

Pericardial Effusion Presenting as Isolated Right Upper Quadrant Pain with Deranged Liver Enzymes a Diagnostic Pitfall in the Emergency Department a Case Report

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Case Report

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Abstract

Background

Pericardial effusion typically presents with chest pain, dyspnoea, and positional discomfort. Presentation with isolated abdominal pain in the absence of cardiopulmonary symptoms is rare and closely mimics acute hepatobiliary pathology. Recognition is critical, since unrecognised pericardial effusion may progress to life-threatening cardiac tamponade.

Case Presentation

A 43-year-old black African woman with virologically suppressed human immunodeficiency virus infection (HIV), ulcerative colitis, and a history of previously treated latent tuberculosis (TB) infection presented to the emergency department with a one-day history of abdominal pain radiating to the flanks and epigastrium. Right upper quadrant tenderness on examination and elevated liver enzymes upon further investigation prompted a provisional diagnosis of cholecystitis. A subsequent chest radiograph showed a globular cardiac silhouette and bedside echocardiography confirmed a large pericardial effusion. Computed tomography (CT) demonstrated an effusion with features of right cardiac impairment, hepatic congestion and pericholecystic fluid, with no evidence of acute cholecystitis. Pericardiocentesis yielded haemoserous fluid, with resolution of symptoms and improvement in liver enzymes. The effusion re-accumulated within 5 days, with right ventricular diastolic collapse and this was surgically managed. The rapid recurrence raised suspicion of a potential inflammatory aetiology; however, the definitive aetiology remains unclear.

Conclusions

Pericardial effusion should be considered in patients with undifferentiated abdominal pain and liver enzyme derangement, even in the absence of typical cardiac symptoms or Beck's triad. This case highlights the potential for cardiac pathology to mimic acute hepatobiliary disease and emphasises the importance of maintaining a broad differential diagnosis in atypical abdominal presentations.

Background

The pericardial space contains 10–50 mL of serous fluid under physiological conditions. It serves as a lubricant, by reducing the friction on the movement of cardiac chambers [1]. Pericardial effusion is described as an abnormal fluid collection conventionally in excess of 50 mL within the pericardial sac [1] and may arise from a myriad of aetiologies including infectious, malignant, autoimmune, and idiopathic causes [2]. In infectious aetiology, tuberculosis represents the leading cause of pericardial effusion, and concurrent HIV infection has been recognised as an important predisposing factor in this context [2].

The clinical presentation of pericardial effusion varies widely and is determined by the curvilinear pressure–volume relationship of the pericardial sac, which governs its haemodynamic consequences. Because the pericardium has limited distensibility, a rapid accumulation of fluid can result in a

disproportionate increase in intrapericardial pressure, particularly once the reserve volume of the sac is exceeded. This can impair cardiac chamber filling and subsequently reduce cardiac output, potentially progressing to cardiac tamponade [3]. Cardiac tamponade is a potentially life-threatening emergency characterised by haemodynamic compromise due to impaired cardiac filling and classically presents with Beck's triad of hypotension, raised jugular venous pressure, and muffled heart sounds [4, 5]. However, the clinical spectrum of pericardial effusion is broad, ranging from an incidental, asymptomatic finding to a large, haemodynamically significant effusion requiring emergency intervention [2]. Symptomatic patients may present with dyspnoea, chest pain, or positional discomfort, while larger or rapidly accumulating effusions are more likely to result in cardiac tamponade [3].

Abdominal pain as the sole presenting complaint of pericardial effusion is rare and presents a significant diagnostic challenge, particularly in the emergency department where more prevalent acute abdominal pathologies, including cholecystitis, pancreatitis, and hepatitis, are encountered far more frequently. Similar cases have been reported in the literature, though they remain uncommon and the consistent theme across reported cases is the absence of classic cardiac symptoms and the presence of abdominal findings sufficient to satisfy an alternative diagnosis [5–7].

The abdominal manifestations associated with pericardial effusion may stem from mechanisms analogous to that of congestive hepatopathy (CH) secondary to right-sided heart failure. Elevated right-sided filling pressures are transmitted retrogradely to the liver, impairing hepatic venous return and producing engorgement of the inferior vena cava (IVC) and hepatic veins, hepatic congestion and stretching of the hepatic capsule, also known as the Glisson's capsule [8]. The resulting alteration in sinusoidal pressure and perfusion causes hepatocyte injury and derangement of liver enzymes [8]. Glisson's capsule is richly innervated by nociceptors and generates pain on distension, which is referred to the right upper quadrant and epigastrium and may radiate to the back or flank [5, 9]. The same haemodynamic consequence produces periportal oedema, pericholecystic fluid and small-volume ascites, further mimicking intra-abdominal disease; irritation of the adjacent diaphragmatic pleura and pericardium may contribute an additional referred component [5].

The risk of anchoring and premature diagnostic closure in this setting is considerable [10]. A patient with right upper quadrant pain, deranged liver enzymes and pericholecystic fluid on imaging will reasonably be assigned a hepatobiliary diagnosis without initially considering a cardiac aetiology. Failure to recognise the effusion risks progression to tamponade and represents an avoidable diagnostic pitfall.

We report the case of a 43-year-old woman who attended the emergency department with isolated right upper quadrant pain and was found to have a large pericardial effusion with radiological evidence of right cardiac impairment, in the complete absence of cardiac signs or symptoms. This case report was prepared in accordance to the CARE (CAse REport) guidelines [11].

Case Presentation

Patient Information

A 43-year-old black African woman presented to the emergency department with a one-day history of abdominal pain that radiated to the flanks and epigastric region. She denied chest pain, shortness of breath, cough, fever, vomiting, diarrhoea, or urinary symptoms. She denied any gynaecological symptoms including abnormal vaginal discharge or pelvic pain. A pregnancy test was negative. She denied any history of prior pericarditis, chest radiotherapy, renal impairment, or thyroid disease. She reported a four-month history of unintentional weight loss, for which her general practitioner had commenced nutritional supplementation.

Her background included HIV infection, type 2 diabetes mellitus, ulcerative colitis, depression, and uterine fibroids, and a history of latent tuberculosis infection treated in 2021. Regular medications were bictegavir, emtricitabine and tenofovir alafenamide as a single-tablet regimen, together with sertraline, atorvastatin, omeprazole and mesalazine in both oral and rectal formulations. She had no known drug allergies and lived alone, had never smoked and did not drink alcohol.

Clinical findings

On examination, her temperature was 37.5°C, blood pressure of 106/72 mmHg, heart rate of 80 beats per minute, respiratory rate of 19 breaths per minute, and oxygen saturations of 97% on air, and a Glasgow Coma Score of 15. Jugular venous pressure was not raised and heart sounds were reported as normal with no appreciable muffling on auscultation. The chest was clear on auscultation. Abdominal examination revealed tenderness in the right upper quadrant without guarding or peritonism, no hepatomegaly was noted. Pulsus paradoxus was assessed but no significant elevation was documented. A provisional diagnosis of cholecystitis was made based on the history and examination findings, however, antibiotics were not administered at this point.

Diagnostic assessment

Urinalysis was unremarkable. Blood tests demonstrated a normocytic anaemia with a haemoglobin of 88 g/L, a white cell count of $4.3 \times 10^9/\text{L}$ and an international normalised ratio (INR) of 1.12. Renal function was normal. Liver enzymes were markedly elevated, with an alanine aminotransferase (ALT) of 162 U/L, aspartate aminotransferase (AST) 189 U/L and alkaline phosphatase (ALP) 177 U/L. Amylase was normal at 84 U/L and C-reactive protein (CRP) was mildly elevated at 15.8 mg/L. The HIV viral load was undetectable at fewer than 50 copies/mL. CD4 count was not available at the time of writing.

A 12-lead electrocardiogram demonstrated sinus rhythm with low voltage QRS complexes across all leads and a prolonged PR interval of 240 ms. Electrical alternans was absent (Fig. 1). Low voltage QRS complexes are a recognised electrocardiographic feature of large pericardial effusion, attributed to the attenuating effect of the surrounding pericardial fluid on electrical signal transmission [12].

A chest radiograph demonstrated a markedly enlarged, globular cardiac silhouette, raising the possibility of pericardial effusion (Fig. 2) [13]. Bedside echocardiography performed in the emergency department confirmed a large circumferential pericardial effusion (Fig. 3). Computed tomography (CT) of the

abdomen and pelvis demonstrated a large pericardial effusion with a maximum depth of 34 mm, containing predominantly low-density fluid with regions of higher attenuation measuring up to 24 Hounsfield units, suggesting proteinaceous content (Figs. 4a and 4b). There was evidence of right cardiac impairment, with a dilated IVC and hepatic veins, periportal oedema, pericholecystic fluid surrounding a collapsed gallbladder and small-volume ascites. Importantly, there was no radiological evidence of acute cholecystitis. The pancreas, spleen, adrenal glands, kidneys and pelvic organs were unremarkable, and no lymphadenopathy or free intraperitoneal gas was identified.

The initial differential was that of acute right upper quadrant pain with hepatocellular injury, and encompassed acute cholecystitis, biliary colic, choledocholithiasis, acute viral or drug-induced hepatitis and, given the history of ulcerative colitis, an inflammatory bowel disease flare with hepatobiliary involvement. Pancreatitis was excluded biochemically. Once the pericardial effusion was identified, the pattern of a dilated IVC and hepatic veins with periportal oedema and ascites, in the absence of gallbladder wall thickening or calculi, reframed the abdominal findings as congestive hepatopathy secondary to impaired right ventricular filling rather than as primary hepatobiliary disease [8].

Therapeutic intervention

An urgent cardiology opinion was sought and the patient was transferred to a tertiary cardiac centre. Pericardiocentesis yielded 300 mL of haemoserous fluid, with complete resolution of her abdominal pain. Repeat liver function tests following pericardiocentesis showed a significant reduction in transaminases illustrated in the Table 1 below:

Table 1: Comparison of liver transaminase values on initial presentation and following pericardiocentesis

Liver Function Test	Initial Presentation (U/L)	Post-Pericardiocentesis (U/L)	Change (U/L)
ALT	162	90	~ 72
AST	189	61	~ 128
ALP	177	117	~ 60

The above table supports a causal relationship between the pericardial effusion and the hepatic dysfunction observed on initial presentation.

Follow-up and outcomes

The patient re-presented to the emergency department five days later with recurrent pain, now accompanied by breathlessness and a raised jugular venous pressure, with features consistent with Beck’s triad. Repeat echocardiography confirmed re-accumulation of a large pericardial effusion with right ventricular diastolic collapse. A pericardial window was created via a subxiphoid approach; intraoperative findings included a thickened pericardium, and 700 mL of fluid was drained. Post-operative echocardiography demonstrated only a trivial residual effusion. The rapidity of re-

accumulation, the haemoserous character of the fluid and the intraoperative finding of a thickened pericardium together favour an inflammatory, exudative process rather than a transudative effusion. The underlying could not be identified despite comprehensive investigation. The sequence of events is summarised in Table 2.

Table 2: *Timeline of clinical events*

Time point	Clinical events	Investigations, interventions and outcomes
4 months before presentation	Unintentional weight loss; nutritional supplementation commenced by the general practitioner.	No cardiorespiratory symptoms reported at any stage.
Day 0	One-day history of back pain radiating to the flanks and epigastrium; right upper quadrant tenderness. No chest pain, dyspnoea or fever. Working diagnosis: acute cholecystitis.	Haemoglobin 88 g/L; white cell count $4.3 \times 10^9/L$; ALT 162 U/L; AST 189 U/L; ALP 177 U/L; amylase 84 U/L; CRP 15.8 mg/L; HIV viral load <50 copies/mL. Chest radiograph: globular cardiac silhouette. Bedside echocardiography: large pericardial effusion. CT abdomen and pelvis: 34 mm pericardial effusion, dilated IVC and hepatic veins, periportal oedema, pericholecystic fluid, small-volume ascites; no cholecystitis.
Day 0–1	Urgent cardiology referral and transfer to a tertiary cardiac centre.	Pericardiocentesis: 300 mL of haemoserous fluid drained. Complete resolution of abdominal pain.
Day 5	Re-presentation with recurrent pain, new breathlessness, raised jugular venous pressure and features of Beck’s triad.	Repeat echocardiography: re-accumulated large effusion with right ventricular diastolic collapse. Pericardial fluid negative for malignant cells, bacterial culture, mycobacteria and fungi.
Day 5–6	Subxiphoid pericardial window performed.	Thickened pericardium noted intraoperatively; 700 mL drained. Post-operative echocardiography: trivial residual effusion.
Follow-up	No definitive aetiology identified despite comprehensive investigation	

Pericardial fluid analysis

Pericardial fluid cytology demonstrated a lymphocyte-predominant reactive effusion with reactive mesothelial cells and macrophages, with no evidence of malignancy. Microbiological investigations were negative, including routine bacterial culture, acid-fast bacilli microscopy, mycobacterial PCR, and fungal microscopy and culture. Overall, the findings did not identify a malignant or infectious cause, and an inflammatory or autoimmune aetiology remained under consideration.

Discussion and conclusions

Pericardial effusion presenting exclusively with abdominal pain, in the absence of chest pain or dyspnoea, is rare and carries a substantial risk of misdiagnosis in the emergency setting [14]. This case illustrates how convincingly a primarily cardiac pathology can mimic acute intra-abdominal disease. A working diagnosis of cholecystitis was reached on entirely reasonable grounds before the underlying aetiology was recognised.

The physiological basis of abdominal pain in this context is well described. Accumulation of pericardial fluid impairs diastolic filling, elevating right-sided pressures and reducing cardiac output [5]. Congestion of the IVC and hepatic venous system follows, and distension of the hepatic capsule manifests as right upper quadrant or epigastric pain that frequently radiates to the back or flank [5]. In this patient the CT findings of a dilated IVC, dilated hepatic veins, periportal oedema, pericholecystic fluid and small-volume ascites provided radiological corroboration of this entire haemodynamic sequence, and simultaneously excluded acute cholecystitis as the primary diagnosis.

The initial clinical picture was misleading in several respects. Right upper quadrant tenderness, markedly elevated transaminases and pericholecystic fluid on imaging are all consistent with hepatobiliary pathology and understandably directed suspicion in that direction. The case therefore demonstrates the hazard of premature diagnostic closure in undifferentiated abdominal pain, and the value of deliberately generating a broad differential even when the initial findings appear internally consistent [10]. A useful discriminating feature, in retrospect, is that pericholecystic fluid accompanied by a collapsed rather than distended gallbladder, in the absence of wall thickening or calculi, is more suggestive of systemic venous congestion than of primary cholecystitis.

This patient did not exhibit Beck's triad at first presentation, and the absence of these cardinal signs contributed materially to the diagnostic difficulty. It is worth emphasising that this is the rule rather than the exception: in contemporary series the full triad is uncommon, hypotension in particular is frequently absent, and dyspnoea is the most consistently reported symptom [14]. Reliance on the classical triad to exclude a significant effusion is therefore unsafe. The case further illustrates that tamponade physiology may evolve insidiously, producing measurable haemodynamic consequences such as hepatic venous congestion and abdominal pain well before overt cardiovascular compromise becomes clinically apparent.

The chest radiograph proved pivotal. A symmetrically enlarged, globular cardiopericardial silhouette with smoothed contours is the classical appearance of a large effusion, although it is neither sensitive nor specific in isolation and typically requires an effusion of at least 200–250 mL to become apparent [13]. Its principal value in this case was as a low-cost prompt to a cardiac investigation that would not otherwise have been requested. Bedside echocardiography then confirmed the diagnosis within minutes. Point-of-care ultrasonography is the recommended first-line modality where pericardial disease is suspected and is associated with earlier drainage of pericardial effusions in the emergency department [2, 3, 15]; this case adds to the argument for a low threshold for a focused cardiac view in patients with unexplained upper abdominal pain and a congestive pattern on cross-sectional imaging.

The aetiology of the effusion merits particular consideration. People living with HIV carry a substantially elevated risk of pericardial disease, including effusion and tamponade, even in the era of effective antiretroviral therapy [16, 17]. This patient had the additional risk factors of a documented history of latent tuberculosis and four months of unintentional weight loss with a normocytic anaemia, a constellation that mandates thorough exclusion of both tuberculous pericarditis and occult malignancy [18–20]. Pericardial fluid analysis was negative for mycobacteria, malignant cells and bacterial and fungal infection; it should be noted, however, that the sensitivity of fluid microscopy and culture for tuberculous pericarditis is limited, and that pericardial biopsy with histology and mycobacterial culture, adenosine deaminase estimation and nucleic acid amplification testing add materially to the diagnostic yield [19, 20]. Rapid re-accumulation, haemoserous fluid and a thickened pericardium found at operation are all consistent with an inflammatory, exudative process and argue against a simple transudate.

The clinical course also illustrates the limitations of pericardiocentesis alone. Simple drainage relieved symptoms immediately but was followed by re-accumulation within five days, at which point tamponade physiology was established. Surgical creation of a pericardial window provides more durable drainage where an effusion recurs or where the underlying process is likely to persist, and additionally offers the opportunity to obtain pericardial tissue for histological and microbiological diagnosis [1, 2, 21].

Comparable presentations have been described, though they remain uncommon. Reported cases include tamponade masquerading as abdominal pain diagnosed by point-of-care ultrasonography in a patient subsequently found to have systemic lupus erythematosus [5], tamponade presenting as an acute abdomen with a preoperative diagnosis of acalculous cholecystitis [6], and right upper quadrant pain as the initial manifestation of a malignant effusion from lung adenocarcinoma [7]. The consistent theme is the absence of classical cardiac symptoms alongside abdominal findings sufficiently persuasive to satisfy an alternative diagnosis, resulting in delayed recognition of the pericardial pathology. The present case extends this literature by describing a patient in whom pericardial effusion presented solely as abdominal pain, subsequently progressed to overt tamponade, and was accompanied by the complete radiological findings of hepatic venous congestion.

The principal limitation of this report is that the aetiology of the effusion had not been established at the time of writing, and that the report describes a single patient; no causal inference can be drawn beyond the temporal association between drainage and symptom resolution, which was nonetheless immediate and reproducible across two interventions.

In conclusion, this case highlights pericardial effusion as a rare but important cause of acute abdominal pain in the emergency department. The absence of classical cardiac symptoms and of Beck's triad, combined with clinical and laboratory findings suggestive of hepatobiliary disease, created a significant diagnostic challenge. Recognition of the globular cardiac silhouette on the chest radiograph and prompt bedside echocardiography were decisive in establishing the correct diagnosis. Clinicians should maintain awareness of cardiac aetiologies when evaluating undifferentiated abdominal pain, particularly

in patients with risk factors for pericardial disease such as HIV infection and previous tuberculosis, in order to avoid diagnostic delay and haemodynamic deterioration.

Abbreviations

ALP: alkaline phosphatase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: C-reactive protein; CT: computed tomography; HIV: human immunodeficiency virus; INR: international normalised ratio; IVC: inferior vena cava.

Declarations

Ethics and consent to participate

Not applicable.

Consent to publish

Written informed consent was obtained from the patient for publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

All data generated or analysed during this study are included in this published article.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

AJ drafted the manuscript. GJ contributed to the drafting of the manuscript and reviewed it. LSD and PV contributed to the design of the report and to the analysis and interpretation of the case, and reviewed the manuscript. All authors read and approved the final manuscript.

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Figures

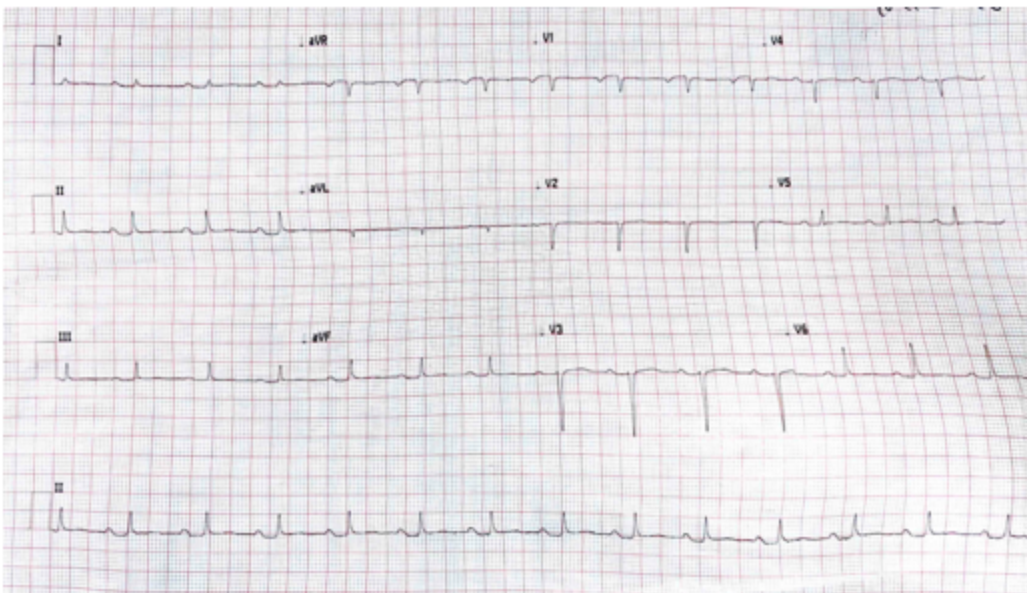


Figure 1

A 12-lead electrocardiogram demonstrating sinus rhythm with low voltage QRS complexes across all leads and a prolonged PR interval. Electrical alternans is absent

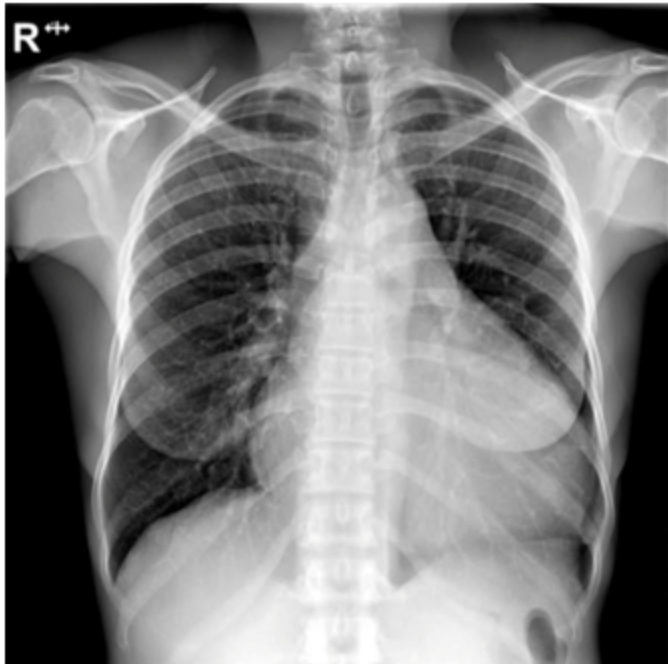


Figure 2

Posteroanterior chest radiograph demonstrating a markedly enlarged, globular cardiac silhouette raising clinical suspicion for a large pericardial effusion.

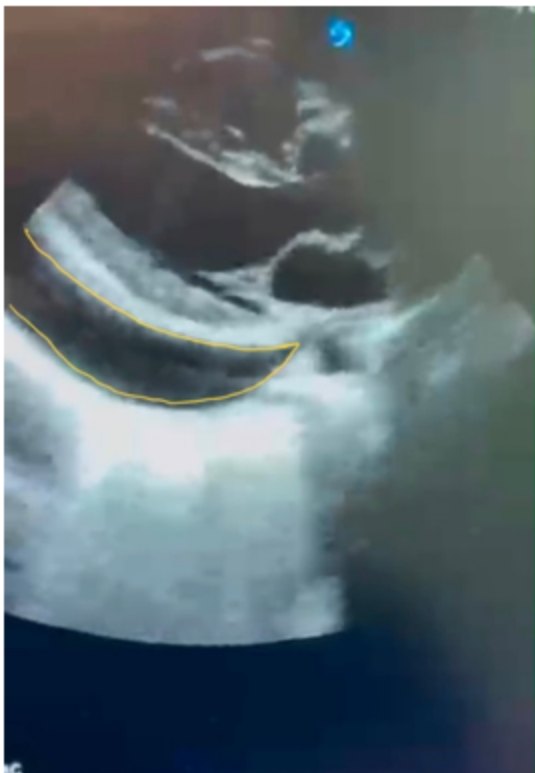


Figure 3

Bedside echocardiographic image demonstrating a large pericardial effusion surrounding the cardiac chambers (outlined in yellow)



a



b

Figure 4

a : *Sagittal CT demonstrating the large pericardial effusion (outlined in black) in relation to the abdominal viscera, illustrating the anatomical proximity contributing to the referred abdominal symptomatology*

4b : *Coronal CT demonstrating the large pericardial effusion (outlined in yellow) with evidence of vascular congestion consistent with right cardiac impairment*

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