MESENTERIC THROMBOSIS

Report of a Case

F. L. SHIVELY, Jr., M.D. and R. J. F. RENSHAW, M.D.

Mesenteric thrombosis is usually a dramatic episode. Recently, a case of mesenteric thrombosis was seen in which the course was not sudden and spectacular but slow and progressive. Because of the unusual duration of the disease and the diagnostic problems encountered a report of this case seemed justified.

The pathema of mesenteric thrombosis is an involvement of the superior or inferior mesenteric vessels either by embolism or by thrombosis. Over 70 per cent of the cases are venous in origin, and the superior mesenteric vessels are more frequently involved than the inferior vessels.[1] The following conditions have been described as causes of mesenteric thrombosis: valvular heart lesions, arteriosclerosis and atherosclerosis, aneurysm of the abdominal aorta or superior mesenteric vessels, blood dyscrasias (polycythemia vera and leukemia), abdominal tumors, trauma (blows, great exertion, straining, parturition), septic foci (diverticulitis, ulcerative colitis, and pelvic infections), predisposing factors (surgery of the stomach, appendix, hernia, and pelvis), and finally a small group in which no primary cause can be demonstrated.

Although the incidence of mesenteric thrombosis is greatest between the third and sixth decades, persons of any age may be affected. Cases have been reported in patients as young as 5 and over 80 years of age. Women are more commonly affected than men.

The clinical picture depends upon the stage at which the patient is seen, extent of infarction, and duration of the process. Usually there is an acute condition of the abdomen, but infrequently the onset may be slow as in the reported case. Pain is the outstanding symptom. It is sudden in onset, colicky in nature, and may occur in paroxysms. Nausea and vomiting follow the pain, whereas in acute intestinal obstruction the opposite is usually true. Constipation is more common than diarrhea, although loose watery stools sometimes follow a period of constipation. Dark blood is frequently passed in or with the stool.

Temperature, pulse, and respiration depend upon the rapidity of onset, extent of infarction, and duration of the process. In acute massive infarction the temperature may be normal or subnormal with symptoms of shock. In slowly developing infarction the temperature may be normal or septic. Shock is usually present in proportion to the degree of infarction and rapidity of onset. Either generalized or local-

ized abdominal tenderness is usually associated with rigidity and rebound phenomenon. Jaundice has been reported in a small percentage of cases. During the early stages the erythrocyte count is within normal limits, but the leukocyte count is between 15,000 and 40,000. Leukocytosis is absolute, the polymorphonuclear leukocytes usually being in excess of 85 per cent. Roentgenologic examination contributes little to the diagnosis.

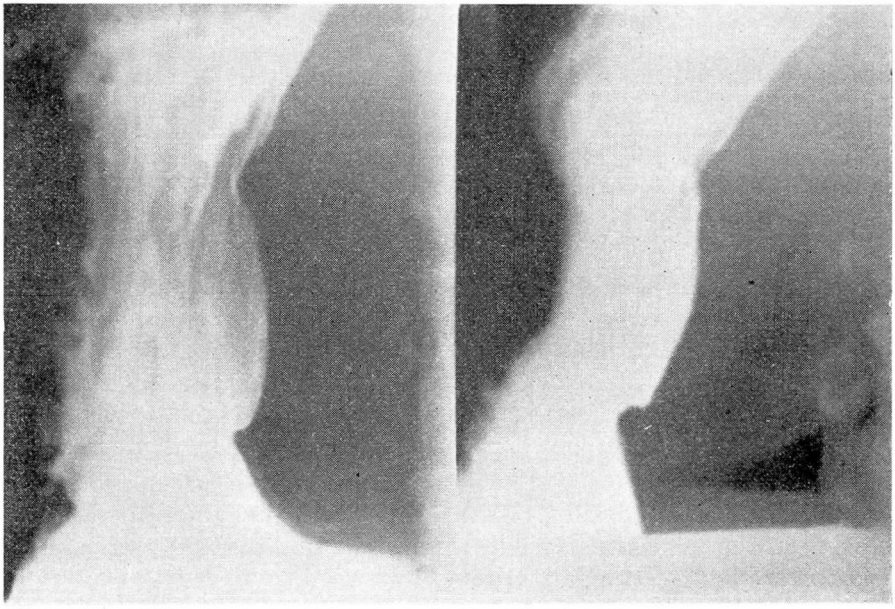

Figure 1

CASE REPORT

A woman, aged 57, was admitted on July 9, 1943 complaining of abdominal pain. The first episode had occurred 13 months prior to admission. The pain was colicky in nature, occurred immediately after meals, and lasted for several hours. Vomiting of frothy material frequently accompanied the pain. There had been no hematemesis nor vomiting of undigested food. Roentgenologic examination of the gastrointestinal tract made elsewhere was reported as being negative. After the initial episode the patient had been relatively free of distress. She had, however, voluntarily for 2 years rigidly restricted her diet and had lost 55 pounds in weight. During the year prior to consultation the patient had subsisted chiefly upon bread and coffee. She especially avoided meat. The appetite had been poor.

Two weeks prior to admission the pain had recurred. The history revealed that she had had two major surgical procedures: A pelvic tumor had been removed in 1907, and a cholecystectomy had been performed in 1931.

The patient did not appear acutely ill. The oral temperature was 98.2 F; pulse 76 beats per minute; systolic blood pressure 115 mm. of mercury and the diastolic pressure 60 mm. of mercury. The skin was dry, unusually wrinkled, and pale, but not icteric. The peripheral blood vessels were sclerotic. Abdominal examination revealed two surgical scars. There was a small postoperative ventral hernia in the lower midline scar. Generalized abdominal tenderness prevented satisfactory or adequate palpation. Rebound tenderness and rigidity, however, were not present. No definite masses nor organs could be palpated. Pelvic examination revealed an atrophic vaginitis. The significance of the postoperative hernia was questioned, because at the first examination it could not be satisfactorily reduced. However, complete reduction at subsequent examinations did not relieve the patient of abdominal distress.

Laboratory examination revealed the following: Urinalysis showed a large number of leukocytes. Hemoglobin estimation was 81 per cent, leukocyte count was 9000. The blood sugar level 3 hours postprandial was 81 mg. per cent. The blood Wassermann and Kahn tests were negative.

A complete gastrointestinal roentgenologic examination showed a gastric ulcer on the upper portion of the lesser curvature. After administration of a barium enema, a lesion found in the sigmoid was interpreted as being inflammatory rather than neoplastic. Roentgenogram of the chest was normal. The tentative diagnosis was gastric ulcer and diverticulosis of the sigmoid colon. The patient was placed on strict ulcer management, antispasmodics, and sedatives.

Sixteen days later on July 28, 1943 the patient was admitted to the hospital as an emergency patient. Since her first visit the abdominal pain had persisted and gradually had become worse. The pain was neither sudden in onset nor had it occurred in paroxysms. For one week prior to admission uncontrollable vomiting had been present. The hospital admitting physician made the following note: "Physical examination revealed a 57 year old woman acutely ill, looking older than her age, dehydrated, and vomiting a dark liquid with a fecal odor." Blood pressure was 140 mm. of mercury systolic and 80 mm. of mercury diastolic. The temperature was 100.8 F, and the pulse 104 beats per minute.

The abdomen was exquisitely tender and rigid but was not board-like. No masses nor organs could be palpated because of the exquisite tenderness. A rectal examination was negative. Laboratory studies revealed a leukocytosis of 16,000. A differential count was not made. The blood urea level was 33 mg. per cent; carbon dioxide combining power 67; blood chlorides 495 mg. per cent; and the blood sugar 169 mg. per cent, apparently several hours after the last intake of food.

The tentative diagnosis was intestinal obstruction with probable perforation of a sigmoidal diverticulum. A Miller-Abbott tube was passed, and parenteral fluid and blood given. Five gm. of sodium sulfadiazine was administered intravenously for 3 consecutive days. On the third hospital day, the patient suddenly became jaundiced, and clay-colored stools were passed. This was attributed to probable sulfadiazine intoxication with hepatitis. The drug was discontinued, and additional blood transfusions and parenteral fluids were administered. The sulfadiazine blood levels rapidly returned to normal; however, the jaundice persisted, and the temperature fluctuated between 98 to 100.5 F.

The subsequent course of the disease raised considerable doubt as to the validity of the diagnosis at hospital admission. The entire clinical picture could not be correlated as one entity. Peritonitis from a ruptured sigmoidal diverticulum did not seem probable because of the increasing leukocytosis with a low grade fever and a soft abdomen. Strict

ulcer management was not continued in the hospital because the pain was not suggestive of peptic ulceration. There was little evidence of a silent common duct stone producing jaundice. A pancreatic malignancy obstructing the common bile duct did not adequately explain the physical and laboratory findings.

Because of conflicting and inconsistent evidence and the presence of persistent deepening jaundice, exploration was advised even though it was realized that the jaundice could have been of a hepatocellular type resulting from sulfadiazine hepatitis.

Exploration of the right upper quadrant was performed on August 13, 1943. Aside from a greatly enlarged yellow-green and swollen liver, the biliary tract was normal. A "T" tube was inserted into the common bile duct and a biopsy of the liver taken. There was no external evidence of peptic ulcer of the stomach. Because of many firm fibrous adhesions and the poor general condition of the patient, further exploration was deemed inadvisable. The patient expired 24 hours postoperatively on the seventeenth hospital day.

By request of the relatives a postmortem examination was limited to the abdomen. The liver presented evidence only of a hepatitis. The stomach was grossly normal, without evidence of peptic ulceration. The entire small and large bowel were gangrenous, and in innumerable areas had undergone lysis. The mucosal continuity of the sigmoid was completely lost for a distance of 10 to 12 cm. The mesenteric arteries and the splenic artery were completely occluded by a severe degree of arteriosclerosis. The abdominal aorta and the left iliac artery were thickened by multiple arteriosclerotic and atheromatous plaques.

The pathologic diagnosis was mesenteric thrombosis secondary to a severe grade of arteriosclerosis involving the aorta and the splenic, iliac, superior and inferior mesenteric arteries.

COMMENT

In retrospect certain points in the history and physical findings are highlighted in view of the pathologic findings. It is purely hypothetical but possible that the initial attack of pain, 13 months prior to consultation, was due to an infarction not large enough to cause the usual spectacular and dramatic phenomena associated with mesenteric thrombosis. During the 13 month interval the arteriosclerotic occluding process was apparently so gradual that symptoms did not occur until massive segments of the gastrointestinal tract had been deprived of sufficient circulation.

Because the arterial occlusion was gradual, the leukocyte count remained normal. We are unable to explain the roentgenologic findings of what appeared to be an ulcerative lesion high on the lesser curvature of the stomach. At the initial fluoroscopic examination the stomach was considered normal, but on the films there appeared to be a shallow ulcer high on the lesser curvature of the stomach (fig. 1). Three days later when the stomach was reexamined roentgenologically by a different examiner, what appeared to be a shallow ulceration was again observed.

The jaundice may have been the result of sulfadiazine intoxication, a toxic manifestation of the extensive degenerative process in the bowel, or a combination of both factors.

Finally, in retrospect, one might ask if treatment should have been different had the true nature of the disease been suspected. We do not believe that mesenteric thrombosis could have been diagnosed or would have been suspected from the initial examination. However, at the time of hospitalization the atypical acute condition of the abdomen might have led to the suspicion, and exploration performed, but in view of the extensive disease no surgical procedure would have been successful. In the treatment of mesenteric thrombosis side-to-side anastomosis is frequently indicated, even though the patient's general condition is poor. On the other hand, exteriorization of the affected segment carries a high mortality rate when parts of the bowel other than the colon are involved.

SUMMARY

A case of mesenteric thrombosis is reported in which the original infarction of the mesenteric vessels presumably occurred 13 months prior to consultation. The differential diagnosis was complicated by the rapid subsidence of the acute condition of the abdomen, by the roentgenologic finding of what appeared to be an ulcerative lesion high on the lesser curvature of the stomach, and by the persistent deepening jaundice.

Postmortem examination revealed the entire small and large bowel to be gangrenous, with innumerable areas having undergone lysis. Continuity of the sigmoid was completely lost for 10 to 12 cm. The mesenteric arteries and the splenic artery were completely occluded by a severe degree of arteriosclerosis.

REFERENCE

1. Moore, Thomas: Mesenteric vascular occlusion. Brit. J. Surg. 28:347-356, 1940.