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Research Letter

Disappearing high velocity severe tricuspid regurgitation following Ventricular septal defect device closure

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ABSTRACT

This report describes the disappearance of severe high velocity tricuspid regurgitation following a small ventricular septal defect device closure, in an eight year old girl.

1. Introduction

Small ventricular septal defects (VSD) have high velocity flow across the defect. These can cause high velocity tricuspid regurgitation(TR) either by impinging on anterior tricuspid leaflet and richoceting as TR,[1,2] or as gerbode defect which can be mistaken as pulmonary hypertension. Sometimes tricuspid septal aneurysm (which is closing large VSD) can distort the tricuspid valve and cause tricuspid regurgitation.[3] We describe an interesting case of high velocity severe tricuspid regurgitation(TR) secondary to venturi effect of small VSD distorting the septal tricuspid leaflet. This severe TR almost disappeared following device closure of the defect.

2. Case report

An eight year old girl child presented to us with incidentally detected murmur during a respiratory illness. Her past history was unremarkable. Clinically her vital parameters were normal. Cardiovascular examination revealed soft S1, 3/6 Pansystolic murmur at left lower sternal border, S2 was wide split and normal intensity. Her echocardiography revealed small 4.5 mm (maximum diameter) upper muscular VSD shunting left to right. The high velocity jet(4m/s) was causing distortion of septal tricuspid leaflet secondary to venturi effect Figs. 1 and 2. This in turn was causing richoceting of high velocity jet in to the right atrium as severe tricuspid regurgitation. The tricuspid regurgitation was initially mistaken as pulmonary hypertension not explainable with small VSD and normal S2. The child was planned for cardiac catheterization followed by device closure if found suitable. The calculated Qp/Qs was 1.8:1 and pulmonary artery pressure was normal.

She was taken up for percutaneous device closure of ventricular septal defect,thinking that closure of VSD should reduce the TR. VSD was closed from the arterial side using 5/6 Amplatzer duct occluder II (ADO II) device. Post procedure there was no residual VSD, trivial TR and no aortic regurgitation Figs. 3 and 4.

3. Discussion

Small VSD can cause TR due to various mechanisms. Firstly it could be caused due to high velocity flow impinging on anterior tricuspid leaflet and richoceting in to right atrium as TR.[1,2] Secondly gerbode defect could be mistaken as high velocity TR. Septal aneurysm closing VSD can also cause TR of varying severity.[3]

Our report highlights an interesting mechanism of TR due to venturi effect of high velocity flow distorting septal tricuspid leaflet, and richoceting as high velocity severe TR. This severe TR was initially mistaken as evidence of pulmonary hypertension. However cardiac catheterization revealed normal pulmonary artery pressure. The TR nearly disappeared after successful VSD device closure. This report highlights the fact that the diligent assessment of the mechanism of TR is the utmost requirement for its definitive management.

Conflicts of interest

None.

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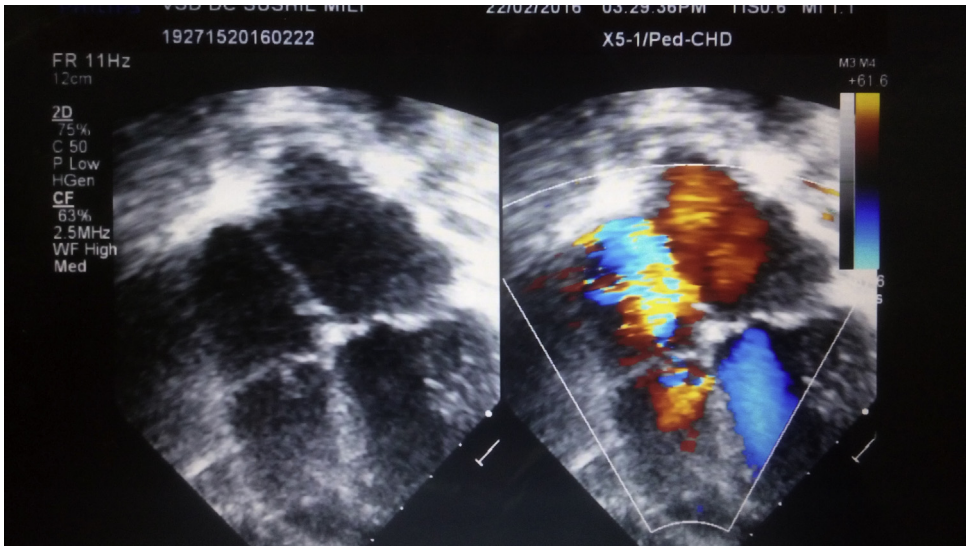

Fig. 1. Apical four chamber view shows dilated left ventricle with small upper muscular VSD and severe Tricuspid regurgitation.

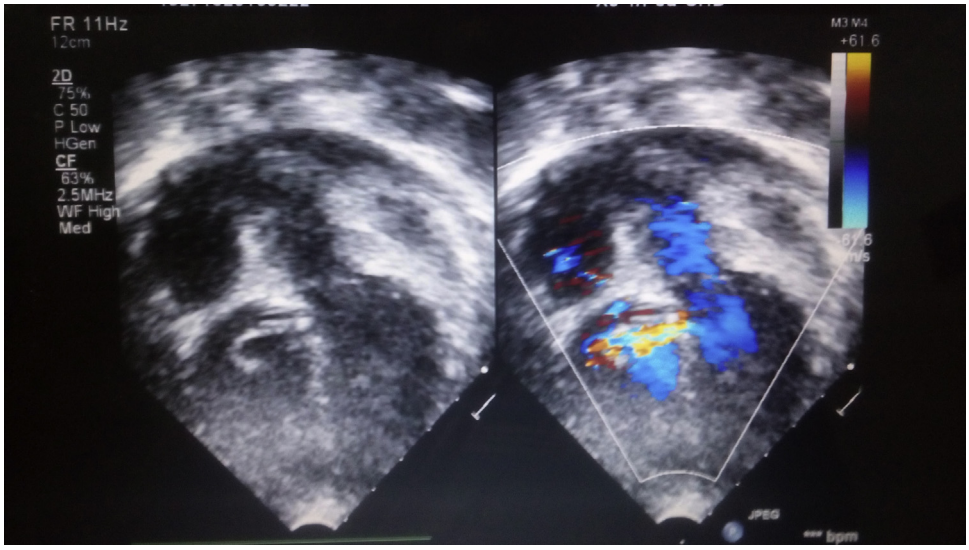

Fig. 2. Apical five chamber view showing distortion of septal tricuspid leaflet with generation of tricuspid regurgitation.

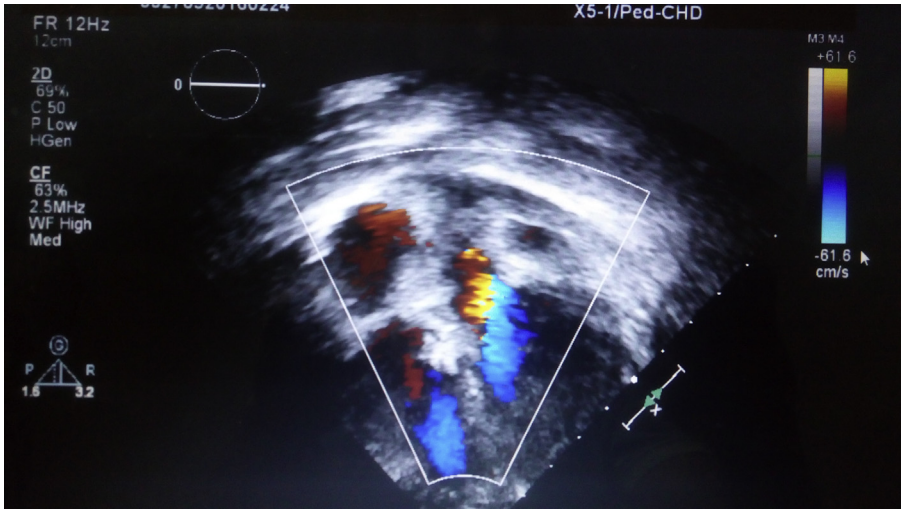

Fig. 3. Apical five chamber view showing VSD device in situ with no residual flow, no tricuspid regurgitation, no Aortic regurgitation.

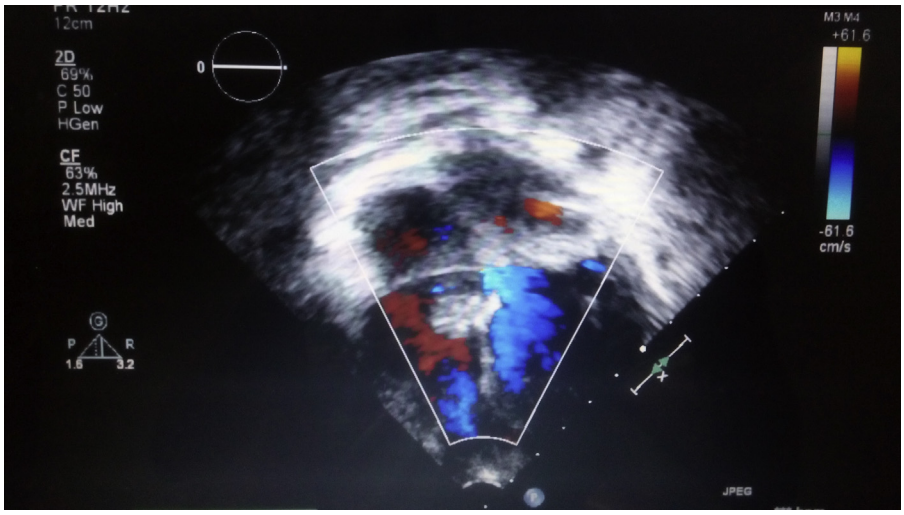

Fig. 4. Apical four chamber view showing VSD device in situ with no residual flow, trivial tricuspid regurgitation.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ihj.2017.01.001.

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