Gastric angina secondary to acute thrombosis of celiac artery

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We report a 48-year-old woman with foregut ischemia with splenic infarct due to isolated celiac artery obstruction. The patient presented with acute-onset pain in the epigastrium 10-15 min after every meal. Investigations revealed obstruction of the celiac artery by atheromatous plaque. This patient had an acute thrombosis, which responded to anticoagulation. [Indian J Gastroenterol 2000; 19:139-140]

Key words: Celiac artery thrombosis, gastric ischemia

Gastric angina, secondary to gastric ischemia, presents with chronic postprandial epigastric pain. The usual causes are multivessel atherosclerotic occlusion involving mesenteric vessels and extrinsic compression of the celiac artery. Isolated acute thrombotic occlusion of the celiac artery giving rise to gastric ischemia has not been reported.

A 46-year-old woman with insulin-dependent diabetes mellitus presented with sudden-onset high-grade fever 3 weeks earlier. Three days later, she developed severe, non-radiating pain in the epigastrium, associated with multiple episodes of vomiting. Examination of the abdomen by a local physician was reported to be normal.

Her WBC count was 13,500/cumm; ultrasonography of the abdomen and ECG were normal. For the next 9 days, the patient had intermittent pain which required injectable analgesics. On day 10, she developed severe epigastric pain and was hospitalized. Physical examination was non-contributory. CT scan showed splenic infarct. Since the pain did not subside, CT scan was repeated 4 days later; this showed splenic infarct with intraluminal thrombus in the abdominal aorta at the level of the celiac axis. Subsequently the patient developed high-grade fever and WBC count increased to 33,000/cumm.

Three weeks after the onset of pain, she was referred to Fig: Spiral CT scan showing aortic plaque with blockage of celiac artery, patent distal splenic artery, and splenic infarct.

On enquiry, the patient stated that she could tolerate only liquids, and would get severe pain which lasted 2-3 hours within 15-20 minutes of intake of solids. On examination, she was febrile and had tachycardia. Abdominal examination was normal except for deep tenderness in the epigastrium.

Investigations: Hemoglobin 11.5 g/dl, WBC 12,500/cumm, ESR 55 mm at 1 hour. The liver and renal profiles and serum electrolytes were normal. She had hypercholesterolemia, with low HDL (21 mg/dl; normal 45-65) and high VLDL (53 mg/dl; normal up to 35). Coagulation profile: normal bleeding time, clotting time, prothrombin time and partial tissue thromboplastin level. Fibrinogen level >5 g/100 ml (normal up to 1.4). FDP >10 mg/ml (normal up to 10). Anticardiolipin antibody, RA factor, antiphospholipid antibody were negative. Protein C, protein S, antithrombin III levels, hemoglobin electrophoresis, and 2D echocardiogram were normal.

Spiral CT scan (Fig) revealed plaque in the aorta, a 1.5 cm long, at the origin of the celiac axis extending into its trifurcation. The origins of the hepatic, splenic and left gastric artery were not visualized. The rest of the splenic artery was seen up to the splenic hilum. The main superior mesenteric artery (SMA) was patent but there was a block in the SMA beyond the origin of the ileocolic branch. Spleen showed infarct in its entire extent. Upper GI endoscopy showed hiatus hernia with pale and flat mucosa in the body and antrum of the stomach. Color Doppler showed no flow across the celiac artery; the SMA was patent.

Her fever responded to antibiotics and pain decreased when she was nil-by-mouth. However, once oral feeding was started, the pain recurred. Digital subtraction angiography showed narrowing of the lumen of the aorta by 60% at the origin of the celiac artery. The plaque was adherent to the wall of the aorta. The celiac artery and its branches were not visualized; the gastroduodenal arcade was seen. Retrograde perfusion of the hepatic arteries via the pancreatic-duodenal arcade was seen. The SMA and the inferior mesenteric artery (IMA) were normal except for the terminal branch of the SMA which was blocked. There was good perfusion of the small bowel.

The patient was started on 1V heparin infusion at 800 U/h and pentoxyphyline (400 mg three times a day). The symptoms improved over 10 days and she could tolerate almost a
normal diet without pain. She was discharged on warfarin 5 mg per day and oral pentoxifylline. Three months later, she is asymptomatic. Repeat color Doppler study showed flow across the celiac artery. Barium study showed good distensibility and emptying of the stomach.

The commonest etiology of gastric angina is celiac artery compression, where 70% of patients may suffer from gastric angina. It is usually seen in the chronic form in patients with multivessel occlusion. Despite the high incidence of atherosclerotic occlusion of mesenteric vessels, most such patients are asymptomatic because of highly efficient collateral circulation.

Our patient presented with acute manifestation due to celiac artery occlusion, probably due to embolic blockage of intramural branches of the left gastric artery in the antral region, secondary to thrombotic occlusion of the celiac artery. This can explain the failure of development of collaterals at the muscular and mucosal level. Spiral CT angiography could establish the diagnosis in this case. Real-time duplex scanning is also useful. Arteriography is done to provide anatomic details for surgical therapy. Thrombolytic or anticoagulant therapy has been described for acute mesenteric thrombosis, but it has not been reported for gastric ischemia as most cases present late. The present case presented early and hence responded to anticoagulation and pentoxifylline.

To conclude, our patient developed gastric angina following thrombotic occlusion of the celiac axis and responded to anticoagulation.

References

Enteric duplication cyst associated with melanosis peritonei

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Melanosis peritonei is usually associated with benign cystic teratomas of the ovary. We describe a one- and-a-half-year-old girl with melanosis peritonei associated with enteric duplication cyst. Melanophages were seen in aggregates in and around the serosal blood vessels, nerve bundles, and scattered within the muscular wall of the cyst. Presence of hyperplastic nerve bundles associated with melanophages suggests their origin from the neural crest. [Indian J Gastroenterol 2000;19:140-141]

Key word: Melanophago

Enteric duplication cyst is a congenital anomaly, which can occur in any portion of the alimentary tract. Diffuse pigmentation of the peritoneum, known as melanosis peritonei, is an extremely rare condition. Only six cases have been reported to date, and four of these have been associated with benign cystic teratoma of the ovary. Only one case of melanosis peritonei has been associated with enteric cyst. We report a case of melanosis peritonei associated with enteric duplication cyst.

A one- and-a-half-year-old girl presented with one-month history of intermittent pain in the abdomen with abdominal distension and occasional constipation, suggestive of intermittent partial mechanical obstruction. There was no history of fever, vomiting, diarrhea or urinary symptoms. On examination, there was a palpable, non-tender, firm mass in the periumbilical region, measuring 2 cm x 2 cm. Bowel sounds were normal. There was no palpable organomegaly. There was no vertebral, skeletal or genitourinary anomaly.

With a clinical impression of extrinsic compression of the intestines by a mass, laparotomy was performed. On exploration, a 2 cm x 2 cm, non-communicating, spherical cyst was found in the ileo-cecal region, attached to the umbilicus by a fibrous cord. Multiple blackish nodules, 1-5 mm in size, were seen on the adjacent peritoneum as well as on the cyst wall. Regional lymph nodes were not enlarged. The cyst was excised. It was covered by serosa with multiple 1-5 mm blackish granules. Wall thickness was 0.8-1 cm and the lumen was empty. The patient had an uneventful postoperative course.

Histologically, the cyst had both colonic and small intestinal lining. In addition, there was neutrophilic hyperplasia on the serosal surface. These nerve bundles and blood vessels were surrounded by collections of macrophages containing brownish-black pigment (Fig.). Similar cells were also seen scattered within the muscularis propria and submucous. A moderate degree of lymphoplasmacytic infiltrate was also seen. The pigment was found to be Schmorl's-positive; however Perl's prussian blue and PAS staining were non-contributory, thereby confirming the pigment to be melanin.

Enteric duplication cysts are found adherent to any portion of the alimentary tract and have smooth muscle wall and a mucosa which may be different from that of the adjacent segment of alimentary tract.

In melanosis peritonei, the melanotic pigmentation can be a result of metastatic or primary malignant melanoma of the ovary or secondary to benign lesions. Till now, only six cases associated with benign conditions