

Management Of Bowel Ischemia Secondary To Mesenteric Vein Thrombosis: A Tight Rope Walk

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Abstract

Acute superior mesenteric vein (SMV) and portal vein (PV) thrombosis can be a complication of hypercoagulable, inflammatory, or infectious states. It can also occur as a complication of medical or surgical intervention. Management of mesenteric and portal vein thrombosis includes both operative and non-operative approaches. Operative interventions include thrombectomy with thrombolysis; this is often employed for patients who present with signs of peritoneal irritation. Nonoperative approaches can be either noninvasive or invasive. Treatment with anticoagulation has been shown to be efficacious, though its rate of recanalization is not as high as with intravascular infusion of thrombolytics. We here describe an interesting case report of a patient who developed ischemic bowel secondary to mesenteric vein thrombosis, who did not qualify for a surgical emergency and provided an interesting challenge in medical management until surgery finally took over the specific management.

INTRODUCTION

Mesenteric venous thrombosis (MVT) is an uncommon cause of mesenteric ischemia. It was first recognized more than 100 years ago by Eliot,¹ who described intestinal gangrene resulting from mesenteric venous occlusion. In 1913, Trotter² reported that 41% of patients with mesenteric ischemia had MVT. It was Warren and Eberhardt's historic report in 1935³ that helped to establish MVT as a distinct clinical entity. They reported a mortality rate of 34%. Additionally, Warren and Eberhardt³ found that only 5% of patients who did not undergo surgical treatment survived. In the last decade, new reports have helped define this rare entity. Kazmers⁴ inferred that MVT is found in as few as one in 1000 laparotomies. In a recent report by Rhee et al,⁵ MVT comprised only 6.2% of all patients treated for mesenteric ischemia.

CASE

A 71 year old Hispanic female with past medical history of hypertension, depression, pelvic tumor resected 50 years back, presented with the complaint of umbilical and hypogastric abdominal pain for past few hours. She suddenly developed this pain which was colicky in nature, continuous, 10/10 in intensity and was associated with nausea and bilious vomiting and 2-3 episodes of non-bloody diarrhea. Apart from occasional 4/10 intensity hypogastric pain, the patient had no other complaint in the recent past.

Physical examination showed mild tenderness of umbilical and hypogastric regions with no rigidity/guarding, no rebound tenderness and normal bowel sounds. A per rectal examination was done which showed yellow stool, with streaks of fresh blood, and a strongly positive guaiac. No Murphy's, Mcburney's or CVA tenderness could be appreciated. Initial labs were unremarkable except for mild increase in WBC (15.1), and increase in Globulin component (9.9 of total protein and 3.3 of albumin).

Initial X ray was not significant for any air under the diaphragm, and CT scan of abdomen and pelvis with IV contrast showed inflammatory changes and bowel wall thickening in multiple lobes of small bowel in right lower quadrant with no involvement of terminal ileum. In addition to this a large clot of Superior mesenteric vein was noted along with a 15 x 11 x 8 cm mass in the pelvis. Ultrasound pelvis confirmed this mass as a complex cystic right ovarian lesion arising from the right ovary. Given the age and presentation, the inflammatory changes of small bowel were most suggestive of ischemic changes of bowel secondary to mesenteric vein thrombosis, which could be attributed to a hypercoagulable state contributed by the potentially malignant tumor of the ovary.

Due to lack of peritoneal signs, the patient was started on the anticoagulation. Blood loss through the GI tract secondary to ischemic bowel was a potential contraindication for

anticoagulation. The established guidelines did recommend starting anticoagulation in spite of active loss of blood from GI tract, provided the patient is losing blood at a rate that can be corrected. So the patient was started on therapeutic heparin and was kept on close watch. Over the next 24 hours the patient did lose Hemoglobin from 12.6 to 10.6. On reassessing the physical condition, the patient was unchanged from previous, with no guarding/ rigidity/ or loss of bowel sounds.

At this point of time, a decision was made to take patient to OR for exploratory laparotomy in conjunction with OBGYN for possible excision of pelvic tumor. During the surgery, 85 cm of ischemic looking small bowel was resected along with the cystic and solid right ovarian mass with smooth borders. Frozen section of the ovarian mass was negative for malignancy and so was the official pathology report which confirmed the mass to be a benign serous cystadenoma. Pathology also confirmed the resected bowel as ischemic, with viable margins and benign nature of peritoneal fluid. The patient's laparotomy was resumed for a second look showing good viability.

The patient had a good post op recovery with no incidents and tolerated PO intake and activity in due course of time. The patient was continued on therapeutic heparin to prevent the progression of thrombus and to prevent any such event again.

DISCUSSION

Mesenteric and portal vein thrombosis is rare, but early diagnosis is important. The only constant finding reported is pain out of proportion to the physical findings and a slow progression. Ultrasonography is helpful and CT should be performed promptly.

The condition is classified into acute and chronic types and the patho-physiological state depends on the speed and extent of thrombus formation. When the veins are occluded completely or rapidly, the symptoms may suddenly exacerbate. The small intestine and mesentery become congested and swollen, and sometimes there is paralytic ileus. The intestine can become ischemic and lapse into hemorrhagic infarction, causing diffuse peritonitis. In contrast, when the thrombus extends slowly and enough collateral vessels develop, the symptoms can be minimal. As in the present case, slow development of thrombus in SMV lead to minimal signs. It is deduced that the thrombus formed sub-acutely because some collateral vessels were found at diagnosis. The sub-acute thrombus may be resistant

to hemolytic therapy. Condat et al. reported that complete recanalization was achieved more frequently when thrombosis involved only the SMV than when it was more extensive⁶. The extent of recanalization may depend on where the thrombus occurs and how long it takes to form.

As for treatment, if the patient is suspected to have bowel infarction, an immediate exploratory laparotomy is needed. And, regardless of whether surgical treatment is needed or not, anticoagulation with heparin and broad-spectrum antibiotics should be begun immediately because they decrease the risk of re-thrombosis and ultimately improve survival. In contrast, Rhee and Gloviczki considered the use of thrombolytic therapy to be controversial because of the increased risk of hemorrhagic complications from an infarcted bowel⁵. However, a small amount of bleeding from ischemic colitis without infarction can be controlled by disobliteration of the bloodstream and successful treatment with thrombolytic therapy has been reported⁷. Yankes et al. described a transhepatic route for the delivery of urokinase directly into the thrombus,⁸ and Rivitz et al. reported using a transjugular-transhepatic route⁹. Poplausky et al. used the SMA as a route for administration of urokinase and showed that it is expedient for SMV thrombosis¹⁰. Thrombectomy is not recommended because of the surgical difficulty and the risks of re-thrombosis. In cases without peritonitis or ongoing bowel necrosis, thrombolytic therapy with anticoagulation can be tried and the approach through SMA is considered the best at this point in time. In the present case, thrombolytic therapy did not open the main trunk, but permitted lysis of the peripheral thrombus and helped to form enough collateral veins. In this case, surgery had to be performed because of the sudden drop in clinical condition of the patient but anticoagulation did help to decrease the extent of the surgery.

Various etiologies of mesenteric and portal vein thrombosis have been reported, including inflammatory states (appendicitis, diverticulitis, pancreatitis and inflammatory bowel disease etc.), malignancy, trauma, portal hypertension, post splenectomy, mechanical compression such as volvulus or pregnancy and oral contraceptive use. As hematological hypercoagulable states, protein C, protein S or antithrombin III deficiency and antiphospholipid antibody syndrome are well known. Recently, several new risk factors associated with venous thrombosis have been investigated, including activated protein C (APC) resistance associated with G1691A, Factor V gene mutation (Factor V Leiden), G20210A prothrombin gene mutation,

hyperhomocysteinemia, a part of which is associated with C677T methylene-tetrahydrofolate reductase (MTHFR) gene mutation, dysfibrinogenemia, elevation of the Factor VIII level and so on¹¹⁻¹³. The current concept is that venous thrombosis results from the concurrence of several factors, such as a genetic disorder and an acquired thrombogenic stimulus and there are racial differences. Ho et al. mentioned that APC resistance resulting from Factor V Leiden, prothrombin G20210A mutation and elevated Factor VIII, which are pointed out as main etiologies of venous thrombus in Caucasian populations, may be rare in the Chinese population¹⁴.

In Short, MVT should be suspected in patients presented with abdominal pains and who have the history of previous abdominal surgeries, hyper-coagulable disorders or deep venous thrombosis, and these patients should undergo contrast enhanced CT scan of the abdomen with close attention to the mesenteric vein phase. Early anticoagulation with heparin followed by Coumadin is usually recommended to limit thrombosis and prevent the recurrence. Properly treated patients with MVT should have a good prognosis.

References

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