Caput medusa: a sign of portal hypertension in case of chronic Budd-Chiari syndrome

Neelam Soni,1 Amrin Israrahmed,2 Priya Singh,2 Hira Lal 2

DESCRIPTION

Portal hypertension can occur due to several presinusoidal, sinusoidal and postsinusoidal causes leading to liver cirrhosis. Gilbert and Carnot initially coined the term 'portal hypertension' in 1902.1 The pathophysiology of portal hypertension in patients of liver cirrhosis is such that; initially there is distortion of blood vessels within the liver parenchyma due to hepatic fibrosis which results in an increase in portal pressure and clinically manifests as portal hypertension. In order to bypass this high pressure system, numerous low resistance vascular circuits become functional leading to portosystemic shunting of blood.2 3 Thus, portal hypertension clinically presents as multiple dilated, tortuous venous channels along the anterior abdominal wall and umbilicus giving rise to the ‘caput medusa’ appearance (figure 1A). The term ‘Caput’ is a Latin word for head. According to Greek mythology, ‘medusa’ was a monster with hair made of snakes, moving in all directions (figure 1B). The term ‘caput medusa’ is used for the appearance of distended and engorged umbilical veins, radiating across the umbilicus. Budd-Chiari syndrome is a type of hepatic venous outflow obstruction which results in an increase in the hepatic sinusoidal pressure causing liver congestion. Thus, it is an important cause of postsinusoidal type of portal hypertension (figure 1C). If the liver congestion (venous outflow obstruction) is not relieved immediately, then it further progresses from acute to chronic stages and results in hepatocytic necrosis and liver fibrosis further resulting in liver cirrhosis.4

We present a case of a 45-year-old man who presented with symptoms of vague abdominal pain and abdominal distension since the last 14 months. Abdominal examination revealed multiple tortuous veins on the abdominal wall giving the caput medusa appearance. Routine laboratory investigations revealed elevated alanine amino transferase 120 U/L, aspartate transaminase 98 U/L, and bilirubin 1.6 mg/dL with a decrease in platelet count of 80,000/µL of blood. Ultrasound abdomen confirmed multiple venous collaterals in the subcutaneous plane of anterior abdominal wall near the umbilicus along with enlarged caudate lobe of liver and moderate ascites (figure 2A,B). Doppler showed a long segment block of the common channel of the middle and left hepatic veins and a short segment block of right hepatic vein near their ostium at the...
Inferior Vena Cava (IVC), thus diagnostic of hepatic venous type of occlusion in Budd-Chiari syndrome (figure 2C). The IVC was normal in calibre. Contrast-enhanced CT abdomen was done for further evaluation, which showed multiple dilated vascular channels in the anterior abdominal wall draining into the peribillioumal veins (figure 2D). Splenomegaly, mild ascites and long segment hepatic vein block was also confirmed on CT. The patient was subsequently put on anticoagulation and scheduled for hepatic vein angioplasty. Endovascular management was performed with balloon angioplasty and stenting of the right hepatic vein (figure 2E,F). A baseline Doppler study done 24 hours after the procedure revealed good flow within the stent with significant reduction in ascites and abdominal wall collaterals. The patient was discharged in stable condition with follow-up advised at 1, 3 and 6 months, respectively, in the first year postprocedure and annually thereafter.

Acknowledgements We would like to thank Dr Kasturi BK Rangan for helping us in obtaining the diagrammatic representation of the mythological creature Medusa. Also, we wish to thank Dr Shivanand G for providing us with his valuable input in the figures section.

Contributors NS was primarily involved in data collection and drafted the final version of the manuscript. AI was involved in acquiring the images and drafting the figure legends. PS helped in data acquisition and HL was responsible for analysing and interpretation of the data. All the authors have participated sufficiently in the submission to take public responsibility for its content. The manuscript and its contents are approved by all authors.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

ORCID iD Hira Lal http://orcid.org/0000-0001-7957-635X

REFERENCES