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CASE REPORT

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Presentation of a residual post-myocardial infarction ventricular septal defect; a literature review based on a case report

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Abstract

Introduction In the era of modern techniques for the early diagnosis and revascularization of myocardial infarction, post-myocardial infarction ventricular septal defect is rarely seen. However, this potentially fatal complication of ischemic cardiac events cannot always be detected and diagnosed in a straightforward pattern of practice. This study presents an initially delayed-presented post-infarction ventricular septal defect.

Case presentation The patient was a 58-year-old white man who presented to the cardiology clinic complaining of moderate exertional dyspnea initiated two months ago and exacerbated by the time. His medical history includes an admission three years prior, where he was diagnosed with COVID-19 pneumonia and a myocardial infarction that was complicated by a ventricular septal defect (VSD) and hemodynamic instability. This condition was managed through urgent surgical revascularization and closure of the defect. Due to his current symptoms, further cardiac investigations were planned. A transthoracic echocardiogram was recommended after detecting a grade 3/6 systolic murmur during the physical examination. The initial assessment using an apical four-chamber TTE appeared normal. However, when performing a modified view with a posterior tilt, a bulging septum was observed, leaning toward the right ventricle. This bulging contained a defect with a left-to-right shunt, identified as a residual defect in the area of the repaired patch, along with a myocardial aneurysm. Due to the inconsequential findings from the echocardiogram study, the patient was scheduled for a follow-up echocardiogram, which showed no changes after six months. Additionally, the patient underwent therapeutic management addressing chronic obstructive pulmonary disease.

Conclusion Although post-infarction ventricular septal defects are rarely seen in the revascularization era, the COVID-19 era was associated with an increase in the prevalence of this complication. It is important to be vigilant for patients who experienced an index event during that time. This potentially fatal complication can present with new issues following the initial event, such as residual defects. Comprehensive imaging studies are necessary to detect the underlying pathology.

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Key clinical message Diagnosing post-infarction ventricular septal defect requires the hypervigilance and precision of the cardiologist, who examines the patient and performs the cardiac imaging. Therefore, comprehensive investigations are crucial in patients with a suspicious history of ischemic cardiac events.

Keywords Myocardial infarction, Ventricular septal defect, Cardiac surgery, Echocardiogram, Case report

Introduction

The COVID-19 pandemic has profoundly impacted the management and outcomes of cardiovascular diseases (CVD), particularly in patients with acute myocardial infarction (AMI) [1]. Post-infarction ventricular septal defect (PIVSD) is a rare but potentially fatal complication that is mostly associated with delayed MI presentation, cardiogenic shock, and high mortality rates if left untreated [2–4]. Recent advancements in revascularization techniques and improved management strategies for acute myocardial infarction (AMI) have significantly decreased the incidence of PIVSD over the past few decades [5]. The incidence of PIVSD has dropped dramatically from 1 to 2% to 0.25–0.3% due to novel thrombolytic therapies and interventional procedures introduced as AMI treatment. Despite these efforts, the mortality rate for medically treated cases of PIVSD remains exceedingly high, exceeding 90%. This highlights the critical need for prompt diagnosis and optimal management of this condition. Unfortunately, there is no consensus on the best treatment approach, and the timing and strategies for managing PIVSD continue to be hotly debated [6–8]. The literature shows conflicting conclusions regarding whether surgical or interventional management is superior, as well as when the procedure should ideally be performed [9, 10]. The latest guidelines from the European Society of Cardiology (ESC) provide a clear protocol that has been endorsed by their surgical intervention and cardiology scientific groups [11]. One significant barrier to the timely treatment of PIVSD is the potential for missed diagnoses, which have been reported due to atypical locations or presentations [12]. Moreover, during the COVID-19 pandemic, there was an unexpected increase in the incidence of post-myocardial infarction ventricular septal defects, likely resulting from delays in patients seeking treatment [13].

The present study reports a complex case of an extremely posteriorly located PIVSD following a late presentation of myocardial infarction (MI), coinciding with COVID-19 pneumonia. The timing of the presentation and the diagnostic process that led to the correct conclusion are unique and could contribute to the existing knowledge about this potentially fatal cardiac condition.

Case presentation

Presentation and physical exam

A 58-year-old white man presented to a tertiary cardiology hospital complaining of moderate exertional dyspnea, which had been initiated two months before his presentation. He reported experiencing cough, sputum production, and pleuritic chest pain during exercise. His medical history was notable for type II diabetes mellitus and a significant smoking history of nearly 40 years (equivalent to 60 pack-years).

A further investigation into the patient's medical records revealed that he had been hospitalized three years earlier due to fever, cough, and shortness of breath. During the pandemic, he was initially diagnosed with COVID-19, confirmed by a polymerase chain reaction (PCR) test. Chest imaging indicated diffuse interstitial pneumonia. On the second day of admission, cardiac ischemic markers (Table 1) were found to be elevated, along with significant changes in the electrocardiogram (Fig. 1), leading to a diagnosis of myocardial infarction (MI). Transthoracic echocardiography (TTE) demonstrated mild systolic dysfunction with an ejection fraction of 45% and inferoseptal hypokinesia. It also revealed a large muscular ventricular septal defect (VSD) measuring 18 mm at the base of the inferoseptal segment.

Due to his COVID-19 condition, coronary angiography (CAG) was performed on the fifth day of admission. This revealed critical three-vessel coronary artery disease (3VD) (Fig. 2), including involvement of the right coronary artery (RCA) (Fig. 3). Given the lack of response to medical support and the deterioration of the patient's condition, he underwent urgent coronary artery bypass grafting (CABG) along with VSD repair.

Intraoperative findings revealed a large necrotic area with loose tissue at the distal end of the basal inferoseptum, surrounding the ventricular septal defect (VSD). Due to the patient's unstable hemodynamics and the close proximity of the necrotic tissue to the tricuspid valve, optimal exclusion of the necrotic tissue and using a standard repair approach was not feasible. As a result, a single patch was used to close the defect, and it was sutured to the available myocardium, despite the absence of sufficient healthy tissue for an ideal repair.

Follow-up TTE on the 15th postoperative day showed no residual VSD and the patient was discharged in stable condition after a total hospitalization of 45 days.

At the current presentation, he appeared tachypneic and anxious on physical examination. The vital sign assessment showed an abnormally elevated respiratory rate and declined oxygen saturation in ambient air (BP: 130/60, HR: 96, RR: 28, T: 36.5, o2 sat: 91%). The cardiorespiratory exam revealed a diffuse rhonchus, wheezing,

Table 1 Laboratory findings of the patient

Test	Result	Reference Range
RBC (10^6/μl)	5.3	4.2–5.5
Hemoglobin (gr/dL)	16	12–16
WBC (per μl)	8000	4.000–11.000
MCV (fL)	89	80–99
Hematocrit (%)	46	37–47
Platelet (per μl)	360	150.000-400.000
Neutrophils (%)	61	40–75
Lymphocytes (%)	42	20–45
MCH (pg/cell)	29	27–31
MCHC (g/dL)	35	32–36
T3(ng/ml)	3	0.7-2.0
T4(mcg/dl)	10	4.5–12.5
TSH (mIU/ml)	3.1	0.83–6.5
Troponin I (ng/ml)	1580	0.3
lipid profile*, *coagulation factors*, *and troponin		
Cholesterol	190	Up to 200
Triglyceride (mg/dl)	130	Up to 150
LDL (mg/dl)	90	Up to 130
HDL (mg/dl)	32	> 45
LDH (Iu/L)	450	235–470
K^+ (meq/lit)	4.7	3.5–5.3
Na(meq/lit)	139	135–148
Creatinine (mg/dl)	1.1	0.5-1.00
Urea (mg/dl)	31	13–43
Uric acid(mg/dl)	6	2.3–6.1
FBS (mg/dl)	145	70–115
CK-MB(u/l)	19	< 24

Abbreviations: RBC: Red Blood Cells, Hemoglobin: Hemoglobin, WBC: White Blood Cells, MCV: Mean Corpuscular Volume, Hematocrit: Hematocrit, Platelet: Platelets, Neutrophils: Neutrophils, Lymphocytes: Lymphocytes, MCH: Mean Corpuscular Hemoglobin, MCHC: Mean Corpuscular Hemoglobin Concentration, T3: Triiodothyronine, T4: Thyroxine, TSH: Thyroid Stimulating Hormone, Cholesterol: Cholesterol, Triglyceride: Triglyceride, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, LDH: Lactate Dehydrogenase, K+: Potassium, Na: Sodium, Mg: Magnesium, Phosphorus: Phosphorus, Ca: Calcium, Alb Serum: Albumin Serum, Creatinine: Creatinine, Urea: Urea, Uric acid: Uric Acid, FBS: Fasting Blood Sugar, CK-MB: Creatine Kinase-MB

and a grade 3/6 systolic murmur along the left sternal border. Therefore, more investigation with echocardiograms was planned.

Diagnostic and therapeutic interventions

Regarding laboratory tests, it is notable that troponin I, infection and inflammatory markers' levels were all negative and NT-proBNP level was 130 pg/ml. Additionally, the ECG revealed no ST-T changes or acute abnormalities. A TTE examination was conducted, which initially appeared normal in the standard apical four-chamber view. However, a modified view with a posterior tilt revealed a bulging septum leaning toward the right ventricle, with expansion occurring during ventricular systole. This bulging contained a defect with a left-to-right shunt. Regardless of the tilt used, the captured image resembled the findings typically associated with a perimembranous ventricular septal defect (VSD), even though detecting the perimembranous structures usually requires an anterior tilt (Fig. 4**).** Ultimately, it was determined that the finding was actually an aneurysmal formation at the basal of the inferoseptum. Color Doppler studies indicated a high-gradient residual VSD, with a peak gradient of 81 mmHg, while pulse wave Doppler analysis showed a nonsignificant left-to-right shunt (Qp/Qs ratio of 1.4).

Conclusion and Follow-Up

The echocardiographic findings indicated a nonsignificant shunt on the Doppler study, a high-gradient jet on color study, and a normal left ventricle size (Table 2). As a result, the patient was advised to pursue medical management, undergo periodic echocardiography follow-ups, and address concerns related to coronary artery disease, chronic obstructive pulmonary disease (COPD), and lifestyle modifications.

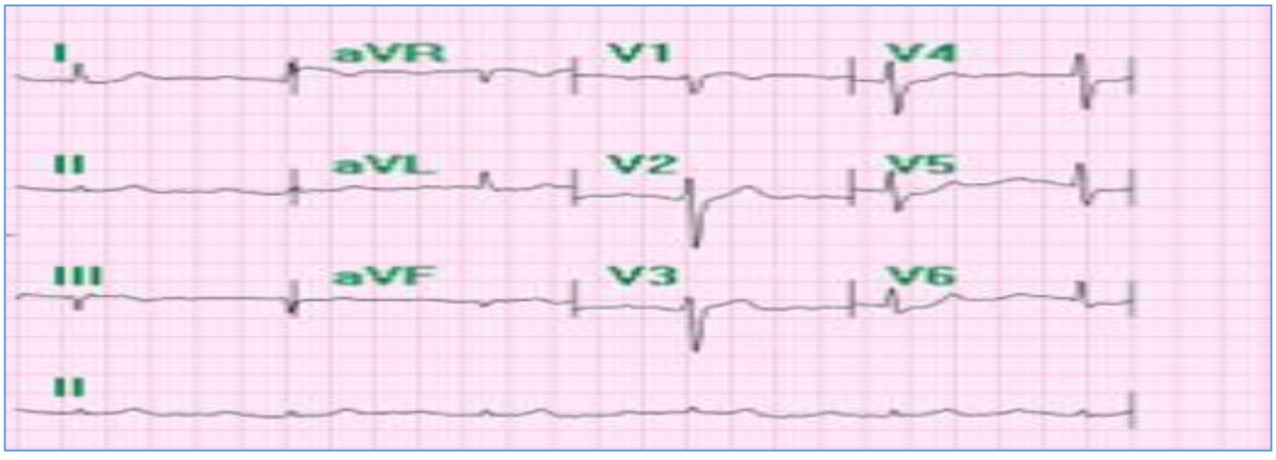

Fig. 1 On the second day of admission, Q waves were detected in leads III and aVF

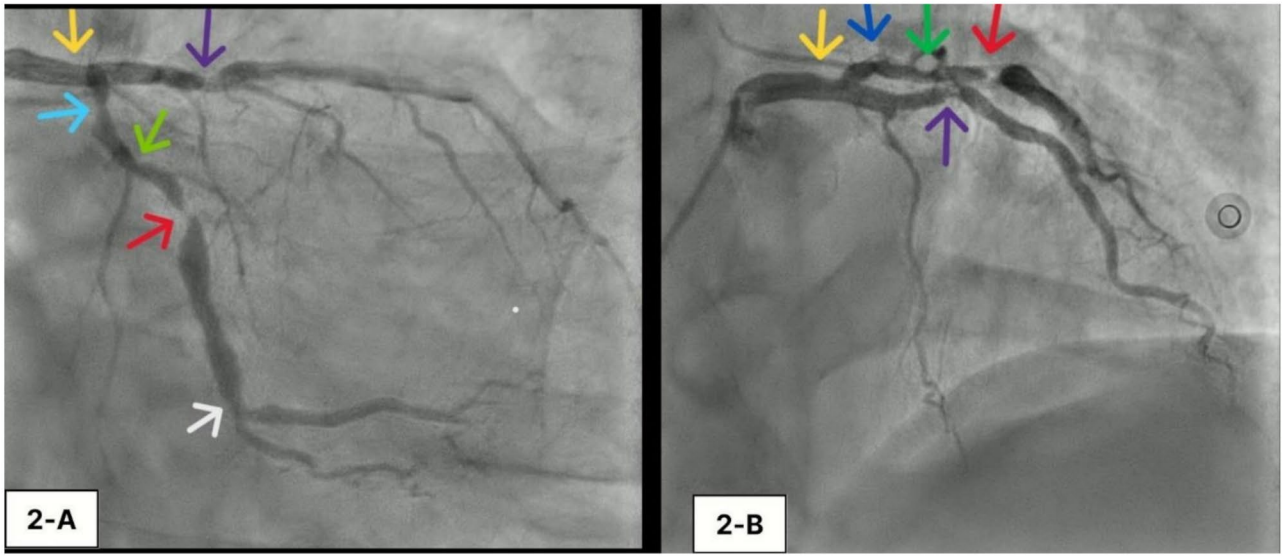

Fig. 2 Right anterior oblique-caudal view, diffuse disease in left main, left anterior descending, anterior descending, left lateral circumflex, yellow: mild stenosis in left main—blue: severe stenosis and at proximal of left lateral circumflex—-green: eccentric plaque with moderate stenosis—-red: severe stenosis at the mid part of left lateral circumflex— white: -moderate stenosis at the distal part of left lateral circumflex. Purple: significant complex lesion at mid of left anterior descending, **B**: Right anterior oblique-cranial, long LM with mild stenosis at distal part(yellow), blue: significant plaque at proximal of left lateral circumflex, green: moderate stenosis at mid part, red: severe stenosis at distal part. Purple: significant complex plaque at mid-part of left anterior descending

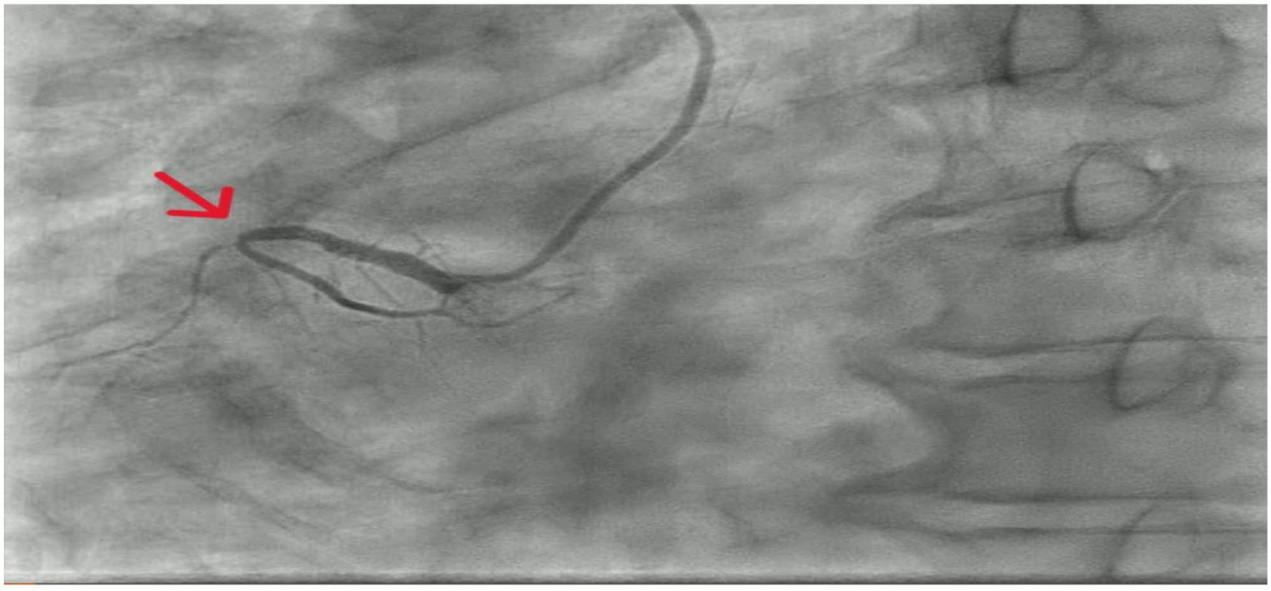

Fig. 3 Coronary angiography of the RCA shows proximal plaques and an abrupt cutoff at the midsection (red arrow), with no antegrade flow

Before discharge, a stress echocardiogram was performed, which showed no evidence of active ischemia. The patient was prescribed inhaler bronchodilators and corticosteroids due to a diagnosis of significant COPD. Furthermore, following a consultation with a pulmonary specialist, the beta-blocker treatment was adjusted to a more specific medication (Bisoprolol 2.5 mg BID) to prevent exacerbation of the respiratory condition.

The patient's condition has stabilized, and both six-month and one-year follow-ups with transthoracic echocardiography (TTE) revealed no significant changes in the patient's clinical or cardiac condition. Furthermore, the patient indicated that they experienced no difficulties carrying out their usual daily tasks.

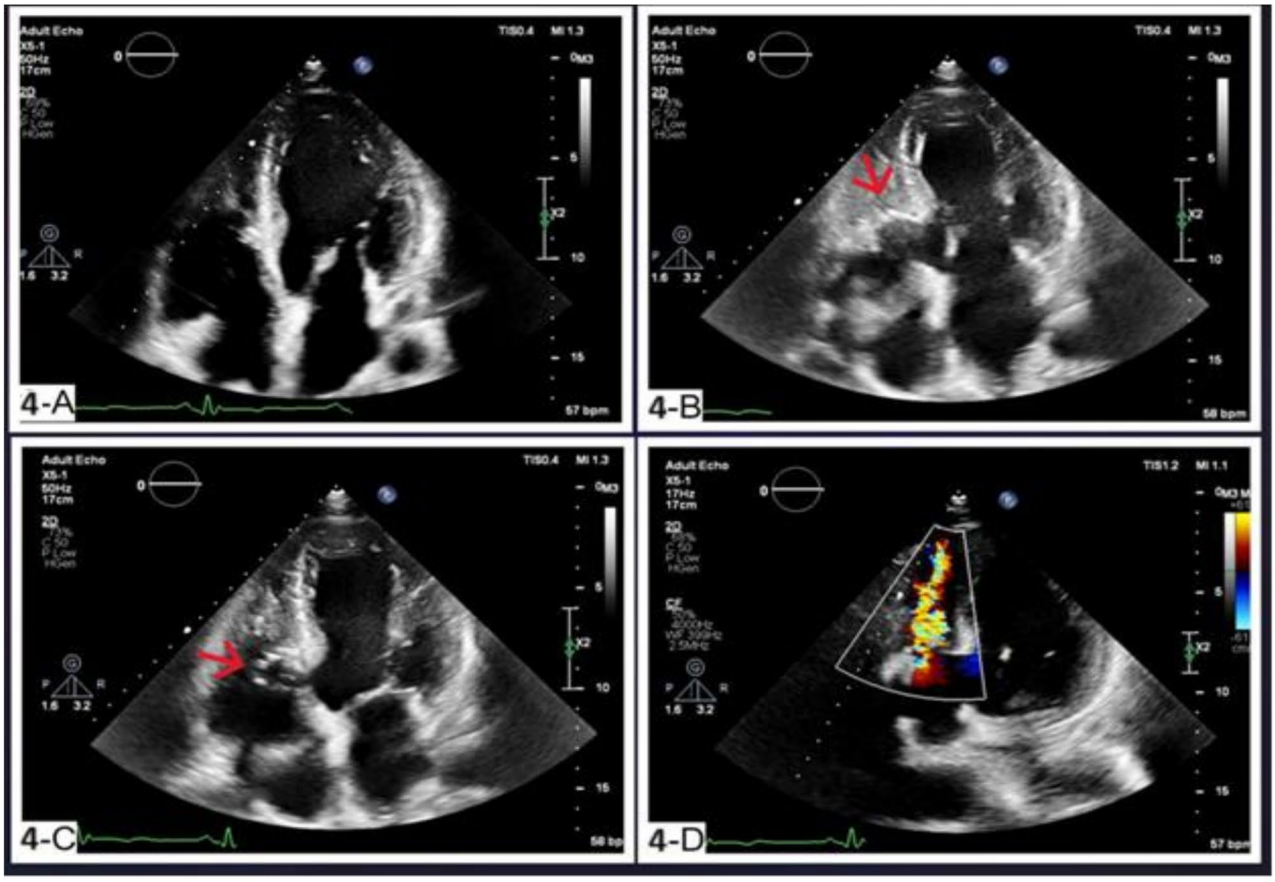

Fig. 4 **A**: The standard apical four-chamber view appears normal with no evidence of any residual defects. **B & C**: A modified apical four-chamber view with posterior tilt shows an aneurysmal formation in the inferoseptum basal segment. A residual defect around the VSD closing patch is visible in systole and diastole. Arrows highlight the defect in systole and diastole, which is more pronounced during systole. The appearance of the finding is similar to a perimembranous VSD, which is detectable by anterior tilt near the aortic valve. **D**: A color study from the modified apical four-chamber view revealed that the left-to-right shunt

Table 2 Echocardiographic findings of the patient

Index	Result
Left ventricle (LV)	Normal LV size and mild systolic dysfunction, ejection fraction (EF): 45%
	Mild Diastolic dysfunction
	Mild septal hypertrophy (LVH)
	Dyskinesia in the base of inferoseptal, small VSD at the basal part of inferoseptal with left to right shunt and peak gradient: 81mmHg, Qp/QS:1.4
Mitral Valve	Mild mitral regurgitation (MR)
	No mitrla stenosis (MS)
Aortic Valve	Trileaflet AV, No aortic stenosis (AS)
	No aortic insufficiency (AI)
Ascending Aorta	Mildly dilated, 3.9 cm
Left atrium	Mildly dilated, LAVI: 37 cc/m2
Right atrium	Dilated (RA area: 22 cm)
Right Ventricle	Mildly dilated (RVEDD:43 mm) with mild dysfunction (TAPSE: 14 mm, Velocity: 8 cm/s, FAC: 32%)
Main pulmonary artery (PA)	dilated (38 mm)
Systolic pulmonary artery pressure (SPAP)	Mildly increased (46 mmHg)
Tricuspid Valve	Moderate TR, no TS

Discussion

This study presents a case that is unique and, more importantly, educational from three aspects: (1) coincidence of AMI with COVID-19 pneumonia, (2) residual PIVSD, which could not be found without attentive evaluation of various views on TTE, which was done three years after Index event and (3) the COVID-19 pandemic heightened anxieties and made medical decision-making significantly more complex, particularly in high-risk cardiac cases, leading to the possibility of suboptimal approaches. Medical professionals should carefully evaluate this group of patients. This report draws attention to the similarities between and also the overlapping symptoms of acute respiratory syndrome and AMI, and the consideration of the different views on TTE to resolve the puzzle of the signs and symptoms.

PIVSD is a severe but rare mechanical complication of myocardial infarction, often linked to delayed MI presentations, cardiogenic shock, and high mortality rates [4, 14]. While its occurrence has declined in the reperfusion era, PIVSD remains a critical complication [15]. The COVID-19 pandemic indirectly contributed to a resurgence in cases by increasing MI risk during acute and post-recovery phases. Additionally, symptoms overlap between COVID-19 and MI, coupled with patients' reluctance to seek timely care, often delaying MI diagnosis and treatment, increasing the likelihood of PIVSD [16, 17]. In this case, COVID-19 symptoms may have concealed the signs of AMI, delayed proper treatment, and allowed a VSD to form. The clinical presentation of PIVSD can vary widely, ranging from an incidental murmur to cardiac shock with circulatory collapse. The left-to-right ventricular-level shunt often (not always) produces a holosystolic murmur along the left parasternal border, underscoring the importance of regular clinical evaluations [18]. In patients with hemodynamic instability that does not align with ventricular dysfunction or ECG findings and enzyme levels, post-MI mechanical complications including PIVSD should be considered [19].

TTE is the primary diagnostic modality and is generally adequate for most cases. However, to confirm the defect's size, location, and hemodynamic impact, it is essential to use color flow Doppler, especially as two-dimensional (2-D) TTE may only reveal subtle septal thinning or abnormal myocardial tissue that can be challenging to detect [20]. In patients who are acutely dyspneic due to experiencing tachypnea and pulmonary edema, the subcostal view is often the best approach [21]. However, all standard and modified views should be assessed to detect probable defects. Additional imaging modalities, such as TEE or ECG-gated cardiac CT, may enhance diagnostic precision [22–24]. Additionally, cardiac MRI also could play a helpful role in decision-making in complex cases [25].

Many factors are associated with poor outcomes in VSD patients and can influence the treatment plan, such as the location and anatomy of the VSD, as well as their gender and age [26, 27]. Anterior PIVSD is generally less complex and typically involves the apical septum. At the same time, PIVSD associated with inferior wall MI is more likely to affect the basal septum or right ventricle, often with a more intricate presentation, such as serpiginous pathways or possible coexisting mitral regurgitation [28]. Additionally, posterior VSD locations pose unique technical challenges during surgery, as proper exposure requires elevating the heart. Important anatomical structures, including the posterior descending artery and the posteromedial papillary muscle, are nearby, necessitating careful surgical planning and execution. These factors complicate surgical repair and can increase the risk of mortality [26, 29].

Furthermore, female and older patients and those with late arrival to the hospital have higher mortality rates [30]. Additionally, there is an ongoing debate about the ideal timing for closure; however, the North American ST-elevation Myocardial Infarction (STEMI) guidelines advocate for emergency surgical repair in all patients, regardless of their hemodynamic status [31]. In contrast, the European acute coronary syndrome guidelines suggest a tailored approach: prompt surgery for patients experiencing refractory shock or persistent right ventricular dysfunction while recommending a delayed intervention for others [11]. Management strategies for PIVSD include medical therapy, surgery, and transcatheter interventions, each selected based on the patient's hemodynamic stability [32]. Initial medical treatment aims to stabilize the patient temporarily. Short-acting inotropic agents like dobutamine or milrinone can enhance cardiac output, while vasopressors like norepinephrine manage hypotension in specific circumstances. In patients with preserved blood pressure, afterload reduction may reduce shunting but carries the risk of hypotension, thus necessitating close hemodynamic monitoring [11, 33]. These therapies are largely temporary, as conservative management yields poor outcomes, with mortality rates exceeding 90% [34].

Based on the ESC 2023, different scientific groups' consensus guidelines state that when the patient is hemodynamically stable, the surgery is performed electively and after a few weeks of continuous monitoring. However, if the patient is not stable, they should first attempt to be stabilized by MSC and medications, and based on the results, the cardiac team will make decisions for the next steps (***Graphical Abstract***, Fig. 5) [11]. The categorizing in this guideline is performed by clinical and hemodynamic evaluation judgment. Patients with small PIVSDs and stable hemodynamics qualify for watchful waiting and continuous monitoring with close hemodynamic

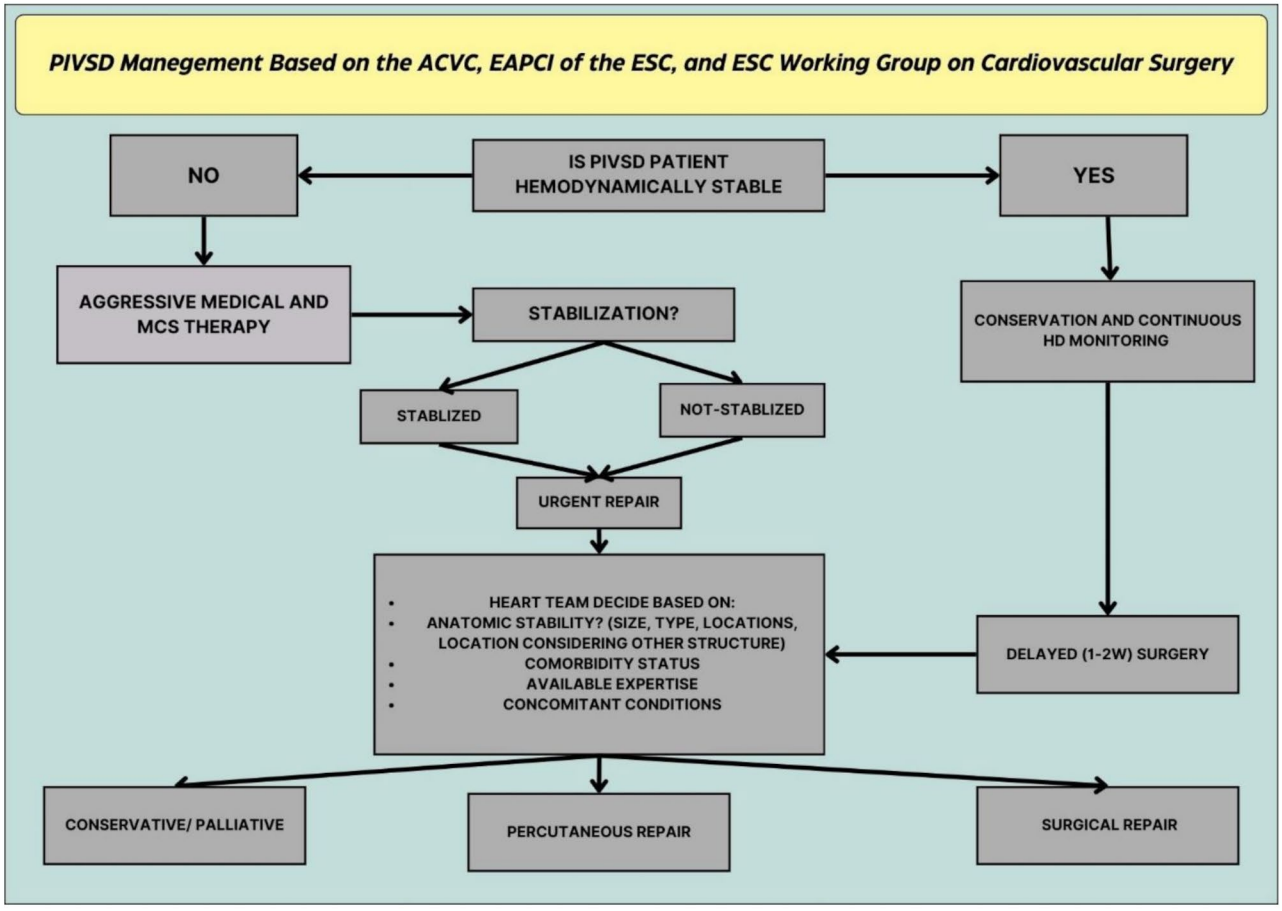

Fig. 5 Recommended protocol by ESC scientific groups Working Group on Cardiovascular Surgery for patients with PIVSD
Abbreviations: ACVC: Association for Acute Cardiovascular Care, EAPCI: European Association of Percutaneous Cardiovascular Interventions, ESC: European Society of Cardiology

tracking until an elective repair after at least 1–2 weeks of medical therapy. However, those with unstable hemodynamic and/or large MI-VSDs with a consecutively high left-to-right shunt require aggressive pharmacologic therapy and possibly MCS treatment. The surgical repair may be delayed if the patient is responsive to treatment. Otherwise, urgent closure within the first few days or as an emergency procedure in cases of refractory cardiogenic shock is recommended [11].

Conclusion

In this context, we emphasize the critical importance of prioritizing patients with myocardial MI or cardiac surgery during the COVID era, as the pandemic has significantly impacted all aspects of healthcare. We highlighted the substantial challenges in managing post-infarction VSDs, particularly regarding the complexity of surgical procedures and the often compromised health status of patients. Effective surgical approaches aim to eliminate abnormal blood flow reliably through various techniques. However, repairing VSDs in areas with limited healthy tissue around the infarcted zone poses significant difficulties, often requiring careful navigation due to the proximity to vital structures such as the tricuspid valve and coronary arteries.

Surgical repair is the cornerstone of PIVSD treatment [35], with patch repair historically reducing mortality compared to medical management [36]. Despite advances in surgical techniques, residual shunts are common and significantly impact long-term outcomes by increasing the risk of heart failure [37]. While the residual shunts have shown to be as high as one-third to half of patients after PIVSD, the development of new techniques has declined this rate to 5–25% of patients following surgical closure detected by intraoperative TEE and TTE [38, 39].

To address the structural complexity of PIVSD, techniques such as infarct exclusion (David technique) that utilizes pericardial or prosthetic material to create a new ventricular septum and infarct excision (Daggett technique), which involves resecting the residual necrotic septal tissue and performing single patch reconstruction of the septum, are frequently employed [40, 41]. In

Table 3 Delayed-diagnosed cases of post-myocardial infarction VSD due to fear of COVID-19 pandemic

First author (YOP)	Number of patients	Presentation	Medical History	Diagnosis	Medical Course and Treatment
Gadre et al. (2021) [49]	1	Worsening back pain	66-year-old woman, with a history of COPD, hypercholesterolemia, and chronic back pain	Inferior STEMI (single vessel disease with RCA occlusion) with no visible VSD at initial evaluation	She became acidotic with respiratory and circulatory failure. Echocardiography revealed a VSD. The family decided to comfort care
Goraya et al. (2021) [50]	1	Worsening SOB and limb edema	53-year-old man, with no known previous disease	Inferior STEMI (multi-vessel disease with RCA occlusion) with a basal inferior wall aneurysm and a small VSD	He underwent removal of mural thrombus, aneurysmectomy, VSD patch repair, and CABG. His hospital course and the 6-month follow-up period were uneventful
Riaz et al. (2023) [51]	1	Acute onset of CP, SOB, and diaphoresis	49-year-old woman, with a history of tobacco use, diabetes, and psychiatric disorders	ST elevations in leads I, II, aVL, and V1-V5 (proximal LAD occlusion) with no visible VSD at initial evaluation	She underwent PCI with DES, but due to the no-reflow phenomenon and hypotension, an IABP was placed. She had a complicated ICU course with COVID-19 pneumonia and cardiogenic shock that was treated appropriately and after improving the symptoms IABP was removed. However, she became hypotensive again and the echocardiography revealed a muscular VSD. She underwent a two-patch technique with bovine pericardium patches for VSD repair that resulted in an improvement of symptoms
Aykent et al. (2022) [52]	1	Worsening SOB and generalized weakness for a week	60-year-old man, with a history of hyperlipidemia and peripheral vascular disease	Inferior STEMI (single vessel disease with RCA occlusion) with large VSD at initial evaluation	An IABP was inserted regarding the management of cardiogenic shock and due to high-risk features, she underwent PCI with DES, and emergent VSD repair was declined. However due to persistent symptoms, ECMO was used and he underwent surgical VSD closure, but unfortunately, 10 days after surgery he expired due to a cardiac arrest
Evans et al. (2022) [53]	1	Worsening SOB and orthopnea	37-year-old man, with a history of hypertension, hyperlipidemia, and hypertriglyceridemia	Anterior STEMI (single vessel disease with LAD occlusion) with large VSD at initial evaluation	He underwent transcatheter VSD closure with an Amplatzer TM post-infarction VSD occlude. The follow-up period was uneventful
Abramo et al. (2022) [54]	7	Acute chest pain	5 men and 2 women with a mean age of 69 (range: 55–78)	Anterior and Posterior STEMI (LAD, LCx, and RCA occlusion). The echocardiographic evaluation demonstrated five posterior and two anterior VSDs.	All patients underwent revascularization (6 PCI and one CABG), surgical VSD repair, and IABP insertion. Most of the patients were discharged uneventfully (unfortunately two patients passed away)
Joshi et al. (2020) [55]	1	Acute onset of CP, lightheadedness, and diaphoresis	72-year-old woman, with a history of hypertension, dyslipidemia, and CAD with prior PCI	Inferior STEMI (single vessel disease with RCA occlusion) with a VSD at initial evaluation	She underwent PCI with DES. Due to poor prognosis and patient wish, VSD surgical repair was declined and she received comfort care.
Okam et al. (2023) [56]	1	Acute onset of CP	92-year-old man, with a history of hypertension, dyslipidemia, CKD, and CAD with prior CABG	Inferior STEMI (RCA occlusion) with second-degree AV block and a VSD at initial evaluation	An IABP was placed for hemodynamic support. However, due to poor prognosis, advanced age, and family decision, VSD surgical repair was declined and he passed away.

Table 3 (continued)

First author (YOP)	Number of patients	Presentation	Medical History	Diagnosis	Medical Course and Treatment
Okam et al. (2023) [56]	1	Acute onset left-sided of CP	62-year-old woman, with a history of hypertension, dyslipidemia, and diabetes.	Inferior STEMI (single vessel disease with RCA occlusion) with no visible VSD at initial evaluation	She underwent PCI with DES. Repeated echocardiography revealed a large VSD in the basal aspect of the inferoseptum. She had a complicated hospital course with tamponade, multiorgan failure, and HIT that was managed appropriately. She underwent a VSD closure using the Amplatzer closure device. Additionally, a right ventricular assist device was inserted on hospital day 15. However, due to acidosis and GIB, she became asystole and passed away
Nasso et al. (2025) [13]	8	Acute onset of CP and SOB	5 men and 3 women with a mean age of 64 (range: 42–79). All patients presented with NYHA class IV	STEMI (LAD, LM, LCx, and RCA occlusion). The echocardiographic evaluation demonstrated seven posterior and one anterior VSD.	All of the patients underwent revascularization (three PCI and six CABG, and one combination of PCI and CABG), surgical VSD repair, and IABP insertion. Additionally, 7 patients required ECMO. Half of the patients were discharged uneventfully (unfortunately four patients passed away)

YOP: year of publication, VSD: ventricular septal defect, COPD: chronic obstructive pulmonary disease, SOB: shortness of breath, CP: chest pain, STEMI: ST-elevation myocardial infarction, IABP: intra-aortic balloon pump, PCI: percutaneous coronary intervention, CABG: coronary artery bypass graft, DES: drug-eluting stent, ECMO: extracorporeal membrane oxygenation, RCA: right coronary artery, LAD: left anterior descending artery, LM: left main, LCx: left circumflex artery, AV: atrioventricular

cases where residual myocardium is sufficient, the David technique's exclusion of necrotic tissue from suture lines offers improved outcomes by reducing the risk of recurrent shunting [42]. In David's repair, the infarcted septum was excluded by a large bovine pericardium patch. There are no unified results in studies that compared these two surgical methods. However, in a prospective study, it has been shown that David is superior in both short- and long-term outcomes compared to the Daggett repair [42].

Another innovative approach is the "triple-patch technique," which offers alternative options for reinforcing the septal repair. This approach aims to prevent suture tension, decrease thrombogenicity, and better preserve ventricular volume remodeling [29] by isolating the defect with multiple patches. Besides surgical management, percutaneous transcatheter techniques for addressing PIVSD have gained increasing acceptance in clinical practice [43]. While challenging, these procedures can offer solutions for anatomically suitable defects. However, this approach may not be amenable to larger or more complex defects, such as those exceeding 35 mm or involving the basal septum near vital structures [44].

During the COVID-19 pandemic, the prevalence of mechanical complications associated with ST-elevation myocardial infarction (STEMI), increased significantly [45]. Indeed, most patients would not come to the hospital for fear of the virus, or logistic difficulties created by lockdowns and healthcare overburdening translated into longer ischemic times with extensive myocardial necrosis and heightened mechanical complications [46]. Tam et al. conducted an observational study that compared the "symptom-to-first medical contact time" before and during the COVID-19 pandemic, revealing that this time was significantly longer during the pandemic, which was associated with poorer patient outcomes [47]. Furthermore, the ischemia injury was exacerbated by the prothrombotic conditions presented by SARS-CoV-2 infection, which raised the likelihood of structural heart damage. Overlapping features of myocardial infarction with clinical manifestations of COVID-19, especially the symptoms of dyspnea, have posed a diagnostic challenge and delayed appropriate intervention [48]. Table 3 represents the delayed diagnosed cases of PIVSD during the COVID-19 era due to fear of exposure to COVID in healthcare settings.

Abbreviations

CVD	Cardiovascular diseases
MI	Myocardial infarction
AMI	Acute myocardial infarction
VSD	Ventricular septal defect
PIVSD	Post-infarction ventricular septal defect
TTE	Transthoracic echocardiogram
2-D	Bi-dimensional
ECG	Electrocardiogram
CABG	Coronary artery bypass graft
3VD	Three-vessel coronary artery disease

COPD	Chronic obstructive pulmonary disease
EF	Ejection fraction
CAG	Coronary angiography
ACVC	Association for Acute Cardiovascular Care,
EAPCI	European Association of Percutaneous Cardiovascular Interventions,
ESC	European Society of Cardiology
RCA	Right coronary artery
STEMI	ST-elevation Myocardial Infarction

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TA, EF, AN, PR, and PE contributed to conceptualization, data curation, formal analysis, project administration, resources, supervision, writing– original draft, conceptualization, data curation, formal analysis, project administration, resources, supervision, writing– original draft. FB, MJ, MK, EF, and AB contributed to supervision, visualization, writing– original draft, writing– review, and editing. All authors have read and evaluated the manuscript and approved it for submission.

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Data availability
Further information will be provided by the corresponding author upon reasonable request due to confidentiality/privacy.

Declarations

Ethical approval
Not applicable.

Consent for publication
The patient provided informed consent for the publication of this report, and the center's ethical policy performed the procedure.

Consent to participate
The patient provided written informed consent to participate in this clinical case report, ensuring that all personal information and medical data will be kept confidential and used solely for research purposes.

Competing interests
The authors declare no competing interests.

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