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Case Report

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Sonographic Bright Band Sign of Splenic Infarction

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Abstract

A 57-year-old woman with diabetes and hypertension presented with acute calculous cholecystitis and cholangitis. Incidentally, two splenic lesions were identified on formal abdominal ultrasound, one demonstrating the sonographic bright band sign. Computed tomography confirmed splenic infarctions and also revealed portosplenic vein thrombosis. Haematological workup, including JAK2 V617F mutation analysis, led to a diagnosis of polycythaemia vera, contributing to cholelithiasis via increased cell turnover and thrombosis due to hyperviscosity. She was started on venesection and hydroxycarbamide and discharged well. However, she defaulted follow-up and re-presented two years later with disseminated intravascular coagulation, eventually succumbing to the illness. This case highlights the utility of the sonographic bright band sign in identifying splenic infarction and distinguishing it from other hypoechoic splenic lesions.

Keywords: Splenic infarction; Ultrasonography; Polycythaemia rubra vera

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INTRODUCTION

As the largest lymphatic organ, the spleen is involved in many systemic pathologies, some of which may be unrecognised and hence undiagnosed. Primary splenic disease is relatively uncommon, and most splenic lesions are secondary to underlying systemic conditions. It is therefore important to investigate for a primary cause whenever a splenic lesion is identified. Conditions such as lymphoma, abscesses, and metastases can involve the spleen. Systemic vascular causes, including arterial thromboembolism and veno-occlusive disease, may also affect the spleen by causing perfusion abnormalities, leading to infarction. Splenic infarction may

be one of the presenting features, or even the sole manifestation, of certain diseases. Its imaging appearance can be variable, necessitating more precise diagnostic tools.

The sonographic bright band sign is a useful feature, as it is seen exclusively in cases of splenic infarction. In our case, the presence of both cholelithiasis and splenic infarction raised suspicion for an underlying haematological disorder, later confirmed to be polycythaemia vera. This case report emphasises the radiological features of splenic infarction, particularly the utility of the sonographic bright band sign.

CASE REPORT

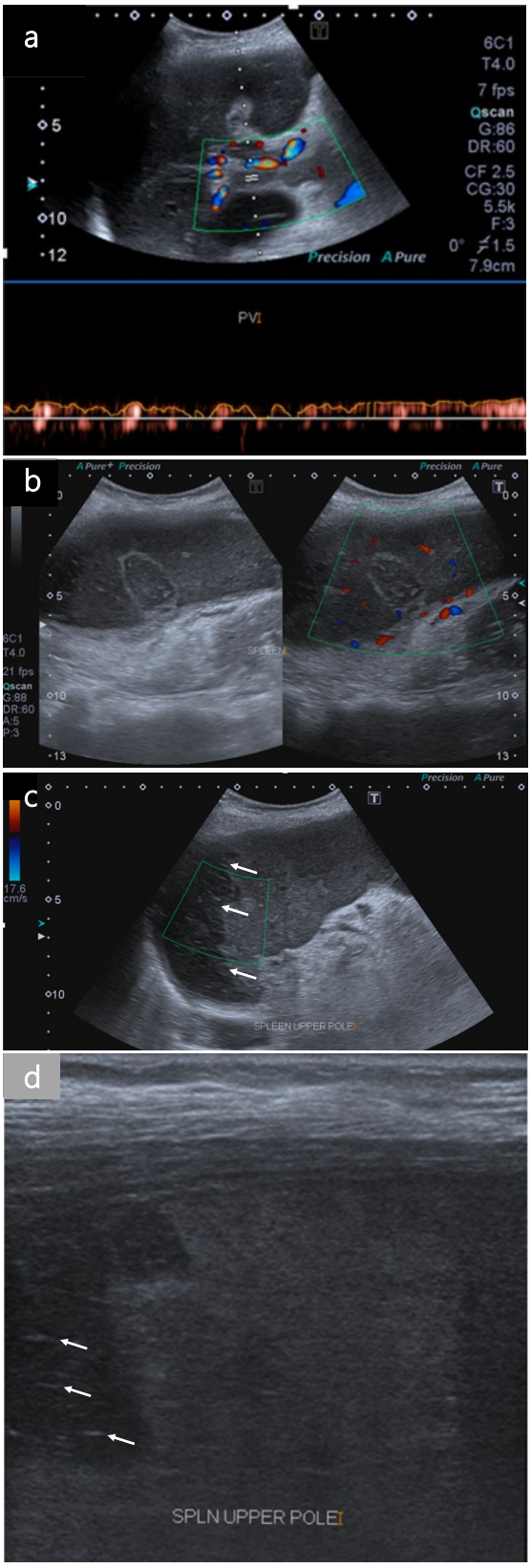
A 57-year-old woman with a background of diabetes mellitus and hypertension presented with a one-week history of general malaise. Her symptoms included mild right hypochondriac pain and loss of appetite, without significant weight loss. She reported dark-coloured urine but denied pruritus, fever, vomiting, or bowel symptoms. Abdominal examination was unremarkable, and her vital signs were stable. There was no personal or family history of malignancy, and no prior hospitalisations.

Initial blood investigations revealed elevated haemoglobin of 17.7 g/dL (normal range [NR]: 11.6 – 15.1 g/dL), haematocrit 56.7% (NR: 35.1 – 44.9%), thrombocytosis 526 × 10⁹/L (NR: 171 – 399 x 10⁹/L), and leucocytosis 18.3 × 10⁹/L (NR: 4.1 – 11.4 x 10⁹/L). Peripheral blood film showed features of polycythaemia with signs of ongoing infection or inflammation.

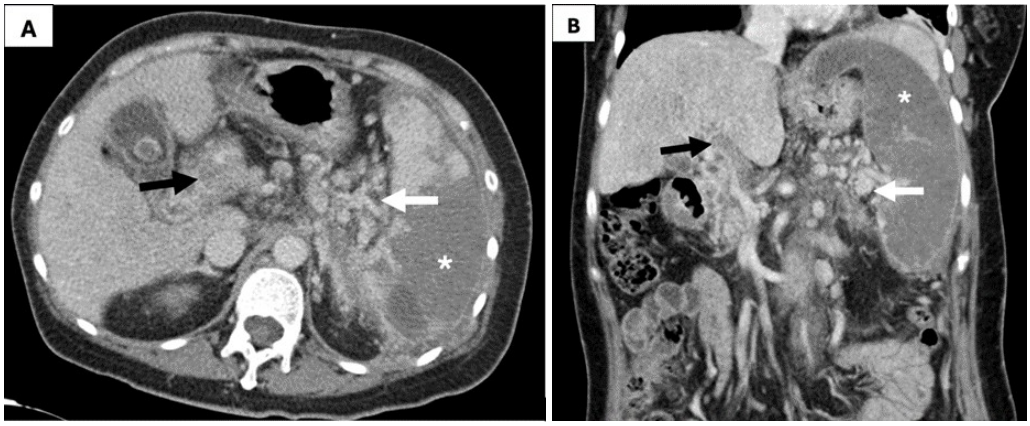
A formal abdominal ultrasound performed in the Radiology Department showed features of cholangitis and calculous cholecystitis, evidenced by periportal hyperechogenicity, gallbladder wall thickening, and cholelithiasis. The portal vein was patent but hyperechoic, with sluggish flow (**Figure 1a**). Two splenic lesions were noted. The second lesion was oval-shaped with peripheral hyperechogenicity and central isoechogenicity (**Figure 1b**), without a bright band sign. One was a peripheral, wedge-shaped hypoechoic lesion (**Figure 1c**), within which hyperechoic parallel lines—consistent with the bright band sign—were identified (**Figures 1c and 1d**).

Contrast-enhanced computed tomography (CT) of the abdomen confirmed near-total splenic infarction and portosplenic vein thrombosis (**Figure 2**).

The JAK2 V617F mutation was positive (ARMS PCR), confirming the diagnosis of polycythaemia vera.



Figs. 1: a) Echogenic portal vein with absence of colour Doppler. Pulse Doppler showed slow flow. b) Oval splenic lesion with central isoechogenicity and peripheral hyperechogenicity; bright band sign absent. c) Multiple hyperechoic parallel lines (Bright band sign) within a large irregular hypoechoic splenic lesion (white arrows) representing infarcted part of the spleen. d) Close-up view of the sonographic bright band sign (white arrows).



Figs. 2: (a) Axial view (b) Coronal view, CT abdomen in portovenous phase. Hypodense splenic parenchyma in keeping with infarction (*). Multiple venous collaterals (white arrow). Portal vein thrombosis (black arrow).

The patient underwent venesection and was initiated on hydroxycarbamide for polycythaemia vera. She was discharged in stable condition after four days, with haematology clinic follow-up arranged. Unfortunately, she defaulted follow-up and presented two years later with disseminated intravascular coagulation (DIC). She ultimately succumbed to the illness during her intensive care unit (ICU) admission.

DISCUSSION

The spleen, being the largest lymphatic organ, is involved in various systemic diseases, many of which may initially go undiagnosed. Its arterial branches are end arteries that do not communicate with each other; thus, any occlusion can result in infarction. Splenic infarction was traditionally thought to present with left hypochondriac pain, tenderness, and swelling, often accompanied by a peritoneal friction rub.[1] However, in the largest case series to date, Antopolsky *et al*. reported that up to 20% of patients with splenic infarction presented without pain.[2] Multiple predisposing factors have been identified, including hypertension (31%), atrial fibrillation (23%), diabetes mellitus (23%), prior thromboembolism (10%) and ischaemic heart disease (19%).[2,3] Notably, 16.6% of patients were previously healthy, with splenic infarction leading to the diagnosis of an underlying systemic disease.[2]

In the present case, the patient presented with right hypochondriac pain and general malaise. Initial ultrasound revealed cholelithiasis (related to increased red cell turnover), slow portal vein flow, and splenic lesions, later confirmed as infarcts. These findings raised suspicion for a vaso-occlusive process with high red cell turnover, subsequently confirmed as newly diagnosed polycythaemia vera via mutation analysis.[4]

Ultrasound remains the initial imaging modality for evaluating splenic pathology due to its wide availability, non-invasiveness, and absence of ionising radiation. It is typically the first-line test performed when splenic lesions are suspected. Although CT is commonly used as a follow-up study and is more definitive due to superior contrast resolution and ability to characterise splenic lesions, ultrasound still plays an essential role in early detection and triage before further cross-sectional imaging is pursued.[5,6]

Typically, splenic infarctions appear as hypoechoic, peripheral lesions with absent colour doppler flow.[3] Diagnostic uncertainty arises in cases with atypical imaging features. Apart from the classic low-echogenicity, wedge-shaped lesion, splenic infarctions may also present as single or multiple lesions, with variable margins (well- or ill-defined), shapes (rounded or wedge-shaped), and echogenicity (anechoic, hypoechoic, hyperechoic, or mixed).[7-9] These variations are attributed to the differing stages of infarction. Differential diagnoses include metastases, lymphoma, and abscesses.[10,11] Once a splenic infarct is identified, thorough investigation for the underlying aetiology is essential.[11]

In atypical cases where splenic infarction cannot be confidently diagnosed, further imaging such as contrast-enhanced ultrasound or CT may be pursued. However, the use of contrast-enhanced CT carries considerations such as allergy risk, radiation exposure, cost, renal function, and clinical stability.

Llewellyn *et al.*, in a nine-year retrospective imaging review, described the sonographic bright band sign,

which was present in 91.9% (n = 34/37) of patients with confirmed splenic infarction and absent in all abnormal and normal controls (n=119),[12] thereby improving the diagnostic yield of standard ultrasound. The sign consists of two or more bright thin, parallel, specular reflectors perpendicular to the ultrasound beam, seen within hypoechoic splenic parenchyma corresponding to infarcted tissue, and reproducible across multiple imaging planes. Histological correlation indicates that these linear echogenic bands represent preserved splenic trabeculae within areas of infarction.[12] Given its high specificity, the bright band sign can aid in differentiating splenic infarction from other hypoechoic splenic lesions, including metastases, lymphoma, and abscesses, which typically do not demonstrate this finding.

In this case, of the two splenic lesions identified on ultrasound, only the lesion with typical wedge-shaped morphology demonstrated the sonographic bright band sign. The second lesion did not show this feature, possibly because very early splenic infarcts may not yet manifest the bright band sign, which is thought to arise from preserved splenic trabeculae that become more conspicuous as the infarct evolves and surrounding parenchymal necrosis progresses. Both lesions were subsequently confirmed as splenic infarcts on contrast-enhanced CT. Notably, the bright band sign is a sonographic phenomenon and is not visualised on CT scan. On CT scan, splenic infarction is characterised by peripheral, wedge-shaped, hypoenhancing regions.

While splenic infarction typically heals without intervention, complications such as splenic rupture can occur. Risk factors include expanding intralesional liquefaction, subcapsular haematoma, peritoneal bleeding, and persistent flow signals within infarcted areas on Doppler imaging. Short-term ultrasound follow-up is recommended for early detection of such complications.[7] As the infarct evolves, follow-up imaging typically shows decreasing size of the lesion, increasing echogenicity, and eventual fibrosis with loss of the previously well-defined hypoechoic wedge-shaped appearance.[9]

CONCLUSION

Splenic lesions are often secondary to underlying systemic diseases; therefore, their presence should prompt evaluation for a primary cause. These lesions may represent a range of pathologies, including metastases, non -liquefied abscesses, infarction, or lymphoma. The sonographic bright band sign is highly specific to splenic infarction and can help differentiate it from other hypoechoic splenic lesions. Accurate identification of splenic infarction is crucial, as the underlying aetiology may be life-threatening if not promptly diagnosed and treated.

Take Home Message

- The sonographic bright band sign is a highly specific and suggestive feature of splenic infarction, helping distinguish it from other hypoechoic splenic lesions.
- The sign consists of two or more thin, parallel, specular reflectors perpendicular to the ultrasound beam, seen within hypoechoic splenic parenchyma corresponding to infarcted tissue, and reproducible across multiple imaging planes.
- The bright band sign is thought to arise from preserved splenic trabeculae that become more conspicuous as the infarct evolves and surrounding parenchymal necrosis progresses.
- Splenic infarction may be the first manifestation of an undiagnosed systemic disease, such as polycythaemia vera.
- Early recognition of splenic infarction is essential, as underlying causes may be life-threatening.

Abbreviations

CT	Computed tomography
DIC	Disseminated intravascular coagulopathy
ICU	Intensive care unit

Declarations

All the authors declared no competing interests.

Ethical Consideration

Written consent was obtained from all patients for publications of the clinical details and accompanying images.

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None

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