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CASE REPORT OF CONGESTIVE HEPATOPATHY: A CLINICAL BUDD CHIARI MIMIC

Radiodiagnosis

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ABSTRACT

Congestive hepatopathy (CH) refers to hepatic abnormalities that result from passive hepatic venous congestion which when prolonged lead to fibrosis and cirrhosis. Liver dysfunction and corresponding clinical signs and symptoms typically manifest late in the disease process. Recognition of CH at imaging is critical because advanced liver fibrosis may develop before the condition is suspected clinically. We report a case of congestive hepatopathy in a 25- year old male presenting clinically with symptoms simulating Budd Chiari syndrome.

KEYWORDS

INTRODUCTION

Congestive hepatopathy (CH) occurs due to passive hepatic congestion, which may be due to any cardiopulmonary disease that leads to increased central venous pressure. Common causes include congestive cardiac failure, constrictive pericarditis, cardiomyopathy, valvulopathy and severe pulmonary hypertension . Hepatic injury in patients with CH is potentially reversible if detected early. Awareness and reporting of this condition by radiologists can facilitate early diagnosis and treatment. The development of hepatic fibrosis has negative prognostic implications for the patient, and imaging may be important for early detection.

CASE REPORT

A 25 year old male with no significant past medical or surgical history presented with abdominal distension and breathlessness since one month but no fever, weight loss or other systemic symptoms. Routine blood investigations revealed no significant abnormality except mildly raised serum bilirubin levels.

Ultrasound of the abdomen was suggestive of mild hepatomegaly with coarse echotexture and moderate ascites suggestive of a liver parenchymal disease. Doppler findings raised a suspicion of Budd Chiari syndrome.

Hence a CT abdominal angiography was performed .It revealed mild hepatomegaly with mild diffuse irregular surface nodularity and hypertrophy of caudate lobe .The liver parenchyma revealed inhomogeneous enhancement on the portal venous phase of the post contrast study which became homogenous on the delayed phase (Fig 1).The infradiaphragmatic and supradiaphragmatic inferior vena cava and hepatic veins were dilated but well opacified with few hepatic veno-venous collaterals but no obvious thrombus (Fig 2).However sections across the lung bases revealed minimal loculated basal pericardial effusion with associated enhancing pericardial thickening and dense plaque like pericardial calcifications(Fig 3) . CT findings were hence highly suggestive of a congestive hepatopathy, secondary to chronic constrictive pericarditis.

A 2-D Echo subsequently performed confirmed the findings of constrictive pericarditis.

Cardiac catheterization was performed which revealed equalization of all diastolic pressures and elevation in filling pressures and other findings confirming constrictive pericarditis. Hepatic venography with transjugular liver biopsy was performed which confirmed dilated IVC and hepatic veins with veno-venous collaterals (Fig 4) and histopathology suggesting liver cirrhosis.

Patient was managed with diuretics in view of congestive hepatopathy and empirically started on anti- Kochs treatment and advised pericardiotomy.

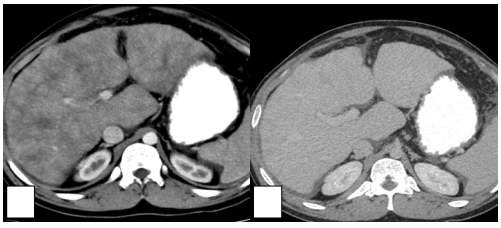

Figure 1: Axial CECT images of the liver reveal inhomogenous enhancement on the portal venous phase(a) which becomes homogenous on the delayed phase(b).

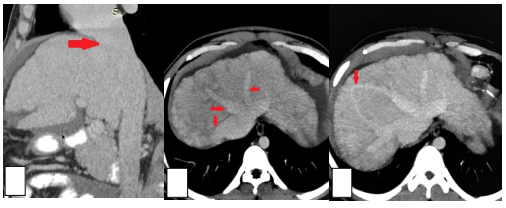

Figure 2 : The supradiaphragmatic and infradiaphragmatic IVC (a) and hepatic veins(b) are dilated but well opacified with no obvious filling defect. Hepatic veno-venous collaterals are seen©

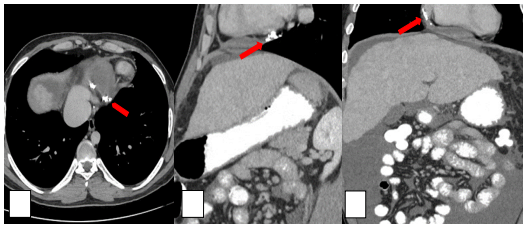

Figure 3 : Axial (a),Sagittal (b) and Coronal (c) CECT images showing minimal loculated basal pericardial effusion with associated pericardial thickening and dense plaque - like pericardial calcifications.

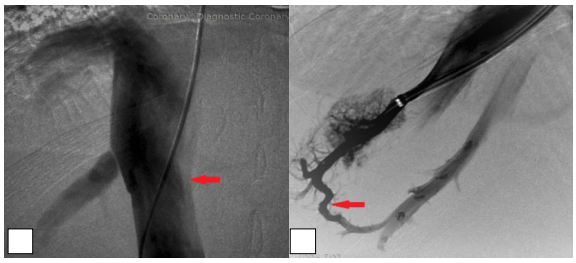

Figure 4: Hepatic venogram showing dilated IVC, hepatic veins (a) and Veno-Venous collaterals (b).

DISCUSSION

Congestive hepatopathy is due to stasis of blood in the hepatic parenchyma due to impaired hepatic venous drainage secondary to cardiac failure . Prolonged exposure to elevated hepatic venous pressure may lead to liver fibrosis and cirrhosis. Patients may present with dull aching pain in the right upper quadrant or hepatomegaly. Lab investigations may reveal elevated bilirubin, serum g-glutamyl transpeptidase enzyme, and serum alkaline phosphatase levels, suggesting cholestasis.

Imaging findings of CH on ultrasound include a dilated inferior vena cava and dilated hepatic veins. The right hepatic vein is normally less than 5.6–6.2 mm in diameter at the origin and dilates in response to elevated venous pressure . The degree of dilatation correlates with the severity of heart failure . Under normal conditions, the inferior vena cava and hepatic veins narrow in caliber as resistance to blood flow returning to the heart decreases during inspiration. Absent or diminished respiratory variation can be seen when there is increased venous pressure . Spectral Doppler US analysis can show loss of the normal triphasic hepatic venous waveform.

CT Findings, reveal hepatomegaly due to acute congestion or an atrophic nodular liver in patients with chronic congestion and cirrhosis. There is dilatation of the inferior vena cava and hepatic veins due to backpressure in constrictive pericarditis. Further long standing CH, results in hepatic vein–to–hepatic vein shunting which may be related to preferential retrograde flow into the right hepatic vein from the inferior vena cava. Due to diminished right sided cardiac output or high right atrial pressure there is reflux of contrast material into the inferior vena cava and hepatic veins. Due to congested hepatic parenchyma the liver shows relatively slow enhancement near the hepatic veins resulting in patchy irregular regions of poor enhancement. This abnormal enhancement pattern is best seen in the portal venous phase . Further, contrast material retention in the hepatic parenchyma reaches a state of equilibrium, resulting in a more homogeneous appearance of the parenchyma in delayed phases.All these findings were also seen in this case of ours. The presence of cirrhosis increases the risk for hepatocellular carcinoma in patients with CH . Nodules that enlarge or show washout enhancement characteristics may require biopsy to distinguish benign nodules from hepatocellular carcinoma.

CONCLUSION

CH manifests with many imaging findings, the most common of which include dilated hepatic veins and heterogeneous parenchymal enhancement. Hepatic injury in patients with CH can be clinically insidious and is potentially reversible if detected early. Awareness of this condition by radiologists can facilitate early diagnosis and treatment. The development of hepatic fibrosis has negative prognostic implications for the patient, and imaging may be important for early detection.

REFERENCES

1. Coakley FV. Pearls and pitfalls in abdominal imaging: pseudotumors, variants and other difficult diagnoses. Cambridge, England: Cambridge University Press, 2010; 34–39.
2. Sherlock S. The liver in heart failure: relation of anatomical, functional, and circulatory changes. Br Heart J 1951;13(3):273–293.
3. Giallourakis CC, Rosenberg PM, Friedman LS. The liver in heart failure. Clin Liver Dis 2002;6(4):947–967, viii–ix.
4. Schiff ER, Maddrey WC, Sorrell MF, eds. Schiff's diseases of the liver. 11th ed. Oxford, England: Wiley-Blackwell, 2011.
5. Wanless IR, Liu JJ, Butany J. Role of thrombosis in the pathogenesis of congestive hepatic fibrosis (cardiac cirrhosis). Hepatology 1995;21(5):1232–1237.
6. Dunn GD, Hayes P, Breen KJ, Schenker S. The liver in congestive heart failure: a review. Am J Med Sci 1973;265(3): 174–189.
7. Shah H, Kuehl K, Sherker AH. Liver disease after the Fontan procedure: what the hepatologist needs to know. J Clin Gastroenterol 2010;44(6):428–431.
8. Tanaka M, Wanless IR. Pathology of the liver in Budd-Chiari syndrome: portal vein thrombosis and the histogenesis of venocentric cirrhosis, veno-portal cirrhosis, and large regenerative nodules. Hepatology 1998;27(2):488–496.
9. Louie CY, Pham MX, Daugherty TJ, Kambham N, Higgins JP. The liver in heart failure: a biopsy and explant series of the histopathologic and laboratory findings with a particular focus on pre-cardiac transplant evaluation. Mod Pathol 2015;28(7):932–943.
10. van Deursen VM, Damman K, Hillege HL, van Beek AP, van Veldhuisen DJ, Voors AA. Abnormal liver function in relation to hemodynamic profile in heart failure patients. J Card Fail 2010;16(1):84–90