

# Portal Vein Thrombosis

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## Abstract

Portal vein thrombosis is a form of venous thrombosis affecting the portal vein which can lead to portal hypertension and reduction in the blood supply to liver. Portal vein thrombosis in its acute stage goes unrecognized, symptoms resolve as collateral channels become established and the end result may be portal hypertension. Procoagulant states are established risk factors for portal vein thrombosis. However, intra-abdominal infection is the most common cause of acute portal vein thrombosis and in adults this is often diverticulitis causing portal pyaemia.

## Introduction

PVT was first reported in 1868 by Balfour and Stewart. It is an important condition because of its serious long term complications. Difficulty in clinical diagnosis occurs because of the non specific nature of the signs and symptoms.<sup>1,2</sup> It being recognised with increasing frequency with the use of ultrasonography. However colour Doppler can accurately diagnose fresh thrombus. CT abdomen may be complementary in the diagnosis.

## Case Report

Fifty five year old post menopausal female patient presented with haematemesis, bleeding per rectum since nine months. Patient was admitted twice in the past and each time two pints packed cells were transfused. Recently she complained of abdominal distension and oedema feet. On examination patient was pale and had icteric tinge. On per abdomen examination liver and spleen were palpable. On investigations there was pancytopenia and indirect hyperbilirubinaemia. Ultrasonography of the abdomen revealed chronic liver disease, portal hypertension and thrombosis of the right portal vein branch (Figs. 1 and 2).

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## Discussion

In PVT there is rapid compensatory increase in the flow through hepatic artery and liver function tests remain unaffected. Symptoms also resolve because of development of collateral channels. Average time for formation of collateral channels is five weeks though as early as 12 days has also been described in literature.

*Incidence* - PVT is relatively a rare condition with an overall incidence of 0.05% to 0.5% in autopsy studies. In cirrhosis the incidence has been reported to vary from 5%-18%. In India extra hepatic PVT has been reported to be more frequent.

*Causes* - In children and neonates the most common aetiology is intra abdominal infection accounting for 50% of all cases in this age group. Appendicitis is a commonly reported risk factor in children with PVT.

Congenital anomalies of portal venous system often associated with cardiovascular anomalies and biliary tract abnormalities have been reported in 20% of children with PVT.

*In adults* - Cirrhosis of liver is the major aetiology accounting for 24-32% of PVT.

Neoplasm is another major cause,



Fig. 1 : Portal vein thrombosis on ultrasonography.

accounting for 21-24% with hepatocellular carcinoma and pancreatic carcinoma causing most of these cases.

Other factors like intra abdominal sepsis and myeloproliferative disorders may be responsible for PVT. However 8-15% of cases have been reported to be idiopathic. Procoagulant states are established risk factors for PVT.

*Clinical presentation* - Acute PVT presents as right hypochondriac pain and/or fever. Other symptoms are increasing ascites, intestinal ischaemia and its associated symptoms and worsening symptoms of PHT.

Chronic PVT patients have signs and symptoms of PHT. PVT is the cause of PHT in 7.8% of cases.<sup>3</sup> In 90% of patients oesophageal bleed is the presentation. In malignancy incidence of bleeding is less. The harsh reality is that they do not survive long enough to develop the sequelae of PHT. On rare occasions, patients with PVT will present with fever of unknown origin.<sup>4</sup>

*Outcome* - In the absence of cirrhosis, the 2 year bleeding risk from oesophageal varices is reported to be 0.25% and of those who bleed the mortality rate is approximately 5%. With



Fig. 2 : Portal vein thrombosis on colour doppler study.

cirrhosis and varices 2 year bleeding risk is 20-30% and a mortality rate of 30-70%.

In adults the 10 year survival rate is 38-60% with most of the death occurring secondary to underlying disease. In children with PVT the prognosis is better with 10 year survival rate greater than 70%.

*Treatment* - when detected early, anticoagulation is recommended. Low molecular weight heparin is started and APTT is achieved between 1.5 and 3.0. This is followed by oral warfarin to achieve INR 2-3. Warfarin is continued for three months. Sometimes it's continued as indicated by their underlying condition. Anticoagulation does not increase the risk of bleeding but reduces mesenteric infarction.<sup>5</sup> Recanalisation after oral anticoagulation is frequently observed.<sup>6</sup>

Transhepatic angioplasty, thrombolysis and/or portosystemic shunt and distal splenorenal shunts are also used. In the face of hepatic dysfunction, shunts increase the incidence of hepatic encephalopathy.<sup>7</sup> Liver transplantation may be necessary in extreme cases.

## Conclusions

In any patient with unexplained acute abdominal symptoms specially in those with signs of sepsis or abnormal liver function test, the possibility of acute portal vein thrombosis should be considered and investigated. Patients should be treated with anticoagulant therapy although long term benefits of the therapy remain to be ascertained. Outcome of portal vein thrombosis is generally good and mortality primarily is associated with consequences of portal hypertension.

## Referances

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### IMPROVING FUNCTION: A NEW TREATMENT ERA FOR MULTIPLE SCLEROSIS?

Robust studies of symptomatic treatments are rare in multiple sclerosis, and so it is encouraging to read the randomized evaluation, by Andrew Goodman and colleagues, of oral fampridine in ambulation and leg strength in *The Lancet* today.

However, although the data presented in today's study show a clinically relevant improvement in function induced by fampridine, it is not easy to extrapolate these findings to daily practice, where a definition of responder on the basis of repeated standardized assessments of a timed walk is unlikely to be feasible.

The results with fampridine in multiple sclerosis are intriguing, both because they show improvement in ambulation and because the patient's perspective on walking is used to validate the primary outcome.

**Alan Thompson, Chris Polman, *The Lancet*, 2009; 373 : 697-98.**