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CASE REPORT

Delayed Giant Splenic Artery Aneurysm Following Splenic Infarction in a Patient with Infective Endocarditis

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Abstract: Infective endocarditis is a severe infectious disease that, in addition to cardiac complications such as heart failure and arrhythmias caused by valve damage, may also lead to extracardiac complications such as septic embolization, metastatic abscesses, and mycotic aneurysms. Here, we report a rare case of splenic infarction resulting from infective endocarditis, subsequently leading to the development of a splenic artery aneurysm three weeks post-surgery confirmed both by CTA and DSA. Following arterial embolization of the aneurysm, the patient eventually recovered completely.

Keywords: infective endocarditis, splenic infarction, pseudoaneurysm

Introduction

Infective endocarditis (IE) is accompanied by a wide range of potential complications, with the likelihood of occurrence influenced by factors such as the infecting pathogen, duration of illness before treatment initiation, and underlying comorbidities.[1] Complications may manifest before, during, or after completion of therapy. Heart failure, perivalvular abscess, pericarditis, and intracardiac fistula are common cardiac complications of IE Septic embolization, metastatic abscesses, and mycotic aneurysms[2] are extracardiac complications of IE mycotic aneurysms that can develop in the cerebral or systemic circulation in the setting of IE, usually at points of vessel bifurcation. Splenic artery aneurysms (SAAs) are uncommon in IE complications, accounting for 1.3–1.4% of all IE complications.[3,4] The larger the diameter of an aneurysm, the higher the risk of rupture and bleeding. Infectious SAAs make up 10% of all SAAs and carry a 37% risk of rupture, significantly higher than the 2–3% risk seen in genuine splenic aneurysms.[3]

Case Presentation

A 36-year-old man presented to the Infectious Disease Department with a 3-month history of fever. Three months prior to admission, the patient reported recurrent high-grade fever (peak temperature 40.0°C) accompanied by chills and myalgia. Self-administered roxithromycin or intravenous etimicin in another hospital resulted in transient fever resolution, followed by intermittent afternoon fevers. Ten days before admission, the patient complained about progressive chest tightness and dyspnea. The patient reports fatigue, night sweats, and a 2 kg unintentional weight loss over a 3-month course of illness. He was previously healthy and there were no dental procedures, valvular disease, IV drug use, or immunosuppression before this event. On examination, patient is general well. A holosystolic murmur (grade 3/6) is audible over the mitral valve area, with no palpable precordial thrill. The spleen is palpable 2 cm below the costal margin, with no tenderness on percussion. Laboratory results showed a normal white blood cell count (9.92×10^9/L), of which 78.8% were neutrophilic granulocytes,

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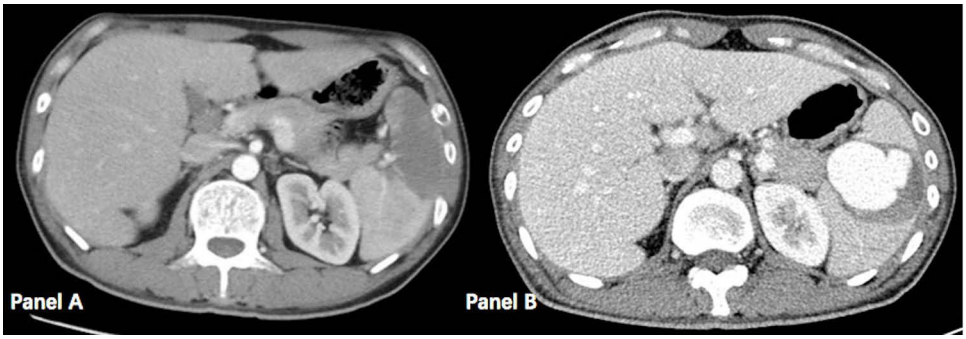

Figure 1 Abdominal computed tomography (Panel A shows splenic infarction, Panel B shows subsequent splenic artery aneurysm in the splenic infarction area).

along with rising levels of procalcitonin (0.23 ng/mL), and ESR (38 mm/H). The pre-admission outpatient chest and abdominal CT demonstrated essentially unremarkable findings without evidence of splenic infarction. After admission, 2 sets of blood culture revealed Streptococcus sanguis. Transthoracic echocardiography showed rupture of the chordae tendineae in the anterior leaflet of the mitral valve, with vegetation formation (18×6mm) on the anterior leaflet of the mitral valve. Intravenous antibiotics (amoxicillin-clavulanate plus amikacin) were administered. On the fourth day of hospitalization, the patient experienced sudden onset of dull abodominal pain in the left upper quadrant accompanied by abdominal distension. Follow-up abdominal contrast-enhanced CT revealed a newly developed splenic infarction (Figure 1. Panel A). According to the Modified Duke Criteria, the patient met 2 major criteria (positive blood cultures, valvular vegetation on imaging) and 2 minor criteria (fever, embolic phenomenon: splenic infarction), leading to a diagnosis of Infective Endocarditis (IE) with splenic infarction, which is considered to be related to splenic embolism caused by the detachment of mitral valve vegetations. Given the large vegetation and the occurrence of an embolic event, the patient underwent mitral valve repair with annuloplasty on the 7th day of hospitalization. The surgery went well, and patient's fever resolved completely together with sterilized blood culture. However, the patient developed abruptly severe left upper abdominal pain again three weeks post-surgery, when his intravenous antibiotics had not yet been discontinued. An abdominal computed tomography angiography (CTA) revealed a newly formed 52mm splenic artery aneurysm (Figure 1. Panel B) in the previously infarcted area, with swirling blood flow observed under digital subtraction angiography (DSA) (Figure 2 and Video S1). Infective endocarditis complicated by a giant splenic artery aneurysm was diagnosed. Giant splenic artery aneurysms are rare complications of IE, carrying a high risk of rupture. Early diagnosis and treatment with embolization or splenectomy can mitigate fatal consequences. The patient underwent coil embolization under DSA, and abdominal pain resolved within 3 days. No further complications were observed during the 3-month follow-up.

Discussion

Infective endocarditis can lead to various extracardiac complications in addition to cardiac complications (such as heart failure, arrhythmias), including septic embolization,[5,6] metastatic abscess, and mycotic aneurysm. Patients with infective endocarditis face a significant embolic risk, with clinical studies demonstrating embolic complications in 13–49% of cases.[7–10] Notably, these embolic events exhibit substantial subclinical prevalence, remaining asymptomatic in up to 50% of affected individuals[11] while still carrying serious clinical implications. Left-sided infective endocarditis is strongly associated with systemic embolization, with the brain and spleen emerging as the primary target organs due to their high vascular susceptibility to septic emboli originating from valvular vegetations. The size of vegetations is an independent risk factor for embolic events in infective endocarditis.[12] Early surgery is associated with a reduced risk of embolization, whereas initial conservative therapy is associated with increased mortality.[13] In this case, the patient was finally diagnosed with infective endocarditis after a prolonged 3-month clinical course. The large size of the cardiac valve vegetations poses a high risk of embolism, which explains the occurrence of splenic embolism and infarction in the patient. Subsequently, the infected emboli invaded the endothelial cells of the splenic artery, leading to the further development of a splenic artery aneurysm. If an earlier diagnosis had been made and cardiac surgery performed promptly, it might have prevented the development of splenic infarction and

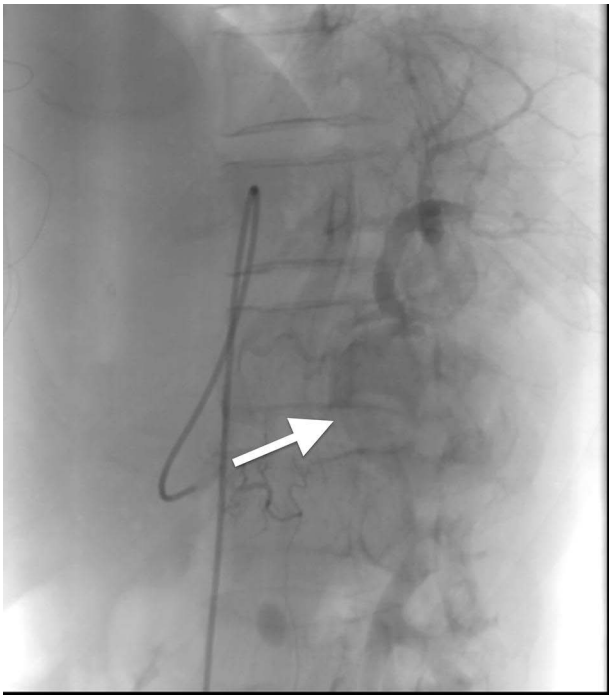

Figure 2 Splenic artery aneurysm (arrowhead) observed under digital subtraction angiography (DSA).

subsequent SAA. It is of great importance of early recognition and imaging surveillance of extracardiac complications in selected cases of IE, particularly given the delayed onset in this case.

SAAs are infrequent occurrences in cases of infective endocarditis, often remaining clinically asymptomatic. Symptoms occur in up to 20% of cases and include upper abdominal pain that can radiate to the shoulder, nausea, vomiting, anorexia, and gastrointestinal bleeding.[14] The size of the SAAs was classified as small (<20 mm), large (20–50 mm), and giant (> 50 mm). Non-infectious splenic artery pseudoaneurysms (SAPAs), with a reported mean diameter of 4.8 cm (range: 0.3–17 cm), are generally larger in size compared to infectious endocarditis-associated SAAs, while true splenic artery aneurysms (SAAs) typically measure less than 3 cm in diameter.[15] Typically, endocarditis-associated SAAs manifest as solitary lesions located near the hilum or intraparenchymal region. The complication of SAA that clinicians are most concerned about is rupture and hemorrhage. As early as 1947, ruptured splenic artery aneurysms (SAAs) associated with infective endocarditis had been documented.[16] The risk of splenic artery aneurysm rupture associated with infective endocarditis is significantly higher than that of ordinary splenic artery aneurysms,[3] with a risk of fatal bleeding in severe cases. Therefore, prompt treatment is necessary upon discovery. The development of SAAs in the context of IE may be delayed, occurring even after appropriate antibiotic therapy and successful cardiac surgery. Regular imaging surveillance is essential for early detection, minimizing the risk of delayed complications. The Society for Vascular Surgery recommends using CTA as the primary diagnostic tool for SAAs.[17] The arterial phase of abdominal CTA was essential for both identifying the location of the SAA, if the aneurysm was patent, and for detecting any potential contrast extravasation, which was a sign of rupture and bleeding.[18]

Digital subtraction angiography is considered the gold standard for diagnosing splenic artery aneurysms as it offers precise localization of the SAA and can also detect other intra-abdominal abnormalities in the context of infective endocarditis. DSA and endovascular intervention were the preferred treatment approach to treat SAAs. However, if there are concerns such as arterial aneurysm rupture, unstable hemodynamics, or local infection, splenectomy should be considered for treatment.[19] Mortality was high for ruptured aneurysms treated with an open approach, with an event rate of 0.29 (95% CI, 0.04–0.71).[20] In this case, as the patient had stable hemodynamics, normal body temperature, and negative blood culture results, intervention with embolization therapy was ultimately chosen.

This case report is inherently limited by its single-patient design, which restricts the generalizability of the findings. Additionally, the absence of long-term follow-up data prevents assessment of the patient's ultimate clinical outcomes and potential late complications. The delayed diagnosis and surgery in this case may have contributed to disease progression and the development of severe complications, suggesting a need for improved early detection protocols. The case also implicates us that patient with infective endocarditis-especially those with embolic events-should undergo continued imaging surveillance even after surgical treatment and resolution of infection, due to the risk of delayed vascular complications like splenic artery aneurysms.

Ethical Approval

This study was approved by the Ethics Committee of Tibet Autonomous Region People's Hospital. According to the Ethics Committee of Tibet Autonomous Region People's Hospital, no additional institutional approval was required for publication of the case details.

Patient Consent Statement

The patient provided informed consent for the publication of both clinical details and images.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no competing interests to declare in this work.

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