Case Report/*Olgu Sunumu*

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Bilateral Popliteal Arterial Occlusion Following Splenectomy in a High-Risk Patient: A Case Report

Yüksek Riskli Bir Hastada Splenektomi Sonrası Gelişen Bilateral Popliteal Arter Oklüzyonu: Olgu Sunumu

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ABSTRACT

Splenectomy is performed for various indications, including trauma, hematologic disorders, infections, and malignancies, and is associated with risks such as infection and sepsis. Increasing evidence suggests that splenectomy may also predispose patients to thromboembolic complications through systemic inflammation, postoperative hypercoagulability, and reactive thrombocytosis. While venous thromboembolic events are well recognized, acute peripheral arterial thrombosis after splenectomy is rare. We report a 63-year-old woman with uncontrolled type 2 diabetes mellitus, hypertension, and obesity (body mass index 38.3 kg m^{-2}) who was diagnosed with a multifocal splenic abscess and underwent emergency splenectomy due to inadequate source control. Standard postoperative care, including prophylactic enoxaparin and early mobilization, was initiated; however, inflammatory markers remained elevated. On postoperative day four, the patient developed acute bilateral lower extremity pain with sensory and motor deficits and absent distal pulses. Computed tomography angiography revealed bilateral popliteal artery occlusion. Emergency bilateral popliteal embolectomy restored arterial perfusion. Cardiac evaluation excluded intracardiac thrombus, infective endocarditis, patent foramen ovale, and atrial fibrillation. Platelet counts showed mild reactive thrombocytosis. This case highlights a rare presentation of acute bilateral peripheral arterial thrombosis following splenectomy and underscores the importance of vigilance for arterial thromboembolic complications in high-risk patients.

Keywords: Arterial thrombosis, postoperative complications, splenectomy, splenic abscess, thrombocytosis, thrombosis

ÖZ

Splenektomi, travma, hematolojik bozukluklar, enfeksiyonlar ve maligniteler dahil olmak üzere çeşitli endikasyonlarla uygulanan, enfeksiyon ve sepsis gibi riskler taşıyan bir cerrahidir. Güncel veriler, splenektominin sistemik inflamasyon, postoperatif hiperkoagülabilite ve reaktif trombositoz aracılığıyla hastaları tromboembolik komplikasyonlara yatkın hale getirebileceğini göstermektedir; ancak venöz tromboembolik olaylar yaygın görülürken, splenektomi sonrası akut periferik arteriyel tromboz oldukça nadirdir. Bu sunumda, kontrolsüz tip 2 diyabet, hipertansiyon ve obezite (vücut kitle indeksi 38,3 kg m^{-2}) tanısı olan ve multifokal dalak apsesi nedeniyle acil splenektomi uygulanan 63 yaşında bir kadın hasta sunulmaktadır. Profilaktik enoksaparin ve erken mobilizasyonu içeren standart postoperatif bakım uygulanmasına rağmen inflamatuar belirteçleri yüksek seyreden hastada, postoperatif dördüncü günde duyusal ve motor kayıp ile birlikte bilateral alt ekstremite ağrısı ve distal nabızsızlık gelişmiştir. Bilgisayarlı tomografi anjiyografi ile bilateral popliteal arter oklüzyonu saptanması üzerine uygulanan acil bilateral popliteal embolektomi ile arteriyel perfüzyon yeniden sağlanmıştır. Kardiyak değerlendirmede intrakardiyak trombüs, infektif endokardit, patent foramen ovale ve atriyal fibrilasyon dışlanırken; trombosit sayımlarında hafif reaktif trombositoz gözlenmiştir. Bu vaka, splenektomi sonrası gelişen nadir bir akut bilateral periferik arteriyel tromboz tablosunu vurgulamakta ve yüksek riskli hastalarda arteriyel tromboembolik komplikasyonlara karşı dikkatli olunmasının öneminin altını çizmektedir.

Anahtar sözcükler: Arteriyel tromboz, ameliyat sonrası komplikasyonlar, splenektomi, dalak apsesi, trombositoz, tromboz

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INTRODUCTION

Splenectomy is a surgical procedure performed for a wide variety of clinical reasons, including trauma, hematological diseases, infections, and malignancies, and affects hundreds of thousands of patients annually (1). Splenic abscess is a rare but potentially life-threatening condition and represents an uncommon indication for splenectomy (2).

Although patients require careful monitoring after splenectomy, especially for postsplenectomy infections and fulminant sepsis, the connection with thromboembolic events has become more evident in recent years. Large national databases have shown an increased occurrence of venous and arterial thrombosis following splenectomy, which may be related not only to infection and inflammation but also to thrombocytosis and heightened coagulation activity that can develop afterward. An essential point in the literature is that women's susceptibility to thromboembolism may differ during the premenopausal period, mainly due to hormonal and autoimmune factors. However, acute peripheral arterial thrombosis following splenectomy remains relatively uncommon and may be underrecognized (3,4).

CASE REPORT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

A 63-year-old female patient with uncontrolled type 2 diabetes mellitus and hypertension weighs 98 kg and is 160 cm tall, with a body mass index of 38.3 kg m^{-2}. The patient had entered menopause at the age of 45, which is within normal limits, and had no history of hormone replacement therapy. She was not taking any antiplatelet or anticoagulant medications. She presented to the internal medicine outpatient clinic with left upper quadrant pain that lasted about one week and worsened over the past two days. She also experienced fatigue, fever, and loss of appetite. Physical examination showed tenderness at the left costovertebral angle and increased pain on palpation. Laboratory tests revealed a C-reactive protein (CRP) level of 426 mg L^{-1} and a white blood cell (WBC) count of 24.600 mm^{-3}. Contrast-enhanced upper abdominal computed tomography (CT) revealed hypodense lesions containing air densities, approximately 5 cm in diameter, located in the inferoposterior and inferoanterior regions of the spleen, accompanied by perisplenic heterogeneity (Figure 1).

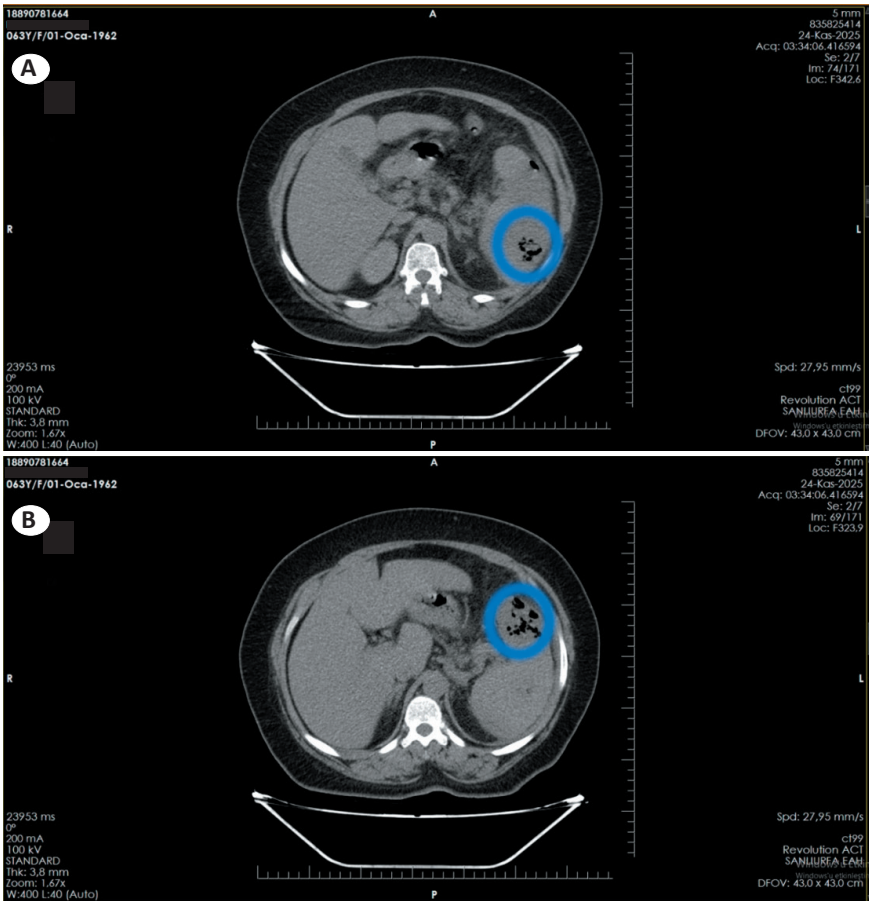


Figure 1. The areas shown indicate abscesses in the two zones of the spleen.

Table I. Postoperative Leukocyte, Platelet, and C-Reactive Protein Trends

Postoperative day	WBC (cells mm^{-3})	Platelet ($\times 10^3$ mm^{-3})	CRP (mg L^{-1})
POD 1	24.000	596 → 520	426
POD 2	42.610	648	318
POD 3	40.650	751	337

Two platelet measurements were obtained sequentially on POD1.
WBC: White blood cell count, **CRP:** C-reactive protein,
POD: Postoperative day.

These findings were consistent with a diagnosis of splenic abscess. Percutaneous drainage was not considered appropriate because imaging demonstrated multiple abscess foci involving different splenic regions, raising concerns regarding inadequate source control. Therefore, based on the general surgery team's assessment, emergent splenectomy was performed for adequate source control.

The patient underwent splenectomy and was admitted postoperatively to the intensive care unit (ICU). The preoperative treatment with piperacillin-tazobactam 3×4.5 g was continued. Enoxaparin 40 mg day^{-1} was initiated for venous thromboembolism prophylaxis, and the patient was mobilized on the second postoperative day. Despite antibiotic therapy, leukocyte counts progressively increased and CRP levels remained persistently elevated, and other acute-phase reactants, such as procalcitonin, showed only modest decreases (WBC 24.000 → 42.610 → 40.650; CRP 426 → 318 → 337 mg L^{-1}). Based on recommendations from infectious disease specialists, antibiotic therapy was escalated to meropenem (1 g three times daily). Intensive care unit admission cultures were obtained as part of the infectious work-up. Urine cultures collected both before and after ICU admission showed no growth. No microorganisms were isolated from the splenic abscess material or surgical wound cultures. Blood cultures yielded growth of coagulase-negative staphylococcus. Following this result, vancomycin was added to meropenem therapy in accordance with infectious disease consultation. Due to the slow progression of the patient's clinical course, intensive care monitoring continued. During this period, triflow breathing exercises and postural drainage were performed. The patient's postoperative platelet counts were monitored at 596, 520, 648, and 751 thousand mm^{-3} (Table I).

The patient was continuously monitored in the ICU, and no episodes of atrial fibrillation or other clinically significant arrhythmias were detected during hospitalization. Examination revealed abdominal distension. A repeat abdominal CT scan was performed, showing gas distension in the intestines; no additional collection or residual abscess was detected at the splenectomy site. Accordingly, a rectal enema was administered, and prokinetic therapy with metoclopramide was initiated.

On postoperative day 4, the patient developed acute pain, bruising, and erythema in the right lower extremity, followed by rapidly progressive sensory loss accompanied by motor deficit. On examination, pulses could not be palpated at the right popliteal and ankle levels, while pulses in the left lower extremity were markedly weak. A cardiovascular surgeon was consulted, and Doppler ultrasound revealed decreased arterial flow in the lower extremities. Subsequently, lower extremity CT angiography was performed, and the report suggested bilateral popliteal artery thrombosis (Figure 2).

Given the acute onset of symptoms, severe neurological deficits, and imaging-confirmed bilateral popliteal artery occlusion, the cardiovascular surgery team decided to proceed with emergency bilateral popliteal embolectomy under general anesthesia. Postoperative Doppler ultrasonography confirmed restoration of arterial flow, and physical examination demonstrated palpable and symmetrical distal pulses in both lower extremities. The patient experienced a marked reduction in ischemic pain following the procedure.

For evaluation of a potential embolic source, the patient was referred to cardiology. Both transthoracic and transesophageal echocardiography were performed, revealing sinus rhythm, a left ventricular ejection fraction of 65%, and normal right and left cardiac chamber dimensions. No intracardiac thrombus, valvular vegetation, or structural cardiac abnormality was identified, and patent foramen ovale was excluded.

Postoperatively, iloprost infusion (20 μg day^{-1}) was initiated as adjunctive peripheral vasodilator therapy and continued for three days. No additional antiplatelet therapy was administered during this period. The patient experienced only a brief, self-limited febrile episode in the postoperative course. Repeated cardiac auscultation revealed no murmurs, and no peripheral stigmata of infective endocarditis were observed. Following the 3-day course of iloprost therapy, the patient was discharged with antiplatelet therapy. Approximately one week after discharge, she developed recurrent arterial occlusion involving both lower extremities, resulting in recurrent impairment of lower-limb perfusion. The patient underwent reoperation at another medical center; however, despite surgical intervention, progressive ischemic necrosis of both lower extremities developed. Consequently, bilateral below-knee amputations were performed at the external center. No further follow-up data were available to our institution after the patient was transferred to the other center.



Figure 2. A) Bilateral blood flow is observed proximal to the popliteal level. **B)** Bilateral arterial thrombosis at the popliteal level. **C)** No contrast enhancement is seen distal to the popliteal level.

DISCUSSION

Venous thrombotic complications following splenectomy, particularly portal or splenic vein thrombosis and deep vein thrombosis or pulmonary embolism, are well-recognized and have been reported with incidences ranging widely from 0.8% to 55%, with more recent large series suggesting rates of approximately 8% (5).

Although arterial thrombotic events are less common, reports exist of cases such as peripheral artery thrombosis, aortic/renal/extremity artery thrombosis after splenectomy; reviews also indicate that splenectomy raises the risk of arterial events (6).

Several mechanisms have been proposed to explain the increased thrombotic risk following splenectomy. These in-

clude reactive thrombocytosis, increased platelet activation and circulating microparticles, infection- or sepsis-related hypercoagulability, and the prothrombotic effects of surgery performed in the setting of active inflammation. The term "inflammatory surgery" refers to surgical interventions undertaken during active infection and systemic inflammatory states, which may further amplify procoagulant pathways (7). In the United States alone, about 16,500 people develop reactive thrombocytosis annually after splenectomy, and around 1,100 patients experience thrombotic events. Studies categorize post-splenectomy thrombocytosis by severity: mild (platelet count 500,000–700,000 mm^{-3}), moderate (700,000–900,000 mm^{-3}), severe (>900,000 mm^{-3}), and very severe (>1,000,000 mm^{-3}) (8). Another study reported that post-splenectomy reactive thrombocytosis is independent of venous thromboembolism development and that previous studies' results are inconsistent with each other (9).

In the present case, several patient-specific factors may have contributed to arterial thrombosis, including active infection with significant systemic inflammation, emergency splenectomy, obesity, diabetes mellitus, hypertension, and postoperative reactive thrombocytosis. Importantly, alternative major causes of arterial thrombosis were carefully evaluated. Continuous cardiac monitoring revealed no atrial fibrillation, and comprehensive echocardiographic assessment, including both transthoracic and transesophageal echocardiography, excluded intracardiac thrombus, valvular pathology, infective endocarditis, and patent foramen ovale. The absence of persistent fever, cardiac murmurs, and peripheral stigmata further reduced the likelihood of infective endocarditis. Additionally, although the literature shows mixed results, reactive thrombocytosis has been linked to both arterial and venous thrombotic events. Hydroxyurea is primarily used as cytoreductive therapy in chronic or clonal thrombocytosis and is not routinely indicated for acute reactive thrombocytosis. However, its use has been reported in selected cases of severe post-splenectomy reactive thrombocytosis, particularly when platelet counts exceed 1,000,000 mm^{-3} (8). In such cases, consultation with a hematologist may be appropriate to assess the thrombotic risk and determine whether cytoreductive therapy is warranted.

In addition to thrombocytosis, splenectomy may cause reactive leukocytosis. In our patient, the marked leukocytosis was likely multifactorial, reflecting both the ongoing infectious/inflammatory process and the post-splenectomy response. Although the independent contribution of leukocytosis to arterial thrombosis remains uncertain, its coexistence with reactive thrombocytosis may have further contributed to the prothrombotic state.

CONCLUSION

Regarding our treatment approach, an emergency bilateral popliteal embolectomy was performed, successfully restoring blood flow. Planning anticoagulation and antiplatelet therapy during the post-splenectomy period requires close monitoring of peripheral pulses, early mobilization, platelet counts, and adjunctive peripheral vasodilator therapy after embolectomy. In addition to thrombotic mechanisms related to splenectomy, other potential embolic sources, including atrial fibrillation and infective endocarditis, should also be considered and appropriately evaluated during the diagnostic work-up. Furthermore, it is essential to recognize that the long-term risk of thrombosis following splenectomy should not be overlooked, and evaluation for potential paradoxical embolism with transthoracic echocardiography remains an important component of comprehensive assessment.

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Informed Consent / Patient Consent: Written informed consent was obtained from the patient for the publication of this report.

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AUTHOR CONTRIBUTIONS

Conception or design of the work: TK
Data collection: SS
Data analysis and interpretation: TK, CG
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Critical revision of the article: CG
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The authors (TK, SS, CG) reviewed the results and approved the final version of the manuscript.

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