Isolated Common Hepatic Artery Branch Thrombosis: Results and Risk Factors

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Abstract- Isolated common hepatic artery branch thrombosis with severe gastric ischemia and duodenojejunal infarction is a rare condition; it usually presents with acute abdomen and may be associated with underlying thrombotic risk factors. We present a 35-year-old man admitted to our hospital with five days history of sudden abdominal pain and deteriorating epigastric pain. He was a driver and had no any past medical history. Explorative laparotomy showed: distal 2/3 gastric, duodenojejunal and papilla vater was sloughed. The stomach subtotal and sloughed duodenum and first 20 cm of jejunum were resected, continuity of the gastrointestinal was preserved with anastomosis of the proximal part of jejunum to gastric stump, pancreatic duct, and CBD repaired to the lateral side of jejunum on the guide of two 18 French feeding tube as an external drain. The patient had a good immediate postoperative recovery. Coagulation checkup after operation revealed isolated Hyperhomocysteinemia.

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Introduction

Common hepatic artery branch occlusion is a very rare condition and carries a high mortality and morbidity when the diagnoses and treatment are delayed (1).

Thrombosis of the coeliac artery when occurs at the vessel origin may result in extensive bowel involvement, liver and spleen necrosis (2) but embolic events which affect distal arterial or isolated branches are limited. Clinical presentation of bowel ischemia is vague with nonspecific abdominal complaints, and its final diagnosis may be delayed (1).

The most important factors of acute mesenteric ischemia include atherosclerosis, congestive cardiac failure, recent myocardial infarction, advanced age, vasculitis, hypergastrinemia and coagulation disorders (3).

Hospital mortality in coeliac artery occlusion is 59-93%. Successful treatment depends on early diagnosis and effective intervention either surgically or endovascular to reestablish blood flow, surgical resection of necrotic parts and good intensive care unit management (3).

In this previously healthy driver man acute mesenteric ischemic event involved 2/3 distal of the stomach, which is in the distribution of right gastroepiploic artery and entirely duodenum and first 20 cm of jejunum which is in the distribution of Gastroduodenal artery, leads gangrene of this organs but spleen and liver were spared. The importance of this case beside of detection of etiological predisposing factors is considering this kind of clinical situation as an acute abdomen, prompt early diagnosis and effective intervention either surgically or endovascularly.

Case Report

A 35-year-old man was referred to our hospital with five days history of sudden abdominal pain, weakness, diarrhea, and fever. He was initially managed as severe gastritis in another hospital. He was previously healthy driver, non-smoker with normal weight, no past medical history of hypertension, underlying disease diabetes or coagulopathy disorders. On physical examination: he was mentally conscious general condition was ill with dry skin. Vital signs were Blood Pressure: 90/60 mmhg Pulse Rate: 100 per min, Respiratory rate: 15 per min, Abdomen was moderately distended with diminished bowel sounds, no muscular rigidity was observed but moderately deep tenderness on palpation was found, worse on the epigastrium and mild rebound tenderness.

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Chest examination was normal and normal peripheral arterial pulses.

Laboratory tests included WBC: 18600 (83.6% granulocytes), glucose was (normal range 65-110 mg/dL), LDH: 556 u/L (normal range 120-230), Na: 130 mmol/L, K: 3.1 mmol/L. Blood gases'results were: pO_2 51 mmHg, pCO2 33.9 mmHg, PH 7.51, SO_2 88.9%.

Color Doppler of mesenteric arteries and vein system revealed relatively high resistance flow in the both origin of the artery. No sign of occlusion was present in the superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) origins (the detectable regions on Doppler ultrasound exam). Pre-operative CT scan of the abdomen on a multi-detector-row spiral scanner after oral contrast administration (meglumine) and intravenous injection of 120 ml of 300 mgI/ml non-ionic contrast medium (Omnipaque) showed edema of the stomach wall with decreased wall enhancement without leakage of contrast (Figure 1). Hypo dense thrombus was found in the gastroduodenal artery; dilatation and wall thickening of the proximal jejunum (edematous change) was noted in LUQ suggestive of functional obstruction/adhesion bands or ischemic process (Figure 2). The rest of small and large bowel was within normal limits. There was also a mild amount of peritoneal fluid on the right side. Contrast-enhanced Spiral Chest CT also showed mild bilateral pleural effusion.

Figure 1. Spiral CT
Gastric wall edema and decreased wall enhancement without contrast leakage

Left gastric and splenic and common hepatic arteries were seen without stricture or obstruction. The gastroduodenal branch of the common hepatic artery was cut off in the proximal portion. There was also no evidence of the right gastroepiploic artery (Figure 3) and (Figure 4).

Following initial resuscitation, NGT, foley catheter insertion and broad spectrum antibiotic administration were performed, the patient underwent an emergency laparotomy: stomach was severely ischemic in upper, and 2/3 distal was necrosis duodenum from first part to treitz ligament entirely was gangrene, pancreas was normal and also first 20 cm of jejunum obviously was gangrenous after it sharply was normal.

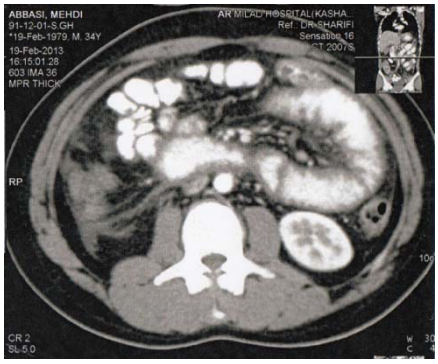

Figure 2. Spiral CT demonstrates dilatation and wall thickening of proximal jejunum

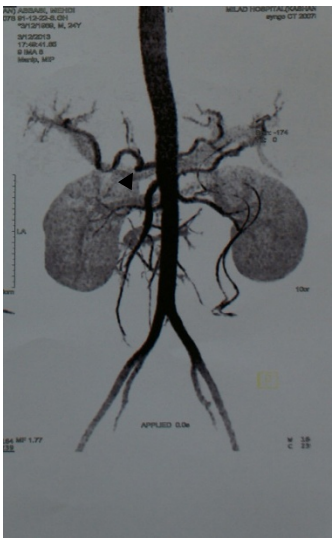

Figure 3. Cut off of the gastroduodenal artery (black arrowhead)

Junction of pancreatic duct with CBD (papilla) was necrotic, free bile-stained peritoneal fluids were found, while the remaining viscera, the spleen, and the liver were normal. The stomach was resected sub totally with extensive debridement (removal of greater and lesser omentum and sloughed duodenum and first 20 cm of jejunum was resected the mucous of the remnant gastric pouch was still pale and ischemic, but muscles were normal since the mentioned organs were gangrene, so vascular reconstruction did not require.

However, the intraoperative investigation showed the celiac trunk and superior mesenteric artery (SMA) had normal flow. Splenic artery and proper hepatic artery as

could as touched were normal. We could not palpable right gastroepiploic artery and gastroduodenal artery.

Figure 4. Volume-rendered 3D color image created from axial contrast enhanced CT data show: cut off of gastroduodenal branch of the common hepatic artery (black arrowhead)

Figure 5. GI study: gastrojejunal anastomotic, pancreatic duct and CBD repairing site. (Whipple procedure)

Continuity of the gastrointestinal lumen was performed with anastomosis of a proximal portion of jejunum to the gastric stump. Pancreatic duct and CBD and after debridement had been separately repaired to the lateral side of jejunum on the guide of two 18 French feeding tube as external drainage it repairs using running absorbable suture in two layers, after trimming the edges of the remnant part exited from 30 cm distally from jejunal loop to abdominal wall. The patient had good immediate postoperative recovery and discharged two weeks after admission, four weeks later drains were extracted. He underwent another gastrograffin study one month later revealed no leakage from the gastrojejunal anastomotic and pancreatic duct and CBD repairing site (Figure 5). He was put on a regular diet, folic acid monthly and B_{12} injections. The immediate cause of thrombosis was evaluated postoperatively: Protein C, protein S and antithrombin III levels were normal, serum homocysteine level was 38.5 mmol/l, (normal under 60 years is 5-15 micmol/l). Fasting blood sugar and lipid profile were normal;Atherosclerosis, aneurysm, atrial fibrillation, intracardiac emboli, and thrombocytosis were ruled out.

Discussion

Isolated common hepatic artery branch thrombosis and infarction of entire duodenum, papilla vater and 20 cm of first proximal jejunal in an active, healthy man is very rare and has not reported yet. It happened as an acute event because, in a chronic case, adequate collaterals developed over time may prevent the infarction (4).

The most mesenteric ischemia are the duo to involving SMA, either from thrombosis of a preexisting stenotic lesion or from embolization which leads to gangrene of total small bowel, parts of the right colon but duodenum and 20 cm of jejunum is spared in most cases of acute intestinal ischemia.

Gastric ischemia also is uncommon, as the stomach has a rich blood supply from branches of the celiac axis, as well as from SMA collaterals (5).

The celiac artery divides into left gastric, splenic and common hepatic artery. It receives collaterals from superior mesenteric artery (SMA) via pancreaticoduodenal arcades and dorsal pancreatic artery. The artery or arc of Buhler is an inconsistent vessel that directly connects the coeliac axis to the SMA (6). Due to these extensive collaterals, the stomach which is mainly supplied by the celiac artery rarely suffers from ischemia. Both celiac and SMA may need to be involved in causing ischemic necrosis of the stomach (7).

In our case diabetes mellitus, hypertension, hyperlipidemia, and smoking were not determined, and other risk factors such as atherosclerosis, aneurysm, dissection, embolization of intracardiac thrombi in atrial fibrillation, thrombocytosis were ruled out. As well as coagulation disorders: protein C, protein S, and antithrombin III levels were normal except hyperhomocysteinemia which may be a major etiologic factor of acute mesenteric ischemia in this patient. Furthermore, thrombus and/or atherosclerotic changes in the descendent aorta and iliac vessels were not observed in the CT-angiography study.

He had no any abuse medication such as ergotamine (8). Possibilities of mesenteric vasculitis with spontaneous thrombosis were ruled out by the histopathological examination of the specimen.

Hyperhomocysteinemia is associated with the development of atherosclerosis as well as venous and arterial thrombosis in numerous epidemiological studies (9-12). However, the mechanism(s) by which elevated plasma homocysteine leads to cardiovascular disease has not been clearly determined. Most studies have focused on the effects of homocysteine on vascular cells. Endothelial dysfunction induced by hyperhomocysteinemia very likely contributes to the enhanced risk of atherosclerosis (13). Enhanced platelet reactivity in hyperhomocysteinemia may contribute to an increased risk of thrombosis (14).

In this referral patient, non-specific clinical presentation prolonged six days before proper diagnosis. Thus early detection of this condition requires high clinical suspicion and elevated amylase, lactate, leukocytosis and ABG sometimes may not help the diagnosis.

The spiral CT of abdomen and pelvis with oral and IV contrast is helpful; it can be used to detect stomach wall thickness, pneumatosis, mucosal or bowel wall enhancement pattern that supports the diagnosis of acute mesenteric ischemia (AMI) (6). In current patient, however, signs of ischemia, such as enhancement and edematous gastric wall and small bowel were present and helped decision making on laparotomy.

Early detection of acute isolated common hepatic artery branch thrombosis requires high clinical suspicion, CT-Angiography of the celiac and mesenteric arteries; contrast enhanced spiral CT of the abdomen may accurately depict gastroduodenal artery occlusion and duodenojejunal infarction. Effective intervention either endovascularly or surgically by radical resection of sloughed bowel resection of necrotic parts and Whiple procedure are optimal managements.

References

1. Cappell MS. Intestinal (mesenteric) vasculopathy. II. Ischaemic colitis and chronic mesenteric ischaemia. Gastroenterol Clin North Am 1998;27:827-60, vi.
2. Ali-Akbarian M, Kahrom M, Kahrom H. Celiac artery trunk thrombosis presenting as acute liver failure. J Coll Physicians Surg Pak 2011;21:301-3.
3. Schoots IG, Koffeman IG, Legemate DA, Levi M, van Gulik TM, et al. Systematic review of survival after acute mesenteric ischaemia according to disease etiology. Br J Surg 2004;91:17-27.
4. Taylor LM, Moneta GL, Porter JM. Treatment of acute intestinal ischemia caused by arterial occlusions. In: Rutherford Vascular Surgery. 5th ed. Philadelphia: WB Saunders Co, 2000:1512-8.
5. LaBerge JM, Kerlan RK Jr. Occlusion of the celiac artery origin and high-grade stenosis of the superior mesenteric artery secondary to compression from the medium arcuate ligament. Society of Cardiovascular & Interventional Radiology. J VascInterv Radiol 1999;10:500-4.
6. Kirkpatrick ID, Kroeker MA, Greenberg HM. Biphasic CT with mesenteric CT angiography in the evaluation of acute mesenteric ischemia: initial experience. Radiology 2003;229:91-8.
7. Mercogliano G, Tully O, Schmidt D. Gastric ischaemia treated with SMA revascularization. ClinGastroenterol Hepatol 2007;5:A26.
8. Papalampros EL, Salakou SG, Felekouras ES, Scopa C, Tsamandas AC, Bastounis E. Ischemic necrosis of gastric wall after long-term ergotamine pill abuse: case report and review of the literature. Dig Dis Sci 2001;46:981-4.
9. Aronow WS, Ahn C, Gutstein H. Increased plasma homocysteine is an independent predictor of new atherothrombotic brain infarction in older persons. Am J Cardiol 2000;86:585-6, A10.
10. Eichinger S, Stumpflen A, Hirschl M, Bialonczyk C, Herkner K, Stain M, et al. Hyperhomocysteinemia is a risk factor for recurrent venous thromboembolism. Thromb Haemost 1998;80:566-9.
11. Lentz SR. Mechanisms of thrombosis in hyperhomocysteinemia. Curr Opin Hematol 1998;5:343-9.
12. Malinow MR. Homocyst(e)ine and arterial occlusive diseases. J Intern Med 1994;236:603-17.
13. Eberhardt RT, Forgione MA, Cap A, Leopold JA, Rudd MA, Trolliet M, et al. Endothelial dysfunction in a murine model of mild hyperhomocyst(e) inemia. J Clin Invest 2000;106:483-91.
14. Durand P, Lussier-Cacan S, Blache D. Acute methionine load-induced hyperhomocysteinemia enhances platelet aggregation, thromboxane biosynthesis, and macrophage-derived tissue factor activity in rats. FASEB J 1997;11:1157-68.