Portal vein thrombosis due to *Candida albicans* associated with hepatic cirrhosis

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**Summary:** A case of portal vein thrombosis due to *Candida albicans* in a patient with alcoholic hepatic cirrhosis in the absence of hepatocarcinoma is described. Infection is a known cause of portal vein thrombosis but thrombosis by *Candida albicans* has not to our knowledge been previously reported.

**Introduction**

Portal vein thrombosis (PVT) is an uncommon complication of hepatic cirrhosis, with an incidence of 0.6%.\(^1\) The most frequent aetiology is invasion from an associated hepatocellular carcinoma.\(^2\) We present a patient with alcoholic hepatic cirrhosis who developed a portal vein thrombosis due to *Candida albicans*. Though infection is a known cause of PVT, it is rare and portal vein thrombosis by *C. albicans* has not to our knowledge been previously reported.

**References**

Case report

A 51 year old male was known to have hepatic cirrhosis with histological confirmation and chronic pancreatitis associated with endocrine and exocrine insufficiency of alcoholic aetiology for 6 years. He presented with epigastric pain radiating to the back, which had appeared one month earlier, together with increased abdominal circumference and oliguria. He also had occasional fever up to 38.5°C. Examination disclosed profound wasting, ascites without peritoneal signs with hard, painless hepatomegaly and mild ankle oedema.

Investigations: haemoglobin 9.8 g/dl, white cell count 14.82 x 10^9/l with normal differential, ESR 110 mm/hour. Serum iron 35 μg/dl (normal 40–160), transferrin saturation 18% (normal 20–55). Serum biochemistry showed glucose 17.3 mmol/l, total proteins 69 g/l, gamma-glutamyl transpeptidase 869 U/l (normal: 639–1,349), alkaline phosphatase 2,419 U/l (98–280), IgG 2,090 mg/dl (639–1,349), and IgA 639 mg/dl (70–312).

Serological markers for hepatitis B and C virus were negative, as were alpha-fetoprotein, antimitochondrial and anti-smooth muscle antibody titres.

Analysis of ascitic fluid showed total protein of 9 g/l, a cell count of 0.156 x 10^9/l mainly polymorphonuclear. Ascites cultures for aerobic and anaerobic bacteria and fungi were negative. *Streptococcus milleri* was isolated from blood cultures and treated with intravenous ampicillin.

Abdominal ultrasonography revealed in addition to ascites hepatomegaly with homogeneous parenchyma. Portal vein thrombosis was detected, involving also both branches of the superior mesenteric vein (Figure 1). In the pancreas, anomalies in the structure of parenchyma with areas of calcification were seen. These findings were confirmed by means of an abdominal contrast computed tomographic scan (Figure 2) and endoscopic retrograde cholangiopancreatography. Given the presence of portal vein block superimposed on a hepatic cirrhosis and the high incidence of hepatocarcinoma in this condition we decided to perform a fine needle aspiration puncture from the area of portal vein thrombosis. A fluid specimen was obtained which showed benign cytology with hyphaes and mycotic spores, growing *C. albicans* in cultures. Immunological studies showed a negative serology for human immunodeficiency virus and normal results for cutaneous tests of delayed hypersensitivity, immunophenotyping of lymphocytes, T4/T8 ratios and *in vitro* functionality of T cells. Serum precipitins to *C. albicans* were positive. Oral panendoscopy revealed lesions consistent with oesophageal candidiasis, which was confirmed by cultures.

The patient was treated with intravenous amphotericin B for 6 weeks with good clinical response, gradual normalization of the anaemia and ESR, and complete disappearance of PVT on abdominal ultrasonography carried out a few weeks later. A further fine needle aspiration failed to demonstrate *C. albicans* in cultures.

Discussion

Extrahepatic or prehepatic PVT of infectious origin was frequent in the preantibiotic era; since then, its incidence has notably decreased. Nevertheless, systemic and intra-abdominal infections are still the most common cause of PVT in children. In contrast, in adults with hepatic cirrhosis, portal invasion from an overimposed hepatocellular carcinoma remains the main aetiology. Formerly, PVT was considered a relatively common complication of hepatic cirrhosis but, nowadays, the incidence of this complication is about 0.6%. It is possible that, even in the absence of tumour,
periphlebitis induced by a bacterial infection may cause PVT in cirrhosis patients as a consequence of the slow portal flow of these patients. At present, better sanitary conditions of the population might play an important role in the decreased frequency of that complication.1

Other causes of PVT in non-cirrhotic adults include abdominal trauma, pancreatitis, intra-abdominal sepsis, splenectomy, porta-caval shunts, or an associated hypercoagulability state (pregnancy, myelofibrosis, tumours).2 In many cases, the aetiology is unknown.6

In our patient, PVT associated with cirrhosis and infection of the portal area by *C. albicans*, led us to consider the possibility that a systemic infection by *S. milleri* had induced a periphlebitis, which in turn would act as a trigger for PVT. Infection by *C. albicans* would therefore be a superadded infection of the thrombus; However, the good response of the patient once treated with amphotericin B with further elimination of the yeast from the portal area, as proven by fine needle aspiration puncture, together with the recanalization of the superior mesenteric vein, led us to suspect that the most important factor maintaining PVT was indeed the associated candidiasis.

We think that though the incidence of PVT of infectious origin is low, when PVT is demonstrated in the absence of tumour, it is wise to perform a fine needle aspiration puncture (given the low incidence rate of complications of this procedure in our experience) with the aim to rule out an overimposed infection which is potentially treatable with specific antibiotic therapy.

References

Keloid of the penis after circumcision

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**Summary:** We report what we believe is the first documented case of keloid formation on the penis following circumcision.

**Introduction**

Keloid formation is a well-recognized complication of both surgical and traumatic skin wounds. However, this has not, as far as we know, been reported after the common procedure of circumcision, even in those otherwise predisposed to keloid.

**Case report**

A 10 year old coloured boy from Sierra Leone underwent a routine circumcision for cultural reasons. The procedure was uneventful and there were no early complications; however, he presented 2 years later with pronounced keloid formation around the base of the glans (Figure 1). He complained of embarrassment and pruritus. He had a previous keloid following a traumatic wound to his axilla but there was no family history of keloid formation.

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