

# Atrioventricular Shunt Post Acute Myocardial Infarction



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## INTRODUCTION

The major mechanical complications of acute myocardial infarction (AMI) include free wall rupture, ventricular septal rupture (VSR), and papillary muscle rupture. Typically VSR results in left ventricular (LV) to right ventricular (RV) shunting. LV to right atrial (RA) shunting is rare. We report a case of a woman with the previously undescribed combination of inferobasal pseudoaneurysm (PsAn) formation with both LV-RV and LV-RA shunting following an AMI.

## CASE PRESENTATION

An 89-year-old female presented to an outside hospital following a syncopal episode. On initial evaluation she was found to have inferior ST segment elevation. She was transferred for urgent cardiac catheterization.

Upon arrival, her blood pressure was 92/40 mm Hg, and her heart rate was 122 bpm and regular. She was tachypneic. Electrocardiogram confirmed an inferior STEMI. On physical examination, her skin was cool and clammy. A loud 3/6 holosystolic murmur was heard at the left lower sternal border with radiation to the midaxilla. A transthoracic echocardiogram demonstrated intact mitral apparatus and no significant mitral regurgitation. There was akinesis of the basal inferoseptal and inferior segments with compensatory hyperkinesis of the distal septum and anterior and lateral segments (Video 1). From the apical four-chamber, steeply modified three-chamber, and subcostal views, the narrow neck of a pseudoaneurysm (PsAn) was noted at the inferior base with typical systolic expansion (Figures 1 and 2 and Videos 2-4). With color Doppler, flow was seen entering both the RV and the RA at the inferior and superior margins of the PsAn (Videos 5-7). There was severe global RV dysfunction.

Based on the echocardiographic findings, cardiothoracic surgery was consulted prior to performing cardiac catheterization. Following discussion with the family, it was felt unreasonable to pursue either surgical or percutaneous intervention, as the mortality risk was deemed unacceptably high. The patient was placed on comfort measures only and succumbed within 24 hours.

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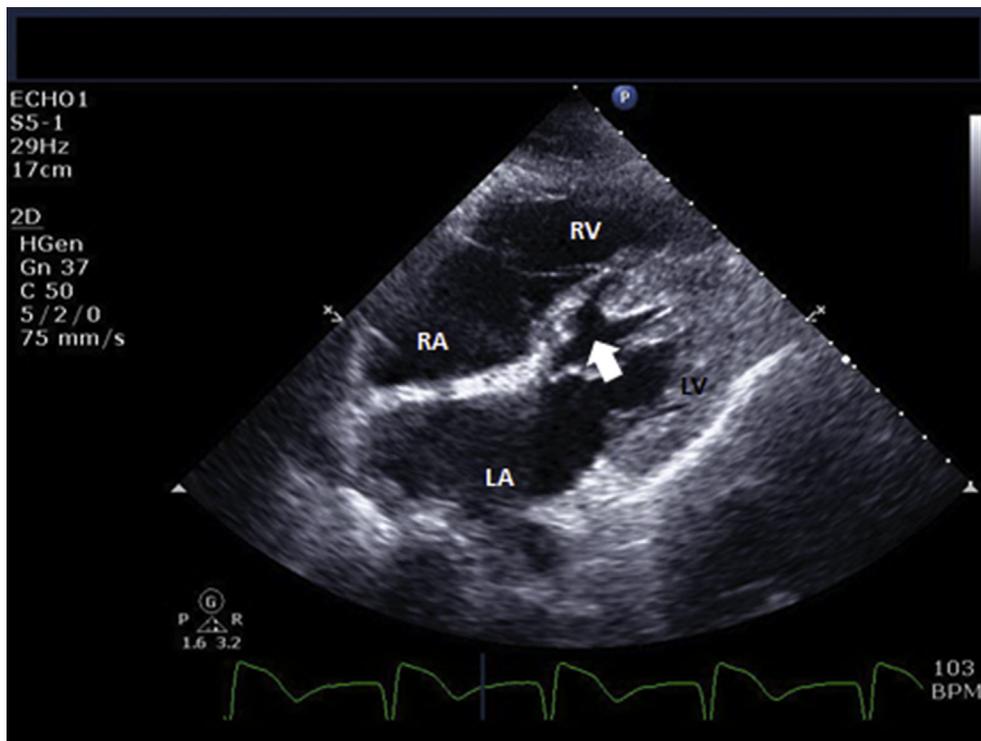
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## DISCUSSION

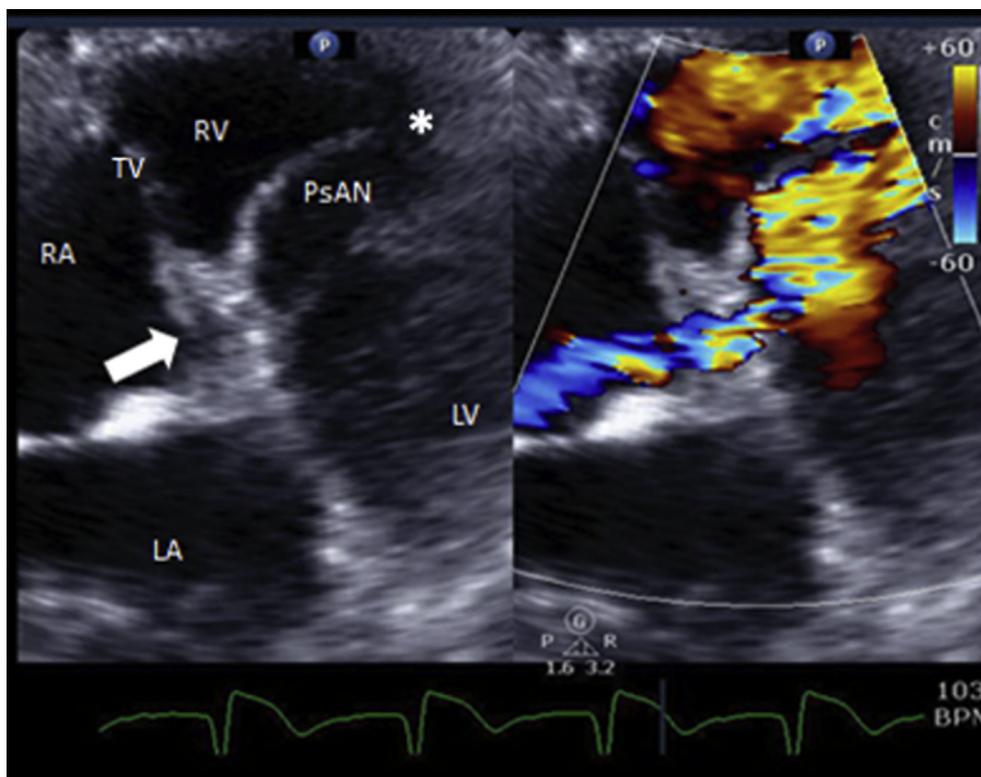
The incidence of mechanical complications of AMI has decreased to less than 1% since the introduction of primary percutaneous coronary intervention, with the incidence of VSR in AMI currently at 0.17%. VSR accounts for 5% of all deaths in AMI; approximately 40% of untreated patients die within the first week and 60%–80% within two months.<sup>1,2</sup> Historically VSR occurred 3–5 days after AMI. With the advent of thrombolytic and percutaneous intervention, VSR occurs earlier, within 24 hours after presentation of the infarct, probably because of myocardial hemorrhage.<sup>1</sup> Prior to the availability of reperfusion options, the risk factors for VSR included hypertension, age, female gender, and absence of angina or previous infarct. As VSR occurs following a transmural infarct, the presence of angina or a previous infarct may result in the development of collateral circulation and myocardial preconditioning, both of which can reduce the risk of a transmural infarct.<sup>2</sup> Two phases of a postinfarct VSR have been identified.

An acute phase occurs within the first two weeks of infarct and is characterized by ongoing myocardial necrosis. Resorption and retraction of infarcted tissue lead to enlargement of the defect. After 3–4 weeks, the chronic phase begins and is characterized by fibrosis and scar formation around the defect; this is the time most appealing for surgical intervention.<sup>3</sup> Simple VSR represents a single “through and through” defect positioned at the same level in both ventricles, usually anteriorly. More complex VSR, as in our patient, can manifest as multiple tortuous hemorrhagic channels with substantial disruption of myocardial tissue and is usually located inferiorly.<sup>4</sup> Typically the physical exam demonstrates a loud, harsh holosystolic murmur at the lower sternal border, commonly associated with a thrill. Surgical intervention is the standard, although percutaneous closure is an alternative to surgery if the anatomy of the defect is favorable. As more large series describing the percutaneous management of VSR become available, it appears that catheter therapy is feasible, regardless of the size of the defect and the time from the index AMI, with cardiogenic shock and high volume shunting being significant risks for death.<sup>5</sup> Surgical mortality is highest in patients with inferobasal septal rupture because of a substantial increase in technical complexity. Few reports are available describing shunting from the LV to the RA as a result of an AMI.<sup>6,7</sup>

In 1949, the Mayo Clinic described autopsy findings in patients with LV to RA communications and abnormal tricuspid valves.<sup>8</sup> In 1955, Stahlman *et al.* described two patients with LV-RA shunting in whom unsuccessful surgical attempts were made; both patients died.<sup>9</sup> In 1956, Charles Kirby, a Philadelphia surgeon, described successful closure of an LV-RA shunt in a 15-year-old girl.<sup>10</sup> In 1958, the first series of five patients to be operated on successfully for an LV-RA shunt was reported by Frank Gerbode, a Stanford surgeon.<sup>11</sup> He described two types of LV-RA communications. One, the more



**Figure 1** Subcostal four-chamber view demonstrating the narrow neck of the PsAn (*arrow*) This view also demonstrates partial extent of the rupture into the distal interventricular septum, which terminates in an apical VSD (see [Figure 2](#)).



**Figure 2** Subcostal zoomed image (*right*) demonstrating flow into the PsAn with flow through the inferobasal margin of the PsAn into the RA. Flow is also noted to migrate distally through the ruptured septal tract, ultimately shunting through the apical VSD into the RV. The *arrow* (*left image*) identifies the LV-RA tract. The site of egress into the RV at the apex is noted with an *asterisk* (\*). TV, tricuspid valve.

common in his original five patients, was a perimembranous defect with an associated defect of the septal leaflet of the tricuspid valve, wherein the shunt is LV-RV and then through the tricuspid defect into the RA (infravalvular type, type 2, indirect). The other type is the direct LV-RA shunt, present in only one of his original cases; this defect occurs above the tricuspid valve (supravalvular type, type 1, direct). While true congenital LV-RA communications are uncommon, acquired defects are being described with increasing frequency. In a recent large review of LV-RA shunts, 121 patients were identified in the literature; of those patients, 88 had acquired defects.<sup>12</sup> The etiologies of those acquired defects were postoperative (59%), infectious (28%), ischemic (5.7%), iatrogenic (4.5%), and traumatic (2.3%). Of the postoperative patients, 39 had valve surgery and 13 had surgery for congenital heart disease, five procedures of which were VSD repair.

Treatment options for Gerbode defects include surgery and, more recently, transcatheter closure. In a recent series from India, 12 patients underwent successful occlusion of a congenital Gerbode defect using an Amplatzer Duct Occluder II (St. Jude Medical, Minneapolis, MN).<sup>13</sup> Transthoracic echocardiographic guidance was used in this pediatric population to make the correct diagnosis (distinguish the defect from tricuspid regurgitation) and to determine the size and efficacy of the device. In this series, 4-mm devices were used in the type 1 defects (six patients), and 6-mm devices were used in the type 2 defects (six patients). Percutaneous closure of acquired LV-RA defects has been described.<sup>14</sup> A recent report documents successful closure of a postinfarct LV-RA communication with a 14-mm Amplatzer atrial septal occluder device (St. Jude Medical, St. Paul, MN).<sup>15</sup> Considerations during device closure include the necessity for meticulous alignment of the device, the potential for conduction system disturbance, and the risk of producing significant tricuspid regurgitation (or stenosis) from leaflet impingement by the device. Transesophageal imaging (although as noted, transthoracic imaging may be adequate in infants and small children) is commonly an integral part of these interventions and helps to define the location and number of defects, confirm device placement, and identify any residual shunt flow.

## CONCLUSIONS

LV-RA communications following AMI are rare. We describe a patient who presented in cardiogenic shock following an inferior infarct, with inferobasal PsAn formation and fistulous tracts from the PsAn into both the RV and the RA. Although percutaneous options are emerging as treatment for postinfarct VSRs, expeditious surgery still provides the highest likelihood for optimal outcome.

## SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.case.2017.01.009>.

## REFERENCES

1. Bajaj A, Sethi A, Rathor P, Suppogu N, Sethi A. Acute complications of myocardial infarction in the current era: diagnosis and management. *J Investig Med* 2015;63:844-55.
2. Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. *Cardiol Clin* 2013;31:519-31.
3. Egbe AC, Poterucha JT, Rihal CS, Taggart NW, Cetta F, Cabalka A, et al. Transcatheter closure of postmyocardial infarction, iatrogenic, and postoperative ventricular septal defects: Mayo Clinic experience. *Catheter Cardiovasc Interv* 2015;86:1264-70.
4. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. *Am J Cardiol* 1984;54:1201-5.
5. Trivedi KR, Aldebert P, Riberi A, Mancini J, Levy G, Macia JC, et al. Sequential management of post-myocardial infarction ventricular septal defects. *Arch Cardiovasc Dis* 2015;108:321-30.
6. Jobic Y, Verdun F, Guillo P, Bezon E, Gilard M, Etienne Y, et al. Postinfarction atrioventricular septal rupture. *J Am Soc Echocardiogr* 1997;10:680-4.
7. Hole T, Wiseth R, Levang O. Post-infarction left ventricle to right atrium fistula diagnosed by transthoracic Doppler echocardiography. *Eur Heart J* 1995;16:866-8.
8. Perry EL, Burchell HB, Edwards JE. Congenital communication between the left ventricle and the right atrium; coexisting ventricular septal defect and double tricuspid orifice. *Proc Staff Meet Mayo Clinic* 1949;24:198.
9. Stahlman M, Kaplan S, Helmsworth JA, Clark LC, Scott HW. Syndrome of left ventricular-right atrial shunt resulting from high interventricular septal defect associated with defective septal leaflet of the tricuspid valve. *Circulation* 1955;12:813.
10. Kirby CK, Johnson J, Zinsser HF. Successful closure of a left ventricular-right atrial shunt. *Ann Surg* 1957;145:392-4.
11. Gerbode F, Hultgren H, Melrose D, Osborn J. Syndrome of left ventricular-right atrial shunt. Successful surgical repair of defect in five cases. *Ann Surg* 1958;148:433-46.
12. Yuan SM. Left ventricular to right atrial shunt (Gerbode defect): congenital vs acquired. *Postep Kardiol Inter* 2014;10:185-94.
13. Vijayalakshmi IB, Natraj Setty HS, Chitra N, Manjunath CN. Amplatzer Duct Occluder II for closure of congenital Gerbode defects. *Catheter Cardiovasc Interv* 2015;86:1057-62.
14. Trehan V, Ramakrishnan S, Goyal NK. Successful device closure of an acquired Gerbode defect. *Catheter Cardiovasc Interv* 2006;68:942-5.
15. Cheng R, Arsanjani R, Tat E, Cox J, Uberoi A, Aron R, et al. Not your typical hole-in-the-wall. Percutaneous closure of an acquired post-myocardial infarction ventriculoatrial Gerbode defect. *JACC Cardiovasc Interv* 2015;8:1905-6.