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Case Report: Splenic vein thrombosis

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Abstract
Splenic vein thrombosis is a blood clot that is obstructing the splenic vein, which is located on the surface of the spleen. Reduced portal blood flow, a hypercoagulable state or vascular endothelial injury, abdominal trauma and after abdominal surgery can lead to splenic vein thrombosis. This study discusses about a 46 year old obese female patient who was presented in the general medicine department with abdominal pain after hysterectomy. CT abdomen shows the presence of fatty liver, small renal calculus and focal partial thrombosis of splenic vein which was diagnosed as splenic vein thrombosis. The study deals with the medical management of splenic vein thrombosis.

Keywords: splenic vein thrombosis, hysterectomy, fatty liver

1. Introduction
Portal vein thrombosis (PVT) refers to thrombosis that develops in the trunk of the portal vein [1]. The portal vein forms at the junction of the splenic vein and the superior mesenteric vein behind the pancreatic head [2]. The pathophysiology of portal vein thrombosis encompasses one or more features of Virchow's triad, viz., reduced portal blood flow, a hypercoagulable state or vascular endothelial injury.
In acute PVT there is a sudden formation of thrombosis within the portal vein that leads to a complete or partial obstruction of the portal vein. Acute PVT in non-cirrhotic non-malignant PVT usually presents with abdominal pain (91%), fever (53%), and ascites (38%) [1]. Sometimes other symptoms of splenomegaly such as thrombocytopenia or pancytopenia and abdominal pain are also manifestations of the disease [3]. We present a rare case of splenic vein thrombosis after hysterectomy.

2. Case report
A 46 year old female after hysterectomy referred to medicine department with pain in the left hypochondrium for past three days. She did not have nausea or vomiting. She had mild tenderness over left hypochondrium. She had a past history of diabetes mellitus and hypertension and was taking T. Metformin 500+Glimepiride 2 mg BD, T. Teneligliptin 20 mg OD and T. Telmisaratan 50 mg OD respectievely. Patient was febrile with stable vital signs. Coagulation profile was normal. Patient blood sugar level found to be elevated 230 mg/dl. CT abdomen reports showed fatty liver, small right renal calculus, post hysterectomy status and focal partial thrombosis of splenic vein. The patient was treated with inj. LMWH 40 mg s/c BD, T. Tramadol 50 mg po TDS, Inj Metronidazole 500 mg iv TDS, Inj. ceftriaxone 1 gm iv BD, T. Paracetamol 500 mg po BD, Inj. Pantoprazole 40 mg iv BD and symptoms reduced within 3 days. Elevated blood sugar levels were managed with Inj. Monotard 6 units and Inj. Actrapid 4 units based on the blood glucose level. The patient was prescribed with T. Acenocoumarol 2 mg po BD and discharged with a good general condition.
Complete or extensive reperfusion can be achieved with early treatment with anticoagulant agents. In conclusion, portal vein thrombosis should be suspected in puerperium period in a patient with abdominal pain and fever to avoid dangerous progression to life-threatening conditions like mesenteric infarction.

3. Discussion
Metin Basaranoglu, demonstrated that the prevalence of nonalcoholic steatohepatitis (NASH) is 3% and 20% in non-obese and obese subjects, respectively.
Mortality in the past was 20%–50% with acute portal vein thrombosis and other splanchnic vessels, but with an early diagnosis, increased clinical awareness, improved diagnostic techniques and use of early anticoagulation the 5 yr survival rate has improved to 85% [4].
Sonia M Najjar, demonstrated that obesity is associated with low-grade chronic inflammation and is strongly associated with chronic macrophage accumulation to the hypertrophied adipose tissue. Adipose tissue macrophages produce pro inflammatory cytokines such as tumor necrosis factor-α, interleukin-6, and C-reactive protein. These cytokines alter insulin signalling by protein kinase C theta, inhibitor κB kinase β, suppressors of cytokine signalling and inducible nitric oxide synthase to contribute to insulin resistance. Similarly, increased fat accumulation in liver alters its inflammatory milieu, thus modifying insulin action. Thus, increased systemic inflammation and increased procoagulant factor levels associated with insulin resistance could explain the higher prevalence of portal vein thrombosis in NAFLD [5].
Elham Naghshineh, documented a case on Portal vein and superior mesenteric vein thrombosis after caesarean hysterectomy which suggests that previous abdominal surgery favors development of PVT in 2/3rd of cases.
Elham Naghshineh, demonstrated. a recent systematic review on the management of acute non-malignant non-cirrhotic PVT demonstrated the variability in location and extent of the thrombus, methods in initiating anticoagulation whether intravenous, subcutaneous or oral.. There is a clear recommendation for the use of anticoagulation in non-

cirrhotic acute PVT with good safety and efficacy data [6].
Yogesh K. Chawla, documented that the aim of the treatment is to reverse or prevent advancement of thrombosis in the portal venous system and to treat complications of established PVT. Low molecular weight heparin has been shown to prevent portal vein thrombosis and liver decompensation in patients with advanced cirrhosis in a recent randomized controlled trial [7].

4. Conclusion

This case report serves as a reminder that a common presenting symptom such as abdominal pain in a young, healthy adult can be a manifestation of a rare diagnosis such as Splenic vein thrombosis. Thrombosis of the portal vein can occur due extra hepatic causes such as portal vein thrombosis. Extrinsic obstructions and inherited hypercoagulable states have been ruled out in our patient. The aim of the treatment is to reverse or prevent advancement of thrombosis in the portal venous system and to treat complications of established PVT. Complete or extensive reperfusion can be achieved with early treatment with anticoagulant agent

5. References

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