Giant Left Atrial V-Waves in Post-Myocardial Infarction Ventricular Septal Defect
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ABSTRACT

A 54-year-old man developed a post–myocardial infarction ventricular septal defect with a 4:1 shunt. The first cardiac catheterization showed left atrial V-waves of 70 mm Hg. Assessment of the presence or absence of mitral regurgitation was not possible because of ventricular irritability and rapid runoff from left ventricle to right ventricle. At the second catheterization two months later, the left atrial V-waves had fallen to 34 mm Hg. The absence of mitral regurgitation was shown by observing the time difference in appearance of indocyanine green in the right ventricle and the left atrium after left ventricular injection. The defect was repaired by right ventriculotomy with subsequent normalization of left atrial V-waves. This case shows that very large left atrial V-waves may occur in postinfarction ventricular septal defects without mitral regurgitation and that these V-waves may decrease with time, probably reflecting increased left atrial compliance.

Bethea and colleagues [1] reported large left atrial V-waves in patients with myocardial infarction complicated by ventricular septal defect without mitral regurgitation. The occurrence of combined ventricular septal defect and mitral regurgitation secondary to myocardial infarction must now be considered extremely rare and previously misdiagnosed. There has been only 1 confirmed case of the combined lesion in the literature [5]. None of Bethea’s 6 patients had mitral regurgitation on left ventricular angiography, and 5 of the 6 had elevated left atrial (or pulmonary wedge) V-waves (24 to 50 mm Hg). Mitral regurgitation in patients with postinfarction ventricular septal defects, therefore, cannot be diagnosed by using right heart hemodynamics as previously suggested by Fleming [3] and by Meister and Helfant [4].

The patient reported here demonstrated left atrial V-waves of 70 mm Hg without mitral regurgitation in conjunction with post–myocardial infarction ventricular septal defect; these V-waves decreased preoperatively with time.

A 54-year-old man was well until April 10, 1977, when he developed severe anterior chest pain. An electrocardiogram the following day was normal. The pain continued intermittently for two more days, and over the next week his predominant complaint was fatigue. On April 16, he developed sudden onset of nausea and diaphoresis, subsequently losing consciousness.

He arrived at an emergency department with ventricular tachycardia and was successfully resuscitated. Diagnosis of acute inferior transmural myocardial infarction was made. Examination revealed a pansystolic murmur at the lower left sternal border accompanied by a thrill. There was a loud third heart sound. Jugular venous pressure was elevated, and there were crepitations at both lung bases. An ECG showed Q-waves in the inferior leads. A chest roentgenogram revealed cardiomegaly and pulmonary plethora.

After initial recovery, the patient continued to have congestive heart failure with mild angina and underwent heart catheterization on June 24. He was found to have a ventricular septal defect with a 4:1 shunt. The mean left atrial pressure (through a patent foramen ovale) was 35 mm Hg with V-waves of 72 mm Hg (Fig 1). Because of severe left ventricular irritability and rapid runoff from left ventricle to right ventricle, it was difficult to assess the presence or absence of mitral regurgitation on angiography. Selective coronary arteriography
showed a blocked right coronary artery, a proximal 75% left anterior descending lesion, and diffuse circumflex disease.

The patient deteriorated on medical treatment and over the next two months developed shortness of breath on exertion, orthopnea, and increasing angina. On August 18, he underwent repeat heart catheterization preoperatively because of uncertainty regarding the presence or absence of mitral regurgitation. The left atrial V-waves at this time were 29 to 36 mm Hg (Fig 1). Indocyanine green dye dilution curves showed a left ventricle to left atrium appearance time of 8.4 seconds, and a left ventricle to right ventricle appearance time of 4.4 seconds (Fig 2). It was concluded that there was no major mitral regurgitation.

At operation, a left atrial line was inserted before the patient was placed on bypass, and left atrial V-waves of 26 mm Hg were recorded. A 2-cm ventricular septal defect was repaired with a Teflon patch through a right ventriculotomy, and a left anterior descending coronary artery bypass was performed. Left atrial V-waves postoperatively were recorded at 12 to 15 mm Hg and remained in that range for three days while left atrial pressures were monitored. Postoperatively, no murmurs were heard, and the patient has remained well.

Comment
In agreement with Bethea and associates [1], we believe that the giant left atrial V-waves simulating mitral regurgitation may be caused by the sudden volume load presented to a non-compliant left atrium following an acute large ventricular septal defect. This case also suggests that the left atrial V-wave may decrease considerably over time, presumably secondarily to increasing left atrial compliance. This is similar to the effect on mean left atrial pressure in chronic severe mitral regurgitation [2].

In this patient, interpretation of the left ventricular angiogram was difficult due to ventricular irritability and rapid runoff from left ventricle to right ventricle. We found that the presence or absence of mitral regurgitation

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Fig 1. Left atrial pressures (LAP) at the time of (A) the first and (B) the second heart catheterizations, approximately two months apart. Giant left atrial V-waves of 72 mm Hg at the first catheterization subsequently fell to 29 to 36 mm Hg at the second.

Fig 2. Representative dilution curves taken from (A) left atrium and (B) right ventricle after left ventricular injection. Dye appears in the right ventricle first, and subsequently in the left atrium after recirculation through the lungs.
could be clarified using dye dilution curves and comparing the appearance time of indocyanine green in the left atrium and the right ventricle after left ventricular injection.

The diagnosis of the combined complications of ventricular septal defect and mitral regurgitation following an acute myocardial infarction should always be questioned. The benefit to the patient of excluding the latter complication is evident with the lesser risk of an easier and shorter surgical procedure.

References

Editor's Note
This case report is basically a confirmation of the report by Bethea and co-workers [1]. However, it does bring the problem to the attention of surgeons who may not have seen the previous publication.