

Case Report

Acute presentation of ascites in association with colon cancer

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Portal vein thrombosis is a relatively rare condition in western countries. In adults it is often related to cirrhosis or hepatocellular carcinoma and does not usually have any acute manifestations other than complications of portal hypertension. We report a patient with portal vein thrombosis presenting as acute ascites associated with a colonic carcinoma.

CASE HISTORY An 80 year old man presented with gross ascites and altered bowel habit. Investigations, including a barium enema and Duplex Doppler ultrasound, revealed a colonic carcinoma and a portal vein thrombosis (figure). The ascites was a transudate and did not contain any malignant cells. The ascites responded satisfactorily to treatment with spironolactone and he then underwent a left hemicolectomy. The ascites recurred a few days after his surgery, and this had to be aspirated. He made a good recovery and was discharged to continue treatment with spironolactone.



Duplex Doppler showing the walls of the portal vein (white lines within the boxed area) with no blood flow within it.

DISCUSSION

The acute presentation of ascites in association with colonic carcinoma could easily have been attributed to metastases. However, it was in fact probably secondary to the portal vein thrombosis. Portal vein thrombosis is a rare condition which affects both children and adults. The overall incidence at autopsy ranges between 0.05% and 0.5%.¹ It does not usually have any acute manifestations, but patients often present with complications of portal hypertension, notably haemorrhage from varices. When it does present acutely, it is usually with sudden onset of ascites,² which tends to resolve spontaneously when a collateral circulation develops. In this case, radiological investigation did not show any collateral circulation.

The commonest causes of portal vein thrombosis are cirrhosis, infection, intra-abdominal inflammation (pancreatitis, appendicitis, cholecystitis, diverticulitis), trauma (including surgery), neoplastic disease (notably hepatocellular and pancreatic carcinoma), myeloproliferative disorders and inherited and acquired hypercoagulable states. Idiopathic cases also occur.¹ In the case presented the first six causes were ruled out by clinical examination, investigation and laparotomy. It was therefore suspected that the underlying factor could have

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been a hypercoagulable state. The patient had no history of thrombosis in the past and was therefore more likely to have an acquired condition. The association of malignancy with hypercoagulable states and thrombosis has long been recognised, and these have been reported in as many as sixty percent of cancer patients.³ Possible contributory causes for thromboembolic disease in cancer include the capacity of tumour cells and their products to interact with platelets, clotting and fibrinolytic systems, as well as their interactions with endothelial cells and tumour-associated macrophages.⁴

If portal vein thrombosis is suspected clinically, ultrasound scanning (preferably with colour Doppler) is the radiological investigation of choice. If this is unhelpful, the next step is to proceed to MRI or CT scanning with intravenous contrast enhancement. If these non-invasive tests are inadequate then portal angiography should be carried out.

Management is often centred around treatment of complications, in this case the treatment of ascites by diuretics. The role of anticoagulation in patients with portal vein thrombosis has not yet been established. There is little evidence that it is of any benefit¹ and it increases the risk of bleeding if varices are present.

This case emphasises the importance of routine ultrasound in the presence of ascites. It should not be presumed that ascites in the presence of a neoplasm is secondary to metastatic disease, as dual pathologies can often coexist.

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