Infective endocarditis on ventricular septal defect complicated by septic pulmonary embolism: A case report

Abstract

Ventricular septal defect (VSD) is a congenital heart disease most likely to cause infective endocarditis. Pulmonary embolism constitutes one of its main complications. Treatment is mostly based on effective antibiotic therapy but can sometimes require additional surgical intervention. We report a case of infectious endocarditis in a 20-year-old patient followed for congenital heart disease such as ventricular septal defect, complicated by septic pulmonary embolism; whose starting point is a vegetation located at the right heart side, with a good clinical-biological evolution under medical treatment only and a complet disappearance of the vegetation.

Keywords: Infectious endocarditis; Ventricular septal defect; Pulmonary embolism

Introduction

Infectious endocarditis (IE) is a serious condition causing significant morbidity and mortality. Among its risk factors are congenital heart diseases, with VSD being the most frequent (1). Patients with VSD have an incidence of 1.67/1000 patient per year, or 11 to 15 times greater risk than the general population (2). Septic pulmonary embolism represents a rare complication, thereby increasing the mortality risk and necessitating appropriate care.

Case Presentation

This is a 20-year-old patient with a history of VSD-type congenital heart disease who consults for dyspnea at rest associated with subacute onset fever that has been ongoing for 2 months. Hemodynamically stable with a systolic blood pressure 111 mmHg and diastolic blood pressure of 61 mmHg, and a temperature of 39.6°C, ecchymotic purpura in the lower limbs, with a perceivedholosystolic murmur at the left sternal border during heart auscultation, the rest of the clinical examination was unremarkable.

Electrocardiogram (ECG) showed a regular sinus rhythm at 107 beats per minute (bpm), fixed PR interval at 120ms, normal heart axis, narrow QRS wave, without repolarization disorders. Transthoracic echocardiography showed good bi-ventricular function, fused mitral profile, the atria were not dilated, no mitro-aortic valvulopathy, presence of a peri-membranous VSD of 11mm long axis with low speed left-right shunt (Figure 1), with a maximum gradient across the VSD at 88mmHg, site of a hyperechoic filiform image measuring 23x12 mm on the lower border of the VSD floating in the right ventricle (Figure 2), non-dilated inferior vena cava (IVC) at 11 mm compliant, no effusion pericardial. Faced with this clinical and echocardiographic picture, the diagnosis of infective endocarditis was retained.

Biologically: hyperleukocytosis at 18030 G/L with predominantly PNN (14170 G/L), C-reactive protein (CRP): 340g/L, renal and hepatic function as well as the hemostasis assessment were correct whereas the 2 blood cultures were positive for staphylococcus aureus. As part of the extension assessment, a chest angio-CT scan was carried out and showed the presence of a bilateral distal pulmonary embolism strongly suggestive of septic emboli (Figure 3), the fundus examination was normal.

The portal of entry was probably dental given the poor oral condition, the patient was put on dual antibiotic therapy based on ceftriaxone 2g/day and gentamycin 160 mg/day with good clinical-biological. Clinical exams: patient was afebrile (T°: 36.8°C), saturation of 98% in open air, with negative CRP value and normal white blood count.

A surgical closure of the VSD was retained taking into account the size of the vegetation and the pulmonary vascular resistance on echocardiography findings which was equals to 1 m/s (Vmax of the TR/pulmonary ITV). The patient was transferred to the cardiovascular surgery department for surgical intervention.

Discussion

Patients with left-right shunts such as VSD present a risk of IE. Indeed, several studies have documented this risk of developing IE in particular on the right side (3,4) due to the high pressure gradient between the two cardiac chambers, source of endocardial erosive lesions on the right side, are generally the site of vegetations.

Active search for pulmonary embolism is always recommended in patients with endocarditis, particularly in patients with large (>10 mm) and mobile vegetations. This link between IE and septic pulmonary emboli is well documented in the literature (5,6). Our patient had septic bilateral distal vegetation embolization which persisted despite appropriate antibiotic therapy. (7,8)

Intravenous antibiotic therapy is the mainstay of treatment for IE, and they typically respond to a 4- to 6-week course of parenteral antibiotics (9,10).

Surgical intervention in right-sided IE is indicated only when caused by difficult-to-eradicate microorganisms, e.g. fungus, bacteremia persisting for more than 7 days despite adequate antibiotic therapy, embolisms recurrent pulmonary heart failure with or without concomitant right heart failure, perivalvular abscess, persistent large tricuspid valve vegetation (>20 mm), or right heart failure secondary to severe tricuspid regurgitation (11,12,13).

Conclusion

Septic pulmonary embolism represents a main complication of endocarditis secondary to VSD. A systematic workout should be carried out in all patients with right heart endocarditis, especially if the vegetation is large and mobile and treated early. Infective endocarditis is a serious condition and must be looked for in the face of any unexplained fever, particularly in patients with congenital heart disease, especially VSD. Echocardiography plays an essential role in the positive diagnosis, monitoring as well as the search for complications and the study of congenital heart disease.

Consent

As per international standards or university standards, patient(s) written consent has

been collected and preserved by the author(s).

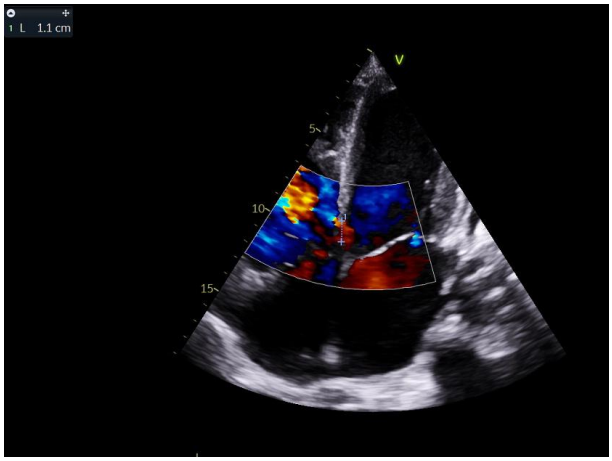

Figure 1 : apical four-cavity cut showing the septal defect (VSD)

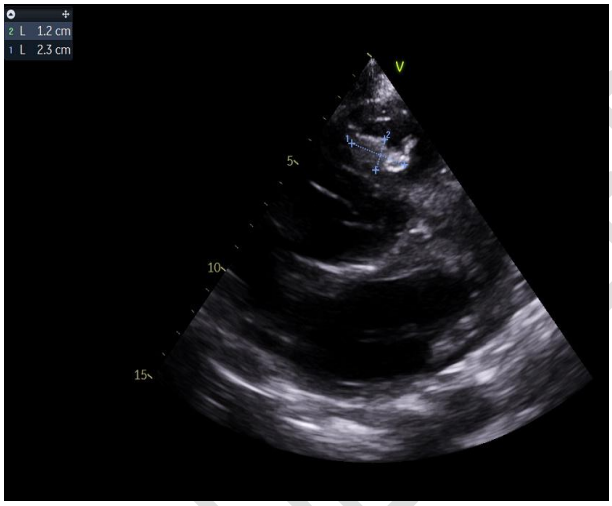

Figure 2 : modified parasternal long axis view showing a hyperechoic filiform image arising on the IVS and floating in the RV (vegetation image)

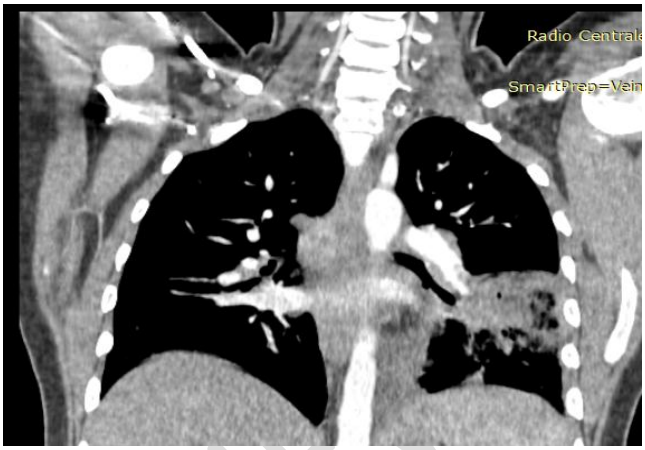

Figure 3 : CT image showing distal pulmonary embolization

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