Case Report

Clinical Outcomes of Ventricular Septal Rupture Complicating ST-Segment Elevation Myocardial Infarction: A Case Series from Siriraj Hospital and Literature Review

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Objective: To access the clinical outcomes of ventricular septal rupture (VSR) complicating acute myocardial infarction (AMI) patients in the Faculty of Medicine Siriraj Hospital, Bangkok, Thailand.

Materials and Methods: The authors retrospectively reviewed the medical records of Siriraj Hospital between January 2005 and December 2021 in line with the International Classification of Disease, version 10 (ICD-10) code I23.2, which revealed 18 patients who matched the search. Among these, three patients were excluded from the present study as one patient could not access the admission data and the other two were not truly VSR.

Results: In total, 15 VSR patients were included in the analysis, comprising eight males and seven females, with mean age of 67.53±7.95 years old. Of all the patients, 60% had anterior ST-segment elevation myocardial infarction. The mean left ventricular ejection fraction was 53.13±14.98%. Coronary angiography was performed in 14 patients (93.37%). In 50% of the patients, left anterior descending artery was the culprit lesion. Ten patients had multi-vessel diseases. Six patients underwent percutaneous coronary intervention (PCI), with the rate of successful reperfusion at 83.33%. For the six patients who underwent PCI, the procedure was performed before the VSR surgical repair. Ten patients underwent coronary artery bypass graft. For the 12 patients who underwent VSR surgical repair, the in-hospital survival rate was 100%. The overall in-hospital mortality rate was 20%. The non-survival patients did not receive the VSR surgical repair. After the median follow-up of three years, no additional mortality rate was reported.

Conclusion: The mortality rate of VSR complicating AMI is high, especially, for patients who are not feasible for VSR repair. However, given the limitation in the number of patients involved in the present analysis, the authors could not identify the risk factors for developing in-hospital adverse outcomes.

Keywords: Ventricular septal rupture; Acute myocardial infarction; Mechanical complications of myocardial infarction

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Ventricular septal rupture (VSR) is one of the lethal mechanical complications in acute myocardial infarction (AMI)[1]. In the modern era of the emergency reperfusion strategy, the mortality rate of AMI has declined as well as the incidence of VSR[2]. Nevertheless, VSR patients still face a grave prognosis, especially patients who are left untreated[3]. The conservative approach, therefore, should be preserved only for inoperable patients.

The surgical closure of the VSR and appropriate hemodynamic support bridging to the operation are mandatory[4]. However, the appropriate timing of the operation is still debated. On one hand, the mortality rate of early surgery is markedly higher compared to delayed surgery[5], while on the other hand, a postponement of the surgery can lead to rupture expansion, refractory cardiogenic shock, multi-organ failure, and even death[6].

To date, percutaneous closure of the VSR serves as an alternative treatment for high-surgical-risk patients[7]. Nevertheless, the early percutaneous intervention still has a bad prognosis as the early surgical correction. As a result, a case-based heart team approach is necessary. The risk of prolonged

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Table 1. Baseline characteristics and clinical presentation

Patient number	Age/sex	BSA	HT	DM	Angina	Referred from other hospitals	Duration from onset to FMC	Clinical diagnosis	ECG localization
1	73/F	N/A	No	No	Yes	Yes	3 days	L-STEMI	Inferior
2	59/M	1.77	Yes	Yes	Yes	Yes	5 days	ADHF	Inferior
3	68/F	1.54	Yes	No	Yes	No	4 days	L-STEMI	Anterior
4	56/M	1.97	Yes	No	Yes	Yes	10 days	ADHF	Inferior
5	71/F	1.61	Yes	No	No	Yes	6 days	ADHF	Anterior
6	70/M	1.53	Yes	No	Yes	Yes	9 days	ADHF	Anterior
7	86/F	1.62	Yes	Yes	Yes	No	N/A	STEMI	Anterior
8	59/M	1.73	Yes	Yes	No	No	5 days	ADHF	Anterior
9	72/M	1.66	No	Yes	Yes	Yes	3 days	L-STEMI	Anterior
10	65/F	1.43	Yes	Yes	Yes	Yes	9 hours	L-STEMI	Inferior
11	78/F	1.39	Yes	No	Yes	No	1 day	L-STEMI	Anterior
12	68/M	1.74	Yes	Yes	Yes	Yes	10 days	L-STEMI	Inferior
13	64/M	1.74	Yes	Yes	Yes	Yes	1 day	L-STEMI	Inferior
14	64/F	1.56	No	Yes	Yes	Yes	8 days	L-STEMI	Anterior
15	60/M	1.66	No	No	Yes	No	3 days	L-STEMI	Anterior

ADHF=acute decompensated heart failure; BSA=body surface area; DM=diabetes mellitus; ECG=electrocardiogram; F=female; FMC=first medical contact; HT=hypertension; M=male; N/A=data not available; L-STEMI=late-onset ST-segment elevation myocardial infarction (onset ≥12 hours); STEMI=ST-segment elevation myocardial infarction (onset <12 hours)

cardiogenic shock against the high morbidity and mortality rates of the early procedure should be taken into account.

In the present manuscript, the authors report a case series of VSR complications in AMI patients at Siriraj Hospital, Mahidol University, Bangkok, Thailand.

Materials and Methods

The author accessed the database of the Faculty of Medicine Siriraj Hospital, Mahidol University and searched between January 2005 and December 2021, using the International Classification of Disease, version 10 (ICD-10) code I23.2 (ventricular septal defect as a current complication following AMI) to identify the study subjects. As a result, 18 patients were found, and their data were extracted. After manually reviewing these on a case-by-case basis, three patients were excluded as two patients did not receive the correct diagnosis and one could not access the patient's admission data. The remaining 15 patients were enrolled in the present study.

The patients' baseline clinical characteristics and clinical presentations were collected. In all cases, the diagnosis of VSR was made by transthoracic echocardiogram (TTE). Coronary angiography (CAG) was either performed at the authors' catheterization laboratory or at the first medical contact (FMC) hospital. The surgical VSR repair was performed by cardiothoracic surgeons at the authors' institute. The decision for the treatment strategy for each patient, including the timing of revascularization or the surgical VSR repair, the procedural technique, and the medication or equipment used, was at the treating physician's discretion. The follow-up data were obtained by retrospective chart review. The Siriraj Institutional Ethical Review Board of the Faculty of Medicine Siriraj Hospital, Mahidol University approved the present study [Protocol number 175/2565(IRB3)]. The need for patient consent was waived as this was a retrospective and anonymized data reviewed.

Results

Baseline characteristics and clinical presentation

The mean patient age was 67.53±7.95 years old. Eight patients were male, and seven were female. Of all the patients, 11 patients had hypertension and eight had diabetes. The mean body surface area was 1.64±0.15 m^2. Of the 15 patients, 13 (86.67%) reported angina at presentation. The median duration from disease onset to the FMC hospital was 1.5 [interquartile range (IQR) 0.75 to 6.0] days. At the time of presentation, 10 patients were diagnosed with ST-elevated myocardial infarction (STEMI), while five patients were diagnosed with acute decompensated heart failure (ADHF). Details of the patients' baseline characteristics, and clinical presentation are listed in Table 1.

Table 2. Investigations

Patient number	Initial troponin (ng/L)	Peak troponin (ng/mL)	LVEF by TTE (%)	RVSP by TTE (mmHg)	VSR localization by TTE	Culprit lesion by CAG	Diseased vessel(s)
1	3.17	5.68	67	24	Inferior septum	RCA	3
2	0.58	1.38	65	55	Inferior septum	RCA	3
3	1.31	1.01	78	50	Apical septum	LAD	3
4	0.98	2.72	61	N/A	Inferior septum	RCA	1
5	0.40	0.39	70	N/A	Apical septum	LAD	1
6	1.90	1.79	55	60	Apical septum	N/A	3
7	575.00	569.00	45	N/A	Apical septum	LAD	1
8	933.60	1398.00	34	56	Apical septum	N/A	N/A
9	9466.00	N/A	54	39	Apical septum	LAD	3
10	4110.00	7653.00	48	64	Inferior septum	RCA	2
11	7853.00	10000.00	53	67	Apical septum	LAD	2
12	1102.00	1345.00	57	25	Inferior septum	RCA	3
13	6001.00	N/A	21	36	Inferior septum	Graft	3
14	N/A	2904.00	54	N/A	Apical septum	LAD	1
15	1729.00	N/A	34	59	Apical septum	LAD	3

CAG=coronary angiography; LAD=left anterior descending artery; LVEF=left ventricular ejection fraction; N/A=data not available; RCA=right coronary artery; RVSP=right ventricular systolic pressure; TTE=transthoracic echocardiography; VSR=ventricular septal rupture

Table 3. Treatment strategies

Patient number	PCI	Timing of PCI after angina onset (days)	CABG/ number of graft(s)	VSR surgical closure	Timing from VSR diagnosis to VSR closure (days)	VSR closure concomitant with CABG
1	Not done	-	Yes/4	Yes	3	Yes
2	Not done	-	Yes/3	Yes	7	Yes
3	Done/successful	4	Yes/4	Yes	9	Yes
4	Not done	-	Yes/1	Yes	2	Yes
5	Not done	-	No	Yes	3	-
6	Not done	-	Yes/2	Yes	N/A	Yes
7	Done/successful	1	Yes/1	Yes	8	Yes
8	Not done	-	Yes/2	Yes	2	Yes
9	Done/failed	3	No	No	-	-
10	Done/successful	1	Yes/3	Yes	1	Yes
11	Done/successful	2	No	Yes	20	-
12	Done/successful	10	Yes/4	Yes	10	Yes
13	Not done	-	No	No	-	-
14	Not done	-	No	No	-	-
15	Not done	4	Yes/2	Yes	12	Yes

CABG=coronary artery bypass graft; N/A=data not available; PCI=percutaneous coronary artery intervention; VSR=ventricular septal rupture

Relevant investigations

All patients underwent TTE. The mean and median left ventricular ejection fraction (LVEF) was 53.13±14.98% and 54%, respectively. Nine patients (60%) had VSR located at the apical-septum area as assessed by TTE. The mean initial and peak troponin T were 1,325.63±748.62 and 1,907.09±1,055.93, respectively. Fourteen patients (93.37%) underwent CAG. Left anterior descending artery (LAD) was identified as the culprit lesion in seven patients, while five patients had right coronary artery as an infarct-related artery. Only one patient had a saphenous vein graft at the culprit lesion. In addition, ten patients had multi-vessel disease. Summaries of the investigations are presented in Table 2.

Timing of revascularization and VSR repair

Table 3 lists the treatment strategies. As seen

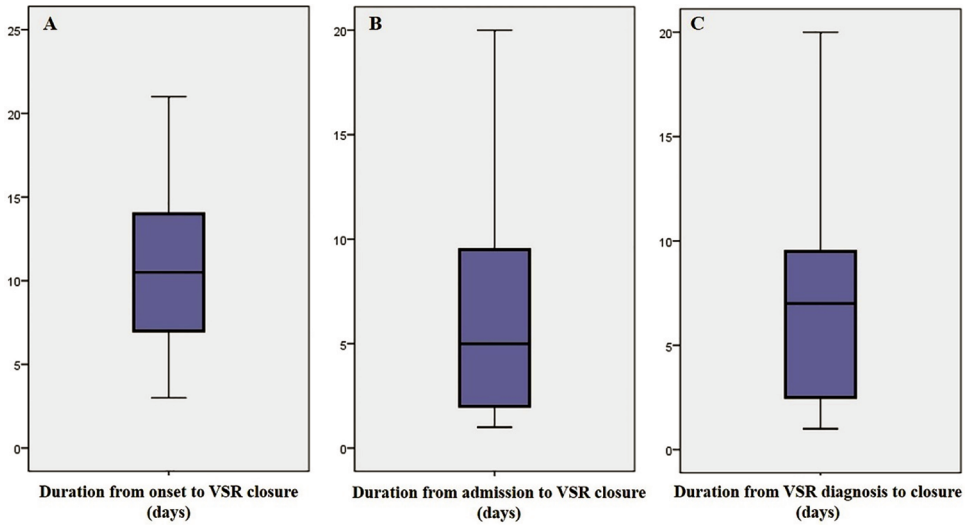

Figure 1. Key timing of surgical VSR closure. (A) The median duration from onset to VSR closure was 10.5 (6.5 to 14.5) days. (B) The median duration from admission to VSR closure was 5 (IQR 2 to 9.5) days. (C) The median duration from VSR diagnosis to closure was 7 (IQR 2 to 10) days.

CABG=coronary artery bypass graft; IQR=interquartile range; VSR=ventricular septal rupture

Table 4. Clinical outcomes

Patient number	Duration of admission (days)	Cardiogenic shock	In-hospital stroke	Hospital-acquired infection	In-hospital outcome	Redo-VSR closure
1	13	No	No	Yes	Survive	N/A
2	14	No	No	No	Survive	No
3	18	No	No	Yes	Survive	No
4	16	No	No	No	Survive	No
5	22	No	No	Yes	Survive	No
6	7	No	No	No	Survive	Yes
7	63	No	No	Yes	Survive	No
8	7	No	No	No	Survive	No
9	1	Yes	No	No	Death (cardiac)	-
10	16	Yes	Yes	No	Survive	No
11	55	Yes	No	Yes	Survive	N/A
12	20	No	No	No	Survive	No
13	24	Yes	No	Yes	Death (non-cardiac)	-
14	28	Yes	No	No	Death (non-cardiac)	-
15	25	Yes	No	Yes	Survive	No

N/A=data not available; VSR=ventricular septal rupture

from the table, six patients underwent percutaneous coronary artery intervention (PCI), which were performed before the VSR repair. For three out of these six patients, the operators decided to perform PCI after the VSR diagnosis was made, prior to the VSR repairment. The median time from onset to PCI and to coronary artery bypass graft (CABG) were 2.5 and 10.5 days, respectively. VSR surgical repair was done in 12 patients. The median time from VSR diagnosis to VSR surgical repair was 7 (IQR 2 to 10) days (Figure 1). The ten patients who underwent CABG, had concurrent VSR repair.

Clinical outcomes and in-hospital adverse events

Table 4 presents the detailed in-hospital clinical outcomes. The median length of hospital stay was 16 (IQR 8 to 24) days. Six patients developed cardiogenic shock during the index hospitalization. An intra-aortic

Table 5. Comparison of the published registries of ventricular septal rupture

Registries	Year of publication	Population	Country	Age (years); mean±SD	Female	Anterior STEMI	LVEF (%); mean±SD	Use of IABP	VSR repair/closure; n (%)	Overall in-hospital mortality rate	In-hospital mortality rate of the patients who underwent VSR closure
Menon, et al.	2000	55	International	72±10	58%	42%	40±11	74.5%	Surgical repair/31 (56.36)	87.27%	80.65%
Tai, et al.	2018	96	China	66±10.70	42.70%	54.17%	50.92±12.20	63%	Device closure/20 (20.83) No surgical repair/0 (0.0)	52.08%	21%
Hua, et al.	2020	55 (non-survival)	China	68±9.1	55%	N/A	46.3±12.4	10.9%	Surgical repair/7 (12.73)	47%	12%
		61 (survival)		62.7±8.1	31%	N/A	49.8±9.5	34.4%	Surgical repair/53 (86.89)		
Zhang, et al.	2021	36 (non-survival)	China	72.3±8.6	66.7%	86.1%	49.6±9.9	97.2%	Device closure/8 (22.2) No surgical repair/0 (0.0)	72%	45%
		14 (survival)		68.7±6.9	57.1%	71.4%	47.50±8.7	78.6%	Surgical repair/3 (21.4) Device closure/10 (71.4)		
The present registries	N/A	15	Thailand	67.53±7.95	46.67%	60%	53.13±14.98	80%	Surgical repair/12 (80.0)	20%	0%

IABP=intra-aortic balloon pump; LVEF=left ventricular ejection fraction; SD=standard deviation; STEMI=ST-segment elevation myocardial infarction; VSR=ventricular septal rupture

balloon pump (IABP) was used in 12 patients (80%) and seven patients had hospital-acquired infections. These patients died during hospitalization, leading to an overall in-hospital mortality rate of 20%. All the non-survival patients did not undergo the VSR surgical repair. The in-hospital survival rate of the patients that underwent VSR surgical repair was 100%.

Follow-up data

The median follow-up period was 36 months, with a maximum of 10 years. Two patients were lost to follow-up. Recurrent VSR leading to the redo procedure occurred in one patient. Two patients developed ADHF during the follow-up period. No mortality or recurrent myocardial infarction (MI) were reported.

Discussion

VSR is one of the lethal mechanical complications of AMI, which comprises ventricular free wall rupture, rupture of the papillary muscle, and VSR[8]. The modern-day timely reperfusion strategy of AMI can markedly reduce the incidence of VSR compared to in the pre-thrombolytic era from 1 to 3% to 0.2%[2,3,9,10]. Nevertheless, patients with a large MI or lack of adequate reperfusion are still at a high risk of developing VSR[11]. As a result, the morbidity and mortality rate of VSR remains high in contemporary studies, from 47.7% to 72%[12,13]. The present study reported an in-hospital mortality rate of VSR of 20%, which is lower than in most other reports (Table 5)[7,12-14].

VSR is caused by necrosis of the ischemic ventricular septum with neutrophil infiltration[4]. This complication can develop at any anatomical location, regarding the vascular territories that supply the affected myocardium. The use of electrocardiography can demonstrate an ongoing ischemic, evolving MI, or an anterior or inferior infarction[11]. An anterior infarction that has LAD as the culprit vessel typically causes an apical rupture and simple lesions, whereas an inferior infarction is more likely to cause a basal inferoposterior rupture, which is more complex in morphology and more challenging to repair, leading to a worse prognosis[1,3,4,8,15]. In addition, basal inferoposterior VSR is commonly associated with secondary ischemic mitral regurgitation (MR). CAG may demonstrate a total occlusion of the culprit vessel without collateral circulation. In the present registry, nine patients (60%) had anterior infarction with apical septal rupture. The mortality rate of the apical rupture group was 22.2%. Of the 14 patients who underwent CAG in the present study, 11 patients (78.6%) had total occlusion of the infarcted artery. Whereas, 57% of patients from the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-1) trial had occluded culprit artery[9].

After the defect has developed, blood would be shunted from the left ventricle, which has a higher pressure, to the lower-pressured right ventricle. The pulmonary blood flow thus increases, and volume overload of the left-sided heart would occur[16].

The clinical course of VSR is defined as a "bimodal distribution"[3]. The incidence of VSR is typically highest on the first day and then between the third and fifth day. In the GUSTO-1 trial, the median time from the onset of AMI to septal rupture was one day[9]; whereas in the "Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK)" trial, it was 16 hours[14]. The

present study reported the median time from the onset diagnosis of VSR as 2.5 (IQR 1 to 7.5) days.

Upon physical examination, in 90% of the patients there would be a harsh pansystolic murmur located at the left sternal border, accompanied by systolic thrill in 50% of patients[17]. A loud pulmonic closure sound, tricuspid regurgitation, and S3 gallop was also heard if the right-sided blood flow was increased. In patients that develop cardiogenic shock, the sign of a low cardiac output would be detected. The clinical presentation would be varied from an isolated murmur, worsening dyspnea, ADHF, cardiogenic shock, or circulatory collapse[8,11]. Interestingly, the presenting symptoms would be only ADHF with silence MI. There was one registry study from China that included 96 VSR patients and reported that only 20% of VSR patients had a history of angina[7]. For the present registry, five out of the present study 15 patients (33%) presented with ADHF without history of angina.

TTE with color flow-Doppler is the gold standard for the diagnosis of VSR[1]. TTE can identify the location of the ventricular septal drop-out, measure the rupture size, and demonstrate the abnormal flow across the VSR. TTE can differentiate VSR from MR caused by a ruptured papillary muscle. In addition, TTE can evaluate the left and right heart function, regional wall motion abnormality, right ventricular systolic pressure, and estimate the degree of the left-to-right shunt[1,3,8,18].

Right heart catheterization with oxygen blood sampling is useful to detect a left-to-right shunt. Here, there would be an oxygen step-up from the right atrium to the pulmonary artery. The ratio of the pulmonary flow and systemic flow can be calculated, which would reflect the degree of the shunt, by using the following formula: Qp/Qs=[Oxygen saturation from systemic artery – mixed venous oxygen saturation] / [Oxygen saturation from pulmonary vein – oxygen saturation from pulmonary artery]. It should be noted that oxygen saturation from the pulmonary vein could be estimated as 100% in a healthy patient[16].

VSR patients are at a high risk of rapid deterioration. Observation in the intensive care unit or transfer the patient to a tertiary care center is advisable[19]. Afterload reduction, including pharmacologic and non-pharmacologic approaches, is required to decrease the left-to-right shunt before definite treatment. IABP insertion should be considered for hemodynamic support. The present registry reported the rate of IABP used as 80%, which is similar to data from the SHOCK trial at 75%[14]. For refractory circulatory collapse patients, extracorporeal membrane oxygenation may be needed for bridging before surgery or heart transplantation.

Conservative treatment alone has a bad prognosis[13,20]. Therefore, medical therapy should only be preserved for inoperable patients. Surgical correction is the definitive treatment for VSR. There are two common surgical techniques for VSR closure, the David and Daggett procedures. The David technique is an infarct exclusion procedure while the Daggett technique places the patch directly over the rupture site[21].

The most appropriate timing of surgery is still debated. Early surgery is associated with worse outcomes, especially in the first 24 hours[22], generally because in the acute setting, the infarcted myocardium is friable and weak, leading to a high chance of unfavorable surgical outcomes[23]. In contrast, delayed surgery could allow for fibrotic healing at the rupture site, resulting in a lower risk of surgical patch dehiscence or recurrent septal rupture[11]. The delay in surgery also reduces the bleeding risk from antiplatelet therapy. However, stable VSR patient may potentially develop rapid clinical deterioration if the surgery was postponed. As a result, a case-based heart team approach is preferred. The present study reported a median time from onset to VSR closure of 10.5 days. Importantly, it should be noted that emergency surgery is commonly performed in unstable and hemodynamically compromised patients. This may cause a selection bias in data analysis, resulting in a higher mortality rate in the early surgery group.

Data from the Society of Thoracic Surgeons National (STS) Database reported the operative mortality rate of VSR repair at 42.9%[5]. Interestingly, the 12 patients in the present registry who underwent surgical VSR repair had an operative mortality rate of 0%.

The revascularization strategy of the infarcted artery is also discussed. The benefit of the immediate restoration of the infarcted artery for salvaging the myocardium against the bleeding risks from anticoagulation therapy should be weighed. However, the involved myocardium is likely to be already infarcted due to the late clinical presentation. Therefore, the role of immediate revascularization prior to surgery is controversial[24]. The 2021 American College of Cardiology (ACC)/American Heart Association (AHA) guidelines for coronary revascularization recommend performing CABG at the time of definite surgery for a survival benefit

(Class of recommendation 1, level of evidence B)[25]. Saphenous vein graft is commonly used for CABG in this clinical setting.

Transcatheter closure of VSR serves as an alternative treatment for inoperable patients[26]. This procedure results in an immediate reduction of the left-to-right shunt. As a result, it instantly improves the hemodynamics. Transcatheter closure can be used for definite treatment, or as a bridging for surgical repair or heart transplantation. However, early intervention still has a high morbidity and mortality rate, which is same as surgical VSR closure[27-29]. This could be explained by the instability of the early infarcted myocardium or serpiginous morphology, resulting in peri-device leakage or device embolization[4].

The 5-year survival of the in-hospital survival patients from the previous literature was 67% to 79%[15,30]; whereas, in the present study, the survival rate of the median follow-up of 12 patients at 36 months was 100%.

Conclusion

VSR is a rare but lethal mechanical complication in AMI. Early detection, adequate hemodynamic stabilization, and appropriate timing of the definite treatment are mandatory to ensure a good outcome. The care of VSR is complex and needs a multidisciplinary approach. Surgical VSR closure accompanied by CABG is the standard treatment. Transcatheter closure is the alternative choice for prohibitive surgical patients. The risk of prolonged cardiogenic shock or rapid clinical deterioration versus the significant mortality rates of an early procedure should be discussed.

What is already known on this topic?

The morbidity and mortality rate of VSR complicating AMI is high. Surgical VSR repair and revascularization are the mainstay treatment.

What this study adds?

To the best of the authors' knowledge, this is the first report of a case series of VSR patients in Thailand. The present study data support the surgical approach for the definite treatment of VSR.

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Conflicts of interest

The authors confirm there are no conflicts of interest to declare.

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