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PORTAL VEIN THROMBOSIS – A STUDY OF RISK FACTORS, CLINICAL PROFILE, COMPLICATIONS AND MANAGEMENT

General Medicine

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ABSTRACT

Background: The portal vein is formed by the confluence of the splenic and superior mesenteric veins. Occlusion of the portal vein by thrombus (portal vein thrombosis [PVT]) typically occurs in patients with cirrhosis and/or prothrombotic disorders/ pancreatitis. Patients with acute PVT have the sudden onset of portal venous occlusion due to thrombus. Patients with acute PVT have not yet developed features of chronic PVT, such as collateral circulation (e.g., cavernous portal transformation) or portal hypertension. Chronic PVT develops when acute doesn't resolve with formation of collaterals. In patients with suspected acute PVT, we typically obtain a contrast-enhanced abdominal computed tomography (CECT) to confirm the diagnosis, evaluate for predisposing conditions (eg, intra-abdominal infection), assess the extent of the thrombosis and the anatomy of collaterals, and detect evidence of intestinal infarction. Doppler and MRI are other alternative imaging modalities. The primary management of acute portal vein thrombosis (PVT) is anticoagulation and, when possible, treatment of predisposing condition. The goal of anticoagulation is to prevent extension of the clot and to allow for recanalization, so that intestinal infarction and portal hypertension do not develop. Unlike chronic PVT, where the role of anticoagulation in patients with cirrhosis is unclear, studies suggest anticoagulation for acute PVT is beneficial for patients with cirrhosis. However, because patients with cirrhosis may have esophageal varices, we typically screen for varices prior to initiating anticoagulation. **Methods:** 60 cases of extrahepatic portal vein thrombosis or intrahepatic portal vein thrombosis were included. All registered diagnoses were based on either ultrasound with Doppler, CT-angiography or MRI. **Results:** In our study, Risk factors were established in 24 cases (80%). When including all risk factors, 16 cases (53.3%) had local risk factor, and 8 cases (26.6%) had a systemic risk factor. Anatomical location was in 26 cases (86%)extrahepatic, (14%) intrahepatic. In addition, patients with extrahepatic PVT also had intrahepatic thrombosis (38%) and/or in the splenic vein (23%). 66% had oesophageal varices, 53% gastric varices, 46% portal hypertensive gastropathy, 26% variceal haemorrhage, and 40% ascites. **Conclusion:** Most patients had a combination of local and systemic risk factors for PVT. Partial/ complete recanalization is more in patients treated with anticoagulation therapy, and that regression of varices was more in patients who were treated with active endoscopy combined with beta blockers.

KEYWORDS

Portal Vein Thrombosis, Venous Doppler, Anticoagulantion

INTRODUCTION

Over the last years, portal vein thrombosis (PVT) is increasingly frequently being diagnosed by wide use of ultrasound-Doppler equipment. Recently, the lifetime risk of getting PVT in the general population is reported to be 1% [1]. Risk factors and complications to PVT have become better defined although most of the information derives from patients with cirrhosis [2]. Based on some case series, anticoagulation may be associated with recanalization of both acute [3] and chronic thrombosis [4]. Band ligation remains central in treating acute variceal bleeding.

Secondary prophylaxis of rebleeding with ligation is effective; however, there are insufficient data on β-blockade alone or in combination with endoscopy [5]. PVT seems most often to develop in the presence of both systemic and local risk factors [6], but the relative importance of these factors for the course and treatment strategy of the condition remains unclear. Thus, there are several unresolved issues regarding PVT that due to the rarity of the condition cannot be clarified by large trials. Therefore, the aim of this study was to describe risk factors, clinical profile, complications, treatment and in patients with PVT in a tertiary hospital.

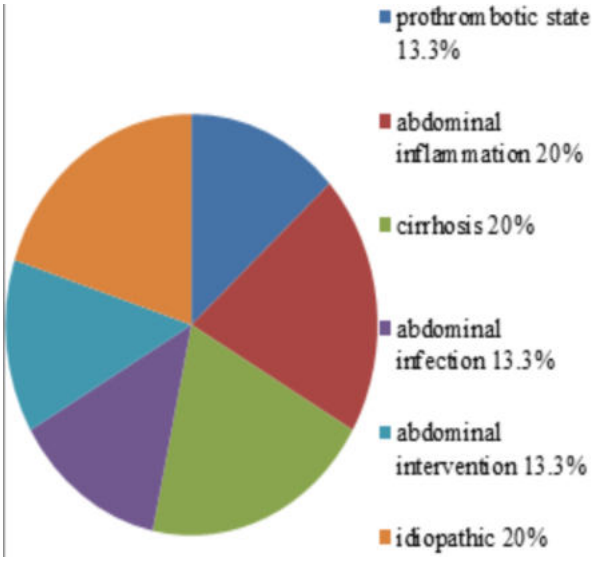
MATERIALS AND METHODS:

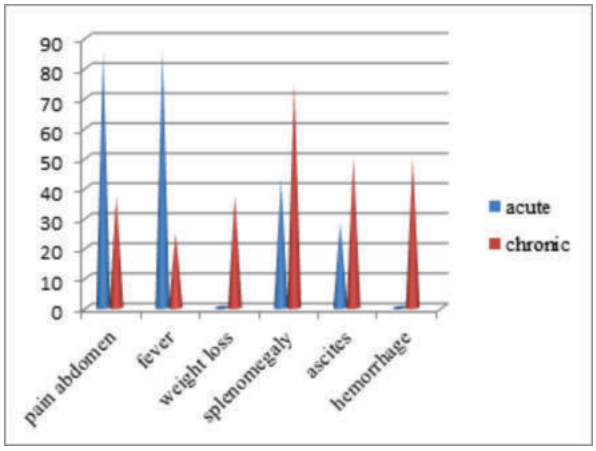
60 cases from January 2017 to august 2022 of extrahepatic portal vein thrombosis or intrahepatic portal vein thrombosis were included. All registered diagnoses were based on either ultrasound with Doppler, CT-angiography or MRI. The following data were used from the clinical records:

- Sex, Age,
- Risk factors (prothrombotic conditions, cirrhosis, cancer,
- abdominal inflammation, infection, surgical abdominal intervention, none),
- Clinical presentation (abdominal pain, loss of appetite, fever, splenomegaly, ascites, haemorrhage)
- Presentation of PVT (acute< 60 days or chronic > 60 days),
- Complications (oesophageal and gastric varices, variceal haemorrhage, portal hypertensive gastropathy, ascites),
- Coagulation disorders,
- Imaging methods (Doppler ultrasound, CT angiography, MRI, and endoscopy),
- Treatment (anticoagulation, sclerotherapy, banding, β-blockade)

RESULTS:

- Mean age at time of admission was 37.8 years (range 27–56). The group included 36 women and 24 men.
- 28 had acute PVT and 32 had chronic PVT.
- There was no difference in age between genders.
- Risk factors – Risk factors were established in 48 cases (80%). Which including all risk factors, 32 cases (53.3%) had a local risk factor, and 16 cases (26.6%) had a systemic risk factor.





IMAGING METHODS:

- In 44 patients, diagnosis was established by means of Doppler ultrasound. Of 16 patients with a negative ultrasound, all were diagnosed by CT angiography.
- Sensitivity was calculated to be 73.3% (44/60) for Doppler ultrasound.
- Anatomical location was in 52 cases (86%) extrahepatic, and in 8 cases (14%) intrahepatic. In addition, patients with extrahepatic PVT also had intrahepatic thrombosis (38%) and/or in the splenic vein (23%).

COMPLICATIONS:

- 36 patients had one or more complications.
- 66% had oesophageal varices, 53% gastric varices, 46% portal hypertensive gastropathy, 26% variceal haemorrhage, and 40% ascites. Oesophageal varices and bleeding were more frequent in patients with chronic PVT.

TREATMENT:

- 36 (60%) patients received anticoagulation therapy (vitamin K-antagonist, low molecular weight heparin or heparin).
- Sixty six percent (20 acute and 4 chronic) of the patients who received anticoagulation therapy improved their portal flow with partial or complete recanalization.
- Patients with oesophageal varices were treated endoscopically with band ligation (VBL) and/or sclerotherapy, in 40% as primary prophylaxis.

DISCUSSION:

- The main limitation of this study is lack of generalizability due to its retrospective nature.
- In accordance with previously published studies, we found that PVT developed because of several risk factors [7,8].
- In the restricted analysis, we found a combination of local (54%) and systemic risk factors (26%).
- Denninger et al. [9] reported a higher incidence of systemic factors (72%).
- The typical presentation of acute PVT was abdominal pain, splenomegaly, fever and ascites, while the presentation of chronic PVT was abdominal pain and splenomegaly together with gastrointestinal haemorrhages and ascites.
- Frequent complications during follow-up were oesophageal and gastric varices, portal hypertensive gastropathy, bleeding from varices and ascites.
- For primary prophylaxis, β-blockade is standard for cirrhotic portal hypertension, but the effect is not documented in PVT, and VBL may be preferable.
- In the acute management of variceal bleeding, vasoactive substances, antibiotics, and VBL remain central. For secondary prophylaxis of re-bleeding we used combined VBL and β-blockade

CONCLUSION:

Most patients had a combination of local and systemic risk factors for PVT. Partial/ complete recanalization is more in patients treated with anticoagulation therapy, and that regression of varices was more in patients who were treated with active endoscopy combined with beta blockers.

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