CASE PRESENTATION

A 60-year-old diabetic man visited our center with an ongoing chest pain started about 12 hours before admission. Inferior ST-elevation myocardial infarction (MI) was evident (Figure 1). At the arrival, a brief physical exam only showed a borderline blood pressure of 100/70 and sinus tachycardia. Emergent coronary angiography revealed three vessel disease including a totally occluded right coronary artery (RCA) from mid-part, significant stenosis of the left anterior descending and left circumflex arteries at mid-part and also significant ostial stenosis of diagonal and obtus marginalis arteries. Primary percutaneous coronary intervention (PCI) was done for the RCA without any complication. The chest pain resolved to a great extent. Later in the coronary intensive care unit, although the patient was free of pain, he seemed to be ill and restless. The physical examination was remarkable for an unresolved sinus tachycardia, slight hypotension, a jugular vein distention, and a holosystolic murmur at the left sternal border radiating to the epigastric region. The repeated ECG showed findings similar to the pre-PCI ECG with no new changes: sinus tachycardia, right-axis deviation, Q-waves, and
unresolved ST-segment elevation in the inferior leads. Transthoracic echocardiography (TTE) revealed a dissected inferior septum forming a serpiginous tract connecting both ventricles at different levels (Figure 2, Videos S1-S3). The patient underwent coronary artery bypass graft surgery (CABGs) and surgical repair of the disrupted septum on the 5th day of admission. Intraoperative transesophageal echocardiography (TEE) showed a dissected septum encompassing an echo-free space which freely communicated with the left ventricular cavity via a large ventricular septal rupture (VSR) at the mid-inferior septum and then reentered to the right ventricle (RV) near the apex (Figure 3, Videos S4-S8). At the time of surgery, a right ventriculotomy was done and the interventricular septum was exposed. By gross inspection, an oblique longitudinal irregular rupture was noticed throughout the septum dividing it into two thin necrotic layers (Figure 4). The necrotic tissues were removed and replaced with a pericardial patch. Post-pump TEE showed a small residual ventricular septal defect. The post-op course was complicated with a prolonged intubation period and renal failure necessitating dialysis. Unfortunately, the patient suddenly arrested while being under dialysis and died.

2 | INTRODUCTION

Myocardial rupture is a rare but fatal mechanical complication of MI. It usually occurs in cases of transmural MI with inadequate collateral tissue and particularly in first episodes of MI. Two types of cardiac rupture have been previously described based on pathologic studies: simple type with a through-and-through direct defect and complex type named intramyocardial dissection (IMD) in which the defect is accompanied with a serpiginous dissecting hematoma remote from the primary site of tear. The hematoma results from entry of blood into the necrotic myocardium, which is initially contained in the left ventricular (LV) myocardium and then tends to separate the ventricular spiral myocardial fibers in the low resistant necrotic area resulting in a neocavitation.
hematoma could further expand via a dissecting tract and rupture into adjacent structures or spontaneously resolve. Therefore, not all dissecting hematomas evolve to a full-blown cardiac rupture. By rupturing in the pericardium, a free wall rupture and/or pseudaneurysm ensues. Rupturing in the RV results in a complex VSR. In this form of VSR, the interventricular defect is not a simple through-and-through direct connection between the ventricles but rather is a serpiginous tract in which the entry and exit site are not at the same plane.

Intramyocardial dissection is a rare complication of MI. Based on a large prospective study on patients with acute MI, it has been reported in only 1% of patients. The occurrence of IMD resulting in VSR is even more rare. The evidence available is limited to few case reports. The optimal management of IMD is controversial due to its rarity and lack of supportive evidence.

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Herein, we first describe a complicated case of IMD with VSR. Then, we systematically review published cases of IMD with VSR in patients with acute MI.

3 METHODS

In accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines, we searched the PubMed and Scopus databases up to Feb 2019 and performed a systematic review of previous cases of LV/RV dissection in the context of ischemia using the following mesh terms in the title: "Ventricular septal dissection," "myocardial dissection," "ventricular septum AND dissection," "myocardial dissecting hematoma," "intramyocardial dissecting hematoma." These terms were combined with the term "myocardial infarction" and "ventricular septal rupture."

Items from the preliminary search were screened for duplicated items and then assessed for eligibility to be included.

The eligibility criteria were as following:

1. Article type of case reports, case series, prospective or retrospective cohorts, and letters
2. Cases of IMD and VSR in the context of acute myocardial infarction
3. Articles published from January 1990 to February 2019
4. Articles published in English with available full text

The exclusion criteria were as following:

1. Cases of IMD due to etiologies other than MI
2. Cases of IMD and VSR in whom the diagnose of acute MI was not confirmed
3. Cases of IMD without VSR or with partial VSR
4. Postmortem case reports

Data of enrolled case reports were collected for further analysis. Collected clinical data were entered into a Microsoft Excel database:
Data were presented as mean ± standard deviation (SD) for continues variables with normal distribution or median and inter-quartile range (IQR) for non-normally distributed data. Categorical data were presented as frequency and percentage. T-test, Mann-Whitney U test, and χ² test were used for comparison of variables between groups. P-value of less than .05 was considered statistically significant. The Statistical Package for the Social Sciences (SPSS), version 24 (SPSS Inc) was used for statistical analysis.

4 RESULTS

A total of 108 reports were identified, of which 22 full-text articles with 36 cases met our inclusion criteria (including 15 case reports, 3 case series, 2 systematic reviews, one prospective study, and one letter).2,7-27 The included studies are summarized in Table S1 of the supplementary material. Our case was added, and we finally analyzed 37 cases. The mean age was 68 ± 8 years (range, 52-82 years). Twenty patients (54.1%) were men. Other baseline characteristics of our study population are summarized in Table 1

Reperfusion therapy was done for 15 (40.5%) cases before the VSR occurred: 7 (18.9%) cases received thrombolytic and in 8 (21.6%) cases PCI was performed.

Regarding the type of approach to the VSR, we observed 3 types of management: surgery, percutaneous closure, and medical therapy. In one case report, the approach to the VSR was not mentioned.8 Three patients underwent percutaneous closure of VSR all of whom had anterior MI with the VSR located at the anterior septum. One of these patients died. Of the 26 patients who underwent surgery, 11 cases died (mortality rate: 42.3%). Of the 7 patients who received medical therapy, 6 cases died (mortality rate: 85.7%).
with a large entry from the LV due to a posterior septal rupture. After tunneling deep through the septum, it reentered RV at the level of apex. The patient succumbed despite our many teamwork efforts to save his life. Late arrival of our case led to an extensive damage and mortality despite nonproximal coronary lesions, prompt diagnosis, and optimal treatment. We decided to conduct an in-depth research in medical literature and study the evidence available relating this rare entity. Thereafter, we designed a systematic review of previous case reports of IMD with VSR.

The critical role of echocardiography in prompt diagnosis of mechanical complications of MI cannot be overlooked. Before the advent of echocardiography technology, IMD was majorly a postmortem diagnosis. In the current era, an antemortem diagnosis of this dreadful complication could be made definitely even with bedside echocardiography. In our case, the ultimate diagnosis of septal dissection was made by TTE in the subcostal view, although more detailed information about the complex anatomy of the “septal neo-cavity” was obtained by TEE. A useful echocardiographic diagnostic criteria has been defined by Vargas-Barron. Intramyocardial dissection is diagnosed based on the presence of at least three of the following signs:

1. Presence of a neocavitation within the myocardium with an echolucent center;
2. Thinned and mobile endomyocardial border surrounding the neocavitation;
3. Myocardium identified in the outer boundaries of the neocavitation;
4. Changes in the echogenicity of the neocavitation suggesting blood content;
5. Partial or complete absorption of the cavity;
6. Continuity between the cavity and one of the ventricles;
7. Communication between the ventricles through the neocavitation;
8. Detection of flow within the cavity based on Doppler imaging

The most important differential diagnosis of IMD is pseudoaneurysm and intracavitary thrombosis (if the dissecting neo-cavity is filled with thrombus). Unlike pseudoaneurysm, myocardial dissection is entirely encompassed with myocardium. Transthoracic echocardiography and/or TEE are usually revealing to differentiate these entities, but sometimes the net diagnosis is confirmed only at the time of surgery.

The study by Pliam and Sternlieb was the first systematic review of 15 IMD case reports (including one original case), 5 of which were postmortem diagnosis. Based on this report, the first survivor of an IMD was a case described by Stewart in 1981 who had been treated by surgery. Ten cases were medically treated with only one survivor. Five cases were surgically treated who all survived. They concluded that surgical treatment of IMD is preferable to medical treatment.

Leitman et al conducted a meta-analysis of 40 cases of IMD (including 2 original and 38 literature case reports). In contrast to our study, they included IMD cases of any etiology regardless of the presence/absence of VSR. They concluded that low LVEF, age more than 60 years, and late diagnosis, are predictors of in-hospital mortality.
Ventricular septal dissection is almost always associated with major coronary artery lesions at more proximal portions. Zhong et al\textsuperscript{27} reported an exceptional case of ventricular septal dissection following a total occlusion of the first septal branch. Our case also showed that this great damage can occur in the context of a small MI and nonproximal coronary lesions. The late presentation of our case contributed to the damage.

As our study results showed, IMD with VSR was more frequent in patients with inferior MI than those with anterior MI (62.2% vs 37.8%, respectively). Based on the review by Westaby et al,\textsuperscript{32} the incidence of VSR is similar for anterior and inferior MIs. However, if we consider the type of VSR (ie, simple or complex), according to the definition suggested by Edwards et al,\textsuperscript{3} complex-type VSRs which are associated with IMD are many more prevalent in inferior MIs. This finding is in line with our study results. Simple-type VSRs which are more common in anterior MIs were not included in our study. But why inferior MI leads to a more complex form of VSR is a concept not yet fully elucidated in the available literature. Our speculation for explaining this situation is that when the rupture occurs in the more infero-basal location of the septum in the context of inferior MI, it should inevitably have a serpiginous trajectory to reach the RV cavity, maybe due to the constraint of the intact noninfarcted anterior septum in the front and also the nonmuscular membranous septum in the base. But as the rupture occurs in a more apical location of the septum in the context of anterior MI, it could "freely" reach the RV cavity via a direct and simple through-and-through trajectory without the need to dissect the septal myocardium.

Another interesting result of our study that should be discussed is the near-normal average LVEF of our study population displayed in Table 1. This result was in line with the study by Menon et al\textsuperscript{33} in which 939 patients who had been suffering from cardiogenic shock following MI were analyzed, comparing 55 patients whose shock was associated with VSR with 884 patients who had predominant left ventricular failure. This study showed that in the group of VSR-associated cardiogenic shock the average LVEF was significantly higher than the LV-failure-associated cardiogenic shock (40% ± 11% versus 30% ± 13%). This finding can be explained by the fact that VSR results in left ventricular (LV) unloading in to the low pressure RV. Consequently, the noninfarcted walls of LV appear hyperdynamic. On the other hand, VSR enhances the preload to the heart, which also contributes to this hyperdynamism.

The management strategy of patients with IMD is based upon multiple factors: the concomitant need of CAGBs, the hemodynamic status of the patient, and whether the IMD reentered to RV or the pericardial cavity. Evident VSR or free wall rupture is a clear indication of surgery. However, in the present systematic review, we found 3 reports on percutaneous closure of VSR, 2 of whom survived.\textsuperscript{16,27} We observed the highest mortality rate in the group who were medically treated. However, the mortality rate of the surgically treated cases was also high. In our systematic review, in order to figure out potential factors affecting outcome in patients with IMD complicated by VSR, we first grouped the total study population to those who died or survived. Then, we analyzed and compared available variables between the two groups by using various statistical methods. As the results are shown in Table 2, we observed no significant difference between the two groups. Of the variables displayed, it is noteworthy to emphasize the time to VSR repair as a potential factor affecting survival. We observed that 5 (38.5%) out of 13 patients died if the surgery was performed earlier than 5 days from the occurrence of VSR, whereas only 1 (12.5%) out of 8 died if the surgery was carried out later than 5 days after the occurrence of VSR. Although this difference seems to be important, it was not statistically significant. Unfortunately, in

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Comparison of variables among patient who survived or died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient group</td>
<td>Total patients N = 37</td>
</tr>
<tr>
<td>Age</td>
<td>67.65 ± 7.90</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>20 (54.10%)</td>
</tr>
<tr>
<td>Female</td>
<td>17 (45.90%)</td>
</tr>
<tr>
<td>MI territory</td>
<td></td>
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<tr>
<td>Anterior MI</td>
<td>14 (38.90%)</td>
</tr>
<tr>
<td>Inferoposterior MI</td>
<td>22 (61.10%)</td>
</tr>
<tr>
<td>Reperfusion therapy</td>
<td>15 (40.54%)</td>
</tr>
<tr>
<td>Management VSR</td>
<td></td>
</tr>
<tr>
<td>Surgery</td>
<td>26 (72.20%)</td>
</tr>
<tr>
<td>Nonsurgery</td>
<td>10 (27.80%)</td>
</tr>
<tr>
<td>Time to VSR closure</td>
<td></td>
</tr>
<tr>
<td>≤5 d</td>
<td>13 (61.90%)</td>
</tr>
<tr>
<td>&gt;5 d</td>
<td>8 (38.10%)</td>
</tr>
<tr>
<td>LVEF</td>
<td>49.27 ± 11.31</td>
</tr>
</tbody>
</table>

Abbreviations: d, day; LVEF, left ventricular ejection fraction; MI, myocardial infarction; N, number; NS, nonsignificant; VSR, ventricular septal rupture.
only 21 cases of our total study population, data were available regarding this important variable and this would erroneously affect the final results. In a retrospective single-center study by Malhotra et al., reporting on 55 cases with post-MI-VSR aiming to identify factors affecting the prognosis of this complication, shorter intervals between VSR and surgery were found to be a strong and probably the most important predictor of mortality. It was suggested that if the surgery could be delayed for more than 3 days after the occurrence of VSR, a 50% reduction in mortality could be expected. However, it is debatable whether this conclusion could be generalized to patients with a combination of VSR and IMD. Finally, as we previously stated, the combination of VSR and IMD is a rare clinical entity. To make a conclusion concerning the effect of time to VSR repair on mortality in the setting of concomitant IMD, a larger population of patients is needed. However, regarding its rarity, conducting such a study seems to be impossible. We recommend to determine the optimal time for surgery according to the individual characteristics of each patient.

The optimal management of IMD confined to the myocardium is more controversial. There are rare reports of spontaneous reabsorption of such hematomas with or without anticoagulation. Some authors recommend a conservative approach in hemodynamically stable patients with small apical IMD without VSR particularly if successful reperfusion therapy has been already implemented. There are also reports of mortality following such a conservative approach. The best strategy should be individualized in every case.

Finally, we affirm the fact that many aspects of the discussed dreadful complication: IMD with VSR is still a matter of debate especially the optimal management of this complication and how to identify at-risk patients. Large-scale high power clinical trials enlightening these dilemmas are highly suggested.

## Conclusion

Intramycocardial dissection with VSR is a rare and dreadful mechanical complication of MI. Echocardiography has a critical role in diagnosing this complication. Key features include neocavitation within the myocardium, communication between the ventricles through the neocavitation and detection of flow within the cavity. Surgery is mandatory in IMD with VSR.

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**References**


**SUPPORTING INFORMATION**

Additional supporting information may be found online in the Supporting Information section.

Table S1. A review of reported cases of intramyocardial dissection with ventricular septal rupture following myocardial infarction

Video S1. Transthoracic echocardiography with color Doppler imaging in the parasternal long axis view: an echo free space in the mid-part of anterior septum blood flow with in the cavity

Video S2. Color Doppler imaging in the subcostal view showing flow across tract and the exit site to the right ventricle

Video S3. Real-time three-dimensional imaging in subcostal view showing the dissected septum (asterisk)

Video S4. Intraoperative transesophageal echocardiography 2D imaging in the two-chamber view showing an abnormal echo-free space in the inferoposterior aspect of the heart (asterisk)

Video S5. X-plane imaging with color Doppler simultaneously showing the dissected septum in two orthogonal planes

Video S6. 2D imaging in the transgastric view showing a large ventricular septal rupture at the level of papillary muscles; the septal rupture directly enters the neo-cavity within the dissected septum (asterisk)

Video S7. Color Doppler imaging in the transgastric view showing free flow from the left ventricle into the neo-cavity via the septal rupture (asterisk)

Video S8. Color Doppler imaging in the transgastric view showing the reentry site of the neo-cavity into the right ventricle (arrow)

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