

Endovascular management of bowel ischemia secondary to portal and mesenteric vein thrombosis

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Portal and mesenteric venous thrombosis (PMVT) is an uncommon disease, but it is a clinically important case of acute mesenteric ischemia. Its diagnosis is often delayed because of nonspecific abdominal symptoms. The treatment of PMVT involves anticoagulation therapy, alone or in combination with surgery. The addition of thrombolytic therapy to the treatment of PVMT may enhance the clearance of thrombus and hasten the clinical improvement. We herein present a case of bowel ischemia due to PVMT that was successfully treated with catheter-directed infusion of urokinase through the superior mesenteric artery and systemic anticoagulation in a patient with ascites and high blood international normalized ratio. We believe that this case demonstrates that slow and steady treatment with transarterial thrombolysis over 13 days can achieve the desired results compared with more aggressive transhepatic or transjugular thrombectomy or surgical thrombectomy, especially in high-risk patients with ascites and high international normalized ratio.

Keywords:

bowel ischemia, intra-arterial thrombolysis, portal and mesenteric vein thrombosis, urokinase

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Introduction

Portal and mesenteric venous thrombosis (PMVT) is an uncommon disease, but it is clinically important because it accounts for 1.5–6.2% cases of acute mesenteric ischemia [1]. Its diagnosis is often delayed because of nonspecific abdominal symptoms; in addition, when this occurs in young individuals without any predisposing factor, the diagnosis may become even more difficult. However, thrombosis can be easily identified by ultrasound (US) or computed tomography (CT). We herein present a case of bowel ischemia due to PVMT, which was successfully treated with catheter-directed infusion of urokinase through the superior mesenteric artery and systemic anticoagulation in a high-risk patient with ascites and high blood international normalized ratio (INR). His clinical improvement suggests that we should adopt slow and steady treatment with transarterial thrombolysis over 13 days than more aggressive transhepatic or transjugular thrombectomy or surgical thrombectomy, especially in high-risk patients with ascites and high INR.

Case history

A 50-year-old male patient presented with severe acute abdominal pain, vomiting, and melena. He had a known case of left-sided hemiparesis with hypertension and had been on irregular treatment since the past 10 years. On examination, his abdomen

was soft with epigastric tenderness and diminished bowel sounds. Biochemical investigations showed low hemoglobin (8 g%), elevated total leukocyte count ($14000/\text{cm}^3$), hypokalemia (3 mEq/l), and elevated prothrombin time-international normalized ratio (PT-INR) (2.14). His symptoms were getting worse. Contrast-enhanced computed tomography (CECT) of the abdomen showed portal and mesenteric vein thrombosis with congestive thickening of the small bowel loops (Figs. 1 and 2). The dehydration was

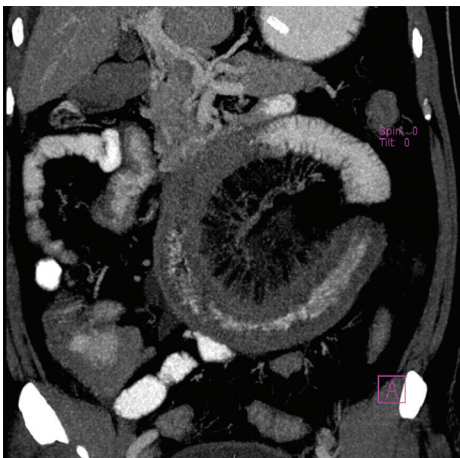
Figure 1



CECT of the abdomen portal venous phase (coronal reconstruction) showing nonenhancing thrombus in the superior mesenteric vein and proximal portal vein with perihepatic ascites. CECT, contrast-enhanced computed tomography.

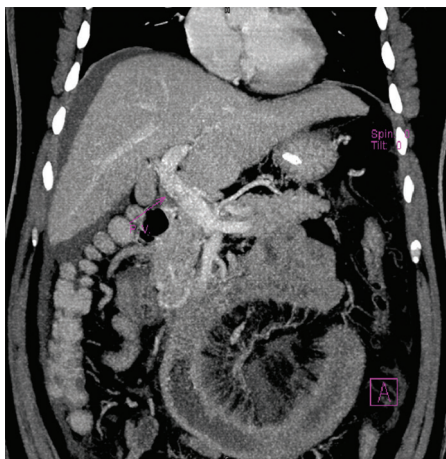
corrected by fluid therapy, whereas blood loss and low hemoglobin were corrected with blood transfusion. Systemic anticoagulation therapy was started, because thrombosis had formed recently, but symptoms continued to worsen. Abdominal digital subtraction angiography was performed through transfemoral arterial access, which showed engorgement of the blood vessels with sluggish flow (Fig. 3). Thrombolysis was performed with a 5 Fr catheter, parked at the orifice of the superior mesenteric artery, through which urokinase and heparin were given as continuous infusion over a period of 3 days with repeated check angiograms (12–24 h) and clinical assessment (a total 60 lakh units of urokinase and 60 000 heparin). After 3 days of infusion, there was significant resolution of PMVT and bowel wall congestive changes (Fig. 4)

Figure 2



CECT of the abdomen portal venous phase (coronal reconstruction) showing diffuse severe concentric mural thickening of the jejunal loops. CECT, contrast-enhanced computed tomography.

Figure 4



CECT of the abdomen portal venous phase (coronal reconstruction) showing significant resolution of the superior mesenteric vein (SMV) and portal vein thrombus with jejunal loops and congestive edema. CECT, contrast-enhanced computed tomography.

and significant clinical improvement in terms of pain, vomiting, and melena. He was discharged after 10 days of hospital stay with shift of anticoagulant therapy to warfarin with targeted PT-INR of 1.5–2.0. On follow-up after 1 month, he was found to be doing well with total resolution of bowel wall congestive changes and near total resolution of PMVT (Fig. 5).

Discussion

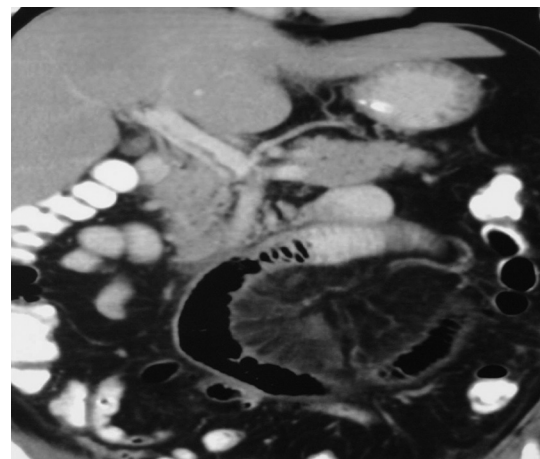
PMVT has been assumed to be an uncommon disease. The incidence of MVT was estimated at 1.8–2.7 per 100 000 person-years [2]. Etiologies of PMVT, when identified, can usually be separated into local intra-abdominal factors and inherited or acquired hypercoagulable states, but most of the time the cause

Figure 3



DSA superior mesenteric injection showing congestion and sluggish inflow. DSA, digital subtraction angiography.

Figure 5



CECT of the abdomen portal venous phase (coronal reconstruction) showing complete resolution of the jejunal loops, congestive edema and near total resolution of the portal and SMV thrombus. CECT, contrast-enhanced computed tomography.

is idiopathic. It presents with nonspecific signs and symptoms, the diagnosis of which requires a high index of suspicion. However, the thrombus can be easily identified with US and/ CECT of the abdomen [3], like in our case where CECT of the abdomen facilitated early and accurate detection and extent of thrombosis and bowel wall ischemic changes, thereby necessitating immediate treatment.

The best management strategy for PMVT is its early diagnosis and immediate treatment [4].

Traditionally, management has been surgical, with assessment of the small bowel for necrosis and/ or resection, followed by anticoagulation [5]. Anticoagulant therapy is recommended in patients with acute MVT to prevent clot formation without increasing the risk of bleeding [6], like in our case. However, when intestinal infarction has not yet occurred, endovascular thrombolysis/thrombectomy is an option, as in our case where early diagnosis was made before bowel infarction started.

The available evidence suggests that treatment should be considered in patients in whom a diagnosis of acute PVT is made. A lot of controversy surrounds the role of anticoagulation in patients with subacute and chronic PMVT [7]. Studies have shown less than 30% usage in chronic PVT and often intermittently. In a recent retrospective analysis of 28 patients who received thrombolysis for acute PMVT, recanalization and rapid improvement in clinical parameters was seen in the 10 patients who received treatment within 14 days of first symptoms. The effectiveness of thrombolysis was significantly reduced in those treated after this period [8]. An INR of equal to or less than 1.5 is considered safe and INR of more than 1.5 is considered relative contraindication for arterial procedures [9]. On extensive searching using Pubmed and Google for words like portal vein thrombolysis or anticoagulation in patients with high INR in varying combination, we could not find any definite reference.

Thrombolytic agents can be introduced either through the superior mesenteric artery catheter or through the transhepatic transportal retrograde approach, whereas mechanical clot lysis can only be achieved through a

retrograde transhepatic venous approach [10]. Probably the safest way to directly access the portal circulation is through the transjugular-transhepatic approach. The direct transhepatic approach can also be adopted but is associated with greater risk of hemorrhage. Because of the high INR and presence of ascites in our case, the transarterial route was preferred over transhepatic with successful recanalization of the superior mesenteric and portal vein with resolution of bowel wall ischemic changes.

Conclusion

PMVT is one of the important and uncommon causes of bowel ischemia and can be easily identified by both US and CT scans. Immediate treatment with anticoagulants and endovascular thrombolytics can improve both the prognosis and the quality of life of the patient.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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