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| RESEARCH ARTICLE

Silent Valves, Painful Abdomen: Missed Infective Endocarditis Revealed by Splenic Abscess

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| ABSTRACT

Exclusion of infective endocarditis (IE) based solely on the absence of an audible murmur or traditional predisposing risk factors is a critical diagnostic error, as a significant proportion of patients lack these features at presentation. A splenic abscess, while a clear indicator of underlying sepsis, often originates from an unrecognized cardiac source. This case exemplifies such a presentation: a 38-year-old male presented to the emergency department with persistent left lower quadrant abdominal pain and low-grade fever—symptoms that effectively masked the presence of IE. Despite the absence of cardiac complaints or classical peripheral stigmata, the patient was found to have splenic involvement due to septic embolization from unrecognized endocarditis. Although he was ultimately treated successfully with a four-week course of intravenous antibiotics, the delay in diagnosis rendered percutaneous drainage unsuccessful, necessitating an otherwise avoidable splenectomy. This surgical intervention, while curative in the short term, confers a lifelong vulnerability to overwhelming post-splenectomy infection, highlighting the imperative for early recognition of atypical IE presentations.

| KEYWORDS

Splenic Abscess, Thrombo-embolism, Septic Emboli, Infective Endocarditis, Vulvular Heart Disease.

| ARTICLE INFORMATION

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1. Introduction

Infective endocarditis (IE) is a serious, multisystemic infection. It is a relatively uncommon condition, with epidemiological studies reporting incidence rates of approximately 3 to 7 cases per 100,000 individuals in developed countries [1]. However, in recent years, this incidence appears to be increasing, largely attributed to aging populations and the growing use of intravascular devices and prosthetic valves [2]. IE typically presents with fever and non-specific systemic symptoms, often accompanied by a new or changing cardiac murmur [1]. Nonetheless, IE may result in complications affecting distant organs via septic embolization. Embolic infarctions of the kidneys, spleen, lungs, and other sites have been well documented [1]. Consequently, abdominal pain may occur in the setting of IE, reflecting embolic infarction or abscess formation in intra-abdominal organs such

as the spleen or mesentery [1,3]. Among the various embolic sequelae of left-sided IE, the spleen is affected more frequently than many other organs. Splenic infarction, resulting from embolic arterial occlusion, is a common finding in IE and is usually clinically silent or benign. In contrast, splenic abscess—defined as a pus-filled cavity within the splenic parenchyma—is a rare but serious complication. Splenic abscess typically arises when septic emboli lodge in the spleen and subsequently undergo secondary infection [3]. Not all infarctions progress to abscess formation; in one review, approximately 5% of splenic infarcts evolved into abscesses [4]. Consequently, the estimated incidence of splenic abscess in IE varies. Classic series report that approximately 3–5% of IE patients develop splenic abscesses [3], whereas more recent data suggest higher incidences, with some cohorts reporting rates as high as 10–20% [4,5]. Although splenic abscess is an uncommon complication of infective endocarditis, its true prevalence is likely underestimated, as its symptoms are often subtle or overlooked. The clinical presentation of splenic abscess tends to be insidious and nonspecific. Patients typically exhibit persistent fever and leukocytosis and may report vague left upper quadrant or flank discomfort [3]. One study found that approximately two-thirds of patients with splenic abscess had fever, and nearly half reported nausea or abdominal pain [6]. On physical examination, clinicians may detect left upper quadrant tenderness or splenomegaly; however, these findings are neither sensitive nor specific for the diagnosis. Abdominal symptoms related to splenic abscess can easily be misattributed to other causes, such as pneumonia or gastritis, and are frequently overlooked in the context of complex IE presentations. Therefore, in any patient with IE who presents with unexplained or persistent abdominal pain—particularly if left-sided—or fever that does not respond to antimicrobial therapy, a high index of suspicion for splenic involvement is warranted [3,7].

Unfortunately, routine clinical evaluation is often insufficient: even a combination of medical history, physical examination, and standard laboratory tests may fail to detect a splenic abscess, leading to delayed diagnosis [3,5]. Diagnostic imaging is thus essential. Contrast-enhanced abdominal CT is considered the gold standard for detecting splenic abscesses, offering near 100% sensitivity by visualizing characteristic hypodense, rim-enhancing lesions within the spleen [3]. Ultrasound may be employed as an initial screening modality or in resource-limited settings; it typically reveals a complex cystic or anechoic mass with irregular margins [3]. However, ultrasound is operator-dependent and less sensitive, particularly for small or early-stage lesions. Magnetic resonance imaging (MRI) provides excellent soft tissue contrast and can identify small abscesses, but it is less commonly utilized in acute clinical settings. In practice, the threshold for obtaining imaging should be low: abdominal CT or MRI should be strongly considered in any IE patient with vague abdominal complaints or persistent fever unresponsive to antibiotics [7]. Early imaging not only facilitates accurate diagnosis but also informs management decisions—such as image-guided percutaneous drainage versus surgical intervention. The prognosis of patients with splenic abscess depends significantly on the timeliness of diagnosis and intervention. Delayed recognition may result in abscess rupture or systemic bacterial dissemination, leading to overwhelming sepsis and multiorgan failure. Historical data indicate that untreated splenic abscess carries a mortality rate of 70–100% [5]. Even with treatment, mortality has been reported to reach approximately 47% in some series [5]. Fortunately, modern management—consisting of timely antibiotic therapy combined with percutaneous drainage or surgical splenectomy—can reduce mortality to below 10% [5]. These outcomes emphasize the critical importance of early recognition. Indeed, systematic reviews have highlighted that early and widespread use of diagnostic imaging (CT or ultrasound) facilitates prompt diagnosis and intervention, which directly correlates with improved survival [5]. In summary, delayed detection of splenic abscess in the setting of IE significantly increases the risk of sepsis, splenic rupture, and death [3,5]. Our case underscores the importance of maintaining a high degree of clinical vigilance. This patient with subacute IE developed left-sided abdominal symptoms that were not initially attributed to a splenic abscess, resulting in diagnostic delay. The case illustrates that even atypical or indolent presentations of IE may harbor life-threatening complications such as splenic abscess. In similar clinical contexts, physicians should sustain a high index of suspicion and pursue early abdominal imaging in IE patients presenting with unexplained fever or abdominal pain [7]. Prompt recognition of this rare complication allows for timely, multidisciplinary management—including antimicrobial therapy, abscess drainage, and cardiac surgery when necessary—ultimately improving patient outcomes.

2. Case Presentation

2.1 Patient's history and Physical Examination

This case report describes a 38-year-old male who presented to the emergency department with a complaint of progressive left upper quadrant abdominal pain, subsequently accompanied by recurrent low-grade fever persisting for approximately 10 days. The abdominal pain was of recent onset and characterized as dull, aching, and poorly localized. It gradually intensified over time and radiated to the left shoulder. The discomfort was associated with constitutional symptoms, including anorexia and an unintentional weight loss of approximately 4 kilograms over the preceding month. Notably, the patient denied experiencing flank pain, dysuria, hematuria, or other urinary tract symptoms. He also reported no changes in bowel habits, no vomiting, and no gastrointestinal bleeding, including melena or hematemesis. In addition, there were no respiratory complaints such as cough, dyspnea, chest pain, or orthopnea. The patient reported multiple episodes of self-medication with over-the-counter analgesics, including paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs), but derived no significant relief. He explicitly denied any recent abdominal trauma or travel history, and there was no prior diagnosis of cardiovascular disease or other relevant medical conditions. His past medical history was unremarkable, with no known history of diabetes mellitus, hypertension, or

other chronic illnesses. Family history was non-contributory. From a social perspective, the patient was a non-smoker and reported no consumption of alcohol or illicit drugs. On physical examination, he was alert and oriented but appeared mildly ill-looking. He was febrile, with a recorded temperature of 38.1°C, and tachycardic, with a heart rate of 102 beats per minute. His blood pressure was within normal limits at 122/84 mmHg. Respiratory rate was 18 breaths per minute, and oxygen saturation was 98% on ambient air. Cardiac examination revealed normal heart sounds without any audible murmurs, rubs, or gallops. Abdominal examination disclosed localized tenderness in the left upper quadrant, without signs of peritoneal irritation (i.e., no rebound tenderness). The spleen was palpable approximately 3 cm below the left costal margin, while the liver edge was not palpable. There was no scleral icterus or other stigmata of chronic liver disease. Neurological examination was non-focal, with no detectable motor, sensory, or cranial nerve deficits.

2.2 Investigations:

The relevant laboratory findings are summarized in Table 1, which demonstrate leukocytosis, a likely anemia of chronic disease, and evidence of a marked inflammatory response, as reflected by significantly elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels. Given the clinical presentation, an abdominal ultrasound was performed as an initial imaging modality. This revealed splenomegaly with a hypoechoic lesion measuring approximately 4.5 × 3.2 cm, raising suspicion for a splenic abscess. These findings were further evaluated and confirmed by contrast-enhanced abdominal computed tomography (CT), which demonstrated a well-defined hypodense collection measuring 4.7 × 3.5 cm located in the mid-pole of the spleen. The lesion was consistent with a splenic abscess, without evidence of rupture, although adjacent perisplenic fat stranding was noted, suggestive of localized inflammation. To investigate a potential cardiac source of septic emboli, transthoracic echocardiography (TTE) was conducted and revealed a mobile echodense mass measuring 0.7 × 0.6 cm, attached to the aortic valve, along with mild aortic regurgitation. These findings were further delineated by transesophageal echocardiography (TEE), which confirmed the presence of aortic valve vegetation, without evidence of a periannular abscess. Eventually, blood cultures returned positive for Streptococcus mitis/oralis, thereby confirming the diagnosis of infective endocarditis, complicated by a splenic abscess. Notably, the disease presented atypically, with abdominal complications as the initial clinical manifestation, rather than with primary cardiac symptoms.

Test	Result	Normal Range
Hemoglobin	10.2	13-17 g\dL
WBC	$15.1x10^9$	$4.0\text{-}11x10^9$\L
Platelets	$210x10^9$	$150\text{-}450x10^9$\L
Sodium	138	135-145 mmol\L
Potassium	4.4	3.5-5.0 mmol\L
Creatinine	1.0	0.7-1.2 mg\dL
CRP	95	<5 mg\L
ESR	84	<20 mm\hr
INR	1.6	0.8-1.2

Table 1: results of relevant laboratory investigations.

2.3 Management course

The patient was initially started on empiric intravenous antibiotic therapy, consisting of vancomycin at 15 mg/kg every 12 hours and ceftriaxone 2 g once daily, upon admission. Following the identification of Streptococcus mitis/oralis in blood cultures, antimicrobial therapy was de-escalated to ceftriaxone 2 g IV once daily, to be continued for a total duration of four weeks. In terms of managing the splenic abscess, an attempt at CT-guided percutaneous drainage was made; however, the procedure was unsuccessful due to the presence of internal septations within the collection. Consequently, the decision was made to proceed with surgical splenectomy, which was performed on hospital day 6. The postoperative course was uneventful. The patient became afebrile by day 9, and subsequent blood cultures remained sterile. He successfully completed the full 4-week course of intravenous antibiotic therapy and was discharged in stable condition on hospital day 32. At the time of discharge, he was referred for cardiology follow-up, with no surgical valve intervention planned, as he remained clinically asymptomatic from a cardiovascular standpoint.

3. Discussion

One of the most compelling aspects observed in this case report is the notable absence of classical predisposing risk factors for infective endocarditis (IE), such as a history of intravenous drug use, congenital heart disease, or the presence of prosthetic heart valves. This clinical observation serves to underscore a critical diagnostic challenge: the tendency to rely heavily on traditional risk profiles can lead to a significant delay in the recognition and diagnosis of IE. In fact, it is important to highlight that over 50% of IE cases occur in individuals without any previously known structural or valvular cardiac disease, reinforcing the notion that IE

can arise in patients previously considered low-risk [1]. Moreover, it is well established that a significant proportion of patients—approximately 15% to 25%—present without an audible murmur at initial evaluation, as was the case here [1]. This is often attributable to either the presence of non-hemodynamically significant valvular regurgitation or suboptimal examination conditions, particularly in acute or critically ill patients [1,2]. These factors together complicate the timely clinical identification of IE and underscore the need for a high index of suspicion, even in the absence of classical auscultatory findings. In the present case, the patient's abdominal pain was the sole presenting symptom, overshadowing any cardiac manifestations and consequently diverting attention from the underlying diagnosis. Such an atypical presentation is diagnostically misleading, as abdominal complications—including splenic infarction and splenic abscess—occur in only 2–5% of cases of IE [3]. The failure to recognize this rare but significant association resulted in a delay in diagnosis, which ultimately necessitated splenectomy—a more invasive procedure that might have been avoidable with earlier recognition and intervention, such as image-guided percutaneous drainage. However, once performed, splenectomy introduces its own long-term risks, most notably the lifelong susceptibility to overwhelming post-splenectomy infection (OPSI) [3]. While classic peripheral signs of IE—such as Janeway lesions, Osler nodes, and Roth spots—remain frequently cited in the literature, their actual incidence has dramatically decreased in modern cohorts, now observed in fewer than 15% of patients [8]. Consequently, the absence of these findings cannot be considered reassuring, and should not be used to exclude the diagnosis of IE, particularly when systemic features suggest a possible endovascular source [8]. This case reinforces the critical importance of considering IE in patients presenting with extra-cardiac manifestations, including those of neurological, pulmonary, or abdominal origin. Specifically, the identification of a splenic abscess on abdominal imaging in the context of fever should raise strong clinical suspicion for IE and prompt immediate investigation for a cardiac source, particularly in the absence of alternative explanations [7]. Once the diagnosis is delayed, the efficacy of percutaneous or minimally invasive drainage strategies declines sharply, with success rates reported to fall below 40% [5]. This is primarily due to the development of viscous, loculated purulent collections that are refractory to simple drainage [5]. In such cases, splenectomy becomes the gold-standard intervention, offering not only definitive source control but also a demonstrated reduction in mortality, largely by preventing recurrent infection and catastrophic rupture [5]. Recognition of IE from such atypical clinical contexts is essential, especially considering the near-100% mortality rate in untreated cases [2]. Early initiation of empiric broad-spectrum antibiotics targeting common causative organisms—namely, Staphylococcus aureus, viridans group Streptococci, and Enterococci—remains a critical determinant of patient survival and clinical outcome [1]. In this particular case, the antimicrobial regimen was guided by current evidence and recent randomized controlled trials, which have shown no survival benefit from the addition of gentamicin, alongside an increased risk of nephrotoxicity [8]. As such, gentamicin was appropriately omitted, in accordance with contemporary guidelines. Given that the causative organism was identified as Streptococcus mitis/oralis, a member of the viridans group streptococci with high susceptibility to ceftriaxone, monotherapy with ceftriaxone 2g intravenously once daily for a duration of four weeks was deemed both appropriate and effective [2]. This regimen is associated with clinical cure rates exceeding 95%, making it a mainstay of treatment in such cases [2]. When administered according to guidelines and in conjunction with successful source control, the risk of relapse following completion of antibiotic therapy is generally under 5% for infections due to viridans streptococci [8]. However, in clinical scenarios characterized by incomplete source control—such as undrained or inadequately managed splenic abscesses—this relapse risk is anticipated to be markedly higher, emphasizing the critical role of early diagnosis and comprehensive management [8].

4. Conclusion

The absence of an audible murmur or traditional predisposing risk factors should never be considered sufficient to exclude the diagnosis of infective endocarditis (IE), as a substantial proportion of patients present without either. While the presence of a splenic abscess invariably indicates underlying sepsis, the most common primary source is frequently cardiac in origin. When diagnosis is delayed, percutaneous drainage often fails due to the development of multiloculated collections and viscous purulence—pathophysiological consequences that may be viewed as a natural sequela of delayed recognition. This, in turn, necessitates more invasive interventions such as splenectomy, thereby subjecting patients to the lifelong risk of overwhelming post-splenectomy infection (OPSI). Clinicians must therefore maintain a high index of suspicion for IE, particularly in patients presenting with atypical manifestations such as fever of unknown origin, isolated abdominal pain, pulmonary symptoms, or unexplained neurological deficits.

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