

Double Rupture of a Tricuspid Papillary Muscle and Ventricular Septum: A Rare Combination after Myocardial Infarction



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INTRODUCTION

Acute myocardial infarction (AMI) in the current era is managed using aggressive reperfusion therapies, including dual-antiplatelet therapy and primary percutaneous intervention, that permit prompt restoration of blood flow to the infarcted myocardium. Such interventions are responsible for the declining rate of ventricular septal rupture (VSR) from the prethrombolytic era to a now comparatively rare incidence.¹⁻⁴

In addition to VSR, other mechanical complications depending on which coronary artery is occluded may ensue. Acute inferior wall ST-segment elevation myocardial infarction (STEMI) contributes to approximately 10% of STEMI's in-hospital mortality, and right ventricular (RV) infarction may occur in up to 50% of inferior wall STEMIs.⁵ Severe tricuspid valvular regurgitation (TR) may follow RV infarction. In such case, acute rupture of papillary muscle within the right ventricle should be high on differential diagnosis.

Ischemic tricuspid papillary muscle rupture is rare, but it has been previously reported.^{6,7} To our knowledge, acute ischemic tricuspid papillary muscle rupture in conjunction with VSR has not been yet reported. Here we illustrate the utility of two and three-dimensional echocardiography in the diagnosis and management of a patient with hemodynamic instability in the setting of VSR and acute tricuspid papillary muscle rupture following an acute coronary event.

CASE PRESENTATION

A 64-year-old man with a history of chronic obstructive pulmonary disease, smoking, patent foramen ovale, and permanent pacemaker presented to his primary care physician with 2 weeks of worsening shortness of breath, lower limb edema, and progressive fatigue. He initially was treated with antibiotics for a suspected upper respiratory tract infection, on which he showed no improvement. Because of unresolved symptoms, he presented to an outside hospital emergency department for evaluation. On arrival to the emergency department he endorsed severe chest pain, shortness of breath, and nausea and vomiting. He also had history of paroxysmal nocturnal dyspnea, orthopnea, and 9 lb of weight gain over several weeks. On electrocardiography, new Q waves were seen in the inferior leads. As part of his

chest pain evaluation, computed tomographic angiography revealed a pulmonary embolism with clot burden in the lower left pulmonary artery. Transthoracic echocardiography showed severe TR with VSR and a bidirectional interventricular shunt (Videos 1-5). Additionally, transthoracic echocardiography revealed severe RV dilation with basal to mid RV free wall hypokinesis and preserved apical contractility (Video 3). Flattening of the interventricular septum during systole and diastole was noted, indicating volume and pressure overload. Overall, the right ventricle had severely decreased systolic function. A mobile echo-density was seen moving to and fro between the right atrium and ventricle, which was suspicious for a tricuspid valve rupture associated with severe TR. The TR was eccentric and directed toward the interatrial septum. Because of the directionality of this jet, it was most likely an anterior leaflet issue. The patient also had a pacemaker that may have affected the septal leaflet. A VSR with bidirectional flow at the base (Video 3) was noted and measured to be 1.5 cm in diameter (Figure 1) on two-dimensional echocardiography. Using three-dimensional echocardiography, the left ventricle was imaged from an apical window, and the VSR was seen en face from the left ventricular perspective. The dimensions were 1.5 × 1.3 cm on a multiplanar reconstructed image (Figure 2).

Because of these findings, left heart catheterization was performed, demonstrating 100% occlusion of the right coronary artery (RCA), 90% left anterior descending coronary artery stenosis, and a pulmonary capillary wedge pressure of 28 mm Hg. After the patient's transfer to Yale-New Haven Medical Center, his condition worsened, necessitating intubation and vasopressors for hemodynamic support.

The patient had severe metabolic acidosis, with a serum bicarbonate level of 16 μmol/L, and evidence of severe end-organ dysfunction with liver function test results >1,000 U/L and a creatinine level of 2.1 mg/dL. Because of cardiogenic shock, he was placed on an intra-aortic balloon pump while the dobutamine drip was continued. Further imaging with transesophageal echocardiography confirmed an anterior tricuspid papillary muscle rupture along with a basal VSR (Video 6). The right ventricle was dilated, with a diameter of 6.2 cm at the base and 5.5 cm at the midcavity.

The patient was then taken to surgery. Surgical interventions included tricuspid valve replacement with a 27-mm Biocor valve (Videos 7 and 8), repair of the postinfarct VSR in the high inferior septum (Video 9), and coronary artery bypass grafting with left internal mammary artery to the left anterior descending coronary artery as well as reversed saphenous vein graft to the patent ductus arteriosus. The RV pacer lead was replaced with a permanent epicardial pacing lead, and the patent foramen ovale was closed. Postoperative transthoracic echocardiography confirmed small residual shunting. The tricuspid valve prosthesis was well seated, with an elevated gradient of 6 mm Hg at 118 beats/min (Figure 3) and no evidence of regurgitation. The patient's postoperative course was complicated by severe hypotension and cardiogenic shock requiring

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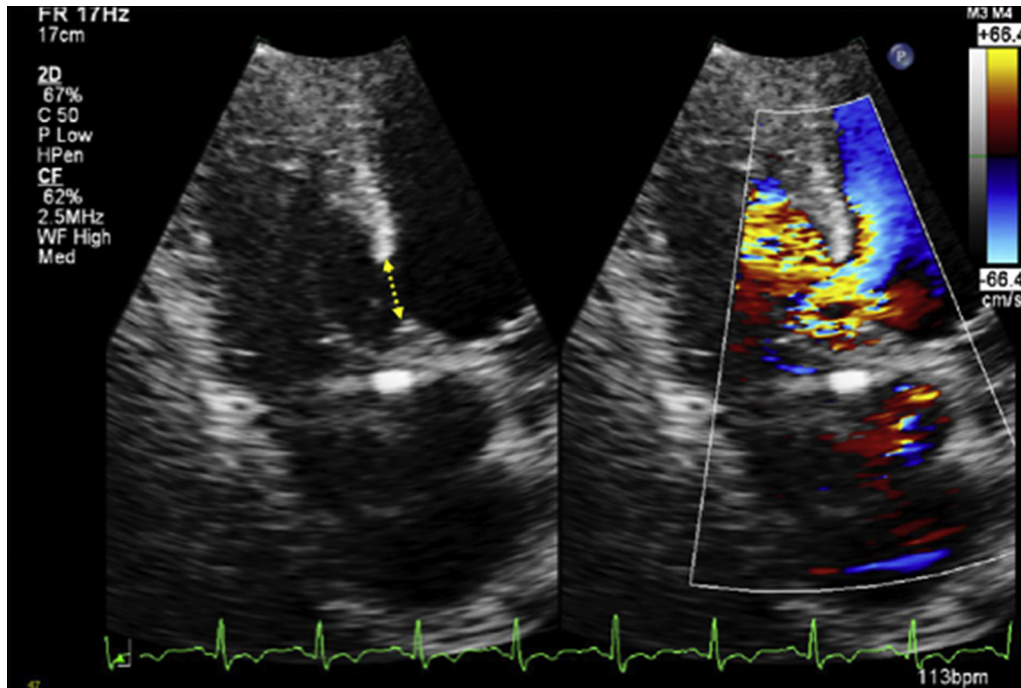


Figure 1 Two-dimensional apical four-chamber view without and with color of the ventricular septal defect measuring 1.46 cm.

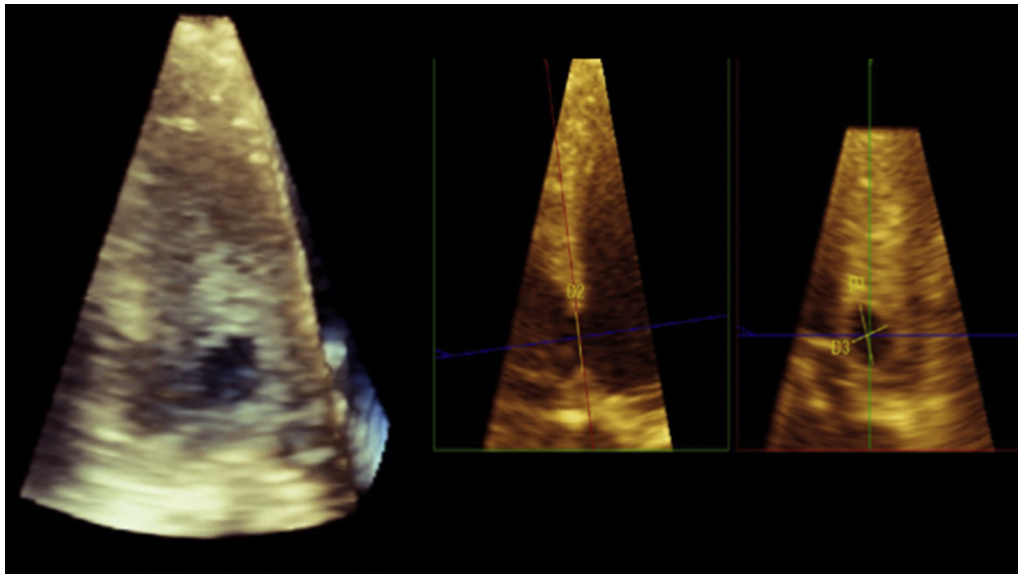


Figure 2 Multiplanar three-dimensional reconstruction of the VSR measuring 1.66 cm in a similar four-chamber cut plane. A unique transection of the rupture provides a measurement of 1.55×1.34 cm.

vasopressor support. Concurrently, he was noted to have shock liver with elevated liver function test results and coagulopathy with an international normalized ratio of 9. Shortly thereafter, he developed acute renal failure that was not amenable to dialysis, and he had continued hypotension and progressive decline of RV function. The patient developed multiple-organ failure leading to his death after 12 days of hospital stay.

DISCUSSION

Tricuspid Papillary Muscle Rupture

RV infarction may ensue in 50% of cases after inferior wall myocardial infarction and is correlated with substantial in-hospital mortality.⁵ Papillary muscle ruptures are more often seen in left ventricular infarction and are usually not associated with RV infarct. Moreover,

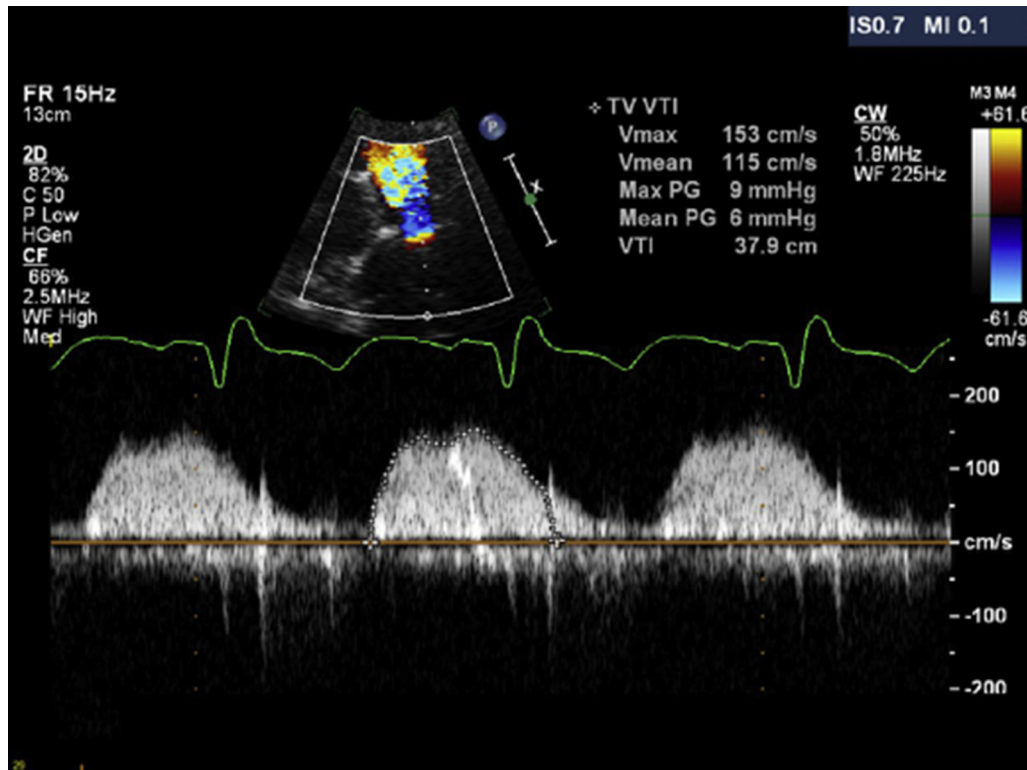


Figure 3 Continuous-wave Doppler across the implanted tricuspid valve with mean pressure gradient of 6 mm Hg.

tricuspid papillary muscle rupture due to infarction leading to acute severe TR is rare. To date, only two cases of isolated tricuspid papillary muscle rupture secondary to myocardial ischemia have been reported.^{6,7} However, unlike this case, neither were accompanied by a VSR.

The tricuspid valve apparatus is composed of three leaflets (anterior, septal, and posterior) supported by the tricuspid annulus, three papillary muscles, and chordae tendineae. The RCA is the main blood supply to tricuspid papillary muscles, and the inferior portion of the interventricular septum is supplied by RCA septal branches.⁵ The interventricular septum constitutes a vital anatomic component of the RV chamber and under normal conditions is responsible for up to one third of RV stroke volume.⁵ Hence, when RV free wall systolic function is reduced, RV stroke volume becomes dependent mainly on septal contraction, and because the right ventricle is unable to generate intracavitary pressure, paradoxical septal motion can be observed resulting from the pressure gradient between the left and right ventricles.

Total occlusion of the RCA and concomitant pulmonary embolism compounded the diagnosis, which led to a delay in diagnosis and could explain the unusual combination of a papillary muscle rupture and VSR. In addition to the ischemia, the rise in pulmonary artery pressure contributed to RV wall strain, dilation, and impairment of systolic function. Consequently, it is not surprising that septal dysfunction due to VSR causing a left-to-right shunt in this case aggravated hemodynamic deterioration associated with RV infarction, eventually leading to cardiogenic shock. Although some cases of functional TR may improve with RV recovery, cases that include frank papillary muscle rupture require surgical intervention.⁸

Incidence, Risk Factors, and Scope of VSR

VSR can ensue after one or more main coronary vessels occlusion as all the coronary vessels give origin to septal branches that supply the interventricular septum.¹ Controversial results about the association of AMI distribution with the incidence of VSR have been reported. From the Myocardial Infarction Data Acquisition System (MIDAS) database, Moreyra *et al*¹ found that anterior wall myocardial infarctions were more frequently observed in patients with VSR. However, the probability of having a VSR from anterior wall myocardial infarction (0.32%) compared with an inferior or lateral wall myocardial infarction (0.21%) was not statistically different ($P = .10$). In contrast, Crenshaw *et al*² reported from the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-I) database that an anterior infarct secondary to left anterior descending coronary artery occlusion is more common in patients with VSR than in those without ($P < .001$). Moreover, Poulsen *et al*⁹ found an equal distribution of AMI between anterior and inferior-posterior locations. Generally, VSR has been found to be more frequently related to single-vessel disease with minimal to no collateral circulation.^{9,10}

Different studies have reported several independent risk factors for VSR incidence in patients presenting with AMI.^{1,2,11} Both the MIDAS and GUSTO-I database were partly in agreement on some of these factors, which include, older age, female gender, and congestive heart failure. Although MIDAS reported a lower VSR incidence with hypertension, GUSTO-I reported that hypertension is an independent risk factor for VSR development. Additionally, patients with VSR in MIDAS were more likely to have chronic kidney disease, while diabetes mellitus was associated with lower VSR incidence, a supported

finding from the SHOCK registry,¹⁰ with an argument that hypertension-induced concentric ventricular hypertrophy and diabetes-associated collateral circulation might decrease VSR rate. However, this could have been attributed to an observational bias, with the possibility that such patients develop cardiogenic shock or fatal rupture.^{1,2,12}

Outcome, Intervention, and Management

Although surgical management of VSR is the mainstay, it remains a daunting intervention, with overall operative mortality of 42.9%.¹³ Thirty-day mortality was associated with older age, female gender, higher serum creatinine, cardiogenic shock, and requirement of mechanical support. From the Society of Thoracic Surgeons National Database, several independent risk factors for surgical mortality were identified. Even though no linear relationship between the timing of surgery and operative mortality was found, surgical repairs that took place within 7 days of myocardial infarction had 54.1% mortality rate compared with 18.4% in case of delayed repair (>7 days).¹³ However, this might denote a selection bias, as early surgical procedures tend to be performed on patients with severe hemodynamic instability.

Even though previously published reports demonstrated better outcomes for delayed VSR repair,¹³ American College of Cardiology and American Heart Association guidelines recommended prompt surgical repair for VSR in the setting of STEMI.⁸ Arguably, in hemodynamically stable patients, delayed surgery might allow steadiness of infarcted myocardium, leading to solidity of repairs. In the presented case, hemodynamic stability was challenged not only by the presence of VSR but also by RV dysfunction and acute severe TR in the setting of STEMI, which promoted development of cardiogenic shock. Cardiogenic shock in conjunction with VSR portends poor surgical outcome with mortality rate up to 90%.^{10,14} The combination of septal rupture and RV dysfunction was particularly found to have a substantial impact on prognosis of patients with VSR.¹⁵ Patients with factors predisposing to operative mortality, such as cardiogenic shock and renal impairment, could potentially benefit from temporizing solutions, including biventricular support or extracorporeal membrane oxygenation to improve hemodynamics as a bridge to definitive surgical repair or heart transplantation as a last resort. Certainly, there could have been a more prompt decision in using extracorporeal membrane oxygenation in the face of cardiogenic shock and await recovery of RV function.

CONCLUSION

This novel case of late presenting inferior wall STEMI complicated by both tricuspid papillary muscle and ventricular septum rupture leading to severe hemodynamic compromise emphasizes the importance of noninvasive imaging to clinicians. Hypotension in the setting of a myocardial infarction should initiate an echocardiographic evaluation for mechanical complications using two-dimensional echocardiography and color Doppler to demonstrate an abnormal flow across an interventricular communication or valvular regurgitation. Three-dimensional echocardiography provides unique en face view, orientation, and accurate measurements of the area of rupture. The role of transesophageal echocardiography in this case provides additional anatomic information and benefits further intervention whether surgical or interventional.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2018.11.009>.

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