Hepatic Artery Aneurysm with Excision of Celiac Axis *

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THE PRESENCE of an intra-abdominal aneurysm is usually an indication for surgical treatment. The grave prognosis of this pathology has been known for many years, but only recently has highly successful excisional type surgery been performed. The abdominal aorta is, of course, the most common vessel involved.

Smaller vessel aneurysms have a similar indication for surgical removal and several cases of splenic artery aneurysms have been successfully resected. The recorded cases of hepatic artery aneurysm also carry the same prognosis as far as death from hemorrhage is concerned. However, the importance of blood with high oxygen content to the liver makes surgery a more serious consideration.

Ligation of peripheral aneurysms had been carried out since the first century.[28] Hepatic arteries have been ligated successfully since the beginning of the 20th century with Kehr's case being the first recorded.[25] Haberer in 1905 outlined the operative procedure for ligation and showed that the hepatic artery could be ligated with impunity proximal to the gastroduodenal artery.[19] The blood was then supplied through the superior mesenteric artery by anastomosis about the head of the pancreas. In aneurysms, partial thrombosis often allows increase in collateral circulation, making ligation of the hepatic artery safe in any area.

The case reported here is the successful complete excision of a hepatic artery aneurysm and is the first case in which complete excision of the celiac axis was necessary.

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Case Report

C. C., a 67-year-old white male, was admitted to the hospital on May 26, 1957, with a nine day history of abdominal pain, nausea, vomiting and fever. There was no past history suggestive of gallbladder disease. He had received treatment for osteoarthritis in the past. Physical examination revealed a blood pressure of 150/90 mm. of mercury, temperature 103° F., pulse of 100 per minute. The sclera was icteric and funduscopic examination revealed a grade II retinopathy. There were crackling rales in both lung bases. The abdomen was tender, especially in the right upper quadrant with a smooth mass palpable 8 cm. below the right costal margin. A diagnosis of acute cholecystitis was made. Treatment with antibiotics, gastric suction and intravenous fluids was started. Response to this treatment was prompt and he was started on a low fat diet on the fourth hospital day. On June 8, 1957, he was released from the hospital.

On July 9, 1957, he was operated upon for interval cholecystectomy. The gallbladder was filled with white, mucoid material and several stones. The gallbladder was edematous and injected. Upon exploring the abdomen a pulsating mass (6 cm. × 4.5 cm.) was found just superior to the body of the pancreas. A cholecystectomy was performed; the common duct was explored and found to be normal. The mass was then further explored and found to be an aneurysm of the hepatic artery beginning at the celiac axis and extending to the gastroduodenal artery (Fig. 1). The right gastric artery arose distal to the gastroduodenal. The aneurysm was partly thrombosed, but was extremely thin on the anterior wall. It was felt that rupture was imminent and surgical treatment was therefore mandatory. Since the gastro-duodenal artery could be left intact, surgical excision was the procedure of choice. However, due to the extension of the aneurysm to the celiac axis, a safe ligation could not be performed distal to the celiac axis. Therefore the celiac axis was entirely dissected with a normally located splenic and gastric artery being identified. The celiac axis was then ligated about 0.5 cm. from the aorta and the splenic and left gastric arteries were also ligated. The spleen was not removed. The aneurysm was then dissected from the pancreas and the portal vein by

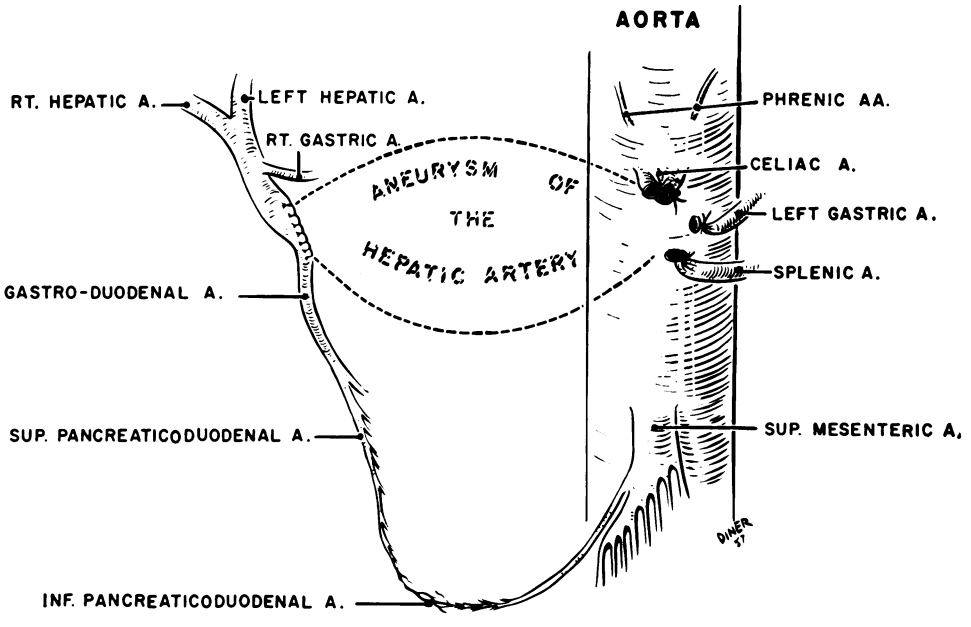

FIG. 1. Diagrammatic illustration of aneurysm, extent of excision and pathway for collateral circulation.

sharp dissection, this being quite difficult due to the firm adherence. A vascular clamp was then placed across the distal portion of the aneurysm, the gastroduodenal artery and the hepatic artery about 0.5 cm. proximal to the gastroduodenal artery. The aneurysm was then removed and a medial wall of the vessel connecting the gastroduodenal and hepatic arteries was constructed by closing the defect with a 6-0 silk over-and-over arterial silk suture. This portion of the aneurysm was pliable and there were no leaks in the suture line. Upon removal of the clamp a good pulse was present in the hepatic artery. The specimen was opened and a laminated clot measuring $3 \times 2 \times 1.5$ cm. found. The abdomen was closed with one drain to the region. He made an uneventful recovery, receiving penicillin and streptomycin for two weeks postoperatively. Microscopic examination of the vessel revealed extensive destruction of the vessel wall with hyalinized fibrous tissue replacement and focal calcification. Liver function tests showed no change from tests taken immediately preoperatively, at which time all were normal. The patient is asymptomatic three months postoperative.

Discussion

The diagnosis of hepatic artery aneurysm is usually impossible preoperatively—indeed, many cases have been missed at laparotomy only to be diagnosed at autopsy.[27, 32, 42] The triad of upper abdominal pain from pressure on adjacent structures, jaundice from direct pressure on the common duct, and hemorrhage, either intra-peritoneal or into the gastro-intestinal tract, has been reported with great frequency, for more than 35 years.[45] The hemorrhage is usually massive, but bleeding into the duodenum, stomach or common bile duct oftentimes is controlled temporarily by clotting; whereas most cases with intraperitoneal hemorrhage from nontraumatic aneurysms exsanguinate rapidly. In the case reported here, it is felt that he may have had pain from the aneurysm in the past, but the present pain was due to the acute cholecystitis. The jaundice was definitely not due to pressure on the common duct as they were not adjacent. Aortogram may occasionally be indicated in diagnosis; [44] but of course, in this case, the finding at surgery was unexpected and immediate treatment carried out without further workup.

The necessity for maintenance of arterial blood supply to the liver has been proven

for many years.[6, 8, 12, 19] Although the portal vein carries about 80 per cent of the blood to the liver, arterial blood has been necessary in animal experiments to preserve life.[30] This supply, of course, does not by necessity require the blood to reach the liver via the hepatic artery.[36] Experimentally, survival can be assured by anastomosing the hepatic artery to the portal vein.[8] This procedure is not feasible as a surgical procedure due to the development of portal hypertension. The question of anomalous blood supply to the right upper quadrant of the abdomen has been the subject of many excellent research problems, with almost 50 per cent of the cases being anomalous blood supply.[10, 24, 33, 41] Michels found a total of 26 different collateral arterial pathways.[33] The inferior phrenic artery is the most constant site of collaterals as would be suspected due to the embryological origin of the diaphragm and liver.[20] This artery will usually preserve the subcapsular portion of the liver after hepatic artery ligation. However, the collateral blood supply in any one case cannot be assurance for survival. Michels has also shown that there are from 20 to 30 terminal branches of the hepatic arteries entering the liver and extensive anastomosis here makes ligation of a right or left hepatic artery at this level possible on occasions. Other anatomists deny this.

The cause of death in dogs after artery ligation has been postulated to be toxins from anerobic organisms.[12, 30] Confirmation was obtained when it was shown that death could be prevented after hepatic artery ligation by use of penicillin or other antibiotics.[31] However, it has been demonstrated that the bacterial content of the liver in dogs is not the same as in humans, as dogs' livers almost always contain large numbers of anerobic bacteria.[12]

The literature lists infection as the most common cause of hepatic artery aneurysm. This is open to serious question as the older literature is filled with cases in which infection is listed as etiology simply because the patient died with a remote infection. No doubt in the pre-antibiotic era, mycotic aneurysms were more common than at present, however. Syphilis is apparently a rare cause, and is consistent with the extremely low incidence of abdominal aortic aneurysms caused by this infection. It is our opinion that arteriosclerosis is the most common cause just as it is in the aorta, femoral, popliteal and splenic arteries. There certainly was no question of our case having this origin. This patient had acute cholecystitis, but it was not adjacent to the aneurysm and the gross and microscopic pathology in no way indicated anything other than arteriosclerosis.

Of the 111 cases of hepatic artery aneurysm in the literature,[1, 3-5, 8, 9, 11, 13-18, 22, 23, 25-27, 29, 35, 37, 42-45] only 12 have survived direct surgical approach. Four have survived ligation,[9, 25, 37, 42] one wiring of the aneurysm,[11] two endo-aneurysmorrhaphy,[15, 16] one rather questionable case of suture to the liver,[1] two wrapped with Cellophane,[44] and two direct excision.[22, 27] The case of Kirlin[22] had a direct surgical excision while Inui's[27] case actually had the false aneurysm excised and ligation of the open ends of the artery thus exposed and ligated. It was obvious that excision was necessary in this case. The dissection revealed the open gastroduodenal artery distal to the aneurysm, thus making excision of the aneurysm safe from hepatic necrosis. However, the encroachment upon the celiac axis presented a problem. It has been shown by Appleby that this vessel can be sacrificed.[2] Dissection of the celiac axis must be carried out with care, however, as occasionally the superior mesenteric arises from the celiac axis in which case a fatality would probably ensue. The question of replacement is, of course, pertinent. This should seldom be necessary except in traumatic cases and then the size of the vessels and the location will make this procedure seldom if ever feasible.

Antibiotics should be used routinely; but this value in man is still open to question.

The experience with purposeful hepatic artery ligation for cirrhosis as started by Rienhoff [39] has been well tolerated as compared to previous reports of accidental ligations by Graham and Connell.[17] There is, of course, a better cause for increased collateral circulation in portal hypertension. In their cases, the portal vein pressure is high, and most deaths probably are due to the basic disease. After the artery is ligated, the vein takes over a portion of the oxygenation of the liver. Poppen [36] showed this very well experimentally, as did Cameron [8] by a different approach many years before.

Summary

A case of hepatic artery aneurysm is presented. It is emphasized that this is a grave disease and excisional therapy should be carried out. Some patients will probably die of hepatic necrosis using the approach; but it is felt almost all patients will die rather promptly unless an aggressive attitude is taken.

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