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# Mycotic Popliteal Aneurysm Due To Infective Endocarditis: Case Report And Review Of The Literature

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### Abstract:

Background:

Infective endocarditis is a serious pathology associated with a heavy morbidity and mortality, affecting approximately 1.7 – 6.2 per 100,000 patients every year. Its management is difficult and requires the heart team endocarditis involvement. Among its complications we note mycotic aneurysms (MA) which preferentially affect the cerebral, abdominal arteries and more rarely the peripheral ones.

Mycotic popliteal artery aneurysm is very rarely reported, its treatment is based on a surgical cure for the underlying heart valve disease with exclusion of mycotic aneurysm. <u>Case summary</u>:

We report the case of a young patient with a sickle cell disease admitted for management of infective endocarditis complicated by a cerebral abscess and a popliteal artery mycotic aneurysm.

Discussion/conclusion:

This case study has led us to conclude that infective endocarditis associated to mycotic popliteal aneurysm is very rare. Frequent complications are acute ischemia or aneurysmal rupture which constitutes a life threatening or even fatal complication this is due to difficulties of diagnosing the mycotic aneurysm.

This observation is crucial to emphasize the role of early diagnosis that improves the prognosis with very favorable outcome.

**Keywords:** infective endocarditis, mycotic aneurysm, popliteal artery.

#### **Introduction:**

Infective endocarditis (IE) is considered to be a serious disease due to its complications and is currently a condition affecting approximately 1.7 - 6.2 per 100,000 patients every year. [1]

Vascular complications are still frequently encountered with IE, despite the development of diagnostic and therapeutic materials in the management of this clinical entity. On the other hand, peripheral vascular complications are rare in this case and are usually the result of Oslerian embolism and mycotic aneurysm.

In actually, Mycotic aneurysm is a dreaded vascular complication that may affect any vascular territory however it is rarely encountered. The first time it was reported was in 1885 by William Osler describing a mycotic aortic aneurysm due to septic emboli from a bacterial endocarditis. *[2]* 

We herein report the case of a young patient with no cardiovascular risk factors, admitted for management of infective endocarditis complicated by a cerebral abscess and mycotic aneurysm of the popliteal artery.

### **Case report:**

A 33-year-old male was admitted to the cardiac emergency room, he presented a dyspnea class II that aggravated, a month and a half prior to his admission, into class III of the NYHA classification associated with frontal headaches, asthenia, vertigo and fever.

He was recently diagnosed with sickle cell disease (3 years ago). He doesn't have any history of repeated angina or acute articular rheumatism, no toxic habits or any drug addiction and does not report any recent dental care.

On admission, he was afebrile, pale, anicteric, blood pressure 107/55 mmHg with tachycardia (100 bpm). The electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy and inferior negative T waves.

Chest X ray was normal. Hemoglobin value was 6.5mg/l, Haptoglobin <0.08, and white blood cell count was 24540/mm3. Presented increased inflammatory markers: C-reactive protein: 91g/l, Ferritinemia elevated to 251. Cytobacterioligical urine exam showed leukocyturia without bacteriuria and microscopic hematuria, multiples blood cultures grew *streptococcus species*. Based on the criteria of DUKES, the diagnosis of infectious endocarditis disease was strongly suspected. We completed then with transthoracic echocardiography **(figure 1A; figure 1B)** and transesophageal echocardiography **(figure 2)** that confirmed the diagnosis by showing several hyperechoic elements on the atrial side of the small mitral valve with variable sizes, the largest one measuring 19x12mm implanted on a dystrophic valve with a false prolapse of P2, with eccentric mitral leakage.

Two days after his admission, the patient presented acute headache, the fundus examination showed a papillary oedema class 1 with bilateral stasis, and crooked ships.

The body CT scan revealed a right frontal brain abscess (likely to be a septic embolism given the context) measuring 14x13x13mm associated with a perilesional edema in glove finger with spontaneously hyperdense areas (hemorrhagic infarction), a hypodense hepatic lesion of segment 5, an asplenia, Horseshoe kidney with lithiasis on the left. **(figure 3A; figure 3B).** We started a double antibiotic therapy with meningeal doses: ceftriaxone 2g t.i.d (100mg/kg/d) + gentamycin 160 mg q.d, rehydration and paracetamol.

On the 7<sup>th</sup> day of hospitalization, the patient presented a red-hot swelling of the left ankle, the venous ultrasound scan of the lower limbs showed no signs in favor of deep venous thrombosis, as well as no soft tissue infiltration, only joint effusion of the left ankle.

The MRI of the left ankle showed talo-calcaneal arthritis with Achilles bursitis. **(figure 4A; figure 4B)** 

The arterial Doppler ultrasound of the left leg showed a ruptured aneurysm of the popliteal artery. The next day the patient was submitted to angiography which revealed a false left popliteal saccular aneurysm centered on the anterior tibial artery. **(figure 5A; figure 5B)** The patient benefited from an ankle joint wash, then transferred for vascular surgery repair.

## **Discussion:**

Mycotic aneurysm (MA) is a rare complication of infective endocarditis, it is a localized, irreversible arterial dilatation caused by the destruction of the vessel wall due to infection,

making the management of these patients more complicated. This complication has been reported to occur in 1.2% to 5.4% of patients [3]

Various pathophysiological mechanisms can contribute in the genesis of the MA such as: invasion of the vessel by a micro-organism migrating from the endocardial site, embolic occlusion, an immunological occlusion by deposit of immune complexes on the vascular wall or by direct or lymphatic contiguous spread from a local infection or a purulent focus and direct bacterial inoculation at the time of trauma. *[1,4,5,6,7]* 

All vascular territories can be affected, from highest to lowest frequency: cerebral arteries, the abdominal aorta then the femoral arteries are the most frequent sites of embolization. However, MA of the popliteal artery is extremely rare. *[8,9]* According to literature, the first case of MA affecting the tibio-peroneal trunk complicating infectious endocarditis was described by Akers et al. in 1992. *[10]* Additionally, Brudon et al. described 21 cases of mycotic aneurysms, within 9 ilio-femoral and 8 popliteal locations. *[11]* 

Trans-thoracic echocardiography and trans-esophageal echocardiography are the most commonly used as predictors of embolic events, as demonstrated by Thuny et al. in a prospective European multicenter study including 384 patients with confirmed infective endocarditis showed that the presence of germs as *staphylococcus aureus* or *streptococcus bovis*, a large vegetation measuring of over 10mm and/or a severe vegetation mobility, are common predictors of embolic risk *[12]*. In the same study, only 3.84% of peripheral artery damage was reported before the introduction of antibiotic therapy *[12]*.

In another retrospective study by Zarzur et al. spanning over a 6-year period and comprising 82 patients, 18 of which developed a vascular complication with only one case of mycotic aneurysm of the popliteal artery[13].

Three main complications of MA are described: acute thrombosis, aneurysm rupture with life-threatening and distanced reinfection after IE episode[13].

The clinical manifestations of peripheral mycotic aneurysms are variable and not specific: they can include pain, fever, redness, acute ischemia of the limb secondary to thrombosis or aneurysmal rupture, sometimes the presentation as deep vein thrombosis may mislead the diagnosis. Peripheral neuropathy by direct compression of the peripheral nerves was reported in very few cases[14].

Indeed, 90% of peripheral aneurysms are easily palpable unlike those affecting the aorta and iliac arteries, which can be undetectable by a simple physical examination and must be detected by echo-Doppler.

In a study by Zarzur et al. 54% of IE were diagnosed 2 weeks after the beginning of antibiotic treatment[13]. However, these vascular complications can occur at a distance from the clinical and biological healing of IE or even after surgical treatment of the underlying valvulopathy [15].

On a bacteriological level, the majority of mycotic aneurysms are bacterial in origin. According to Magilligan and Quinn, among the infectious agents responsible of peripheral mycotic aneurysm complicating bacterial endocarditis on native valve, we note the *alphahemolytic streptococcus viridans group* (22%), *staphylococcus aureus* (20%) followed by *streptococcus feacalis* and *staphylococcus epidermidis* (14% and 11%)[16]

On one side, studies made by Oz et al and Malouf et al, isolated the organisms involved in MA who are represented mainly by cocci-gram + with 45% of cases *staphylococcus aures* and in 10% of cases *streptococcus*[17,18].

On the other, fungal mycotic aneurysms, represent 1% of the MA according to the Brown SL team, and are generally encountered with immunocompromised patients. The most frequently described fungi are: *Candida Albicans, Aspergillus* and more rarely *Penicillum* or *Histoplasma [19]*. These fungal mycotic aneurysms are associated with a very high mortality rate *[20]*.

According to the literature, only 50% of blood cultures are negative and this should not exclude the diagnosis of a mycotic aneurysm *[21,22]*.

The examination of choice for the diagnosis of MA remains the echo-doppler which is non-invasive and readily accessible. During this examination, the physician must: locate the site of the aneurysm, determine its diameter, its type and content, visualize a possible parietal thrombus and study the peripheral vascularization to evaluate the quality of the arterial network upstream and downstream. Thus, in case of suspicion of popliteal artery aneurysm, the CT-angiography is systematically performed to guide the operative approach [23,24].

Treatment is based on the administration of a well-adapted parenteral antibiotic for long periods of time associated with a surgical cure of the underlying valve disease. The treatment of mycotic popliteal artery aneurysm (MPAA) consists of an exclusion of this formation, ligature associated with a wide resection of the infected area then restoration flow by a graft. It may be either an autologous vein (internal saphenous vein or superficial vein of the lower limb) [22] or prosthetic in situ (poly-tetra-fluro-ethylene-expanded prosthesis (PTFE)) that is more resistant to infection compared to Dacron prostheses according to Christides et al. publication. These anastomoses should be performed at a distance from the infected area to reduce the risk of suture loosening [23,25].

Surgical treatment should allow the control of hemorrhage, complete excision of the infectious site to avoid any infectious recurrence from an unsterilized aneurysm as well as the restoration of revascularization of the downstream territory [26].

Endovascular treatment such as stent placement and embolization may be an alternative to surgical treatment [26].

### **Conclusion:**

Mycotic aneurysm of the popliteal artery complicating bacterial endocarditis is rare. Frequent complications are acute ischemia or aneurysmal rupture which is considered a fatal and life threatening complication, hence the necessity of early detection, adequate and urgent surgical management, that can consequently improve the functional and vital prognosis.

#### **Competing interests**

The authors declare no competing interest.

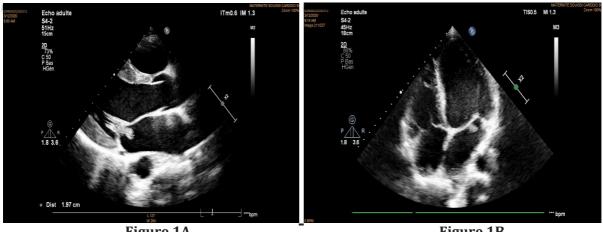
#### Authors' contributions

JCR Doctors Samih, Kadiri have examined and supported the patient. Doctor Faid has contributed to the research and supported the patient. Professors Zarzur and Cherti have read and corrected the manuscript.

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#### **Figures**



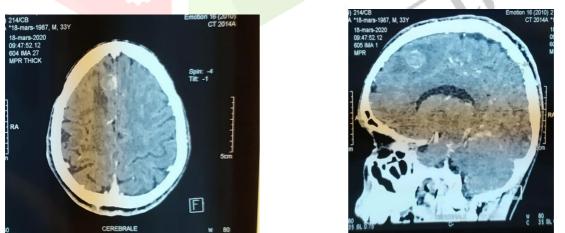
**Figure 1A** 

Figure 1B

Figure 1A, 1B: Echocardiography showing a hyperechoic element appended to the distal end of the large mitral valve on the ear slope measuring 19.7x11.5mm.



Figure 2: Transesophageal echocardiography showing hyperechoic elements on the atrial side of the small mitral valve with a false prolapse of P2.



**Figure 3A** Figure 3B Figure 3: Cerebral CT scan showing a right frontal brain abscess.





Figure 4AFigure 4BFigure 4:MRI of the left ankle showing talo-calcaneal arthritis with Achilles bursitis.



Figure 5AFigure 5BFigure 5CFigure 5:Angiography of lower limbs showing a false left popliteal aneurysm.

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