Surgical treatment of a huge hepatic artery aneurysm without revascularization—Case Report

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1. Introduction

Visceral artery aneurysms are rare and account for approximately 1–2% of all vascular diseases [1,2]. The rate of hepatic artery aneurysms (HAA), defined as an artery having at least a 50% increase in diameter compared with the expected normal diameter of the artery [3], is approximately 20% [4]. Furthermore, a huge HAA is rarer and presents as a life-threatening emergency. Because of a high risk of rupture, the aneurysm can easily cause high morbidity and mortality. We describe in line with the SCARE criteria [5] a novel case of successful surgical treatment of a huge hepatic artery aneurysm without complete revascularization in a male patient.

2. Case report

A 68-year-old man presented frequent urination that had begun 1 week before referral to our hospital. The patient had a past medical history of hypertension and constipation and was a former smoker. During the initial physical examination, there was a pulsatile large mass in the epigastrium. A blood test on admission showed that all chemical parameters were within the normal range (Table 1). However, an initial computed tomographic (CT) scan revealed a huge HAA (67–84 mm diameter), which displaced the common hepatic artery in an upwards direction and the gastroduodenal artery in a forwards direction (Fig. 1a–d). In addition, although the CT scan showed that the primary arteries around the pancreas and spleen were intact, the bilateral proper hepatic arteries were narrowed. After the patient underwent laparotomy, a pulsatile and huge HAA was confirmed and narrow bilateral proper hepatic arteries were identified (Fig. 2a). The pulsation in the aneurysm disappeared where there was a cluster of the common and proper hepatic arteries (Fig. 2b). Beyond the cluster, intraoperative ultrasound (IOUS) showed pulsatile inflow to the left hepatic lobe in addition to portal blood flow. Infow and backflow of blood to the aneurysm was confirmed (Fig. 2b). The aneurism was resected, and the common hepatic artery and base of the proper hepatic artery were ligated; however, revascularization of the proper hepatic artery was not performed due to anatomical limitations. A liver vasculature drug (glycyrrhizin acid) and prostaglandin E1 were administered from day 1, and stopped on day 10 and 14, respectively. A CT scan on day 7 showed cholecystitis and hepatic...
infarction of the medial segment of the left hepatic lobe (Fig. 3). Percutaneous transcatheter gallbladder drainage was performed. The recovery of symptoms and postoperative blood tests progressed satisfactorily (Table 1), and he was discharged 30 days after the operation. After two months of monitoring him post discharge, open surgery for cholecystectomy as a second operation was performed from the previous upper abdominal wound. After detaching strong adhesion of intestinal tract to liver, atrophied gall bladder was confirmed and resection was performed.

3. Discussion

HAA is not a common vascular disease, for approximately only 20% of all visceral artery aneurysms are HAA [5,6]. Previous studies indicated that aneurysm rupture was observed in 21–80% of all HAA cases, and one report showed that operative mortality was 33% in the group with ruptured aneurysms and 0% in the group who underwent elective surgery [7]. For patients with a 2–5 cm diameter HAA, electing for the surgical intervention may be more controversial, while a diameter ≥5 cm indicates a huge HAA, and usually some surgical treatment or intervention is required. Unfortunately, over half of patients with non-ruptured HAA did not have any specific symptoms that could suspect the existence of HAA. Previous studies have indicated that 8–43% and 3–5% of all patients with non-ruptured HAA had abdominal pain (or right upper quadrant pain) and obstructive jaundice, respectively [7,8]. Although our patient with a huge HAA presented “frequent urination”, there are no studies and reports on the correlation between various symptoms, including urination, and the existence of HAA.

HAA is manifested most commonly in men and associated with arteriosclerotic diseases, hypertension, collagen vascular diseases, and fibromuscular dysplasia [9]. This patient had past medical history of hypertension, which suggested that he had an inducible factor causing HAA. Although little is known about the relationship, it is reasonable to think that past smoking history could have affected the incidence and deterioration. However, so far there have not been any reports on the etiological correlation between incidence of HAA and other diseases.

Since 1903 when Kehr et al. reported the first successful ligation of an HAA [10], many therapeutic alternatives have been reported for repairing HAA. The differences in surgical strategy are due to the complex anatomy of the visceral artery and sensitivity of the patient to hepatic ischemia [7]. Options for open surgical treatment of HAA include ligation, excision, grafting and hepatic resection [7,11]. Open surgical repair is the traditional method for treating HAA; however, many patients with visceral artery aneurysm develop severe pancreatitis and hepatobiliary disease, which may require high-risk laparotomy and eventually result in high mortality. Also, it is extremely hard to explore the aneurysm and the source of bleeding from hematoma by laparotomy for a ruptured aneurysm, and these patients may require additional operations. For these reasons, endovascular surgery is becoming increasingly popular in selected patients. In select patients with common hepatic artery aneurysms, coil embolization could be applied safely if adequate collateral circulation ensures hepatic perfusion [8]. However, one report on endovascular intervention for visceral artery aneurysm demonstrated that 30–40% of patients treated by endovascular treatment and embolization developed major complications such as late recurrence requiring open surgical repair, splenic infarction and severe pancreatitis [12]. Embolization itself can cause an increased compensatory vascular flow, which may induce progression of heterotopic aneurysm. Also, irregularity of the HAA lumen and narrowness of the outflow vessels may result in prolongation of the irradiation time. Furthermore, whether hepatic artery revascularization is needed after exclusion of HAA is also a con-

Fig. 1. Computed tomography (CT) scan before surgical operation. (a) Branch section of celiac artery. (1) common hepatic artery. (b) Maximum of a huge hepatic artery aneurysm (HAA) with 67mm–84mm. (c) The origin of a HAA. (2) the entry of common hepatic artery. (c) 3-D reconstruction CT imaging for a HAA.
Table 1
Pre- and postoperative blood test.

| Variables | Normal value | 30 Oct 2015 (Admission) | 3 Nov 2015 (Operation) | 4 Nov 2015 (1POD) | 6 Nov 2015 (3POD) | 10 Nov 2015 (7POD) | 23 Nov 2015 (20POD) | 3 Dec 2015 (30POD) |
|-----------|--------------|--------------------------|------------------------|-------------------|-------------------|-------------------|---------------------|---------------------|
| WBC (× 10^9/μL) | 3300–8500 | 8200 | 16700 | 18600 | 16800 | 12800 | 7000 | 8300 |
| RBC (× 10^12/μL) | 425–555 | 445 | 350 | 375 | 340 | 335 | 310 | 357 |
| Hb (g/dL) | 13.7–16.8 | 13.7 | 10.6 | 11.6 | 10.6 | 10.5 | 9.4 | 10.5 |
| Ptt (× 10^5/μL) | 15.8–34.8 | 21.2 | 14.4 | 17.6 | 10.8 | 21.8 | 36.1 | 36.3 |
| AST (U/L) | 1–30 | 20 | 49 | 337 | 190 | 35 | 17 | 22 |
| ALT (U/L) | 40–42 | 23 | 60 | 337 | 493 | 100 | 17 | 26 |
| D-Bil (mg/dL) | 0.0–0.4 | 0.05 | 0.06 | 0.05 | 0.24 | 0.32 | 0.09 | 0.06 |
| I-Bil (mg/dL) | 0.0–0.5 | 0.80 | 0.86 | 1.03 | 1.00 | 0.86 | 0.50 | 0.55 |
| LDH (U/L) | 124–222 | 111 | 206 | 614 | 283 | 233 | 110 | 118 |
| γGTP (U/L) | 13–64 | 22 | 16 | 15 | 33 | 174 | 43 | 33 |
| TP (g/dL) | 6.6–8.1 | 6.9 | 5.4 | 5.6 | 5.0 | 5.6 | 6.5 | 7.0 |
| ALB (g/dL) | 4.1–5.1 | 3.7 | 3.3 | 3.4 | 2.4 | 2.2 | 2.4 | 3.0 |
| CK (U/L) | 59–248 | 53 | 112 | 286 | 182 | 62 | 23 | 26 |
| BUN (mg/dL) | 8–20 | 15.4 | 13.7 | 12.6 | 18.0 | 10.6 | 17.0 | 9.7 |
| Cre (mg/dL) | 0.65–1.07 | 1.09 | 0.89 | 0.79 | 0.78 | 0.74 | 0.70 | 0.83 |
| Na (mEq/L) | 138–145 | 140 | 140 | 137 | 136 | 136 | 131 | 133 |
| K (mEq/L) | 3.6–4.8 | 4.5 | 4.2 | 3.9 | 3.9 | 3.6 | 4.5 | 4.8 |
| Ca (mg/dL) | 8.8–10.1 | 8.4 | 8.1 | 8.1 | 8.1 | 7.8 | 8.4 | 9.0 |
| CTR (mg/dL) | 0.00–0.14 | 0.05 | 0.05 | 0.94 | 6.02 | 24.05 | 13.72 | 3.71 |

Fig. 2. HAA under operation. (a) bilateral proper hepatic arteries shown as (1), and (b) inside of the aneurysm after clump and incision, (2) common hepatic artery and (3) inlet of the aneurysm.

Fig. 3. CT scan on day 7 after aneurysm resection. Cholecystitis and hepatic infarction of medial segment of left hepatic lobe were confirmed.

Controversial matter. Revascularization of the proper hepatic artery was not performed in our case because of anatomical limitations and the problem with long-term patency of grafts [7]. One case series showed that resection of giant HAA without revascularization did not deteriorate the biochemistry markers of liver function [13], and a more recent case report mention that revascularization was not considered due to the satisfactory hepatic arterial flow by intra-operative ultrasound [14]. Nonetheless, in this case, although collateral circulation via the hepatic left lobe as shown by IOUS was confirmed, the patient developed postoperative cholecystitis. Thus, if there is no revascularization of the right proper artery, cholecystectomy should be considered as an additional treatment.

In summary, open surgical treatment was an effective procedure to treat a huge HAA, and cholecystectomy should be considered as an additional treatment option in patients with no revascularization. Our study indicates that reconstruction of hepatic arterial inflow after resection should always be considered even if there is evidence of arterial collateral circulation.

Conflicts of interest

The authors declare that they have no conflicts of interest, financial, personal, or otherwise which could influence bias.

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Ethical approval

Ethical approval was not required for this case report in our institution.

Consent

Written consent was from the patient to write this case report and accompanying images. Identifying details have been omitted.

Author contribution

Dr. Tomohiro Imazu — corresponding author; reviewing patient notes, writing articles, analysing images, approving final submission.

Dr. Masateru Uchiyama — writing article and critical analysing data.

Dr. Shigefumi Matsuyama — carrying out the research and collecting the data and images.

Dr. Mitsuru Iida — revising article and collecting the data and images.

Dr. Tomoki Shimokawa — major contributing in writing the article and approving final submission.

Registration of research studies

N/A (This case is not clinical trial).

Guarantor

Dr. Tomohiro Imazu

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