MALABSORPTION AND MESENTERIC ISCHEMIA

Report of a Case

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ACUTE mesenteric ischemia is a catastrophic event if not treated. Chronic mesenteric ischemia is less dramatic but is just as lethal. The patient may have no symptoms or there may be gradations to and including those of typical abdominal angina. The occurrence of diarrhea, constipation, and weight loss, associated with cramping postprandial abdominal pain, may be misdiagnosed as evidence of functional gastrointestinal disease unless the possible presence of mesenteric arterial insufficiency is considered. Typical abdominal angina is characterized by excruciating abdominal pain that occurs from 20 to 30 minutes after eating and lasts from one to two hours. To avoid pain, the patient does not eat and severe weight loss ensues. Impaired gastrointestinal motility and malabsorption contribute to the loss in weight and cause diarrhea.

Although occlusive vascular disease as a cause of mesenteric infarction has been stressed for 25 years, nonocclusive causes such as polycythemia vera,[1] congestive heart failure,[2-4] acute hypotension,[5] and aortic insufficiency,[6] have been reported only in the last 10 years. Because relatively few cases of malabsorption associated with mesenteric insufficiency have been described in the literature,[7-11] the following report of such a case is presented.

Report of a Case

A 44-year-old white man was admitted to the Cleveland Clinic Hospital on December 16, 1964. In March 1962, he had a posterior myocardial infarction, and after a three-week recovery period he had no symptoms of angina pectoris. In October 1964, he began to experience severe retrosternal pain associated with physical exertion and emotional stress, as well as nocturnal pain that awakened him four to six times a night.

The physical examination on admission to the hospital revealed a thin apprehensive man. Grade 2 arteriosclerotic changes were present in the ocular fundi. The heart and lungs were normal. The abdomen was soft, without masses, tenderness, or bruits. The liver was palpable two fingerbreadths below the right costal margin. No pedal edema was present. An electrocardiogram revealed changes of an old posterior myocardial infarction. A roentgenogram of the chest was normal. Coronary arteriography demonstrated severe coronary artery disease and a small aneurysm of the ventricular septum. On December 22, 1964, a Vineberg internal mammary artery implantation and omentopexy were performed.†

The postoperative course was satisfactory except for moderate distension of the abdomen and two or three greenish-brown bowel movements per day. On the third day after operation, there were five similar bowel movements. The abdomen was moderately distended, with hyperactive bowel sounds and moderately tympanitic. No rebound tenderness was present. By the fifth postoperative day, on December 27, the greenish-brown bowel movements (without blood) had increased to eight per day. Severe abdominal cramps occurred intermittently at that time

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and were not associated with the ingestion of food. A roentgenogram of the abdomen taken with the patient supine revealed distended loops of jejunum and air in the right transverse colon. On the evening of December 27, laparotomy was performed* because of continuance of abdominal pain and distension despite the fact that two electrocardiograms after the Vineberg operation revealed an acute anteroseptal infarction. At laparotomy, all of the small intestine beginning four feet from the ligament of Treitz appeared congested and purple. A diagnosis of "small-bowel syndrome" after a cardiac operation was made.

Although the arterial blood supply to the small intestine was insufficient, no vascular occlusion was present. The bowel appeared to be viable, so the abdomen was closed without the removal of any tissue.

Except for the persistence of two or three liquid green stools per day, the patient seemed to recover satisfactorily. Diphenoxylate hydrochloride with atropine sulfate† was used for the treatment of the diarrhea but without much benefit. On January 12, 1965, coagulase-positive Staphylococci were cultured from the rectum. One gram per day of cephalothin‡ was given intravenously for nine days. The temperature postoperatively ranged between 100 and 102 F. and became normal during the cephalothin therapy. However, the patient continued to have several greenish bowel movements per day. On January 26, hypoalbuminemia was noted (1.73 gm. of albumin per 100 ml. of blood). The total serum protein value was 5.5 gm. per 100 ml. Leukocytosis was persistent, the leukocyte count ranging between 13,000 and 14,900 per cubic millimeter. On February 1, d-xylose and serum carotene values were 1.0 gm. per 5 hours and 15 μg. per 100 ml., respectively. On February 2, the serum values of magnesium, calcium, and phosphorus were 1.9 mg., 7.7 mg., and 3.2 mg. per 100 ml., respectively. The prothrombin time was 19 seconds (normal, 13 seconds) and the serum concentrations of sodium, chloride, potassium, and carbon dioxide were normal. Serial roentgenographic studies of the small intestine after barium swallow revealed multiple areas consistent with partial obstruction beginning in the proximal jejunum (*Fig. 1*). The most severely obstructed loop appeared to be in the midportion of the ileum, which retained barium long after most of the contrast material had passed into the colon. After the intravenous injection of 28 μc. of I^{131}-tagged polyvinylpyrrolidone, stool specimens were collected for three days. Unfortunately, the stool collected in the first 16 hours of the three-day collection period was lost. The fecal fat content and the amount of radioactivity were 13.3 gm. and 1.5 percent, respectively. These are normal values for a 72-hour collection, but since the important first specimen was not available, they indicate increased loss of fat and polyvinylpyrrolidone.

On February 8, the total serum protein value was 4.5 gm. per 100 ml. and the albumin fraction was 1.53 gm. per 100 ml.; the serum calcium content was 8.5 mg. per 100 ml. On February 9, the patient suddenly became hypotensive. An electrocardiographic tracing was interpreted as ventricular fibrillation. Despite resuscitative measures, the patient died seven hours later.

At necropsy, there were fibrinous adhesions covering the serosal surfaces of most of the ileum and part of the jejunum. The serosal surface of the terminal 50 cm. of ileum, cecum, ascending, transverse, and descending colon appeared reddish blue. An area of perforation of the ileum, 1 cm. in diameter, was present approximately 20 cm. from the ileocecal junction. The mucosal surface of the terminal ileum was dark reddish pink, with four superficial ulcers, the largest being 0.3 cm. in diameter. The mucosal surface of the cecum and of the ascending and the transverse colon was hemorrhagic. Microscopic examination of sections of the terminal ileum and of the colon revealed acute hemorrhagic necrosis, serositis, and pseudopolyposis. An atheromatous plaque at the ostium of the superior mesenteric artery reduced the lumen 30 percent. No other obstruction was present in the distal branches of the vessel. The celiac artery was not occluded and the inferior mesenteric artery was hypoplastic. The final pathologic diagnoses were: thrombosis of internal mammary artery implant, complete; severe coronary arteriosclerosis with segmental occlusion of the anterior descending and right coronary arteries; healed transmural infarct of the posterior wall of the left ventricle; hemorrhagic necrosis of the terminal ileum, cecum, ascending, transverse, and descending colon with acute serositis; and moderate arteriosclerosis of the superior mesenteric artery at its origin.

Discussion

The blood supply to the small bowel and the large bowel is primarily derived

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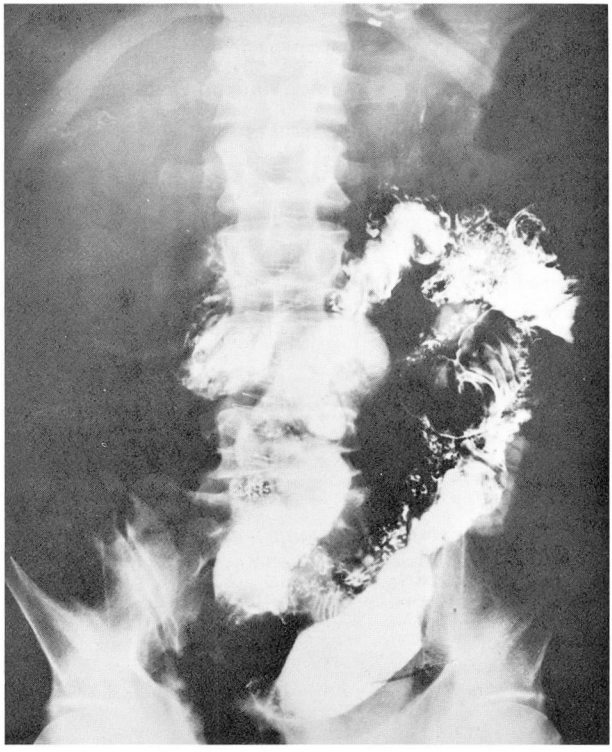

Fig. 1. Roentgenogram of the small intestine after barium swallow reveals multiple areas consistent with obstruction. A large dilated loop of ileum retained barium long after contrast material passed into the colon.

from three sources: the celiac artery, the superior mesenteric artery, and the inferior mesenteric artery. There is an extensive collateral communication among these arteries. The inferior and superior pancreaticoduodenal arteries provide avenues for communication between the celiac artery and the superior mesenteric artery. The inferior mesenteric artery communicates with the superior mesenteric artery via Riolan's arch.[12] Atheromata at the origins of the celiac and superior mesenteric arteries are the most common cause of mesenteric ischemia. However, mesenteric ischemia may occur secondary to vasoconstriction of the splanchnic bed.[13] It is widely known that in congestive heart failure cardiac output decreases, resulting in compensatory vasoconstriction that redistributes blood from the skin, the muscle, and the splanchnic bed to more vital organs. Injections of procaine hydrochloride into the root of the mesentery in patients having nonocclusive mesenteric ischemia have been reported[13] to restore viability to ischemic bowel presumably by relief of vasospasm.

Ende[3] reported six cases in which bowel infarction occurred without vascular

occlusion. Five of the patients had congestive heart failure and one patient had syphilitic aortic insufficiency. It was the opinion that nonocclusive mesenteric ischemia could cause mesenteric infarction in a manner similar to that in which nonocclusive peripheral gangrene may occur as a complication of myocardial infarction.[3] Berger and Byrne[5] reported 23 cases of nonocclusive massive bowel infarction, the predisposing causes of which were said to be: congestive heart failure, myocardial infarction, and hypotension. Two cases of aortic insufficiency and nonocclusive bowel infarction have also been reported.[3, 6]

Adequate circulation is necessary for gastrointestinal cells to carry on active transport. If mesenteric ischemia exists, various degrees of cell malfunction occur, which may result in malabsorption. Guthrie and Quastel[13] reported that there was great impairment of active absorption of sugars and amino acids in isolated intestine after experimental shock. Joske, Shamma'a, and Drummey[11] described two cases in which mesenteric ischemia and prolonged malabsorption of fat, carbohydrate, and protein occurred after temporary occlusion of the superior mesenteric artery. A decrease of serum albumin content and the leakage of I^{131}-labeled polyvinylpyrrolidone into the intestine of a dog were observed after occlusion of the superior mesenteric artery.[14] Villous atrophy,[15] mucosal ulceration,[12] and cicatricial stricture due to segmental mesenteric ischemia may also occur in human beings and in animals.[11, 15]

It is postulated that in the patient whose case we presented, mesenteric vascular insufficiency developed as a consequence of the myocardial infarction incurred shortly after the operation on the heart. This resulted in diarrhea, abdominal distension, and cramps, with associated intestinal obstruction according to roentgenographic evidence. At laparotomy the vascular insufficiency was confirmed. Subsequently, a relative insufficiency of the mesenteric circulation continued with a consequent derangement of function of the small intestine, manifested by diarrhea, malabsorption, and increased permeability of the intestinal wall to serum albumin and polyvinylpyrrolidone. Roentgenographic examination of the small intestine one month after the laparotomy showed loss of tone of the small bowel and disordered motility. The vascular insufficiency was still quite evident at the time of necropsy about six weeks after the beginning of the abdominal symptoms. It is believed that small-bowel ischemia due to a diminished blood flow was present throughout this time, even though from day to day there may have been variations in the quantity of the blood reaching the intestine. Mesenteric infarction without vascular occlusion is not unusual as an acute event. This case is unique in our experience because of the long duration of the mesenteric vascular insufficiency and because of the roentgenographic demonstration of the effects of such insufficiency. Probably many factors helped to produce the inadequate mesenteric blood flow, the most important one being the congestive heart failure and a failing myocardium. The partial occlusion of the superior mesenteric artery was thought to play a relatively small part in producing this patient's disease.

Summary

Myocardial infarction occurring after a cardiac operation was thought to have caused relative mesenteric arterial insufficiency in a 44-year-old man. Intestinal malabsorption with diarrhea, weight loss, and hypoalbuminemia was manifest over a six-week period. Laparotomy three days after the myocardial infarction demonstrated hypoxia of the entire small intestine; roentgen examination one month later showed a dilated, atonic, small bowel; hyperemia, gangrene, and perforation were found at necropsy six weeks after the onset of the disease.

References

1. Schwartz, C. J.; Acheson, E. D., and Webster, C. U.: Chronic mid-gut ischaemia with steatorrhoea in polycythaemia rubra vera. Am. J. Med. **32:** 950-953, 1962.
2. Sullivan, J. F.; Ezzo, J. A.; Fitzgerald, W. T., and Mack, R. E.: Mesenteric vascular inadequacy. Geriatrics **13:** 97-101, 1958.
3. Ende, N.: Infarction of bowel in cardiac failure. New England J. Med. **258:** 879-881, 1958.
4. Sullivan, B. H., Jr.: Vascular lesions simulating acute abdomen. Am. J. Cardiol. **12:** 358-360, 1963.
5. Berger, R. L., and Byrne, J. J.: Intestinal gangrene associated with heart disease. Surg. Gynec. & Obst. **112:** 529-533, 1961.
6. Hoffman, F. G.; Zimmerman, S. L., and Cardwell, E. S., Jr.: Massive intestinal infarction without vascular occlusion associated with aortic insufficiency. New England J. Med. **263:** 436-440, 1960.
7. Morris, G. C., Jr., and DeBakey, M. E.: Abdominal angina—diagnosis and surgical treatment. J.A.M.A. **176:** 89-92, 1961.
8. Mavor, G. E., and Michie, W.: Chronic midgut ischaemia. Brit. M. J. **5095:** 534-537, 1958.
9. Shaw, R. S., and Maynard, E. P., III: Acute and chronic thrombosis of mesenteric arteries associated with malabsorption; report of two cases successfully treated with thromboendoarterectomy. New England J. Med. **258:** 874-878, 1958.
10. Orr, T. G., Jr.; Lorhan, P. H., and Kaul, P. G.: Mesenteric vascular occlusion: report of 2 cases. J.A.M.A. **155:** 648-650, 1954.
11. Joske, R. A.; Shamma'a, M. H., and Drummey, G. D.: Intestinal malabsorption following temporary occlusion of superior mesenteric artery. Am. J. Med. **25:** 449-455, 1958.
12. Reiner, L.: Mesenteric arterial insufficiency and abdominal angina. Arch. Int. Med. **114:** 765-772, 1964.
13. Guthrie, J. E., and Quastel, J. H.: Absorption of sugars and amino acids from isolated surviving intestine after experimental shock. Arch. Biochem. **62:** 485-496, 1956.
14. Zuidema, G. D.; Turcotte, J. G.; Wolfeman, E. F., Jr., and Child, C. G., 3d: Metabolic studies in acute small-bowel ischemia. Arch. Surg. **85:** 130-135, 1962.
15. Laufman, H.: Gradual occlusion of mesenteric vessels; experimental study. Surgery **13:** 406-410, 1943.