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Case Report

Radiology Section

Giant Non-traumatic Splenic Pseudocyst Mimicking a Pancreatic Pseudocyst: A Case Report

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ABSTRACT

Splenic pseudocysts are rare non-parasitic cystic lesions of the spleen characterised by the absence of an epithelial lining. Most occur following trauma, whereas non-traumatic splenic pseudocysts are uncommon. A 45-year-old woman presented with a one-year history of chronic postprandial vomiting, early satiety, abdominal fullness, weight loss (6-7 kg), and recent-onset breathlessness. Initial ultrasonography suggested a pancreatic pseudocyst because of the apparent proximity of the lesion to the pancreatic tail and poor visualisation of the spleen. However, normal serum amylase and lipase levels together with unexplained cytopenias prompted further evaluation. Contrast-Enhanced Computed Tomography (CECT) revealed a large splenic cystic lesion with peripheral calcifications and internal septations, establishing its splenic origin and prompting revision of the initial diagnosis of pancreatic pseudocyst. The patient subsequently underwent open splenectomy. Histopathological examination confirmed a splenic pseudocyst. This case highlights that a presumed pancreatic pseudocyst in the absence of pancreatitis and with normal serum amylase and lipase levels should prompt consideration of alternative diagnoses, including splenic pseudocyst.

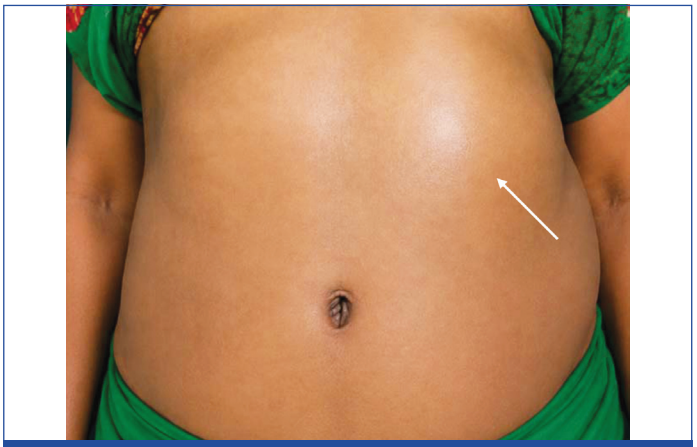
Keywords: Computed tomography, Cystic splenic lesion, Histopathology, Hypersplenism, Splenectomy

CASE REPORT

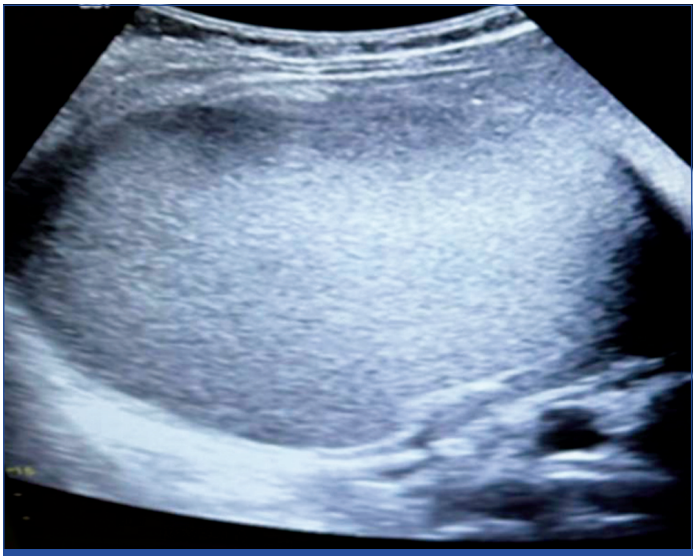
A 45-year-old woman presented with a one-year history of recurrent postprandial vomiting, early satiety, reduced appetite, progressive upper abdominal fullness, and an unintentional weight loss of 6-7 kg. The vomiting was non-bilious, non-projectile, consisted of recently ingested food, and was not associated with haematemesis. She also reported progressive exertional dyspnoea {New York Heart Association (NYHA) Class II} for the preceding 10 days, without chest pain, cough, fever, or orthopnoea. No history of abdominal pain, no alcohol intake, no fever, no jaundice, no gastrointestinal bleeding, no abdominal trauma, no history of tuberculosis, and no previous abdominal surgery. The patient had been a known case of type 2 diabetes mellitus for one year and was on oral hypoglycaemic agents, including glimepiride 1 mg twice daily and pioglitazone 15 mg once daily. She had been diagnosed with hypothyroidism one month before admission and was on levothyroxine (Thyronorm) 25 µg once daily. She was also diagnosed with systemic hypertension two weeks prior to presentation and was receiving nifedipine 10 mg three times daily and enalapril 5 mg once daily. The patient reported regular compliance with her prescribed medications.

On examination, she was haemodynamically stable. Abdominal examination revealed a smooth, non-tender swelling in the left hypochondrium, measuring approximately 10 x 10 cm. The overlying skin was normal [Table/Fig-1]. The remainder of the systemic examination was unremarkable.

Ultrasound of the abdomen demonstrated a large, well-defined unilocular cystic lesion measuring about 13×12×13 cm. There was internal echogenic debris with Brownian motion likely representing proteinaceous material, cholesterol debris, haemosiderin-laden macrophages, and cellular debris. A few smooth thick septations were seen within. The lesion seemed to be in close relation to the pancreatic tail and greater curvature of stomach. No definite solid component was observed [Table/Fig-2]. Based on the sonographic appearance, a provisional diagnosis of pancreatic pseudocyst arising from the tail of the pancreas was considered. Doppler assessment was not performed because the lesion demonstrated unequivocally cystic morphology on grayscale ultrasonography.



[Table/Fig-1]: Clinical photograph demonstrating a smooth left hypochondrial swelling measuring approximately 10×10 cm, with normal overlying skin (white arrow).

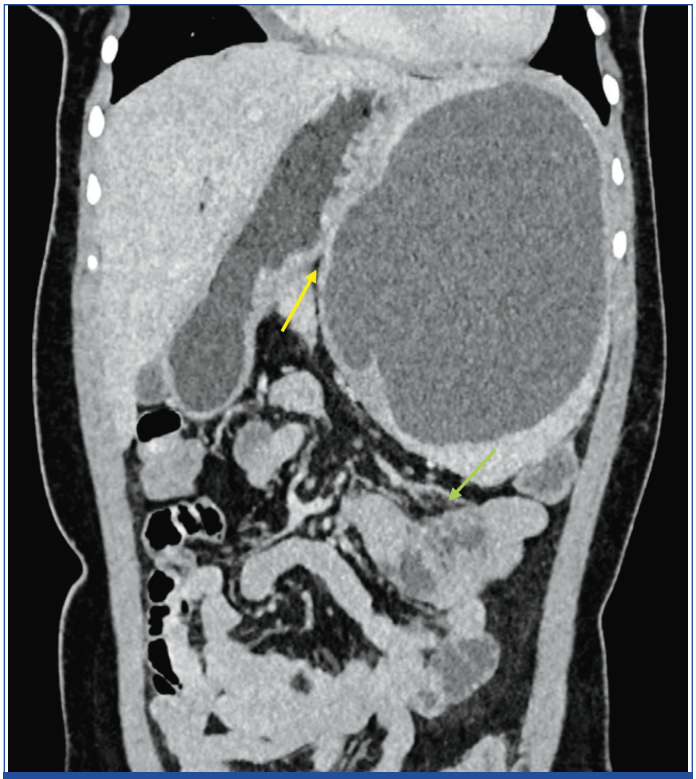


[Table/Fig-2]: Ultrasonogram of the left hypochondrium showing a large well defined unilocular cystic lesion with echogenic debris.

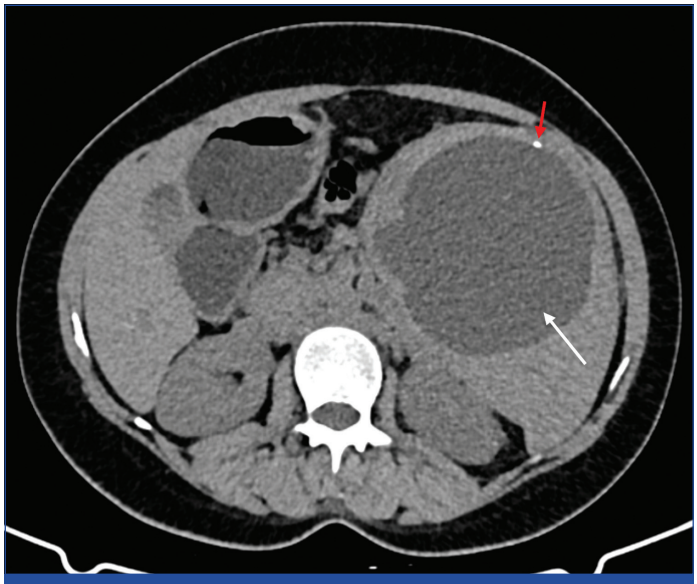
Haemoglobin was 9 g/dL (normal: 12-15 g/dL), platelet count was 0.91 lacs/mm^3 (normal: 1.5-4.5 lac/mm^3), and total leucocyte count was 3,870 cells/mm^3 (normal: 4,000-11,000 cells/mm^3). The combination of anaemia, thrombocytopenia, and leucopenia was unusual in a pancreatic pseudocyst and suggested an underlying splenic pathology with hypersplenism. Coagulation parameters were within normal limits. Peripheral blood smear revealed normocytic normochromic anaemia with normal leucocyte morphology and no haemoparasites. Liver function tests were unremarkable, making hepatic dysfunction or coagulopathy unlikely contributors to the observed cytopenias. Furthermore, the patient had no documented history suggestive of pancreatitis. Serum amylase and lipase levels were 54 U/L (normal: 30-110 U/L) and 77 U/L (normal: 0-160 U/L), respectively, both within normal limits. These findings did not support the diagnosis of pancreatic pseudocyst.

In view of the discrepancy between the ultrasonographic impression and the clinical-biochemical findings, CECT of the abdomen was performed. CECT demonstrated a well-defined non-enhancing cystic lesion measuring approximately 13×11.5×14 cm arising from the lower pole of the spleen. A few peripheral calcific specks and a few thin incomplete septations without nodularity were noted. Mild enhancement was confined to the septations on post-contrast images. No mural nodules, irregular wall thickening, or enhancing solid components were identified. These findings favoured a benign non-neoplastic cystic lesion. The lesion exerted significant mass effect on the stomach, explaining the patient's longstanding symptoms of early satiety and postprandial vomiting. Splenomegaly was also evident on CT [Table/Fig-3-5].

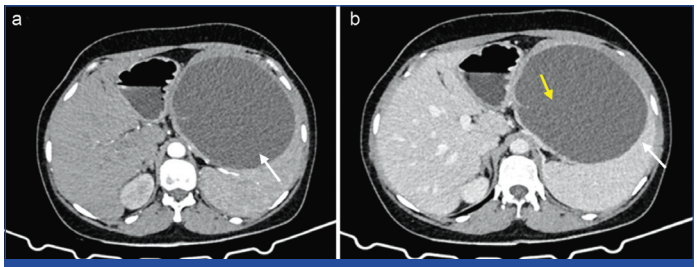
The lesion measured 13×12×13 cm on ultrasonography and 13×11.5×14 cm on CECT, demonstrating good radiologic concordance. At this stage, the diagnosis was revised from pancreatic pseudocyst to a primary splenic cystic lesion. Hydatid cyst remained a consideration because of the large size and peripheral calcific foci. There were no daughter cysts, detached membranes, or other radiological features characteristic of hydatid disease. A foregut duplication cyst was less likely because the lesion was clearly intraparenchymal within the spleen. A true splenic cyst could not be excluded radiologically. Due to the large size of the lesion and the persistence of symptoms and haematological abnormalities suggestive of hypersplenism, surgical management was planned. Open splenectomy was planned because of the large size of the lesion.



[Table/Fig-5]: Coronal Contrast enhanced CT of venous phase showing gross splenomegaly with pseudocyst showing an internal incomplete enhancing septation. Mass effect on stomach medially (yellow arrow) and displacement of bowel loops inferiorly (green arrow).



[Table/Fig-3]: Axial non-contrast CT demonstrating a fairly well-defined hypodense cyst of size ~ 13 x 11.5 x 14 cm seen occupying the lower pole of splenic parenchyma (white arrow). A peripheral calcific spec seen (red arrow). No internal calcifications/solid components.

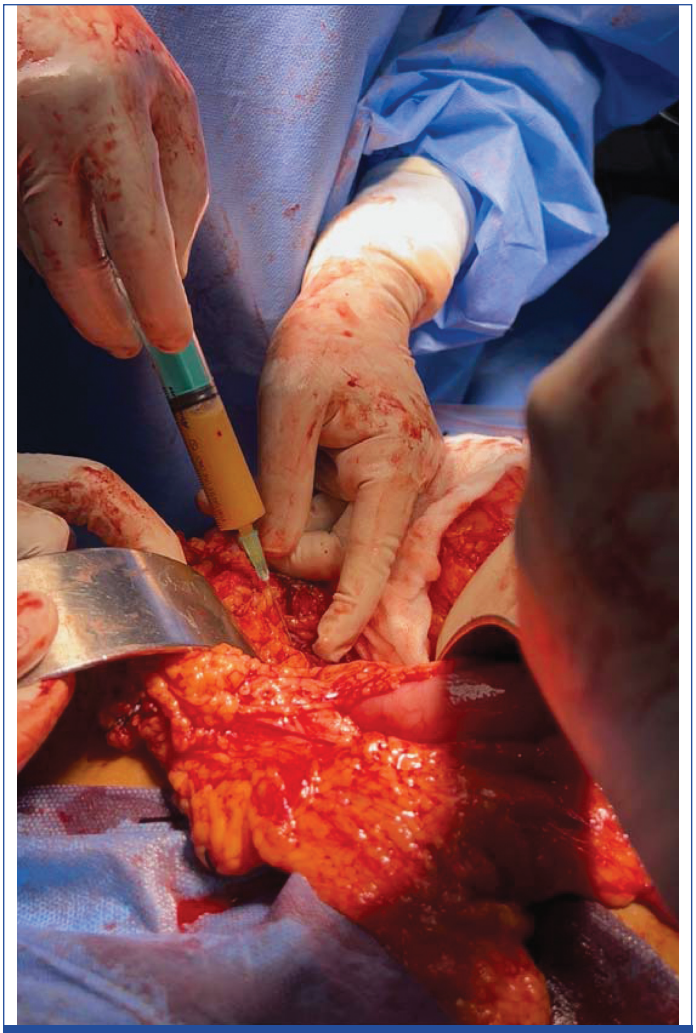


[Table/Fig-4]: Axial contrast-enhanced CT of arterial (a) and venous (b) phases demonstrating a fairly well-defined non-enhancing cyst at the lower pole of the splenic parenchyma (white arrows). An incomplete septation is noted within (yellow arrow), which on post-contrast images shows mild enhancement. No internal calcifications or solid-enhancing components.

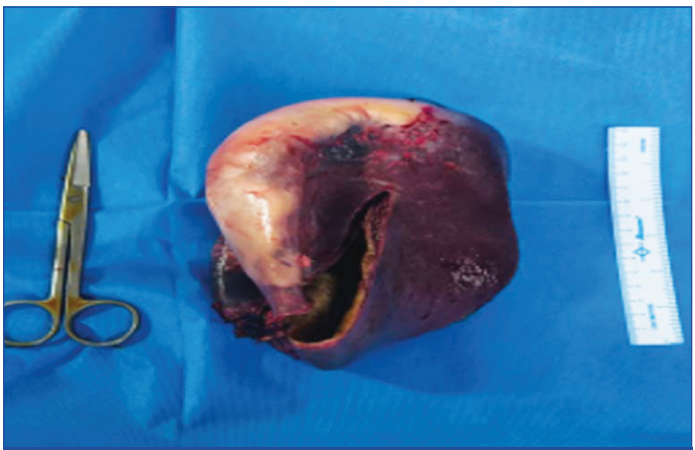
On intraoperative findings, a giant cyst involving much of the spleen was identified. Greenish-yellow fluid was aspirated, and a cytology specimen was submitted [Table/Fig-6]. Gram stain demonstrated a few pus cells with occasional Gram-positive cocci in pairs, while culture showed no growth. Ziehl-Neelsen staining for acid-fast bacilli was negative, and GeneXpert (CBNAAT) did not detect *Mycobacterium tuberculosis*. Cytology identified scattered neutrophils, lymphocytes, and cyst macrophages within an inflammatory background. No atypical or malignant cells were seen. The greenish-yellow appearance of the fluid was considered most likely secondary to chronic degeneration of blood products, cholesterol-rich debris, and inflammatory cellular contents within the longstanding cyst.

On gross examination, a splenectomy specimen measuring 16×15×8 cm was identified. On cut examination, a multiloculated cyst measuring 11×10×8 cm was detected [Table/Fig-7]. The slightly smaller pathological dimensions likely reflect postexcision collapse of the cyst cavity following drainage of its contents and specimen fixation.

Histopathological examination demonstrated marked congestion of the splenic parenchyma with dilated sinusoids and veins and prominent haemosiderin deposition. The cyst wall was markedly thickened and fibrotic, containing foamy histiocytes, siderophages, focal calcification, and cholesterol clefts. Despite extensive sampling, no epithelial lining was identified, confirming the diagnosis of a splenic pseudocyst [Table/Fig-8].



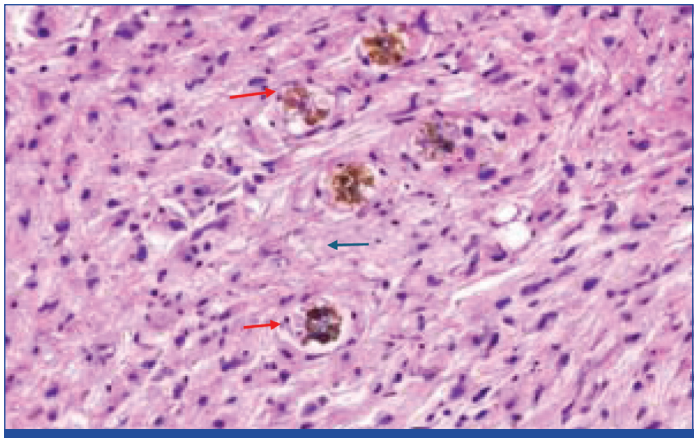
[Table/Fig-6]: Intraoperative image of the greenish yellow aspirate of the splenic pseudocyst.



[Table/Fig-7]: Gross photograph of the splenectomy specimen measuring 16×15×8 cm and demonstrating a large splenic cyst.

The postoperative course was largely uneventful. The patient was managed with adequate analgesia, incentive spirometry, breathing exercises, and chest physiotherapy beginning on postoperative day 3. Blood pressure monitoring was performed regularly in view of pre-existing hypertension. On postoperative day 4, she developed elevated blood pressure readings up to 170/90 mmHg and was evaluated by the cardiology team. Antihypertensive therapy was optimised with continuation of nifedipine and enalapril, along with intravenous labetalol as required. Blood pressure subsequently stabilised.

The patient was discharged on postoperative day 8 in stable condition. The patient had received preoperative vaccination with Pneumococcal Polysaccharide Vaccine (PPV23), meningococcal vaccine, and *Haemophilus influenzae* type b (Hib) vaccine. At approximately four weeks of follow-up, the patient reported symptomatic improvement.



[Table/Fig-8]: Histopathological section (H&E stain, X400) of a splenic pseudocyst demonstrating numerous haemosiderin-laden macrophages (siderophages) (red arrows) with focal foamy histiocytes (blue arrow). No epithelial lining is identified, supporting the diagnosis of a splenic pseudocyst.

Repeat haematological evaluation demonstrated improvement in haemoglobin levels (10.1 g/dL), normalisation of total leucocyte count (9200 cells/mm^3), and resolution of thrombocytopenia with a platelet count of 4.75 lacs/mm^3, supporting hypersplenism as the cause of the preoperative cytopenias.

DISCUSSION

Splenic pseudocysts are uncommon and constitute a small proportion of non-parasitic splenic cysts. They are mostly said to develop after trauma, resulting in the liquefaction and organisation of intrasplenic haematoma. Non-traumatic pseudocysts are relatively rare and less understood [1]. Clinical presentation would depend on the size of the cyst. Small cysts are asymptomatic, whereas large cysts can cause abdominal fullness, early satiety, vomiting, respiratory symptoms, or a palpable abdominal mass due to compression of other organs [2,3].

The exact pathogenesis of non-traumatic splenic pseudocysts remains incompletely understood. Unlike traumatic pseudocysts, which develop following organisation and liquefaction of intrasplenic haematomas, non-traumatic pseudocysts have been associated with spontaneous intracystic haemorrhage, infarction, infection, and degeneration of pre-existing splenic lesions [1,4]. In the present case, the absence of trauma together with the histopathological findings of a thick fibrotic wall, siderophages, cholesterol clefts, and focal calcification suggests chronic organisation of a previous intracystic haemorrhagic event as the most likely mechanism [4]. Epithelial lining was absent, ruling out a true splenic cyst and a foregut duplication cyst. No hydatid membranes or parasitic elements were seen, and hydatid disease was ruled out. The cytology was negative for malignancy, but no evidence of neoplastic involvement was found in the biopsy. Based on these findings, a final diagnosis of non-traumatic splenic pseudocyst was established.

Management depends on cyst size, symptoms, and complications. Small asymptomatic cysts may be managed conservatively with periodic imaging follow-up. Surgical intervention is generally recommended for symptomatic cysts, lesions larger than 5 cm, rapidly enlarging cysts, or those complicated by haemorrhage, rupture, infection, or compression of adjacent organs [1,4]. Although spleen-preserving procedures such as cyst fenestration, marsupialisation, partial splenectomy, and cystectomy have been described, total splenectomy remains appropriate for giant cysts that occupy a substantial portion of the splenic parenchyma or for cases associated with hypersplenism [4].

Abu Sabha MR et al., reported a giant non-traumatic splenic pseudocyst successfully treated with cyst aspiration and partial cystectomy [4]. Similar to the index patient, the lesion was large and symptomatic; however, hypersplenism-related pancytopenia and initial diagnostic confusion with a pancreatic pseudocyst were not

prominent features. A recently reported giant splenic pseudocyst on Radiopaedia also demonstrated a large cystic splenic lesion with internal debris and peripheral calcification [5]. Foregut duplication cyst was not considered because lesions are bounded by gastrointestinal epithelium [6], which is absent from our specimen. A thorough histologic examination did not reveal an epithelial lining, ruling out a true splenic cyst.

Another important finding was the presence of anaemia, thrombocytopenia, and leucopenia. Normal coagulation parameters and liver function tests excluded alternative causes of cytopenias, while histopathological evidence of marked splenic congestion provided a plausible explanation and supported the diagnosis of hypersplenism. The marked improvement in haemoglobin, leucocyte count, and platelet count at four-week follow-up following splenectomy further supported hypersplenism as the cause of the preoperative cytopenias.

CONCLUSION(S)

Non-traumatic splenic pseudocysts are uncommon and can present challenges with diagnosis due to their resemblance to pancreatic pseudocysts. In cases of presumed pancreatic pseudocysts, normal serum amylase and lipase levels should warrant a consideration of alternate diagnoses. For cystic lesions in the left upper quadrant, CECT can identify the organ of origin and guide management. However, the definitive diagnosis can only be made with histopathological examination.

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