Thrombosis of the Superior Mesenteric Vein

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A case of extensive thrombosis of the superior mesenteric vein treated surgically is reported. Selective arteriography may lead to earlier diagnosis and thrombectomy may be successful in salvaging the involved intestine.

INTRODUCTION

Thrombosis of mesenteric veins, with or without infarction of the intestines, is generally considered to be an uncommon entity. Diagnosis is often delayed and mortality rates remain high. Reports of instances of mesenteric venous thrombosis occurring in persons taking contraceptive medication, as well as the availability of improved methods of diagnosis and treatment are reasons for continued review of the information pertinent to this condition.

CASE REPORT

E. M., a 53-year-old white woman, was admitted to Presbyterian Hospital complaining of mild, lower abdominal, postprandial cramping pain of one week's duration. The pain was partially relieved by defecation or enemas. Five days prior to admission, physical findings, complete blood count and urinalysis were considered normal. One day prior to admission, repeat physical examination revealed hyperactive bowel sounds and mild generalized abdominal tenderness. WBC was 9,000/mm³ with a normal differential. Urinalysis, chest and abdominal x-ray films were interpreted as normal. Eighteen hours before admission, the abdominal pain became constant. Nausea, vomiting and diarrhea ensued and gross blood was noted in the stool. She was admitted as an emergency.

The patient had previously been in good health and reported no prior surgical procedures. She had taken an estrogen (Premarin, 2.5 mg daily) for eight years for postmenopausal symptoms but was on no other medication. Family history was not contributory.

Physical Examination: The patient was a well-developed, thin, white woman who appeared acutely ill. Pulse was 100/min and regular, blood pressure was 115/85 mm Hg, respirations 16/min. and temperature was 96.6°F. Pertinent findings included slight guarding of the abdomen, especially in the lower quadrants, and hypoactive bowel sounds. Rebound tenderness was not present and no abdominal masses were palpated. Rectal examination showed the presence of blood in the ampulla. The remain-
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der of the physical findings were unremarkable.
Laboratory Data: Hgb 17.2 gm/dl, Hct 55%, WBC 19,200/mm³ (15 bands, 77 segs, 4 lymphs, 4 mons), BUN 27 mg/dl, Na+ 138 mEq/L, K+ 4.0 mEq/L, Cl—106mEq/L, CO₂ 18mEq/L. X-rays of the chest and abdomen were unremarkable.
After hospitalization, nasogastric suction and intravenous fluid therapy were instituted and analgesics administered. Shortly after admission, the patient became hypotensive and complained of increased abdominal pain. The abdomen was distended, bowel sounds were absent and rebound tenderness was present. Surgical consultation was obtained and the patient underwent immediate exploratory laparotomy. Upon entering the peritoneal cavity, approximately 300 cc of serosanguineous fluid was found. The small bowel was edematous and deeply cyanotic. Patency of the arterial supply was demonstrated by opening a small artery in the peripheral mesentery. Numerous thrombi were found in the superior mesenteric vein extending into the very small veins near the intestinal wall. A segment of infarcted bowel, 350 cm in length, was resected and primary anastomosis made between the remaining 25 cm of proximal jejunum and a similar length of terminal ileum.
Intravenous heparin, 10,000 units, was administered in the early postoperative period and continued at a rate of 5,000 units every six hours. Moderately severe upper gastrointestinal hemorrhage began on the third postoperative day and heparin therapy was discontinued. Bleeding responded to iced saline lavage. Radioisotopic pulmonary perfusion studies performed after the onset of pleuritic chest pain showed large defects compatible with pulmonary emboli. Heparin therapy was reinstituted. Frequent loose stools and cramping abdominal pain began after the fifth postoperative day but responded to paregoric and a diphenoxylate-atropine preparation (Lomotil). Upper GI series two weeks after operation confirmed the small bowel length to be less than two feet, with a small bowel transit time of five minutes. Fecal fat content prior to discharge from the hospital was 12 grams/24 hours on no dietary restriction (normal upper limit, 7 grams/24 hours). Three weeks following admission, the patient was discharged on oral anticoagulant therapy and Lomotil for the control of diarrhea.
One year after hospitalization, the patient reported one to three solid stools daily on an unrestricted diet. Her weight had stabilized at 111 pounds and she had resumed normal activities. Her only medication was Lomotil taken four times daily. Sixteen months after the intestinal resection, she was hospitalized for treatment of severe thrombophlebitis of the right leg from which she recovered satisfactorily. She is maintained on coumadin.

DISCUSSION

Obstruction of mesenteric veins with resulting changes in the intestine, ranging from edema to gangrene, is commonly encountered.

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Venous obstruction, with or without thrombus, is an early event in strangulated hernia and is observed when mechanical distention of the intestine becomes severe. In some instances, thrombosis of mesenteric veins is associated with recognized systemic processes such as polycythemia, cirrhosis and heart failure. Those instances of mesenteric venous obstruction due to recognized mechanical events (hernia, volvulus, neoplasms) or systemic diseases (polycythemia, etc) have been termed “secondary” venous obstruction and will not be further discussed. Of greater interest are those cases in which mesenteric venous obstruction occurs in the absence of recognized mechanical cause or predisposing disease. This has been termed “primary” or “agnogenic” mesenteric venous obstruction and the case presented is considered to be an example of this category. It is quite unlikely that the process is the result of a single factor but, for the present, a recognizable pathologic, pathophysiologic and clinical entity can be described.

The incidence of primary mesenteric venous thrombosis cannot be calculated with any degree of confidence from existing data. It is common enough that every surgeon should be aware of the entity. The history of its description has been recorded by Berry and Bougas. Collective experience with this process allows several observations that may be related to cause. A high incidence of previous peripheral venous disease is commonly reported. One family with multiple episodes of mesenteric venous thrombosis is of interest. Recently, reports of mesenteric venous occlusion in patients taking contraceptive medication have appeared. A detailed discussion of the relationship between thromboembolic phenomenon and contraceptive medication is beyond the scope of this communication, but knowledge of the suspected role of contraceptive medication in mesenteric venous disease is obviously important.

Typically, thrombosis begins in a branch of the superior mesenteric vein. The pattern of propagation of thrombus obviously determines the severity and extent of bowel involvement. Pathological studies of the condition have revealed thrombus in varying stages, suggesting that the process is an evolving one. Microscopic changes suggesting vasculitis have been described by Van Way, et al. Grossly, there is edema of the mesentery and bowel wall. Discoloration of the bowel ranges from dusky to purple-black. There is usually a moderate amount of serosanguineous fluid in the peritoneal cavity. Perforation of the bowel occurs quite late and death of the patient is likely before perforation has occurred.

The principal pathophysiologic change in mesenteric venous obstruction is loss of fluid and blood into the mesentery and bowel. Hypovolemia, hemoconcentration and acidosis result with sepsis playing a late role. Mesenteric venous thrombosis has been produced experimentally by Polk and the pathophysiologic changes recorded. Untreated animals died within a few hours of the production of extensive mesenteric venous thrombosis.

A diagnosis of mesenteric venous thrombosis is often delayed because of a lack of distinctive clinical features. A prodromal period, consisting of vague episodic abdominal discomfort, may last days to weeks before the onset of acute symptoms. The acute episode usually consists of persistent, severe, generalized abdominal pain. Vomiting is common. Bloody diarrhea may occur. Ileus with abdominal distention is usually present after 24 to 48 hours.

Physical findings during the prodromal period usually are not remarkable. After onset of severe pain, examination often reveals no more than poorly localized abdominal tenderness. Later, deteriorating vital signs, abdominal distention, diminished bowel sounds and rebound tenderness signal development of an acute abdomen.

Laboratory studies show leukocytosis with a left shift. Elevations in hemoglobin and BUN compatible with hemoconcentration may be found. X-ray films of the abdomen may show a paucity of bowel gas and a “ground glass” appearance suggestive of fluid collection within the peritoneal cavity. Free air due to perforation is an uncommon finding. A high incidence of air-filled loops of bowel suggestive of obstruction has been reported. Polk performed selective arteriograms in animals with experimental superior mesenteric venous thrombosis. These studies demonstrated that injection of contrast material into the superior mesenteric artery resulted in reflux into the aorta, a markedly prolonged arterial phase and opacification of thickened bowel wall. Venous filling was absent. On the basis of these studies, Polk sug-
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gests that selective arteriography of the superior mesenteric artery can be a valuable diagnostic tool, particularly in distinguishing between arterial and venous occlusion.

In the great majority of cases of mesenteric venous thrombosis reported to date, treatment has consisted of surgical removal of the involved intestine with re-establishment of continuity or temporary enterostomy. Appropriate preoperative fluid and electrolyte replacement and improved general surgical support has resulted in a decreasing mortality rate. There is evidence that earlier diagnosis and operation might permit thrombectomy with salvage of the intestine. This has been demonstrated experimentally by Polk, and such a procedure has been carried out by Inahara. The use of heparin in the immediate postoperative period is strongly supported by retrospective studies. Most authors advise the use of sodium warfarin (coumadin) for several weeks after heparin has been discontinued, but the value of such a regimen has not been convincingly substantiated. The role of antibiotics in the management of patients with mesenteric venous obstruction has also not been completely clarified. Most authors have used broad spectrum antibiotics.

The mortality rates in the management of mesenteric venous occlusion, although reduced, remain appreciable, ranging between 40-60% in most series. It is likely that this figure could be reduced by increased awareness of the condition, earlier diagnosis and aggressive surgical treatment.

References


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