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Cardiology

Ischemic Stroke with Aortic Infective Endocarditis Complicated by Pulmonary Artery Abscess

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Abstract | **Case Report**

The initial lesions of infective endocarditis (IE) are mainly valvular, but sometimes may cause other cardiac or extracardiac complications. Cerebral involvement is the most frequent site of extracardiac complications caused by IE. They might be the mode of revelation in 10 to 20% of patients. These complications increase morbidity and mortality. We report the case of a patient with IE complicated by a fistulised abscess in the pulmonary artery leading to a stroke.

Keywords: Ischemic stroke, infective endocarditis, abscess.

INTRODUCTION

Infective endocarditis (IE) manifests itself with signs affecting almost all organs and is usually divided into cardiac and extracardiac complications. The disease begins with the formation of vegetations in the heart, which might increase in size and number, leading to degradation and destruction of valvular and perivalvular tissue with or without extracardiac extension. We report a patient with IE complicated by a fistulised pulmonary artery abscess leading to a stroke.

PATIENT AND OBSERVATION

44 year old patient admitted for ischemic stroke associated with prolonged fever, with a history of smoking and chronic alcoholism. On admission the patient was febrile at 39°C; tachycardic at 101 beats per minute, his blood pressure was 126/76 mm Hg, a right pyramidal syndrome and aphasia were noted.

Cardiac auscultation found a high-pitched, crescendo-decrescendo midsystolic murmur, with an early diastolic soft decrescendo murmur at the aortic focus, and bilateral basithoracic crepitus rales.

The biological assessment showed a positive inflammatory syndrome, with a CRP of 260mg/l, a hyperleukocytosis of 33,000 with a predominance of polynuclear cells. viral serologies, blood cultures and 24-hour proteinuria tests were negative, and the cytobacteriological examination of the urine showed leukocyturia without any germ.

Echocardiography (transthoracic and transoesophageal) showed a left ventricle of normal size and systolic function, the aortic valve is remodeled, tricuspid without commissure fusion, presence of vegetations (Figure 1) the largest of which measured 10 mm, a peri-annular abscess discharged into the pulmonary artery (Figure 2), severe multijet aortic insufficiency. There is a poorly restrictive perimembranous septal defect (VSD) measuring 7 mm. The right heart chambers are noted to be dilated, with a well-functioning right ventricle.

Medical imaging (MRI) of the brain showed hypodensity of ischemic appearance in the deep left sylvian territory, sinus radiography and fundus of eye were normal.

The patient was treated with ceftriaxone and gentamycin as a probabilistic treatment for infective endocarditis. The evolution was favourable until the 25th day of treatment, when the patient developed acute dyspnea with desaturation, tachycardia and murmur on pulmonary auscultation. The electrocardiogram showed SIQ3, the chest X-ray showed cardiomegaly with a snowflake appearance of the lung. Death occurred the following day.

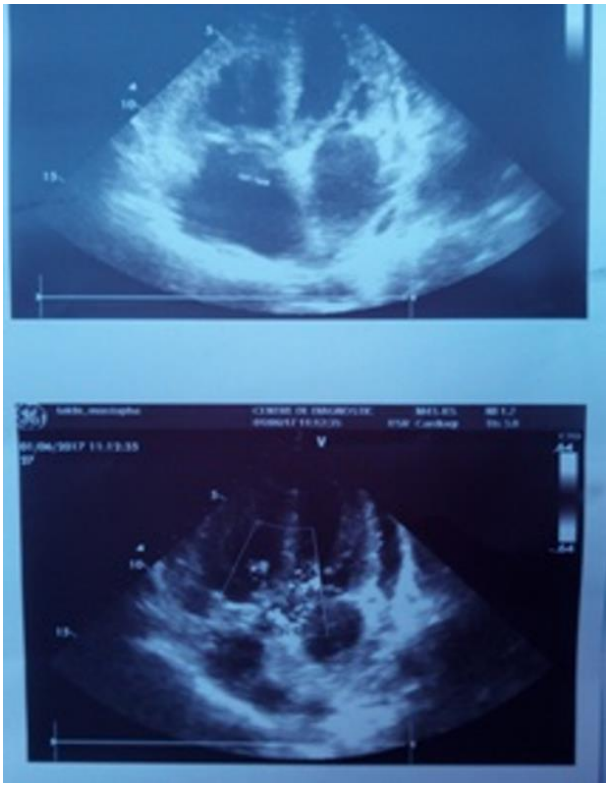

Figure 1: TTE showing vegetation on aortic valve

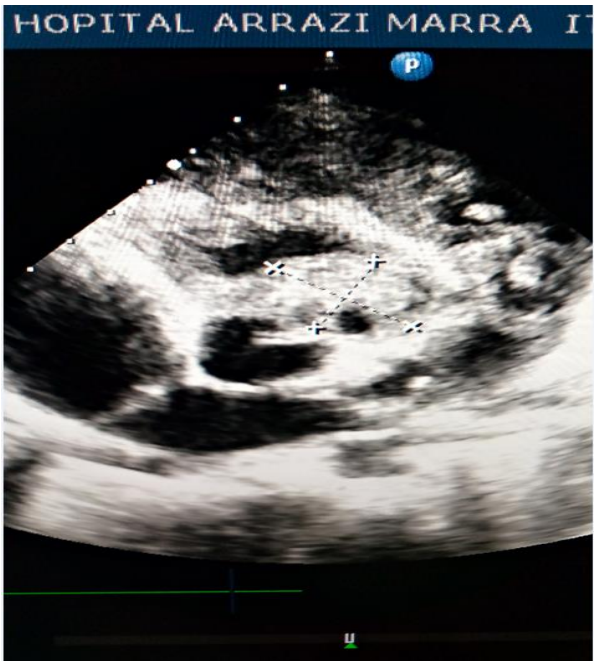

Figure 2: TTE showing the periaortic abscess fistulized in the pulmonary artery

DISCUSSION

The initial lesions of IE are predominantly valvular, and may be complicated by perivalvular extension in the absence of appropriate treatment. Bacterial adhesion and growth on the damaged valvular endocardium or on intracardiac material results in vegetation, which is the initial lesion of IE. Anatomical damage most often results in functional damage. Valve leaks are the most common, and may be related either to a coaptation defect when the vegetation impedes normal valve movement, or to perforation, in which case they will be eccentric, arising from the valve body [1].

Progression of the infection in the heart results in an increase in the size and number of vegetations, with destruction of valvular tissue and perivalvular extension. The perivalvular extension of the infection is particularly to the valve annulus. Abscesses are more frequent at the peri-aortic level where they can drain into one of the 4 cardiac chambers depending on their location. Peri-valvular damage occurs in 20% to 40% of cases, more frequently in the aortic valve and in the presence of a valve prosthesis. Fistulisation is rarer, diagnosed in about 3% of cases [2].

IE can cause numerous extracardiac manifestations through vascular and/or immunological phenomena. Many of these complications are minor criteria in the diagnostic classifications of IE. Cerebral complications are the most frequent complications of IE. Their prevalence varies between 10% and 65% depending on the study and the imaging technique used [3].

The presentation of neurological complications of infective endocarditis is broad. It includes ischaemic and haemorrhagic strokes, transient ischaemic attacks (TIAs), asymptomatic (or silent) strokes and infectious complications such as infectious aneurysms, brain abscesses and meningitis. Ischaemic strokes related to septic emboli constitute the majority of cerebral complications of endocarditis [4]. These emboli are most often manifested by a focal neurological deficit (hemiparesis, hemianesthesia, hemianopsia, etc.) that is sudden, permanent or transient, and occurs in a febrile setting [3].

There is no specific medical treatment for ischemic strokes in IE . Intravenous thrombolysis cannot generally be considered, given the high risk of haemorrhagic transformation of the lesions. Symptomatic management is similar to management of stroke in the acute phase, avoiding any secondary cerebral insult [5].

CONCLUSION

Infective endocarditis is a serious condition that must be diagnosed and treated promptly. Urgent assessment is therefore required, including microbiological analysis and imaging techniques, to assess the risk of severe complications and death. Once a diagnosis has been made, prompt action should be taken to reduce the risk of complications, including the use of probabilistic antibiotic therapy and, if necessary, surgery. These interventions can reduce the risks and improve the patient's prognosis.

Conflicts of Interest

The authors declare no conflicts of interest

Author Contributions

All authors contributed to the drafting of the manuscript, all authors read and approved the final version of the manuscript

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