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| RESEARCH ARTICLE

Rare Coexistence of Abdominal Aortic Aneurysm and Pericardial Effusion: A Case Report

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| ABSTRACT

We report the case of a 59-year-old male who presented to the emergency department with a two-week history of progressive shortness of breath, intermittent fever, fatigue, and reduced exercise tolerance. He later developed non exertional central chest discomfort, vague abdominal pain, and poor appetite with unintentional weight loss. There was no history of recent invasive procedures or known cardiac disease. On presentation, he was febrile, tachycardic, and mildly hypoxemic, with elevated jugular venous pressure, bilateral basal crackles, and a subtle early diastolic cardiac sound. Abdominal examination revealed a suspected pulsatile mass. Initial investigations showed raised inflammatory markers and leukocytosis. Chest radiography demonstrated cardiomegaly, while bedside ultrasound revealed a moderate pericardial effusion and a fusiform infrarenal abdominal aortic dilatation. Computed tomography of the abdomen confirmed an abdominal aortic aneurysm with features suggestive of infective involvement. Blood cultures later grew a streptococcal species consistent with infective endocarditis. The patient was managed with targeted intravenous antibiotics after initial empirical therapy, along with close hemodynamic monitoring. Cardiology input supported conservative management of the pericardial effusion as there were no signs of tamponade. Vascular surgery recommended initial non operative management of the suspected mycotic aneurysm with strict blood pressure control and prolonged antibiotic therapy. Multidisciplinary care was central to management, including infectious diseases, cardiology, and vascular surgery teams. This case highlights an unusual presentation of infective endocarditis complicated by both pericardial effusion and abdominal aortic aneurysm, emphasizing the importance of early imaging, blood culture diagnosis, and multidisciplinary coordination in detecting and managing systemic infectious vascular complications.

| KEYWORDS

Aortic Aneurysm, Pericardial effusion, Arteritis, Vasculitis, Serositis, Pericarditis, Infective Endocarditis

| ARTICLE INFORMATION

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Introduction

Infective endocarditis is a serious and potentially life threatening condition caused by microbial infection of the endocardial surface of the heart, most commonly involving the cardiac valves. Despite advances in diagnostic imaging, antimicrobial therapy, and surgical techniques, it remains associated with high morbidity and mortality, particularly when diagnosis is delayed or complications develop. The disease is characterized by a wide spectrum of clinical manifestations ranging from subacute constitutional symptoms to acute fulminant sepsis with multiorgan involvement [1,2]. The clinical course is often complicated by systemic embolization and metastatic infection, which may involve multiple vascular territories and distant organs.

One of the most important complications of infective endocarditis is systemic embolization, which occurs due to fragmentation of friable vegetations on the infected valve. These emboli can travel to various organs, leading to infarction, abscess formation, or infected aneurysm development. The risk of embolic events is highest in the early phase of the disease and is strongly associated with vegetation size, mobility, and causative organism [15]. Embolic phenomena may involve the brain, spleen, kidneys, coronary circulation, and peripheral vasculature, making infective endocarditis a true multisystem disease [3,12].

Abdominal aortic aneurysm in the setting of infection, also referred to as a mycotic aneurysm, is a rare but severe complication of bacteremia or septic embolization. The term mycotic is historical and does not imply fungal infection, but rather reflects the mushroom like appearance of the aneurysm. Mycotic aneurysms result from microbial invasion of the arterial wall, leading to destruction of the media and adventitia, weakening of the vessel wall, and subsequent aneurysmal dilatation. In the context of infective endocarditis, septic emboli can lodge in the vasa vasorum or arterial wall, initiating this destructive process [6,7].

The abdominal aorta is one of the less commonly affected sites, but when involved, the condition carries a high risk of rupture and mortality. Patients may present with nonspecific symptoms such as abdominal or back pain, fever, or signs of systemic infection. In some cases, the aneurysm is detected incidentally during imaging performed for evaluation of endocarditis or embolic complications. The coexistence of abdominal aortic aneurysm and infective endocarditis should raise strong suspicion for a septic vascular process [5,6].

Pericardial effusion in the setting of infective endocarditis is less common but clinically significant. It may result from several mechanisms, including direct extension of infection, hematogenous spread, immune mediated inflammation, or complications of septic embolization. In severe cases, purulent pericarditis may develop, which is associated with rapid clinical deterioration and high mortality if not promptly treated. Even non purulent effusions can contribute to hemodynamic compromise, particularly when combined with other cardiovascular pathology [10,12].

The coexistence of abdominal aortic aneurysm and pericardial effusion in a single patient is unusual and suggests a disseminated infectious process with widespread vascular involvement. This combination should prompt clinicians to consider an underlying source of persistent bacteremia, with infective endocarditis being a leading cause. The systemic nature of endocarditis allows it to seed multiple vascular beds, leading to complex and atypical clinical presentations that may initially obscure the diagnosis [1,3].

The diagnosis of infective endocarditis is based on a combination of clinical, microbiological, and echocardiographic findings. The modified Duke criteria remain the standard diagnostic framework and include major criteria such as positive blood cultures and evidence of endocardial involvement on echocardiography [1]. Transesophageal echocardiography is particularly useful in detecting vegetations, abscesses, and complications such as valve perforation or fistula formation. Advanced imaging modalities, including computed tomography and positron emission tomography, have also improved the detection of extracardiac complications such as vascular infection and aneurysm formation [14].

The management of infective endocarditis requires prolonged intravenous antibiotic therapy tailored to the causative organism, along with careful monitoring for complications. Empirical therapy is initiated in patients with high clinical suspicion while awaiting culture results. Once identified, targeted antimicrobial therapy is continued for several weeks. Surgical intervention may be required in cases of heart failure, uncontrolled infection, or prevention of embolic events, particularly in the presence of large vegetations or structural cardiac damage [1,13].

Management of mycotic abdominal aortic aneurysm is complex and often requires a combination of prolonged antibiotic therapy and surgical intervention. Options include open surgical repair with debridement of infected tissue or endovascular repair in selected high risk patients. However, endovascular approaches carry a risk of persistent infection due to retained infected material. Early recognition is therefore critical to prevent rupture, which is often catastrophic [6,7].

Pericardial effusion associated with infective endocarditis requires careful evaluation of its hemodynamic impact. In cases where tamponade physiology develops, urgent pericardial drainage is necessary. In less severe cases, treatment of the underlying

infection may lead to resolution of the effusion. However, close monitoring is essential, as rapid progression can occur in the setting of ongoing systemic infection [10].

The coexistence of these complications highlights the aggressive nature of infective endocarditis and its potential for widespread vascular involvement. It also underscores the importance of early diagnosis, as delayed treatment is associated with increased risk of embolic and structural complications. The presence of multiple vascular abnormalities should prompt immediate investigation for a unifying infectious source [2,3].

From a pathophysiological perspective, infective endocarditis represents a dynamic interaction between microbial virulence and host immune response. Bacterial adherence to damaged endocardium leads to vegetation formation, which consists of fibrin, platelets, and microorganisms. These vegetations serve as a continuous source of bacteremia and embolization. In addition, circulating inflammatory mediators contribute to endothelial dysfunction and vascular injury, facilitating complications such as aneurysm formation and serosal inflammation [12,15].

This case is important because it demonstrates an unusual combination of abdominal aortic aneurysm and pericardial effusion as manifestations of underlying infective endocarditis. Such a presentation can easily be misinterpreted as separate pathological processes unless a high index of suspicion is maintained. Recognition of this pattern is essential, as early diagnosis allows timely initiation of antimicrobial therapy and consideration of surgical intervention, which can significantly improve outcomes [1,6].

In summary, infective endocarditis is a multisystem disease with a wide spectrum of complications resulting from septic embolization and hematogenous spread. The development of mycotic abdominal aortic aneurysm and pericardial effusion reflects advanced systemic involvement and indicates a severe disease state. Early recognition, prompt imaging, and coordinated multidisciplinary management are essential to reduce morbidity and mortality. This case underscores the importance of considering infective endocarditis in patients presenting with unusual combinations of vascular and cardiac findings, as timely diagnosis remains the key determinant of prognosis.

Case Presentation

This case involves a 59 year old male who presented to the emergency department with progressive shortness of breath, intermittent fever, and generalized fatigue over a period of approximately two weeks. He had been in his usual state of health until about 14 days prior to presentation, when he developed low grade fever associated with chills and reduced appetite. This was followed by generalized malaise and reduced exercise tolerance, which gradually worsened. He initially attributed his symptoms to a viral illness and did not seek medical attention. Over the following days, he developed increasing breathlessness on exertion, which progressed to dyspnea at rest.

About one week prior to admission, he began to notice intermittent episodes of dull central chest discomfort, not related to exertion and not radiating. He also reported vague abdominal discomfort and early satiety. There was no history of cough, sputum production, hemoptysis, or pleuritic chest pain. He denied palpitations, syncope, or previous similar episodes. He also noted poor appetite and unintentional weight loss over the same period. There was no history of recent dental procedures, invasive interventions, or known source of infection.

His past medical history was notable only for well controlled hypertension diagnosed five years earlier. He was not known to have diabetes mellitus, ischemic heart disease, or chronic kidney disease. He was not on anticoagulants or antiplatelet agents other than low dose aspirin prescribed for primary prevention. He had no known history of valvular heart disease, congenital cardiac abnormalities, or prior hospital admissions for cardiac conditions. He was a non smoker and did not consume alcohol. There was no history of intravenous drug use.

He lived independently and was functionally active prior to this illness. There was no recent travel history, no exposure to sick contacts, and no known contact with tuberculosis. His vaccination history was unclear. There was no family history of aneurysmal disease, sudden cardiac death, or known connective tissue disorders.

On the day of presentation, his symptoms worsened significantly, with marked shortness of breath even at rest. His family noted that he appeared visibly fatigued and was unable to complete full sentences without pausing to breathe. This prompted presentation to the emergency department.

On arrival, he appeared ill and in moderate respiratory distress. He was alert but fatigued and slow to respond. His temperature was 38.2°C, heart rate 118 beats per minute, blood pressure 102 over 64 millimeters of mercury, respiratory rate 24 breaths per minute, and oxygen saturation 91 percent on room air. He was placed on supplemental oxygen via nasal cannula with improvement in oxygen saturation.

Cardiovascular examination revealed a tachycardic rhythm with normal first and second heart sounds. No obvious murmurs were initially appreciated. However, there was a subtle early diastolic sound suggestive of possible valvular involvement on repeat auscultation. Peripheral pulses were palpable and symmetrical. Jugular venous pressure was mildly elevated.

Respiratory examination showed reduced air entry at the lung bases bilaterally with fine inspiratory crackles. There were no wheezes or signs of focal consolidation. The trachea was midline.

Abdominal examination revealed a soft but mildly distended abdomen with tenderness in the periumbilical region. A pulsatile abdominal mass was suspected on deep palpation, raising concern for underlying aortic pathology. There was no guarding or rebound tenderness. Bowel sounds were present. No organomegaly was appreciated.

Neurological examination was unremarkable, with the patient fully oriented and no focal neurological deficits. Skin examination showed no petechiae, splinter hemorrhages, or Janeway lesions at the time of initial assessment.

Initial bedside ultrasound was performed in view of suspected cardiovascular pathology. This revealed the presence of a pericardial effusion of moderate size without clear signs of tamponade physiology at that stage. In addition, a fusiform dilatation of the infrarenal abdominal aorta was noted, raising suspicion for an abdominal aortic aneurysm. Given the combination of findings and the febrile presentation, an underlying infectious etiology involving the cardiovascular system was strongly suspected.

The overall clinical picture was therefore suggestive of a systemic infectious process with cardiovascular involvement, raising concern for infective endocarditis complicated by vascular infection and pericardial involvement. The patient was admitted urgently for further evaluation, blood cultures, advanced imaging, and initiation of empiric intravenous antibiotic therapy.

Diagnostic Workup:

Following initial assessment in the emergency department, the patient was admitted for urgent evaluation due to concern for systemic infection with cardiovascular involvement. Although initially hemodynamically stable, he remained febrile with persistent tachycardia and progressive symptoms of breathlessness. Given the suspicion of infective endocarditis with possible embolic complications, he was closely monitored with continuous cardiac and oxygen saturation monitoring. Serial vital signs showed intermittent low grade fever and borderline blood pressure, although no frank shock was present at the time of admission.

Baseline laboratory investigations demonstrated elevated inflammatory markers with leukocytosis and neutrophil predominance. C reactive protein and erythrocyte sedimentation rate were markedly raised. Renal function tests were within normal range initially, with no evidence of acute kidney injury. Liver function tests showed mild elevation of transaminases without significant cholestatic pattern. Serum lactate levels were not elevated on admission. Cardiac enzymes were within normal limits, reducing the likelihood of acute coronary syndrome as a cause of chest discomfort.

Electrocardiography showed sinus tachycardia without ischemic changes or conduction abnormalities. Given the presence of fever and suspected endocarditis, multiple sets of blood cultures were obtained prior to initiation of antimicrobial therapy. These were sent for aerobic and anaerobic bacterial growth.

Chest imaging was performed early in the diagnostic workup. Chest radiograph demonstrated an enlarged cardiac silhouette consistent with cardiomegaly, with no focal pulmonary consolidation or pleural effusion. These findings are shown in Figure 1. The cardiomegaly raised concern for underlying structural cardiac involvement, including possible pericardial effusion or valvular disease in the context of infective endocarditis.

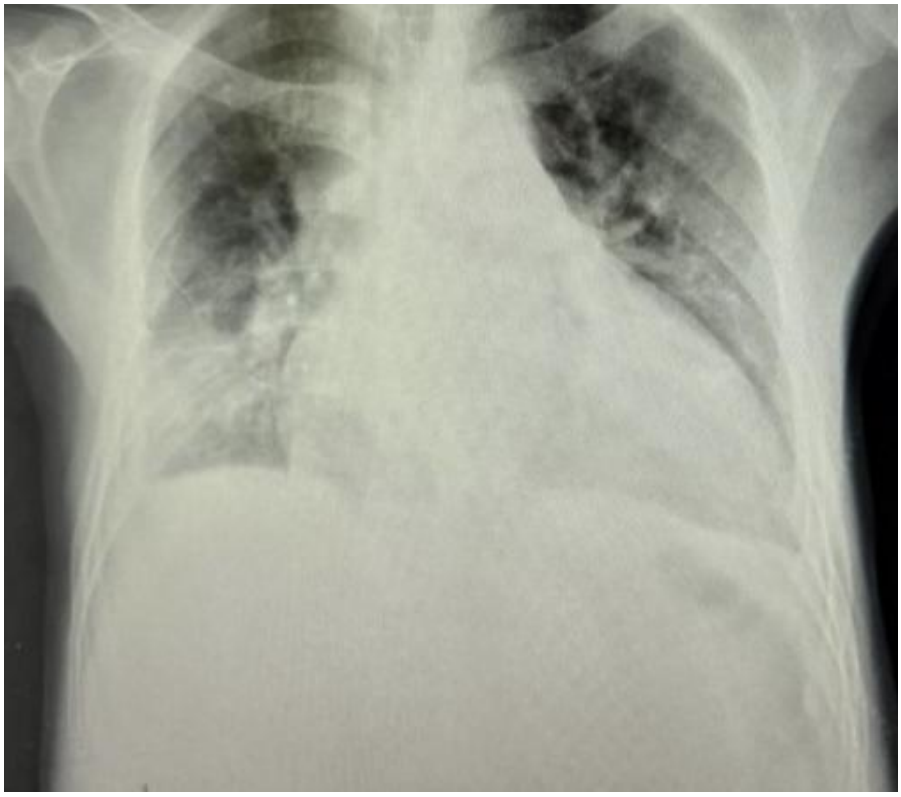


Figure 1: Chest X ray showing cardiomegaly with enlarged cardiac silhouette.

Given the radiographic findings and ongoing clinical suspicion, bedside transthoracic echocardiography was performed. This demonstrated a moderate circumferential pericardial effusion without clear evidence of tamponade physiology at the time of examination. There was no obvious valvular vegetation identified on transthoracic imaging, although image quality was limited. The pericardial effusion findings are shown in Figure 2.

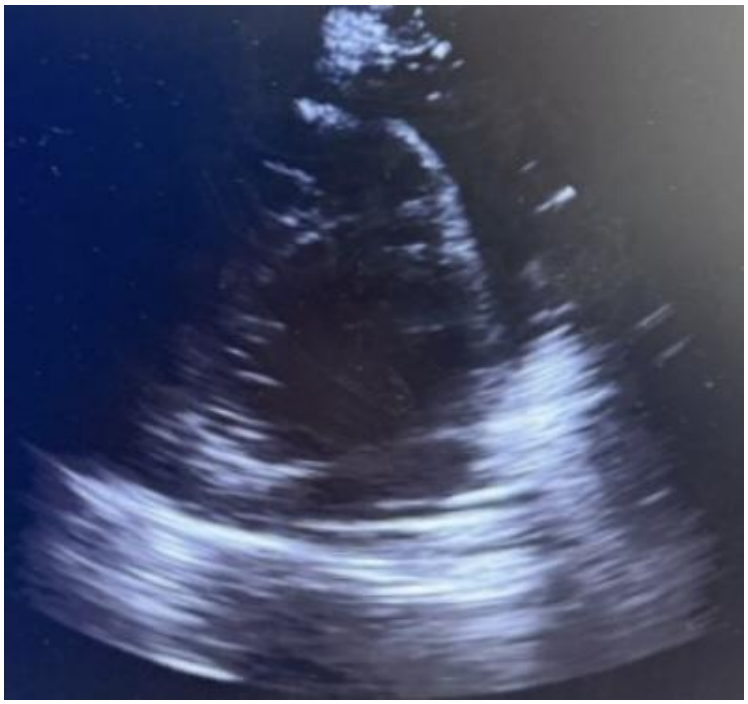
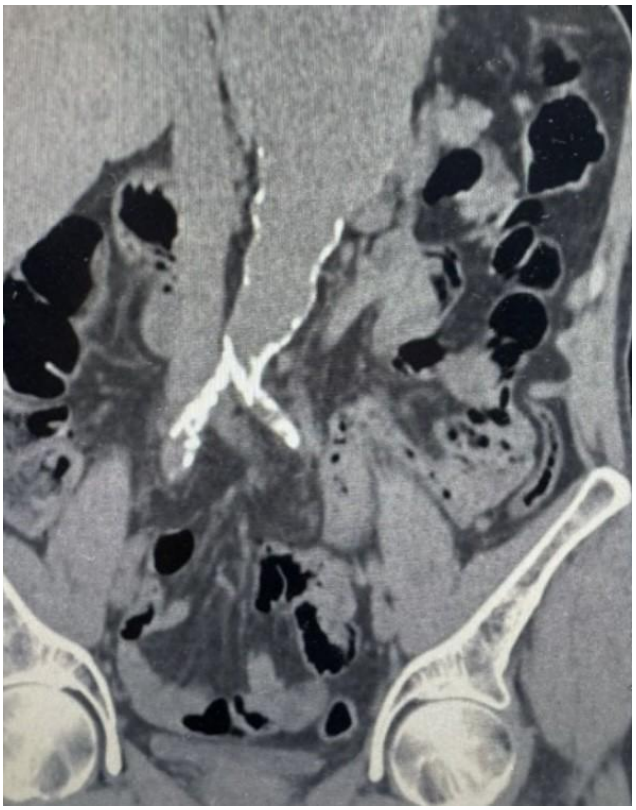


Figure 2: Ultrasound image demonstrating moderate pericardial effusion.

In view of suspected infective endocarditis and possible embolic phenomena, further imaging was undertaken to evaluate for systemic vascular involvement. Computed tomography of the abdomen with contrast was performed. This revealed a saccular infrarenal abdominal aortic aneurysm with surrounding inflammatory changes, highly suggestive of an infective or mycotic aneurysm rather than a degenerative process. There was no evidence of rupture or retroperitoneal hemorrhage at that stage.



These findings are shown in Figure 3.

Figure 3: CT abdomen with contrast showing infrarenal abdominal aortic aneurysm.

Following these imaging findings, the diagnostic impression strongly favored infective endocarditis complicated by systemic embolization with vascular seeding. The combination of pericardial effusion and abdominal aortic aneurysm in the setting of fever and elevated inflammatory markers supported a disseminated infectious process. Blood cultures later became positive for a Gram positive organism consistent with streptococcal species, further strengthening the diagnosis of infective endocarditis.

Repeat laboratory testing over the subsequent 48 hours showed progressive rise in inflammatory markers. Renal and liver function remained stable during the early phase of admission. There was no initial evidence of coagulopathy. Serial clinical assessment continued to show persistent low grade fever and intermittent tachycardia despite supportive care.

Overall, the diagnostic workup demonstrated evidence of infective endocarditis with systemic vascular complications, including pericardial effusion and mycotic abdominal aortic aneurysm. The imaging findings played a central role in establishing the extent of disease and guiding further management decisions, including the need for prolonged intravenous antibiotic therapy and surgical evaluation.

Management course

Management focused on rapid stabilization, treatment of severe infection, and control of complications related to infective endocarditis with systemic vascular involvement. The patient was admitted under close monitoring in a high dependency setting due to the risk of clinical deterioration related to ongoing sepsis, pericardial effusion, and suspected mycotic abdominal aortic

aneurysm. Continuous cardiac monitoring and frequent vital sign assessment were initiated. Two wide bore intravenous lines were secured, and strict fluid balance monitoring was commenced.

Empirical intravenous antibiotic therapy was started immediately after blood cultures were taken. Given the high clinical suspicion of infective endocarditis with systemic embolic phenomena, broad spectrum coverage was initiated and later adjusted according to culture results. Once Streptococcus species was identified as the causative organism, antibiotics were narrowed to targeted intravenous beta lactam therapy in line with sensitivity results and current infective endocarditis management recommendations [1,2].

Initial hemodynamic management included cautious intravenous fluid administration to maintain adequate perfusion. However, care was taken to avoid fluid overload given the presence of pericardial effusion and early signs of cardiac strain. Blood pressure remained relatively stable initially and did not require vasopressor support at admission. Serial lactate measurements and clinical perfusion markers were used to guide ongoing resuscitation.

Given the presence of a moderate pericardial effusion, cardiology input was obtained early. The effusion was initially managed conservatively as there were no overt signs of tamponade physiology. Repeat bedside echocardiography was performed regularly to monitor for progression. Drainage was not immediately required as ventricular filling remained preserved.

The abdominal aortic aneurysm identified on computed tomography was reviewed urgently with vascular surgery. Imaging findings were highly suggestive of a mycotic aneurysm in the setting of infective endocarditis [6,7]. Given the absence of rupture or leak, initial management was conservative with strict blood pressure control and continuation of intravenous antibiotics. Surgical intervention was deferred pending stabilization and further evaluation, as early surgery in unstable infection carries significant risk.

Anticoagulation and antiplatelet agents were avoided due to the risk of septic embolization and potential hemorrhagic complications. Serial inflammatory markers were monitored to assess response to therapy. Although there was some initial improvement in fever and systemic symptoms, inflammatory markers remained elevated during the early course of treatment.

As part of ongoing evaluation, repeat echocardiography was performed, which continued to show pericardial effusion without significant progression. No clear vegetation was visualized on transthoracic imaging; however, transesophageal echocardiography was planned for further assessment of valvular structures once the patient was more stable.

Multidisciplinary input was central to management. Infectious disease specialists guided antimicrobial therapy duration and choice, while cardiology and vascular surgery teams were involved in decision making regarding potential surgical intervention. The possibility of delayed surgical repair of the abdominal aortic aneurysm was discussed depending on response to medical therapy.

Over the subsequent days, the patient showed partial clinical stabilization with reduction in fever and improvement in inflammatory markers. However, the underlying vascular complications persisted, and close monitoring was continued for potential progression of aneurysmal disease or development of tamponade physiology.

The patient was maintained on prolonged intravenous antibiotic therapy with a planned extended course consistent with guidelines for complicated infective endocarditis [1,3]. Regular clinical reassessment and serial imaging were planned to evaluate resolution of pericardial effusion and stability of the abdominal aortic aneurysm.

Before discharge planning could be finalized, arrangements were made for continued inpatient monitoring due to the high risk of complications. Long term follow up with cardiology, infectious diseases, and vascular surgery was planned to assess response to therapy and determine need for delayed surgical intervention.

This case illustrates the importance of early recognition and coordinated multidisciplinary management in infective endocarditis complicated by both pericardial effusion and mycotic abdominal aortic aneurysm. Even in the absence of immediate hemodynamic instability, such patients require close surveillance due to the unpredictable nature of disease progression and the risk of sudden deterioration.

Discussion

This case represents a severe and atypical manifestation of infective endocarditis complicated by systemic embolization, vascular infection, and pericardial involvement. It highlights several important clinical issues including the protean nature of infective endocarditis, the pathogenesis of mycotic aneurysm formation, and the diagnostic challenge posed by concurrent cardiovascular complications. Infective endocarditis remains a life threatening disease caused by microbial infection of the endocardial surface, most commonly affecting native or prosthetic heart valves [1,2]. Despite advances in diagnostics and antimicrobial therapy, it

continues to carry significant morbidity and mortality, particularly when diagnosis is delayed or complications are present at initial presentation [3].

The clinical presentation of infective endocarditis is highly variable and often nonspecific. Fever, fatigue, weight loss, and malaise are common early symptoms, but these may be subtle and easily overlooked, particularly in subacute disease [2]. In this case, the patient initially presented with low grade fever and nonspecific constitutional symptoms, followed by progressive shortness of breath and chest discomfort. Such presentations are consistent with the well described delayed and insidious onset seen in subacute streptococcal endocarditis [4]. The absence of a clear predisposing risk factor in this patient, such as prosthetic valve, intravenous drug use, or known structural heart disease, further contributed to diagnostic complexity, as a significant proportion of cases may occur in patients without obvious risk profiles [1].

Systemic embolization is a hallmark complication of infective endocarditis and results from fragmentation of friable vegetations on infected valves [15]. Emboli may lodge in multiple vascular territories, leading to infarction, abscess formation, or infected aneurysm development. The risk of embolic events is highest in the early phase of infection and is strongly associated with vegetation size and organism virulence [15]. In this case, the presence of an abdominal aortic aneurysm in the setting of suspected infective endocarditis strongly suggested septic embolization to the arterial wall, leading to vascular infection and aneurysmal degeneration. This process is well described in the literature as a mycotic aneurysm, which results from destruction of the arterial media and adventitia following microbial invasion [6,7].

Mycotic abdominal aortic aneurysms are rare but clinically significant complications of bacteremia and infective endocarditis. Although they account for a small proportion of all abdominal aortic aneurysms, they carry a disproportionately high risk of rupture and mortality [6]. The abdominal aorta is an uncommon site of involvement, but when affected, it often reflects advanced systemic infection. The pathophysiology involves hematogenous seeding of the arterial wall or septic embolization to the vasa vasorum, leading to localized infection, inflammation, and progressive weakening of the vessel wall [7]. In this case, computed tomography findings were highly suggestive of an infected aneurysm rather than a degenerative process, particularly in the context of systemic infection and pericardial effusion.

Pericardial involvement in infective endocarditis is less common but clinically important. It may result from direct extension of infection, hematogenous spread, or immune mediated inflammatory response [10]. Pericardial effusion in this context can range from small, clinically silent collections to large effusions with tamponade physiology. Although tamponade was not present in this case, the presence of a moderate effusion reflected significant systemic inflammatory burden and potential pericardial involvement by infection. Purulent pericarditis is a rare but severe complication of bacteremia and is associated with high mortality if not promptly recognized and treated [12].

The coexistence of abdominal aortic aneurysm and pericardial effusion in a single patient is unusual and should prompt consideration of a disseminated infectious process. This pattern strongly suggests ongoing bacteremia with systemic vascular seeding. Streptococcal species, particularly viridans group streptococci, are well recognized causes of subacute infective endocarditis and are capable of producing both valvular infection and metastatic complications [2]. The identification of Streptococcus in blood cultures in this case supported the diagnosis and aligned with classical epidemiological patterns described in infective endocarditis literature [4].

Echocardiography remains the cornerstone of diagnosis in infective endocarditis. Transthoracic echocardiography is often the first imaging modality, although transesophageal echocardiography offers superior sensitivity for detecting vegetations and complications such as abscess formation [1,14]. In this case, no clear vegetation was identified on initial transthoracic imaging, which is not uncommon in early or subacute disease. The absence of visible vegetations does not exclude the diagnosis, particularly when there is strong clinical and microbiological evidence [1]. Advanced imaging modalities, including computed tomography, played a crucial role in identifying extracardiac complications such as the abdominal aortic aneurysm, underscoring the importance of multimodal imaging in complex cases [14].

The management of infective endocarditis requires prolonged intravenous antibiotic therapy tailored to the causative organism [1,13]. Empirical therapy is initiated early in suspected cases, with subsequent adjustment based on culture results. In this case, targeted beta lactam therapy was appropriate once streptococcal infection was confirmed. Duration of therapy is typically four to six weeks depending on the organism and presence of complications [1]. Early involvement of infectious disease specialists is essential to optimize antimicrobial selection and treatment duration.

Management of mycotic abdominal aortic aneurysm remains challenging and often requires a combination of prolonged antibiotics and surgical intervention [6,7]. Open surgical repair with debridement of infected tissue is considered the standard approach in many cases, although endovascular repair may be considered in high risk patients. However, endovascular techniques carry a risk of persistent infection due to retained infected material. In this case, conservative management was initially favored due to clinical stability, with plans for delayed intervention depending on response to therapy.

The presence of pericardial effusion also required careful monitoring. While many small effusions resolve with treatment of the underlying infection, larger or progressive effusions may require drainage [10]. In this case, conservative management was appropriate given the absence of tamponade physiology. Serial echocardiographic assessment is essential in such patients to detect progression early.

This case also highlights the importance of multidisciplinary management in complex infective endocarditis. Coordination between cardiology, infectious diseases, and vascular surgery teams is essential to guide both medical and surgical decision making [1]. Such collaboration is particularly important when multiple organ systems are involved and when treatment strategies may conflict, such as balancing infection control with timing of vascular surgery.
From a prognostic perspective, infective endocarditis complicated by systemic embolization and mycotic aneurysm carries a significantly worse outcome compared to uncomplicated cases [3]. Mortality is influenced by organism type, extent of embolic disease, and timing of diagnosis. Streptococcal endocarditis generally has a better prognosis than staphylococcal disease, but the presence of vascular complications significantly worsens outcomes [2,3].
In conclusion, this case illustrates a rare but clinically important presentation of infective endocarditis complicated by both abdominal aortic aneurysm and pericardial effusion. It emphasizes the need for high clinical suspicion in patients with unexplained systemic infection and vascular abnormalities. Early diagnosis, multimodal imaging, prolonged antimicrobial therapy, and coordinated multidisciplinary care are essential for optimal outcomes. Reporting such cases contributes to greater awareness of the wide and sometimes atypical spectrum of infective endocarditis complications.

Conclusion

This case highlights the importance of considering infective endocarditis in patients presenting with persistent fever and unexplained systemic inflammatory signs, even when initial findings are non specific. Early symptoms such as fatigue, low grade fever, and breathlessness can easily be mistaken for less serious conditions, leading to delayed diagnosis. Careful attention to subtle clinical features and early use of blood cultures are essential to avoid missing the diagnosis at an early stage.
The presence of vascular complications such as abdominal aortic aneurysm and pericardial effusion should raise immediate concern for a disseminated infectious process. These findings are not typical in isolated cardiac disease and should prompt urgent evaluation for septic embolization and mycotic aneurysm formation. In this case, early imaging played a key role in identifying the extent of disease and guiding further management decisions.
Pericardial effusion in the setting of infective endocarditis should always be closely monitored, as it may progress and lead to hemodynamic compromise. Similarly, detection of an abdominal aortic aneurysm in the context of bacteremia should be treated as potentially infectious until proven otherwise. Delayed recognition of these complications can significantly worsen outcomes due to risk of rupture or cardiac tamponade.
This case also reinforces the importance of early multidisciplinary involvement, including cardiology, infectious diseases, and vascular surgery teams. Management of infective endocarditis with systemic complications is complex and requires coordinated decision making regarding antibiotic therapy, imaging follow up, and timing of surgical intervention. Prolonged intravenous antibiotics remain the cornerstone of treatment, but structural complications often require individualized surgical planning.
Despite advances in antimicrobial therapy and imaging, infective endocarditis with systemic embolic and vascular complications continues to carry significant morbidity and mortality. Early recognition, high clinical suspicion, and prompt initiation of treatment are essential to improving outcomes. This case emphasizes the need for clinicians to maintain a broad differential diagnosis when encountering unexplained fever with cardiovascular abnormalities, as early diagnosis remains the most important factor influencing prognosis.

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References

1. Habib G, Lancellotti P, Erba PA, et al. 2023 ESC guidelines for the management of infective endocarditis. *Eur Heart J*. 2023;44(39):3948-4042.
https://academic.oup.com/eurheartj/article/44/39/3948/7243105
2. Cahill TJ, Prendergast BD. Infective endocarditis. *Lancet*. 2021;397(10278):788-802.
https://www.sciencedirect.com/science/article/pii/S0140673621000584
3. Baddour LM, Wilson WR, Bayer AS, et al. Infective endocarditis in adults. *Circulation*. 2015;132(15):1435-1486.
https://www.ahajournals.org/doi/10.1161/CIR.0000000000000296
4. Murdoch DR, Corey GR, Hoen B, et al. Clinical presentation, etiology, and outcome of infective endocarditis. *Arch Intern Med*. 2009;169(5):463-473.
https://jamanetwork.com/journals/jamainternalmedicine/fullarticle/414667
5. González-Urquijo M, Salgado-Garza G, et al. Infective native visceral artery aneurysm: systematic review. *J Vasc Surg*. 2025.
https://journals.sagepub.com/doi/10.1177/1358863X251326537

6. Sörelius K, Budtz-Lilly J, Mani K, Wanhainen A. Systematic review of mycotic aortic aneurysms. *Eur J Vasc Endovasc Surg*. 2019;58(3):426-435.
https://pubmed.ncbi.nlm.nih.gov/31320247/
7. Sörelius K. Mycotic aortic aneurysms: a review. *J Cardiovasc Surg*. 2020.
https://www.sciencedirect.com/science/article/pii/S1934592518304465
8. Ito F, Inokuchi R, et al. Clostridium septicum infected aortic aneurysm: case report and review. *J Med Case Rep*. 2017;11:268.
https://link.springer.com/article/10.1186/s13256-017-1422-0
9. Liphaus BL, Goldenstein-Schainberg C, et al. Abdominal mycotic aneurysm in infective endocarditis. *Arq Bras Cardiol*. 2007.
https://pubmed.ncbi.nlm.nih.gov/17364110/
10. Matsuda B, Hoo A, Teerasukjinda O, et al. Mycotic aortic aneurysm with purulent pericardial effusion. *Case Rep Crit Care*. 2014.
https://pmc.ncbi.nlm.nih.gov/articles/PMC4175936/
11. Osler W. The Gulstonian lectures on malignant endocarditis. Historical reference on septic emboli and aneurysms.
12. Mylonakis E, Calderwood SB. Infective endocarditis in adults. *N Engl J Med*. 2001;345:1318-1330.
https://www.nejm.org/doi/full/10.1056/NEJMra2002328
13. Prendergast BD, Tornos P. Infective endocarditis. *BMJ*. 2017;358:j3777.
https://www.bmj.com/content/358/bmj.j3777
14. Holland TL, Baddour LM, Bayer AS, et al. Infective endocarditis. *Nat Rev Dis Primers*. 2016;2:16059.
https://www.nature.com/articles/nrdp201659
15. Choudhury S, Reddy YNV, et al. Embolic complications of infective endocarditis. *Circulation*. 2019.
https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.119.041433
16. Bonow RO, Mann DL, Zipes DP, Libby P. *Braunwald's Heart Disease*. 12th ed. Elsevier; 2022.
17. Jameson JL, Fauci AS, Kasper DL, et al. *Harrison's Principles of Internal Medicine*. 21st ed. McGraw-Hill; 2022.
18. Kumar V, Abbas AK, Aster JC. *Robbins & Cotran Pathologic Basis of Disease*. 10th ed. Elsevier; 2020.