CASE REPORT

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A Rare Case of Post-partum Ascites

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Abstract

Introduction: Budd-Chiari syndrome (BCS) is defined as obstruction of the hepatic venous outflow. This obstruction might be located anywhere between the small hepatic veins to the suprahepatic inferior vena cava (IVC). This syndrome occurs in 1/100,000 in the general population.

We report a case of Budd-Chiari syndrome with onset in post-partum period. She presented with sudden onset pain in abdomen and ascites. On investigation, BCS was radiologically diagnosed and she was anticoagulated.

Key words: Budd-Chiari syndrome, ascites in pregnancy, puerperial ascites.

Introduction

Budd-Chiari syndrome (BCS) is an uncommon disorder characterised by obstruction of hepatic venous outflow. The obstruction may be, thrombotic or non-thrombotic, anywhere along the venous course from the hepatic venules to junction of the inferior vena cava (IVC) to the right atrium. BCS is a heterogeneous clinical condition – it may be curable or potentially lethal. BCS occurs in 1/100,000 of the general population worldwide[1]. Most patients with BCS have an underlying condition that predisposes to blood clotting. Obstruction is mainly caused by primary intravascular thrombosis. It was found that at least one hereditary or acquired hypercoagulable state could be identified in 75% of patients; more than one aetiologic factor may play a role in 25% of patients[2].

Case report

A 22-year-old lady came with chief complaints of abdominal distension and pain in abdomen since 3 days. she had delivered vaginally six months back. She did not report any fever, loose stools, vomiting, jaundice and had normal sleep, appetite and bladder/bowel functions. There were no addictions and no significant past or family history. She had not taken oral contraceptive pills in the recent past.

Patient was afebrile and vitally stable. On examination, pallor, icterus, oedema, cyanosis, clubbing or lymphadenopathy were absent. Per-abdomen examination: revealed abdomen was soft and distended, with mild hepatomegaly. Shifting dullness was present but no fluid thrill. Other systems were normal.

Laboratory investigations: Haemogram, LFT, RFT: WNL, total serum protein: decreased, Pt-INR/APTT: 1.7/39.8, viral markers: negative and pro thrombotic profile was normal.

Ascitic fluid: Clear, pale yellow. No clots, coagulum or cobweb R/M: zero cells, total proteins/albumin: 2.5/1.4, sugars: 128 mg/dl, ADA 3.8. Ascitic fluid suggestive of transudative nature with SAAG = 1.61.

Radiological investigations: Chest X-ray was normal, USG A/P: liver 16 cms enlarged, with normal echogenicity. Portal veins-normal, pseudo gall bladder wall thickening, spleen, pancreas, kidneys were normal. Free fluid noted in pouch of Douglas, pelvis, perihepatic and perisplenic region.

Colour Doppler of portal system: Liver 18 cms, relative atrophy of left lobe of liver with enlargement of caudate lobe of liver. Hepatic veins - right hepatic vein appears thrombosed/thick hyper echoic. Middle hepatic vein appears patent and shows normal hepatofugal flow. Left hepatic vein is thrombosed. IVC – normal, no thrombosis Portal vein – 7 mm with reversal of flow, Collaterals - intrahepatic venovenous collaterals seen with no flow s/o thrombosis. Conclusion: Budd-Chiari Syndrome with portal hypertension.

CECT of abdomen and pelvis: Hepatomegaly with nutmeg appearance of liver with poor opacification of hepatic veins-veno occlusive disease with moderate ascites s/o Budd-Chiari syndrome (Fig. 1).

On Upper GI endoscopy mild portal hypertensive gastropathy and nodular pangastritis.

Diagnosis: Budd-chiari syndrome – most probable post-partum aetiology.

Treatment:

Injection: Enoxaparin 0.6 cc BD for 5 days and overlapped

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with Tablet: Acenocoumaral 2 mg OD on day 3 of enoxaparin

Tablet: Furosemide/spironolactone 20/50 mg BD

Tablet: Propranolol 40 mg OD

Follow-up after one month: Patient had symptomatically improved. She was continued on anticoagulation medication and INR was maintained between 2 - 3. On USG A/P: no evidence of ascites.

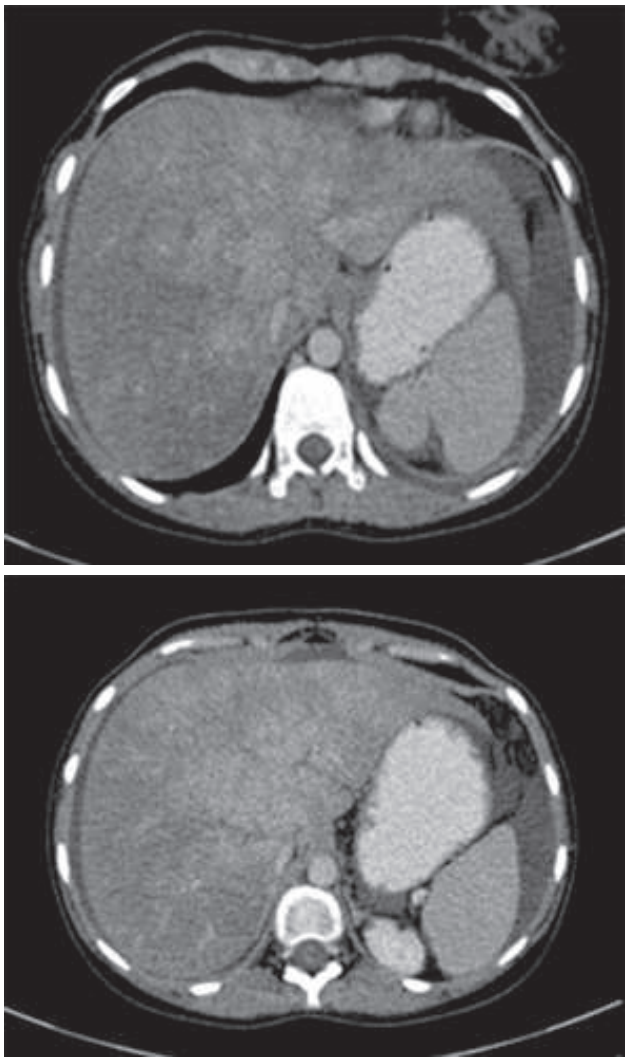

Fig. 1: *CECT abdomen.*

Discussion

Budd-Chiari syndrome is a rare condition affecting one in a million persons. It presents with the classical triad of abdominal pain, ascites and liver enlargement.

Pregnancy is a hypercoagulable state with increased serum clotting factors, predominantly originating from the liver. Thus, the concentrations of clotting factors in HVs and IVC might be higher in pregnant or puerperal women, resulting in their increased chance of developing thrombosis within the hepatic venous outflow. An important finding in one study was that the prevalence of pregnancy-related BCS is 6.8%[5]. Another study showed prevalence of 13.1%, suggesting that pregnancy might be a relatively common aetiology of BCS[7]. It appears that the prevalence of pregnancy-related BCS (6.8%) is similar to that of other common risk factors, such as inherited antithrombin (2.3%), protein C (3.8%), and protein S (3.0%) deficiencies, prothrombin G20210A mutation (3.0%), and factor V Leiden mutation (12.0%) in BCS. Therefore, pregnancy must not be neglected as the aetiology of BCS is being assessed[6].

The courses of puerperium may be variable among countries. In some Asian countries, prolonged rest postpartum is followed by late and slow mobilisation after childbirth. According to a report by Dilawari *et al*, in rural India, puerperal women often experience up to 30 or 40 days of confinement and fluid restriction. Thus, the combination of increased clotting factors, lack of activity, and dehydration may produce a condition conducive to venous thrombotic complications, potentially leading to a higher prevalence of pregnancy-related BCS (13.1%).

BCS should be suspected in patients with: (1) Abrupt onset of ascites and painful hepatomegaly; (2) Massive ascites with relatively preserved liver functions; (3) Sinusoidal dilation in liver biopsy without heart disease; (4) Fulminant hepatic failure associated with hepatomegaly and ascites; (5) Unexplained chronic liver disease; (6) Liver disease with thrombogenic disorder.

Doppler ultrasonography of the liver, with a sensitivity and specificity of 85% or more, is the technique of choice for initial investigation when BCS is suspected[3]. Imaging of hepatic veins without flow signal, and with spider-web appearance, collateral hepatic venous circulation and stagnant, reversed or turbulent flow are indicative of BCS. CT scanning may be recommended for imaging the vascular anatomy and the configuration of the liver when a transjugular intrahepatic portosystemic shunt is considered. Unvisualised hepatic veins are suggestive of disease on CT[4].

Budd-Chiari syndrome after pregnancy is an extremely rare disease. It is recommended that physicians should be vigilant for pregnancy-related BCS. Further research should explore the risk factors for pregnancy-related BCS and their potential mechanisms.

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