

Case report

Acute lower Limb Ischemia Caused by Fungal Infective Atrial Thrombus: A Case Report

Nasser Malekpour¹ , Maryam Abbasi², Faezeh Shams², Atoosa Gharib³ , Sara Besharat^{4*} 

Abstract

Septic thromboembolic events have two main consequences, ischemic and infection. This paper discusses a case with acute lower limb ischemia caused by fungal infective atrial thrombus. The patient underwent anticoagulant therapy. A 43-year-old female patient who suffered from valvular heart disease (history of mitral and tricuspid valve replacement) was referred to the vascular surgery department of Modarres hospital with acute left limb ischemia. She underwent a successful emergency surgical thrombectomy. The pathological report of thrombus demonstrated fungal infection. After surgical thrombectomy to control the acute lower limb ischemia, antimicrobial treatment of septic emboli is necessary.

1. Critical Care Quality Improvement Research Center, Tehran, Iran.
2. Clinical Research and Development Center, Shahid Modarres Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
3. Department of Pathology, Clinical Research and Development Center, Shahid Modarres Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
4. Department of Radiology, Shahid Beheshti University of Medical Sciences, Tehran, Iran

Corresponding Author:

Dr. Sara Besharat, Department of Radiology, Shahid Beheshti University of Medical Sciences, Clinical Research and Development Center, Shahid Modarres Hospital, Tehran, Iran.

Email: besharat.sara@gmail.com

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Introduction

Septic thromboembolism is an occlusion of a blood vessel, commonly caused by an infected thrombus that moves through the bloodstream far from the infectious source and occludes a blood vessel. Septic thromboembolism has two consequences, early ischemic due to vascular occlusion and the infection that leads to inflammation and probable abscess formation (1). Infective endocarditis is the most common cause of septic emboli (2). Generally, septic emboli start depending on the anatomical location of the infection and the vasculature surrounding that area. The clinical manifestation of peripheral vascular

emboli varies in severity, from transient ischemia to acute limb ischemia, requiring surgical intervention from thrombectomy to amputation. Acute lower limb ischemia has also occurred after MRSA pneumonia (3). This paper discusses a case with acute lower limb ischemia caused by the fungal infective atrial thrombus.

Case Report

A 43 -year-old female patient who suffered from valvular heart disease consulted with the vascular surgery department of Modarres Hospital with 2 days



Figure 1. Excised thrombi.

history of pain and numbness of the left lower extremity. Past medical history reported chronic renal failure, heart failure, atrial fibrillation rhythm, and valvular heart disease. She also had a history of



Figure 2. Excised thrombi in the second thrombectomy.

cerebrovascular accidents (CVA), mitral valve replacement, and Tricuspid valve repair about two months before admission. Taking warfarin was notable in her drug history.

Vital signs revealed blood pressure 140/88 mmHg, body temperature 36.5 °Celsius, and heart rate 98/min. Physical examination demonstrated that her left lower extremity was exceedingly painful, associated with a pulse deficit of dorsal pedis, tibialis posterior, and femoral arteries. The laboratory data are shown in Table 1. Ultrasonography demonstrated thrombosis in the left common iliac artery.

She underwent emergency surgical thrombectomy (Figure 1). Immediately after the surgery, blood flow and backflow were established, and distal pulse was palpable. However, five hours after the surgery, signs, and symptoms of ischemia returned (pain, pallor, pulse deficit, and poikilothermia). She underwent emergency surgical thrombectomy again (Figure 2). After the second surgery, she did not experience an ischemic attack. The pathological report of thrombus demonstrated fungal infection (Figure 3). Broad-spectrum intravenous anti-fungal therapy was initiated. To investigate the source of fungal infection, she underwent Transthoracic echocardiography (TTE). TTE showed a left ventricular ejection fraction (LVEF) of 45% and a well-seated bioprosthetic Mitral without vegetation on the bioprosthetic valve. The lab tests revealed anemia (with hemoglobin of 6.8 g/L), impaired coagulation tests, prerenal uremia, and increased markers of inflammation; while blood culture and urine culture were done. A chest x-ray and a lung CT scan were normal. After all workups, we found positive urine culture for yeast.

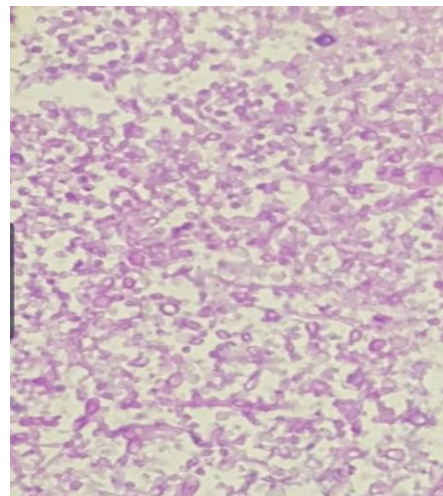


Figure 3. Fungal elements (PAS staining).

Discussion

A source of infection may cause septic thromboembolism. Infective endocarditis (2), tunneled hemodialysis catheters (4) and Cardiac implantable electronic devices (CIED) (5) are the most causes of

septic emboli in order of prevalence.

Septic emboli might also be originated from the most common microorganism such as *Staphylococcus aureus*, coagulase-negative *Staphylococcus*, streptococcal group, enterococcus group, polymicrobial infections, and less frequently, gram-negative organisms. Fungi, mostly *Candida* and *Aspergillus*, may be found in septic emboli (6). Septic emboli have an extensive range of manifestations from asymptomatic to severe with high mortality. Bellomy et al. (7) explained a case where femoral occlusive septic emboli were identified by point-of-care ultrasound after mitral valve replacement.

To detect the cause of septic thromboembolism, we obtained at least three blood cultures. Arterial and venous duplex studies are required to pursue arterial emboli and venous thrombophlebitis. Echocardiography is necessary to detect valvular vegetation. Transesophageal echocardiography leads to a better outcome for the detection of vegetations than transthoracic echocardiography. To diagnose septic pulmonary emboli, a chest CT scan with IV contrast is required. The CT scan shows multiple peripheral nodular lungs infiltrates, with or without cavitation. Ultrasound may detect hypochoic lesions in the spleen or kidneys (8). Two main steps to manage septic emboli are source control and prolonged antimicrobial therapy.

The antimicrobial therapy in the control of septic emboli targets the causative organisms, the involved organs, and the pharmacokinetics and pharmacodynamics of the available drugs. The management would change based on the location of the septic embolus and the size of the surrounding infarct or inflammatory area (8).

In our test case, we did not find any source for fungal thromboembolism. Her blood culture was negative. Her echocardiography did not show any evidence for endocarditis. Her lung CT scan did not show any evidence of pneumonia or pulmonary embolism. In our check-up, we just found a positive urine culture. Surgical thrombectomy was done to treat her acute limb ischemia, anticoagulant therapy

continued, and broad-spectrum intravenous anti-fungal therapy was initiated.

Conclusion

PVE could cause peripheral thromboembolism in patients under therapeutic anticoagulant treatment. After initial surgical management of acute peripheral ischemia, treatment of endocarditis could lead to sufficiently good results in selected patients.

Acknowledgment

None.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

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