

EDİTÖRE MEKTUP /LETTER TO THE EDITOR

Superior mesenteric venous thrombosis combined with portal and splenic thrombosis in an elderly patient successfully treated with anticoagulation alone

Antikoagulasyonla başarılı bir şekilde tedavi edilen portal ve splenik trombozla kombine superior mezenterik ven trombozu

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Dear Editor,

It is difficult to diagnose mesenteric vein thrombosis early and severe ischemia can develop. Diagnosis of a chronic portal/mesenteric vein thrombosis based on clinical findings is often more challenging than acute form¹. Patients may be asymptomatic or may present with unspecific abdominal pain. We present a widespread mesenteric venous thrombosis combined with splenic and portal vein thrombosis treated with anticoagulation in a 72-year-old man.

A 72-year-old male patient presented to a peripheral hospital with complaints of gradually increasing abdominal pain. His abdominal pain was bluntskilled and spreading throughout the abdomen and was not related to nutrition. Vomiting, diarrhea and constipation were also not accompanied to abdominal pain. Upper endoscopy and colonoscopy performed due to his complaint revealed no abnormality. The patient was then transferred to our center without any diagnosis. On his physical examination, there was mild abdominal tenderness and muscular defense. Laboratory tests revealed no abnormality except for lactate dehydrogenase: 385 u/l, fibrinogen: 623 mg/dl. Doppler ultrasound revealed that superior mesenteric vein was dilated with no flow in the lumen, and that portal vein showed cavernous transformation, and increasing echogenicity secondarily to inflammation was present in mesenteric fatty tissue. CT angiography showed partial thickening of the

small intestinal walls secondarily to widespread thrombosis of portal, superior mesenteric and the proximal splenic vein and recanalization with luminal filling in the portal vein (Figure 1a-d).

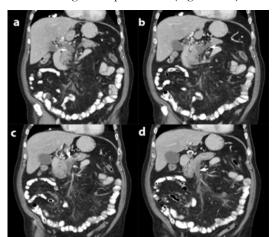


Figure 1 (a-d). A coronal CT angiography showing complete thrombosis of portal vein (a), the proximal splenic vein (b), portomesenteric confluence (c) and superior mesenteric vein (c-d) (white arrows).

Because intestinal infarction and peritonitis were not developed, angiographic and surgical interventions were not attempted. The patient was treated with low molecular weight heparin (enoxaparine, 1 mg/kg) during the admission and chronic anticoagulation therapy (warfarin 2.5 mg/d, INR 2-3) after discharge. The patient's symptoms and

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clinical condition ameliorated progressively after initiation of anticoagulant treatment. In the genetic and biochemical analyzes conducted to uncover the etiology, Factor 2 prothrombin G20210A and MTHFR C677y were found as homozygous. The protein C and S levels were also lower than normal. Patient's symptoms decreased completely during 20 days of hospitalization, and he was discharged without intervention. A Doppler ultrasound six months later showed an improved portal flow with complete re-canalization and without ascites. At his 1-year follow-up visits, the patient was still asymptomatic.

Superior mesenteric venous thrombosis combined with portal and splenic venous thrombosis is much rarer than superior mesenteric vein thrombosis, but can present nonspecific signs and severe symptoms, which can be potentially destructive². Delayed in the diagnosis and treatment of thrombosis because of the nonspecific symptoms can often have negative impact on prognosis. The only strong finding is pain which is disproportionate to physical findings³. In the event of suspicious findings, computed tomography scan should be performed immediately.

Exploratory laparotomy is usually required in patients with signs of peritonitis, transmural bowel infarction, or hemodynamical instability⁴. If complete occlusion of the veins develops acutely,

the symptoms may arise suddenly and congestion in the small intestine and mesenterium develops as in this case. Prothrombin and MTHFR gene mutation and decreased protein C and S activity were determined to be the risk factors in this patient. In patients with no known risk factors for thrombosis, hematological and genetic researches for primary hypercoagulable states should be required.

In conclusion, early diagnosis of widespread thrombosis and urgent anticoagulation should be pre-emptively initiated irrespective of whichever surgical intervention is chosen.

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