Coeliac Artery Thrombosis: An Uncommon Cause of An Acute Abdomen

K Saravana, A A Zainal, S K Lee

Department of General Surgery, Hospital Kuala Lumpur, Jalan Pahang, 50586 Kuala Lumpur.

SUMMARY

Coeliac artery thrombosis with ischaemia is a rare condition, which usually presents with severe peptic ulcer disease symptoms. It is usually associated with risk factors for thrombosis or embolism. The manifestation is rare because of large number of collaterals between the coeliac and superior mesentery artery. Early detection and intervention is required to prevent the progression of its complications that includes gastric ischaemic necrosis.

KEY WORDS:
Coeliac artery thrombosis, gastric ischaemic necrosis

INTRODUCTION

Coeliac artery occlusion is an uncommon condition. Autopsy studies have shown that the incidence of a more than 50% stenosis in at least one mesenteric artery occurs in 6-10% of population[1]. Risk factors for arterial thrombosis in general includes, low flow states, atherosclerosis, congestive cardiac failure, recent myocardial infarction, advanced age, vasculitis and intra-abdominal malignancy. Unlike embolic events which affect distal arterial branches and results in limited bowel ischaemia, thrombosis of coeliac artery occurs at the vessel origin and may result in extensive bowel involvement including the liver and spleen1. This case report illustrates an example of an acute mesenteric ischaemic event involving the coeliac artery distribution.

CASE REPORT

A 48 year old woman presented to us with severe epigastric pain of 10 days duration for which she was initially managed as severe gastritis in another hospital. There was no associated diarrhea, hematochezia or malaena. Despite being on intravenous proton pump inhibitors, intravenous dextrose saline hydration and analgesia (paracetamol and tramadol) her condition did not improve. She had no known medical illness, a non-smoker with no recent history of NSAIDS or traditional medication usage. She was visiting her relative at the onset of abdominal pain and ate routine home cooked food there. On her arrival to our centre she had a temperature of 37·8°C, tacchycardic (110 beats per minute), dehydrated and a tender guarded upper abdomen. Her white blood counts revealed marked leucocytosis and she was anaemic (Hb 7.1g/dl) with thrombocytosis (510x109 cells/L). She was hyperglycaemic (23mmol/L) with a raised lactate dehyrogenase and amino transferase levels with metabolic acidosis. Serum amylase and urinary diastase were not significantly elevated. Semi-erect Chest x-ray (as patient was unable to be propped up due to pain) revealed air under diaphragm and no cardiomegaly. Abdominal x-ray also showed a few dilated proximal loop of jejunum. She underwent a contrasted CT scan abdomen that revealed a large loculated (14cm by 13cm by 12cm) collection with pneumoperitoneum. The spleen was not visualized and there were infarcts in segments 4 and 7 of the liver. The coeliac trunk was thrombosed. (Fig 1 and 2)

She underwent an emergency laparotomy. The whole stomach was gangrenous with a large perforation (6cm by 8cm), the spleen was necrotic and areas of infarct in the liver. A total gastrectomy with Roux en Y esophageal-jejunal anastomosis with extensive debridement (removal of greater and lesser omentum, slough from splenic bed area) and peritoneal lavage was done. Post operatively she was started on anticoagulant. She underwent another laparotomy and debridement due to persistent fever and discharge a few days later. She had a prolonged stay in ICU with duodenal stump leak which was managed conservatively. A gastrograffin study at 1 month demonstrated an esophageo-jejunal anastomotic leak. Both were managed conservatively and she was kept on total parenteral nutrition. The histopathological report showed ischaemic stomach with no evidence of malignancy, inflammatory bowel disease or vasculitic changes. Echocardiogram revealed no mural thrombus, valvular or wall defects or vegetations. ECG showed no tachyarrythmias.

She underwent another gastrograffin study at 2 months which revealed no major leakage from the esophageo-jejunal anastomotic site. She was started on oral fluids, which was well tolerated. She was able to take soft diet over the next few days and she was discharged well on day 72 of hospital stay. She was put on elemental diet combined with normal diet and 3 monthly B12 injections.

On follow-up 2 months later she was well and her weight had increase by 6 kg. Blood investigations to assess her fasting blood sugar, lipid profile, thrombhophiliac screening subsequently revealed that they were normal. Currently she has no symptoms suggestive of mesenteric ischaemia.

DISCUSSION

The coeliac artery arises from the ventral aspect of aorta at the level of T12-L1 vertebral body and divides into left gastric, splenic and common hepatic artery. It receives collaterals from superior mesenteric artery (SMA) via pancreatico-

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Corresponding Author: Zainal Ariffin Azizi, Head of Department of General Surgery, Kuala Lumpur Hospital, Jalan Pahang, 50586, Kuala Lumpur
Email: mrzainal@hkl.gov.my

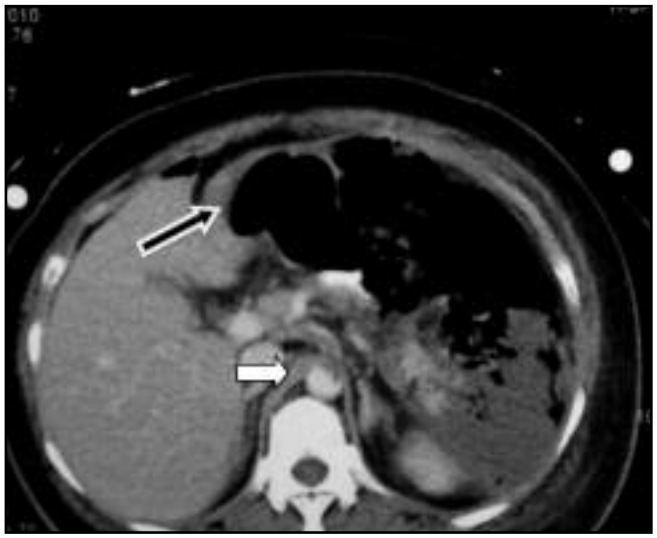

Fig. 1: CT Abdomen with contrast(oral &IV) showing celiac artery thrombosis (white arrow) and pneumoperitoneum (black arrow)

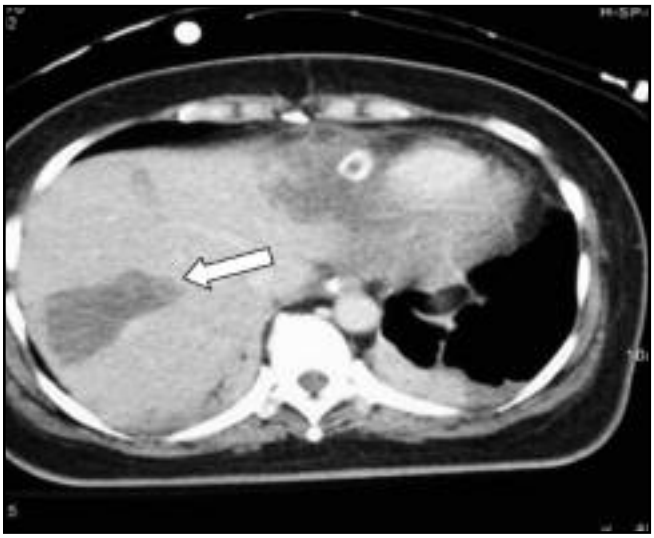

Fig. 2: CT Abdomen with contrast(oral & IV) showng and infarct segment of the liver (white arrow).

duodenal arcades and dorsal pancreatic artery. The artery or arc of Buhler is an inconsistent vessel that directly connects the coeliac axis to the SMA[2]. Due to these extensive collaterals, the stomach which is mainly supplied by the coeliac artery rarely suffers ischaemia. Both coeliac and SMA may need to be involved to cause ischemic necrosis of the stomach[2]. The incidence of asymptomatic coeliac artery or SMA stenosis of more than 50%, in patients undergoing arteriography is about 27%[3]. In our patient, the thrombosed coeliac artery was probably an acute event with minimal or no collaterals. In acute on chronic case, adequate collaterals which developed over time may have prevented the catastrophe.

Even for the patients with asymptomatic mesenteric arterial lesions, the suggested natural history of mesenteric occlusive disease is progressive and potentially morbid. The degree of reduction in blood flow that the bowel can tolerate is remarkable. Only 1/5 of mesenteric capillaries are open at any given time and normal oxygen consumption can be maintained with only 20% of maximal blood flow[4].

In our case with the background history of no prior medical illness and as the patient presented with overt sepsis, the immediate cause for the risk factor(s) for ischaemia would have to be partly reevaluated post operatively. Her hyperglycaemia may have been due to systemic mediated immune response syndrome resulting in metabolic derangement. This leaves the aetiology to other causes of non-occlusive or occlusive mesenteric ischaemia. Possibility of inflammatory bowel disease or mesenteric vasculitis with sponataneous thrombosis were ruled out by the histopathological examination of the specimen. Most literature discuss on the mesenteric ischaemia involving SMA and rarely on celiac arterial distribution. Visceral arterial embolism preferentially occurs at superior mesenteric artery (SMA) because it emerges at aorta at an oblique angle. Of these 15% of arterial emboli occur at origin of SMA and 50% occur at the first branch of SMA which is the middle colic artery[4]. Acute mesenteric thrombosis (occurring in 25-30% of all mesenteric ischaemia) almost all occur in the severe atherosclerotic setting and usually result in larger area of infarction than emboli[4].

Early detection of this condition requires high clinical suspicion and may be assisted with a few biochemical results such as elevated amylase, lactate, leucocytosis, aspartate transaminase and lactate dehydrogenase. Duplex ultrasonography can be used to visualize the coeliac trunk and SMA but may be obscured by gas filled bowel in the acute phase. Multi detector row CT arteriography(CTA) with accurate timing of contrast and 3D reconstruction can show good images of the vessels for both acute and chronic mesenteric ischaemia. The scan can be used to detect stomach wall thickness, pneumatosis, mucosal or bowel wall enhancement pattern that supports the diagnosis of acute mesenteric ischaemia (AMI). Overall sensitivity 100% and specificity of 89% achieved for the detection of AMI5. CTA studies are judged to be satisfactory up second order branches of both the coeliac and SMA[5].

Conventional selective angiography is the gold standard. It can be used as a diagnostic and therapeutic tool in management of AMI. However it is more invasive than CTA.

The treatment options, for coeliac artery occlusion depends on severity of the occlusion, the development of collateral circulation and the patient's clinical condition. Initial treatment includes volume resuscitation, correction of acidosis and intravenous antibiotics. Endovascular treatment of coeliac and mesenteric stenosis has a low incidence of complications, lower morbidity and high technical success rate2. However local expertise has to be available. This was not an option in our patient as she presented as an acute

abdomen with peritonitis. Laparotomy will show the extent of bowel ischemia. Embolectomy via a longitudinal arteriotomy is done with size 3 or 4 fogarty catheter if embolus is suspected and in thrombosis, a bypass is required using autologus vein. All non-viable ischemic bowel should be resected and second look laparotomy in 2-3 days should be carried out if indicated.

CONCLUSION

Coeliac artery thrombosis with ischaemia of the visceral organs is uncommon but a high degree of clinical suspicion and radiological imaging is essential for early detection and treatment to ensure a successful outcome.

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