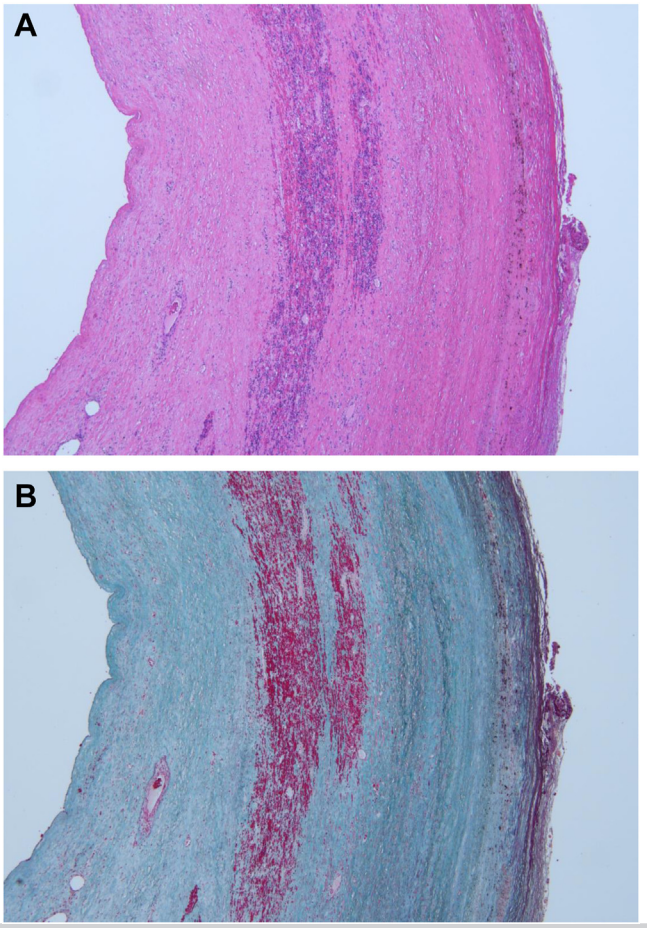

Fig 5. Histological examination. Histological examination revealed that the lesion was composed of stratified fibrous connective tissue with lymphocyte aggregations, with minimal presence of elastic fibers or smooth muscle fibers, leading to a diagnosis of pseudoaneurysm. **A,** Hematoxylin-eosin stain; **B,** Elastica-Masson stain.

patient maintained stable hemodynamics; thus, priority was given to resolving the jaundice. A patient's overall health status and the anatomy of the aneurysm determine whether open or endovascular repair is appropriate. Despite their rarity, liver necrosis and gangrenous cholecystitis can develop after endovascular embolization. In the preoperative judgment, open surgery was selected over endovascular therapy for insufficient liver blood flow and revascularization evaluation. Intraoperatively, bypass surgery was considered as a revascularization modality. However, the great saphenous vein was deemed too narrow, and the infection risk associated with prosthetic conduits made its use unadvisable. Suturing of the rupture site ensures an anterograde blood flow from the CHA to the LHA and MHA. In addition, ligation of the GDA at the point of origin of the pseudoaneurysm ensures that the SMA to the RHA via the collateral vessels is secured.

Our patient's aneurysm perforated both the duodenum and the common bile duct, which has not been previously reported. As the perforation was discovered incidentally during careful removal of the mural thrombus, careful inspection of the aneurysm's interior is necessary in cases of a giant CHA aneurysm. In imaging studies preoperatively, no signs of infection or inflammation were observed, despite slightly elevated C-reactive protein levels. Simple closure of the small fistulae in the duodenum and biliary tract was sufficient for reconstruction. Reconstruction may be complex if there is significant inflammation or necrosis.

CONCLUSIONS

A giant pseudoaneurysm of the CHA with perforation of the duodenum and common bile duct was successfully repaired with open aneurysmoplasty. This is the first documented case of a giant, chronic CHA pseudoaneurysm larger than 10 cm. Patients with HAA and hepatic dysfunction require adequate collateral circulation to avoid serious complications. Additionally, adjacent organ perforation must be assessed.

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