Acquired ventricular septal defect due to infective endocarditis

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ABSTRACT

Acquired intracardiac left-to-right shunts are rare occurrences. Chest trauma and myocardial infection are well-known causes of acquired ventricular septal defect (VSD). There have been several case reports describing left ventricle to right atrium shunt after infective endocarditis (IE). We present here a patient found to have an acquired VSD secondary to IE of the aortic and tricuspid valves in the setting of a known bicuspid aortic valve. This is the first case reported of acquired VSD in a pediatric patient in the setting of IE along with literature review of acquired left-to-right shunts.

Keywords: Endocarditis, infective endocarditis, ventricular septal defect

INTRODUCTION

Acquired intracardiac left-to-right shunt is a rare occurrence, but it has been occasionally described in the literature as a possible sequel of bacterial endocarditis. Aortic valve (AoV) root abscess has been more commonly described as a complication of infective endocarditis (IE), but a fistula is largely uncommon.[1] Anatomically, this type of communication may occur secondary to surgery, trauma, or myocardial infarction much more commonly than IE.[2] Acquired ventricular septal defect (VSD) secondary to IE has not been reported before in the pediatric population. We present here a case of acquired VSD with infective endocarditis.

CASE REPORT

A 9-year-old female patient, with known bicuspid AoV and aortic stenosis with moderate aortic regurgitation, presented to an outside hospital with new headaches, emesis, left foot pain, and fever. Laboratory workup showed elevated liver enzymes as well as a normal white blood cell count and acute kidney injury. Blood cultures grew methicillin-sensitive Staphylococcus aureus. The patient was started on antibiotics of Vancomycin and Rocephin; however, due to the progression of symptoms (continued fever with worsening headache and nausea), she was transferred to our institution.

On arrival at our institution, the patient had continued complaints of headache and left foot pain. On examination, there was a III/VI loud systolic murmur heard best at the right upper sternal border from her aortic stenosis. There were purpuric lesions on the index finger, left palm, and sole of the left foot. Transesophageal echocardiogram (TEE) was performed given her cutaneous findings along with fever, positive blood culture, and known history of bicuspid AoV.[3] TEE showed a large mobile vegetation in the ascending aorta near the sinotubular junction, likely associated with the AoV. There was also a second mass/vegetation seen associated with the septal leaflet of the tricuspid valve and the interatrial septum [Figure 1]. No communication across the ventricular septum was observed [Figure 2].

The patient underwent treatment for endocarditis with resolution of cutaneous findings, headaches, and foot pain. She also had multiple negative blood cultures following antibiotic therapy which was deescalated to intravenous nafcillin based on blood culture sensitivities.[4] One month after the

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diagnosis of IE, with continued antibiotic therapy, the patient was readmitted for rash secondary to peripherally inserted central catheter line dressing. On examination, she was noted to have a new medium-high frequency holosystolic murmur. Echocardiogram at that time showed a new VSD (left ventricle [LV] to right ventricle [RV] to anterior septal commissure of the tricuspid valve to right atrium) [Figure 3]. This appeared to be a restrictive defect with left-to-right flow near the membranous/posterior muscular septum and just inferior to the right noncoronary commissure of the AoV. There was also markedly worsened aortic insufficiency from moderate to severe and dilation of the LV as compared to the previous study. These findings were confirmed on TEE as well as confirmation of residual mass/vegetation associated with the septal leaflet of the tricuspid valve.

The patient underwent surgical VSD closure with a bovine pericardium patch. Intraoperative findings showed a vegetation near the septal leaflet of the tricuspid valve with VSD [Figure 4] and erosion of the noncoronary cusp of the AoV [Figure 5]. In addition, given the vegetative damage to the tricuspid valve leaflets, as well as the worsening of the congenital pathology in the aortic position, bioprosthetic valves were placed in both positions (Tricuspid – Bioprosthetic St. Jude Epic 29 mm, Aortic – Bioprosthetic St. Jude Epic 23 mm, St. Jude Medical Inc., One St. Jude Medical drive, St. Paul, MN 55117-9983, USA). There were no acute postoperative complications, and follow-up echocardiogram shows no evidence of residual shunt.

**DISCUSSION**

VSDs are the most common congenital heart defect. They have been a well-described lesion that increases the risk of IE in the adult population,[6] but there is very little discussion of VSD as a complication of IE reported in the literature. Only 3 adult papers were found in an English-language PubMed review. One case described an acquired VSD due to IE present on the AoV with subsequent perforation and worsening
acquired VSD due to IE. The regurgitant jet was aimed at the ventricular septum, thus responsible for the acquired VSD.[7] The second case described an acquired VSD forming as sequela of an aortic root abscess in the setting of a mechanical AoV.[8] The third case described a case of IE with destruction of the mitral and AoVs, left ventricular outflow tract abscess, and subsequent VSD.[9] There are no reported cases of new VSD secondary to IE in the pediatric population currently reported.

We contemplated two mechanisms regarding the formation of VSD, as our patient did not have an AoV abscess. There was never a VSD seen on echocardiogram, nor was there a wind-sock appearance of the membranous portion of the ventricular septum inferior to the AoV. There is a possibility that there was previously a VSD that was spontaneously closed by the growth of tricuspid valve tissue. With the infection, there would have been destruction of the septal valve leaflet of the tricuspid valve, thus reopening of a VSD. There is also the possibility of a LV to RV subaortic fistula forming as a result of the aortic and tricuspid valve involvement. There was suggestion of an aortic abscess on some echocardiogram images, but this was never confirmed.

While the mechanism of VSD formation in our patient is unknown, the destruction from the IE was apparent, as well as obvious aortic and tricuspid valve destruction. Surgical repair was the best option in this instance, and our patient did remarkably well following the procedure.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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