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CASE REPORT

Unusual abdominal wall Varicosity: A Diagnostic Dilemma

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ABSTRACT

Abdominal wall varicosity manifests as enlarged, winding veins on the anterior abdominal wall that is prominent enough to assess blood flow direction. While this condition can stem from several underlying causes, careful clinical examination can help distinguish between cirrhosis with portal hypertension and obstruction of the inferior vena cava (IVC) or superior vena cava (SVC).

Clinical Assessment

To determine blood flow direction in these veins, clinicians should follow a systematic approach:

1. Identify a vein segment free of branches for at least 3 cm
2. Place two fingers close together over the middle of this segment
3. Move both fingers in opposite directions to "milk" and completely empty the vein
4. Release one finger and observe the speed and direction of blood flow
5. Repeat the procedure in the opposite direction

Budd-Chiari Syndrome Connection

Budd-Chiari syndrome (BCS), while uncommon, should be considered when evaluating abdominal wall varicosity. This condition occurs when hepatic venous outflow becomes obstructed. The underlying causes typically fall into two categories:

- Inherited hypercoagulable states
- Acquired prothrombotic conditions

Over half of BCS cases stem from acquired prothrombotic states, particularly myeloproliferative disorders including:

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- Polycythemia vera
- Paroxysmal nocturnal hemoglobinuria
- Essential thrombocytosis
- Agnogenic myeloid metaplasia
- Myelofibrosis

Clinical Presentation

The manifestation of BCS varies significantly, ranging from asymptomatic cases to acute hepatic failure. The severity depends on two key factors:

- The extent and speed of hepatic vein occlusion
- The development of venous collateral circulation to decompress liver sinusoids

Notably, when large abdominal veins (typically the inferior vena cava or portal vein) become thrombosis, collateral vessels may develop in the abdominal wall. These collaterals can lead to visible abdominal varicosities during physical examination, serving as an important diagnostic indicator.

KEYWORDS

• Abdominal wall varicosity • Budd-Chiari syndrome (BCS) • Polycythemia vera (PV) • JAK2V617F tyrosine kinase

INTRODUCTION

Abnormally dilated tortuous abdominal wall veins indicate portal, hepatic, IVC or SVC obstruction. Budd-Chiari syndrome (BCS) is one of them and commonly associated with hypercoagulable state. Polycythemia vera (PV) is one of the most common chronic myeloproliferative disorder in which overproduction red blood cells along with white blood cells and platelets in the absence of an identifiable physiologic stimulus. It occurs in 2.5 per 1 lac population, in adult age group. Mutation in the JAK2V617F tyrosine kinase has an important role in the pathogenesis of PV. WHO criteria for the diagnosis of PV include-

Major criteria:

1. Increased haemoglobin (>16.5 g/dl in men or >16 g/dl in women), hematocrit (>49% in men or >48% in women), or other evidence of increased red cell volume
2. Bone marrow biopsy showing trilineage hypercellularity
3. JAK2 V617F mutation

Minor criteria:

Subnormal serum erythropoietin level.

Diagnosis of PV requires either all three major criteria or first two major and the minor criteria

PV can cause venous or arterial thrombosis of any vessel, out of that cerebral, cardiac, and mesenteric vessels are most commonly involved. PV should be suspected in any patient with increased red blood cell mass or increased haemoglobin/hematocrit or in patients with mesenteric, portal, or splenic vein thrombosis. Budd- Chiari syndrome (BCS) represents a rare hepatic condition characterized by vascular obstruction of the efferent hepatic flow at a site that may vary from the small hepatic veins up to the place where the inferior vena cava enters the right atrium. More than 50% of BCS cases may have acquired prothrombotic states such as myeloproliferative disorders e.g., polycythemia vera.[2-4]

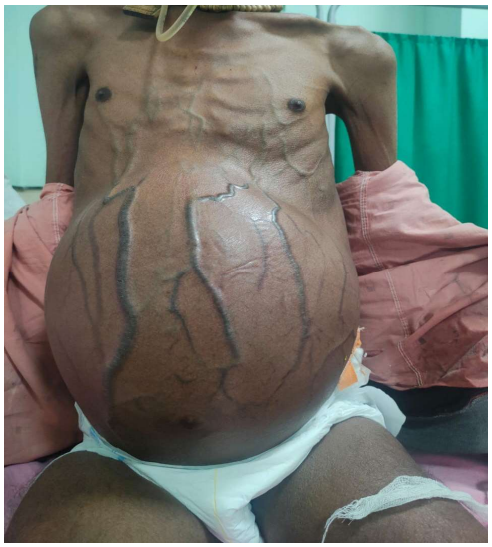
CASE DESCRIPTION

A 29 years old male patient presented with abdominal distension since last 1 year, which was insidious onset, gradually progressive and has further increased in last 15 days. Patient also has altered mental status since last 3 days, in terms of reduced responsiveness and irrelevant talking, associated with increased day time sleepiness. No past H/O Blood in vomiting, blood in stool, blackish stool, yellowish discoloration of eyes, decreased urination & cutaneous bleeding manifestations. On examination Patient was drowsy and not oriented to time, place & person. His vitals were BP - 124/88 mmHg, PR-75 bpm regular, RBS - 126 mg/dl, SpO2 - 95% ra, RR - 18cpm. Patient was emaciated, conjunctiva was congested

bilaterally, icterus, cyanosis, clubbing, pedal oedema were absent, no palpable lymph nodes, neck veins not engorged, JVP not visualised,Flapping tremors were present. On per abdominal examination-abdomen grossly distended, umbilicus downward & slit like, multiple engorged dilated superficial veins extending up to posterior aspect of abdominal wall, direction of filling of veins below upwards, nontender hepato-splenomegaly were present. Fluid thrill was present.

Investigations: The hemogram showed persistent erythrocytosis (Hb 18.8 & hematocrit 60) with other cell counts being normal limit. The basic metabolic panel showed mildly deranged RFT (urea 85, cr. 1.59), hyperbilirubinemia (bilirubin total 2.41), and hypoalbuminemia (albumin 2.21). Ascitic fluid analysis was suggestive of a high SAAG low protein with no SBP and negative CBNAAT. Viral markers were found to be negative. USG abdomen confirmed the findings suggestive of CLD, collapsed all three hepatic veins and intrahepatic IVC, Gross ascites, Cholelithiasis. CT abdomen showed thrombosis of right, middle, left hepatic veins, intrahepatic IVC, SMV and portal veins S/O Budd chairi syndrome.

Evaluation for erythrocytosis showed normal EPO level, JAK 2 mutation was found to be positive.



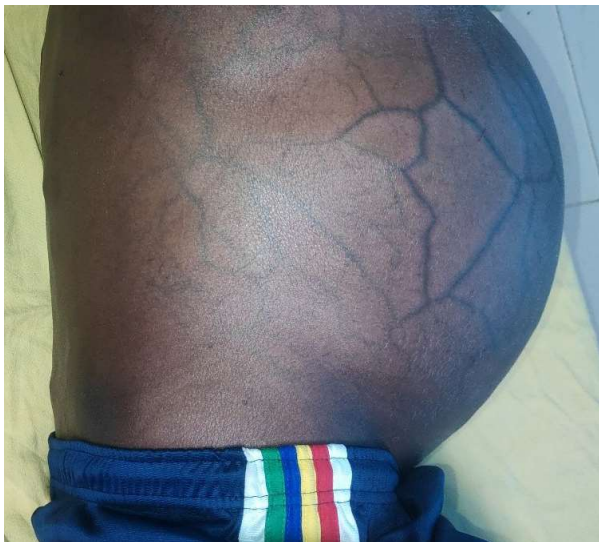
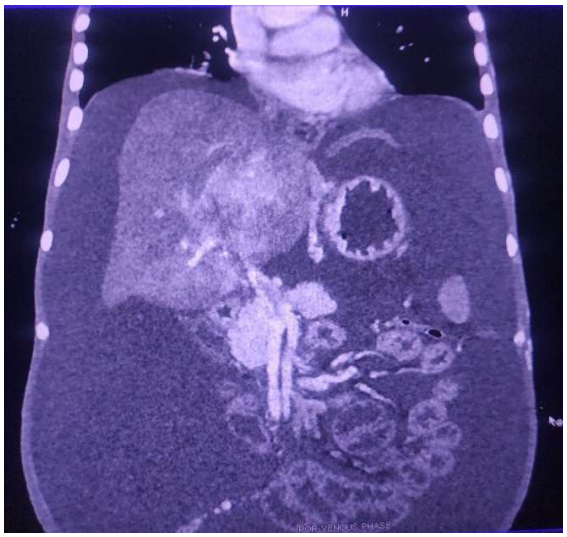


Fig. 1, 2: Engorged veins all around the abdominal wall (anterior & posterior as well)



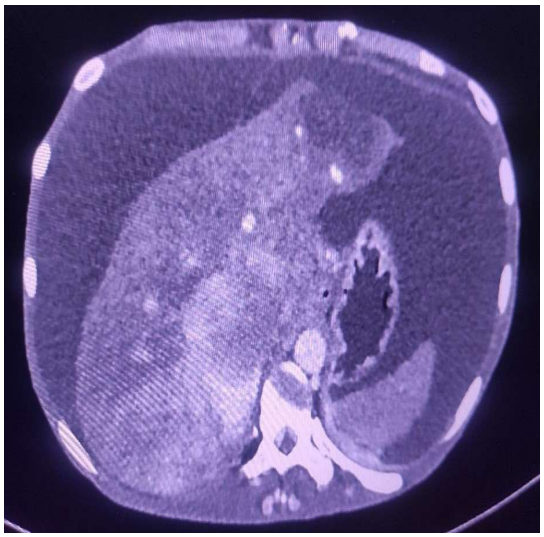


Fig. 3 & 4: Non-visualization of hepatic veins and intrahepatic IVC on CECT abdomen

DISCUSSION

When any patient comes to us with unusual clinical presentation, we have to examine thoroughly and find out underlying etiology. Extensive abdominal varicosity is rare, but when it is present and extending up to back gives clue for obstruction of portal, hepatic or IVC k/a Budd chairi syndrome. Up to 75% of cases, there is an underlying hypercoagulability disorder, associated with **Myeloproliferative disorders** with a high prevalence of the *JAK2V617F* mutation. Among the associated MPNs, polycythemia Vera (PV) is the most frequent, corresponding to about half of the cases. BCS is a potentially life-threatening condition in which hepatic venous outflow obstruction causes hepatic congestion and subsequent fibrosis. The elevated sinusoidal pressure leads to ascites, portal hypertension, and the development of collaterals.[2]

BCS is associated with high mortality up to 80% in three years. Treatment should focus on restoring hepatic flow and monitoring the consequences of hepatic congestion, along with managing the underlying prothrombotic condition. Thus, the prompt initiation of diuretics and anticoagulant therapy is essential and may give in following combination:

(1) Antiplatelet therapy with or without cytoreduction

(2) Oral anticoagulation with or without cytoreduction

(3) Oral anticoagulation plus antiplatelet therapy with or without cytoreduction

(4) cytoreduction only or no antithrombotic or antiplatelet treatment. Treating the underlying cause, with hydroxyurea and phlebotomy. Timely treatment is crucial to restore hepatic venous flow, and in extreme cases, intervention endovascular surgery & liver transplantation may be necessary.[3]

CONCLUSION

CLD is a serious health concern in India. Recognizing relatively uncommon causes like hepatic vein obstruction is crucial for specific treatment that can improve mortality &morbidity. BCS presents a particular challenge due to the absence of typical signs until cirrhosis occurs. Prominent engorged veins all around the abdominal wall give a unique clinical clue. Understanding and underlying factors of BCS highlights the importance of proactive management strategies to prevent complications. Myeloproliferative conditions like polycythemia Vera are particularly concerning and should be evaluated.

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