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Isolated Complete Occlusion of the Abdominal Vasculature Corrected by Surgery

KEYWORDS: Mesenteric, intestinal angina, thrombosis, revascularisation

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ABSTRACT *This report depicts a case of mesenteric artery thrombosis developing in a chronic smoker with relative sparing of the rest of the body vasculature, and showing an unprecedented improvement after surgical revascularisation. We have highlighted the rare occurrence of extensive atherosclerotic involvement of the superior mesenteric artery (SMA), inferior mesenteric artery (IMA) and celiac vessels, with no symptomatic disease in the coronary, carotid or cerebral vessels, most probably due to smoking. There was 100 % occlusion of the celiac trunk, SMA and IMA in the absence of vasculitis. The significant improvement of the patient as measured by his increased appetite and weight gain after surgery was remarkable.*

INTRODUCTION

Chronic mesenteric ischemia (CMI) is uncommon, accounting for less than 5 % of intestinal ischemic diseases; it is almost always caused by mesenteric atherosclerosis, although rare causes such as collagen vascular disease and inflammatory vasculopathy are known. There is no specific association between CMI and smoking, although 75% of patients have a history of smoking.

CASE STUDY

A 44-year old man hailing from Bihar, working as a farmer, with a 40-pack year history of smoking and no comorbidities, came with chief complaints of abdominal pain, loose stools, malaena, weight loss and decreased sleep for the past one and a half year. He complained of severe colicky episodic post-prandial pain, intermittent malaena and loose motions which occurred 7-8 times per day. The patient reported a weight loss of 30 kilograms over a period of one and a half years and insomnia due to the severe abdominal pain at night followed by 2-3 loose motions in the morning. On general examination, he was grossly emaciated and malnourished. There was loss of the buccal pad of fat with wasting of the extremities. Systemic examination was unremarkable.

Routine investigations showed a haemoglobin of 14.5 gms/dl, ESR of 28 mm/hour, white blood count (WBC) of 10,500 cu/mm, total proteins of 6.2 gm %, cholesterol of 123 mg % , LDH of 184 mU/ml, SGOT of 59 mU/ml, SGPT of 41 mU/ml , ALP of 139 mU/ml, urea of 7 mg% ,a sodium level of 131 mEq/L and calcium oxalate crystals in the urine. On day 2 of admission, the haemoglobin dropped to 12.5 due to the ongoing malaena. The apparently high haemoglobin level (12.5) noted on admission was probably secondary to polycythemia secondary to the patient's chronic smoking. No tachycardia or orthostatic hypotension was noted. On further investigation, in view to rule out a vascular abdominal pathology, a CT Angiography of the abdomen was done which showed severe stenosis of the celiac trunk (CA) and occlusion of the superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) origins with narrowing of bilateral renal artery at its origin, circumferential soft tissue thickening of the abdominal aorta with few specks of calcification. Upper gastrointestinal endoscopy showed gastric erthyema. Colonoscopy showed patchy mild active ileitis with a normal mucosal study. Vasculitis was suspected but the tests for ANA, ANCA were negative. Malabsorption workup (Anti-TTG) was negative. Thyroid function tests were within normal limits and vitamin D levels were decreased at 18.2 ng/ml (Vitamin D deficient). The patient was diagnosed with chronic mesenteric ischemia secondary to mesenteric artery thrombosis most probably due to chronic smoking. The patient was started on intravenous pantoprazole, vitamin D3 supplements and total parenteral nutrition. After exhausting the medical line of management, a digital subtraction angiography (diagnostic and therapeutic) was done but radiological intervention could not be performed due to the presence of 100 % stenosis. The patient was referred to the vascular surgeons who performed a retrograde mesenteric bypass in which a saphenous venous graft (SVG) conduit was taken from the left thigh whose proximal end was anastomosed to the right iliac artery and its distal end was anastomosed to the SMA. Another SVG conduit was taken in a 'Y' fashion to the first conduit and anastomosed to the drummond artery, (a large collateral between the SMA and the IMA). Good pulsations were observed in the SMA and IMA intra-operatively. The post-operative course was uneventful and the patient demonstrated a significant improvement in his diet on account of complete resolution of his previous post-prandial pain and demonstrated a weight gain of 2 kilograms in seven days.

Figure 1: CT Angiography of the abdomen demonstrat-

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ing narrowing of the superior mesenteric artery (SMA)

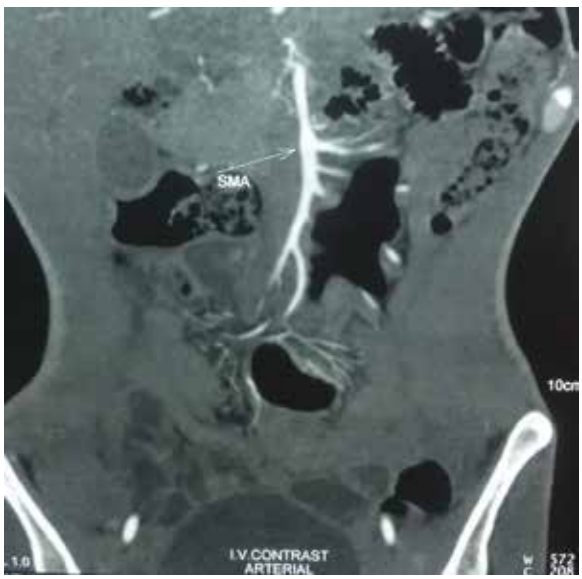

Figure 2 : CT Angiography of the abdomen demonstrating narrowing of the inferior mesenteric artery (IMA)

DISCUSSION:

Abdominal pain is likely caused by ischemia in the small intestine as blood is stolen from this organ to meet the increased demands for gastric blood flow as food enters stomach. This rationale for why the pain occurs so soon after eating, when food still remains in the stomach, is preferable to the historic explanation that a fixed and limited supply is incapable of meeting the increased metabolic demands of the small intestine during digestion.

The cardinal clinical feature of CMI is abdominal cramping discomfort that usually occurs within 30 minutes after eating, gradually increases in severity, and then slowly resolves over 1 to 3 hours. Although minimal at first, abdominal pain progressively increases in severity over weeks to months. The association of pain with meals leads to fear of eating with resultant weight loss. Nausea, bloating, episodic diarrhea and malabsorption or constipation may occur, but it is the weight loss and relation of the abdominal pain to the meals that charactise this syndrome. Early in the course of the disease, if patients do not eat, they remain pain free; pain occurs only after eating or during a meal. Later, pain becomes continuous, and this portends intestinal infarction. Uncommon presentations of CMI include antral ulcerations that are unassociated with Helicobacter Pylori (H. Pylori) and which do not heal on therapy with PPI's; gastroparesis (that resolves with revascularisation) and acalculous cholecystitis. Approximately one-third to half of patients have some evidence of cardiac, cerebral, or peripheral vascular disease. Physical findings are usually limited, but patients with advanced disease may appear cachectic. The abdomen typically remains soft and non-tender even during painful episodes, although distension may be appreciated. An abdominal bruit is common but nonspecific.

Diagnosis of CMI is difficult because of the vague nature of the complaints and the lack of a specific diagnostic test. Abdominal plain films and CT scans are usually negative for pathology, although vascular calcification may be present. Endoscopic inspection of the gastrointestinal tract reveals it to be normal; and random biopsies of the upper tract may show only non-specific abnormalities; a diagnostic clue may be antral ulcerations unassociated with H. Pylori, that do not heal on acid-suppression therapy. Barium studies are normal or show non-specific evidence of malabsorption or a motility disturbance. Rarely, radionuclear emptying tests may show delayed gastric emptying. A duplex ultrasound (USG) can be used to identify splanchnic artery stenoses but not to establish the diagnosis of CMI. Elevated peak systolic velocity in the SMA and CA of 275 and 200 cm/sec respectively, is a reliable sign of at least 70 % stenosis of these vessels. Duplex USG and phase-contrast cine MR imaging of the SMA and CA have been used to measure the effect of eating on mesenteric blood flow, based on the principle that eating normally increases blood flow to the small intestine, whereas in CMI, this fails to occur. However post-prandial studies are no better than fasting examinations, especially at lesser degrees of vascular stenosis. More experience with these provocative tests is needed before firm conclusions about their diagnostic usefulness can be made. Duplex USG, MR Angiography and traditional mesenteric angiography all merely reveal anatomic limitations of splanchnic blood flow and do not establish the presence or absence of intestinal ischemia.

In the absence of a specific and reliable diagnostic test, diagnosis of CMI is based on clinical symptoms in combination with radiologic demonstration of an occlusive process of the splanchnic vessels, and to a great measure, the exclusion of other gastrointestinal disorders. Angiography should show the occlusion of two or more splanchnic arteries to allow the diagnosis of CMI; however, such occlusions, even of all three vessels, do not by themselves make the diagnosis of CMI, because they may be present with no corresponding clinical symptoms. In most patients with CMI, at least two of the three splanchnic vessels either are completely obstructed or severely stenosed. In a review of series of patients with CMI, 91 had occlusion of at least two vessels and 55 % had involvement of all three; 7 % and 2 % had isolated occlusion of the SMA and CA, respectively.

Surgical revascularisation has been the traditional method of therapy for patients with CMI. CMI is not considered to require urgent surgery, although acute complete occlusion of the gastrointestinal blood supply may occur if thrombosis is superimposed on already narrowed arteries. Since the early 1980s, percutaneous transluminal mesenteric angioplasty (PTMA) alone or with stent insertion has been used as alternative therapy but is reported only in small numbers of patients. Whether surgery or PTMA is better will be determined by their relative success in relieving symptoms and the durability of such relief. The results of surgical revascularisation for CMI vary in different reports, depending on the nature of the operations used, the number of vessels revascularised, and whether concurrent operations such as aortic reconstruction are performed.

The true efficacy of surgical revascularisation and PTMA is difficult to determine because of the varied criteria used by different investigators to define a successful outcome. Thus, some authors use graft or vessel patency rates, whereas others define success by relief of symptoms, recurrence rates, or long-term survival. Most recent series have reported mortality rates below 10%, success rates of more than 90 %, and recurrence rates generally lower than 10 %. Several long-term studies have shown that patients surviving surgical revascularisation have cumulative 5-year survival rates of approximately 80 % to 90 %.

The initial success rates of PTMA are similar to those of

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surgical revascularisation. The experience with PTMA is more limited but has been achieved in patients often considered too high-risk for a surgical procedure. More recently, intra-luminal stenting has been added to PTMA in an attempt to decrease the incidence of recurrent stenoses. Too few patients have been treated in this fashion to permit a conclusion as to its long-term value in managing patients with CMI. If however, the use of stents proves to reduce the recurrence rates of PTMA close to those of surgical revascularisation, PTMA may become the treatment of choice in the future.

CONCLUSION:

A patient with the typical pain of CMI and unexplained weight loss whose diagnostic evaluation has excluded other gastrointestinal disease and whose angiogram shows occlusive involvement of at least two of the three major arteries, should undergo revascularisation. Patients with CMI who are otherwise relatively healthy should be treated with surgery; poorer risk patients should have an initial attempt at PTMA with or without stenting to relieve symptoms.

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