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Superior mesenteric vein thrombosis: how a family medicine physician's knowledge of a patient helped save a life

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This case illustrates the clinical difficulty in making the diagnosis of mesenteric ischemia and in particular superior mesenteric venous thromboembolism. There is no definitive sign, symptom, laboratory or diagnostic criteria to establish the diagnosis, rather a constellation of data along with clinical judgment must be utilized for quick recognition and treatment. In this case although the delay in care was minimal and the patient was taken to surgery in a timely manner he still suffered the comorbid complications of respiratory and hepatic failure. Familiarity of the patient and high clinical suspicion still remain the most vital tools when diagnosing mesenteric ischemia. The family physician's knowledge of, and long-term relationship with the patient, certainly averted a possibly morbid outcome.

Mr. Y is a 56-year-old male who was diagnosed with type 2 diabetes in 2000. He was somewhat noncompliant with diet and remained under poor control with oral hypoglycemics. Insulin therapy was initiated in 2004, mainly secondary to dietary indiscretion. He also was diagnosed with hypertension, hyperlipidemia, and fatty infiltration of the liver. He was followed in the office regularly and his liver functions remained within normal limits. His HbA1C level remained around 9.5 %. Mr. Y was found to have low platelets in early 2007 in the range of 70,000 u/L. This was monitored and there was no specific medication interaction attributed to this.

In the fall of 2008, he developed abdominal pain and hematemesis and was admitted to the intensive care unit; upper endoscopy revealed esophageal varicies. These were successfully ligated and the patient was discharged. Other admission diagnostics included a computed tomography (CT) scan demonstrating liver cirrhosis with abdominal and pelvic ascites.

Three to four months later he presented to the emergency department with severe abdominal pain. Initial workup included an abdominal ultrasound and gastrointestinal (GI) consultation. Esophagogastroduodenoscopy was scheduled for the next day. The GI specialist did not request a CT scan based on the finding of esophageal varices on previous admission.

During family medicine rounds it was noted that the patient was in severe abdominal pain, despite medication with morphine. Because the patient was well known to the primary care physician, it was felt that this was out of character for him. His symptom complex was worrisome enough that the family medicine physician ordered a stat abdominal CT. This revealed an acute large superior mesenteric vein thrombosis. Vascular and general surgery were consulted and the patient was started on intravenous heparin. Surgical consultation recommended emergent exploratory laparotomy. After discovering mesenteric and bowel wall ischemia, a small bowel resection was performed.

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Discussion

Superior mesenteric vein thrombosis accounts for 5% of all mesenteric ischemia.[1,2] Other causes of ischemia include superior mesenteric artery embolism (50%), superior mesenteric artery thrombosis (15-25%), and nonocclusive ischemia (20-30%).[1,2] Risk factors for the development of mesenteric venous thrombosis include hypercoagulable states, portal hypertension (which was the case with this patient), abdominal infections, blunt abdominal trauma, pancreatitis, splenectomy, and malignancy in the portal region.[3]

Symptoms associated with acute mesenteric ischemia include rapid onset of severe peri-umbilical abdominal pain, which is out of proportion to findings on physical examination. Nausea and vomiting are common, and pains with forceful bowel evacuation should increase suspicion. Mesenteric vein thrombosis may have a more insidious presentation of symptoms that may occur over weeks to months.[4,5] Nonspecific abdominal pain may be the only feature of patients that present with bleeding from esophageal or gastric varices as a result of portal or splenic vein thrombosis. Features of the pain and its presentation can provide clues to distinguish small bowel from colonic ischemia. Severe pain is more likely with small bowel involvement. Pain will often precede vomiting in patients with small bowel obstruction, leading to ischemia. Pain is thought to be more sudden in embolic disease versus thrombotic and vasculitis causes. Lower abdominal pain associated with hematochezia is more likely associated with colonic ischemia. Despite these symptoms, the abdominal examination is usually normal initially and may only reveal abdominal distention.[6]

The timely diagnosis of mesenteric ischemia is almost totally dependent on high clinical suspicion, especially in patients with risk factors. Rapid diagnosis is essential because of the high morbidity and mortality rates associated with intestinal infarction.[7] Once the diagnosis is suspected, the initial management should be directed at correction of underlying factors contributing to the ischemia (e.g., hypovolemia and/or hypotension, cardiac arrhythmias). A CT scan should be ordered when mesenteric vein thrombosis is suspected. Angiography is the gold standard and may be helpful in identifying the site of vascular compromise before surgery, and it can be used to relieve mesenteric vasoconstriction with infusion of papaverine. Immediate exploratory laparotomy is the diagnostic and therapeutic modality of choice in patients who are unstable because of mesenteric ischemia.[8]

Laboratory studies are nonspecific in the diagnosis of mesenteric ischemia. There may be a leukocytosis with an increased amount of immature white blood cells. An elevated hematocrit is consistent with hemoconcentration and metabolic acidosis. There is no specific laboratory test for early diagnosis of mesenteric ischemia, and most laboratory abnormalities become elevated once the ischemia has progressed to necrosis.[9] Serum lactate was found to be 100% sensitive but not very specific because of the many other conditions that lead to elevated lactate such as shock, diabetic ketoacidosis, and renal and hepatic failure.[10] Elevated serum amylase levels exist in about half of patients with intestinal ischemia.[11,12] Lactate dehydrogenase (LDH) was shown to be 73% sensitive for bowel infarction but was not able distinguish between ischemia and infarction.[13]

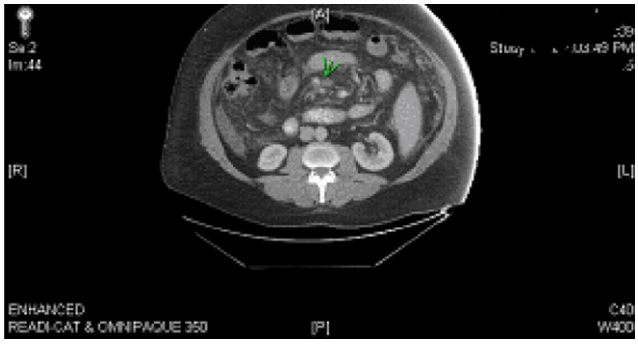

Figure 1 CT scan showing bowel wall edema and ischemia.

The goal of treatment with mesenteric ischemia is to restore intestinal blood flow as rapidly as possible. Management should be focused on hemodynamic monitoring and support, correction of underlying metabolic acidosis, initiation of broad-spectrum antibiotics, and bowel rest and decompression with placement of a nasogastric tube.[1] Mesenteric venous thrombosis should receive initial treatment with heparin anticoagulation and immediate resection of necrotic bowel.[14] It should be noted that anticoagulants should be given even in patients who have gastrointestinal bleeding, if the bleeding risk is outweighed by the risk of bowel infarction.

Case resolution

Mr. Y's postoperative course included an episode of hepatic failure, with his ammonia level rising to 90. He required respiratory support in the intensive care setting for seven days postoperatively. He also was found to have increased fever and, upon investigation, mitral valve vegetations, which required a prolonged course of antibiotic therapy. His underlying hepatic cirrhosis progressed to liver failure and the cause was believed to be nonalcoholic steatohepatitis confounded by his diabetes and metabolic syndrome. He was placed on a liver transplant list and in April of 2009 received a donor liver. He currently has recovered from his transplant and continues to improve (Figure 1).

Conclusion

This case illustrates the clinical difficulty in making the diagnosis of mesenteric ischemia and, in particular, superior mesenteric venous thromboembolism. There are no definitive signs, symptoms, laboratory, or diagnostic criteria to establish the diagnosis; rather, a constellation of data along with clinical judgment must be used for quick recognition

and treatment. Mr. Y was assessed in the emergency department and thought to have recurrence of his gastric varices and further assessed by the gastroenterologist who also did not suspect more serious disease. It was the family physician who raised clinical suspicion towing to knowledge of the patient's previous pain threshold that prompted ordering of the CT scan that made the definitive diagnosis. This patient's pain pattern was out of proportion to clinical examination based on knowledge of the patient. He did have a nonspecific pain distribution that is consistent with splenic vein thrombosis, which was later proven on CT scan. The delay in care was minimal and the patient was started on anticoagulation therapy and taken to surgery in a timely manner. Despite this treatment, he had comorbid complications of respiratory and hepatic failure. Ultimately, he did recover fully after a liver transplant; however, this illustrates the severe course that usually takes place even with timely recognition and treatment of mesenteric ischemia. Familiarity of the patient and high clinical suspicion still remain the most vital tools when diagnosing mesenteric ischemia. The family physician's knowledge of and long-term relationship with the patient certainly averted a possibly morbid outcome.

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