Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Surgical Intensive Care Unit

An Epidural Abscess Complicating an Infectious Endocarditis: Case Report

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DOI: 10.36347/sjmcr.2023.v11i04.083 | **Received:** 05.03.2023 | **Accepted:** 19.04.2023 | **Published:** 30.04.2023

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Abstract **Case Report**

Infective endocarditis is responsible of high intra-hospital morbidity and mortality rates. This can be explained partially by the diversity of its complications, which are often difficult to diagnose and manage therapeutically. We report the case of a 63-year-old woman, admitted to the intensive care unit for a sepsis due to infective endocarditis. On her second day of admission, she presented rapidly worsening paraparesis that leaded to flaccid paraplegia, for which she underwent a spinal MRI showing an extensive epidural abscess from T8 to T12 with spinal cord compression. The patient benefited from an emergency laminectomy, and the intraoperative bacteriological sample found the same germ isolated in the blood cultures (staphylococcus aureus methicillin sensitive). This case report emphasizes the importance of clinical and paraclinical evaluation of patients with infective endocarditis, in order to detect complications as early as possible, because if unnoticed, their management become very delicate.

Keywords: Infectious Endocarditis, complication, epidural, abscess, staphylococcus aureus.

INTRODUCTION

Infective endocarditis is characterized by a microbial infection, most often bacterial, of the endocardium [1]. It frequently affects the valve apparatus of the left side of the heart. Infective endocarditis is responsible of high mortality rate [2] explained by the various complications making therapeutic management very difficult.

The osteoarticular manifestations of infective endocarditis are dominated by spondylodiscitis and septic arthritis. We report a case of a peridural abscess complicating a Staphylococcus aureus infective endocarditis.

CASE REPORT

A 63-year-old woman was admitted to the intensive care unit (ICU) to manage a sepsis secondary to infective endocarditis. In her medical history, we found arterial blood hypertension under calcium channel blockers and heart failure diagnosed 3 months before, under beta-blockers and an angiotensin II receptor antagonist. The patient was also being treated for end-stage chronic renal failure, for which she was undergoing dialysis via a left humerocephalic arteriovenous fistula for 2 years.

One month before her admission to ICU, the woman presented an aneurysm at the level of the arteriovenous fistula, for which she underwent a flattening and placement of a prosthesis as well as a tunneled dialysis catheter in the right internal jugular vein.

Six days before her admission, the patient presented a persistent 39°-40°C fever, associated to a tunnelitis in the path of the dialysis catheter with pus issue. The catheter was removed, send for bacterial analysis, as well as blood cultures. An empirical antibiotic therapy based on teicoplanin was started. Because of the lack of improvement and the worsening of her clinical condition, the woman was admitted to ICU.

At that time, the patient was febrile at 39.5°C, tachypneic (30 cycles/min) with a blood oxygen saturation of 88% on room air. Hemodynamically, she had a Mobitz I type 2 atrioventricular block at a rate of 40-45 beats/minute and a blood pressure of 120/80mmHg, with no signs of peripherical hypoperfusion. Neurologically, she was conscious, with no deficit on initial examination. The insertion site of the left femoral temporary dialysis catheter was clean.

A chest CT angiography was performed, showing multiple septic pulmonary emboli without parenchymal involvement. The blood cultures (three in total), came back all positive for methicillin-sensitive Staphylococcus aureus. The initial transthorasic ultrasound showed a suspicious image at the small mitral valve, which led us to complete with transesophageal ultrasound, showing a multilobed mobile mass, inserted on the atrial side of the small mitral valve, measuring 24mm x 7mm. According to the modified DUKE criteria (3), the diagnosis of Infective endocarditis was retained.

On her second day after admission, the patient presented rapidly worsening paraparesis that leaded to flaccid paraplegia, for which she underwent a spinal MRI showing an extensive epidural abscess from T8 to T12 with spinal cord compression. The patient benefited from an emergency laminectomy, and the intraoperative bacteriological sample found the same germ isolated in the blood cultures (staphylococcus aureus methicillin sensitive).

In the follow-up, the patient presented a deterioration of her neurological state with a GCS going from 15 to 6, associated with respiratory distress attributed to nosocomial pneumonia. Thus, the patient was intubated based on neurological and respiratory criteria. The lumbar puncture performed came back negative. The cerebral MRI objectified a cerebral ischemic stroke. Given her clinical condition, and the worsening of biological markers, the spectrum of antibiotic therapy was enlarged, adding cefepime in combination with teicoplanin.

The surgical indication for the treatment of Infective endocarditis was retained initially, but the procedure could not be performed given the severity of her neurological condition.

The patient was extubated on day 17 of her hospitalization, after she regained her basic neurological state and improved of her pulmonary exchange. Despite this, she kept a low muscle strength (2/5 in the lower extremities). Control spinal MRI showed thickening of the compressive meningeal bundle, for which the patient was put on corticosteroid therapy for 7 days but without clinical improvement.

22 days after her admission, the patient was transferred to a cardiovascular surgery unit for further management.

DISCUSSION

We can subdivide the complications of infective endocarditis (IE) into intracardiac and extracardiac complications. For the cardiac one, they can be of the type of heart failure, myocardial infarction, pericarditis, intracardiac abscess, or rhythm and conduction disorders. These can manifest in several forms, including atrioventricular block, which signs an extension of the infection and the formation of an abscess interrupting the intracardiac conduction pathways]1].

As for the extracardiac complications, they can be generated either by an immunological process, or by a vascular damage. Embolic accidents are generally the consequence of migration of emboli from the valvular vegetations. They represent septic thrombi, which are most often implanted on the upstream side of the valve leaves, which corresponds to the low pressure zones where the rheological conditions enable platelet aggregation on the pathological valve tissue or on a foreign material, stimulated by pro-inflammatory cytokines [4]. These vegetations will eventually send emboli, either to the lung for IE of the right heart, or through the systemic circulation for IE of the left heart, to reach the brain most often, or the abdominal locations (spleen, kidney).

The beginning of antibiotic therapy plays a decisive role in the frequency of these embolic accidents, with an increase of their incidence the days before and a rapid decrease the days following the start of antibiotic therapy [5]. However, the major problem relies on the difficulty of detecting these emboli, since they can be clinically asymptomatic, as they can be clinically speaking, or even constitute the revealing mode of the IE. This may explain the great variability in the percentage of embolic complications reported in the literature [5]. Indeed, cerebral emboli represent more than 50% of the cases reported in the literature [6]. This can be explained by the clinical impact caused, thus making their diagnosis obvious. Unlike peripheral emboli, which may go unnoticed, explaining their low percentage in the literature. As an example, in the French epidemiological study of 2008, they realized systematic complementary imaging for 87% of patients, the embolic episode was observed in 45% of patients, and it was symptomatic only in the 2/3 of cases [6].

Moreover, musculoskeletal symptoms and rheumatological manifestations may be the first manifestations of IE and can delay its diagnosis. Peripheral arthritis occurs in about 14% of cases, and the prevalence of spondylodiscitis in patients with IE is about 1.8–15% [3].

No particular clinical risk factor for infectious spondylodiscitis has been identified during infectious endocarditis. The prognosis of infectious spondylodiscitis during infectious endocarditis appears to be identical to that of isolated infectious spondylodiscitis, with a similar mortality rate, about 8% [7]. An increase in neurological complications due to the presence of epidural abscesses has been described [8].

Epidural abscess is a very rare complication, reported in only 2 patients in the last four decades [9, 10]. Both cases presented since admission a neurological symptomatology made of progressively worsening low back pain with paraparesis of the lower limbs. The first case [9] was due to an L4-L5 epidural abscess, revealing left heart IE (streptococcus pneumoniae), while the second case [10] had an L5-S1 epidural abscess, complicating an IE due to methicillin-resistant Staphylococcus aureus.

The exact pathophysiological mechanism is difficult to determine. It can be caused by an embolization of the vertebral artery, a septic necrosis of the vertebral body, or even the consequence of immunological deposits [10] .

It appears that the pyogenic propensity of the underlying organism causing IE is at least in part responsible for the development of spondylodiscitis and secondary epidural abscess [8]. The prognosis depends on the rapidity of the diagnosis and the implementation of the treatment.

The clinical case reported above corroborates these data. A prolonged antibiotic therapy is generally required until no signs of inflammatory activity are detected [3].

CONCLUSION

Infectious endocarditis is responsible of a high mortality rate, and this is mainly due to its various complications and their difficult diagnosis. Therefore, a detailed clinical examination while maintaining a unified diagnostic approach, is necessary, in order to ensure appropriate therapeutic management.

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