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A Perfect Storm: Navigating the Complexities of Post-myocardial Infarction Ventricular Septal Defect in Metabolic Syndrome

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Abstract

The co-occurrence of rare and Lethal complications of myocardial infarction (MI) in metabolic syndrome (MetS). A 70-year-old man with hypertension and diabetes presented to our emergency department with a 3-day history of sudden onset of severe chest pain that is associated with diaphoresis, shortness of breath, feeling of impending doom, nausea, and unrecordable blood sugar on admission.

Meticulous physical examinations revealed pansystolic murmur loudest at left sternal edge, loud pulmonary component of second heart sound (P2), bi-basal coarse crepitation. While focus investigations revealed unrecordable random blood sugar (RBS), electrocardiography (ECG) significant ST segment elevation in leads II, III and aVF, elevated cardiac markers and NT pro BNP, echocardiography established ventricular septal defect, and derange urea, electrolyte and creatinine. A definitive diagnosis of inferior wall ST-elevation myocardial infarction (STEMI) complicated by acute heart failure (HF), ventricular septal defect (VSD), cardiogenic shock (CS), and cardiorenal syndrome (CRS) with co-occurring hyperosmolar hyperglycemic state (HHS) in MetS was established. He was stabilized with appropriated medical therapy and referred to heart surgery capable hospital.

This case underscores the potential for severe, life-threatening complications of myocardial infarction in metabolic syndrome particularly in resource constrained settings. Early recognition and timely referral to cardiac surgery capable centre are crucial for improving outcomes in such cases, especially in resource-limited settings.

Keywords: Post infarction ventricular septal defect; Heart failure; Cardiogenic shock; Cardiorenal syndrome; Metabolic syndrome

Introduction

Myocardial infarction (MI) can lead to life-threatening complications like HF, VSD, CS, CRS [1] and co-occurring HHS. These complications are particularly serious in patients with metabolic syndrome and can be difficult to diagnose and treat, especially in resource-limited settings. The rising prevalence of hypertension, diabetes, and metabolic syndrome in sub-Saharan Africa, coupled with the adoption of Western lifestyles, is increasing the incidence of acute coronary syndrome (MI) [2,3]. This trend is likely to lead to more cases of MI with severe complications like ventricular septal defect (VSD), which may be misdiagnosed and mismanaged due to limited resources and expertise in the region.

Post-MI VSD is a serious complication that can occur few days after interventricular septal transmural ischemic necrosis. This life-threatening complication often develops within days of MI and can significantly worsen patient outcomes. Without surgical intervention, the mortality rate can be as high as 94%, while with surgical intervention is about 47% during the first 30 days of infarction [4]. In resource-limited settings, mortality rates may be even higher.

Several risk factors influenced the development of post-MI VSD which include. Gender (women), older individuals, and those with history of hypertension in 90% of cases are more susceptible [5,6]. Anterior wall myocardial infarction [5,6], and worse MI Killip class at presentation also increase the risk [5]. Delayed treatment and a lack of previous MI history are additional factors [6]. Prompt recognition and treatment of MI are crucial to minimize the risk of post-MI VSD and its associated complications.

The pathophysiologic mechanisms of post-MI VSD include occlusion of coronary artery supplying the interventricular septum leading to transmural ischemic necrosis. The weakened, necrotic septum ruptures under the pressure of the left ventricle, creating a VSD that allows blood to flow from the left to the right ventricle. This abnormal blood flow leads to right ventricular enlargement, reduced cardiac output, pulmonary hypertension, pulmonary edema, and potentially, cardiogenic shock.

Post-MI VSD can present with severe complications like HF, CS, CRS, life-threatening arrhythmias, and even death. In this regard, early diagnosis and treatment are crucial to improve outcomes. The management of this catastrophic complication involves immediate medical attention. Early surgical intervention is the primary treatment to restore hemodynamics and has better outcomes compared to delayed intervention [1,7,8], although device closure is an emerging option [9]. Timely treatment is crucial to stabilize the patient and prevent further complications.

People with metabolic syndrome (MetS) are at an increased risk of MI due to several interrelated factors. MetS is characterized by a cluster of conditions, including high blood pressure, obesity, abnormal cholesterol levels, and insulin resistance. These factors contribute to the development of MI [10]. On the other hand, MetS was also documented to be a prothrombotic, procoagulant and hypofibrinolytic states leading to cytokine dysregulation triggering chronic tissue and organ-specific inflammation and in the vascular system leading to endothelial dysfunction, vascular remodeling and thrombosis [11].

The association of MetS components and underlying pathophysiologic insulin resistance, chronic inflammation, endothelial dysfunction, and increased thrombogenicity contribute to the atherosclerotic process, leading to an increased risk of acute coronary events [12,13]. Metabolic syndrome patients with excess body weight particularly central obesity have higher risk of MI and cardiovascular events [12], Furthermore, individuals with MetS who experience MI often have larger areas of heart muscle damage and a higher risk of complications compared to those without MetS. In summary, the combination of risk factors associated with MetS increases the likelihood of MI and worsens its outcomes, particularly in individuals with central obesity. We present a unique case of inferior wall STEMI with catastrophic complications HF, post-MI VSD, CS, CRS and HHS in MetS with diagnostic and management challenges in resource constrained settings of sub-Saharan Africa, successfully stabilized with medical therapy and referred to heart surgery capable hospital.

Case Presentation

A 70-year-old man with hypertension and diabetes diagnosed 15 years ago but not regular with medication and clinic visit presented to our emergency department with a 3-day history of sudden onset of severe chest pain with associated diaphoresis, shortness of breath, feeling of impending doom and nausea. He had past history suggestive of recurrent unstable angina (stable angina) 10 days prior to presentation where he was seen at peripheral hospital with diagnosis of acid peptic disease and administered anti acid peptic disease medications with no improvement. He neither smoke no take alcoholic beverages and cannot ascertained family history of hypertension and diabetes.

Physical examination findings at presentation revealed respiratory rate (RR) of 26/minute, SPO2 85% at room air, anicteric, acyanosed, afebrile (T=37.2oC), tachycardia with pulse rate (PR) of 136/minute, blood pressure (BP) 115/75mmHg, Heart sounds (HS) were S1, S2, pansystolic murmur, Chest: bi-basal coarse crepitation middle to lower zone bilaterally, Abdominal and neurological examination were unremarkable. Admitting random blood sugar was unrecordably high. A provisional diagnosis of acute coronary syndrome ACS in Killip class III with co-occurring HHS on background MetS was entertained with differential diagnosis of pulmonary embolism (PE). He was administered Aspirin, Clopidogrel, Clexane, Rosuvastatin, insulin therapy, frusemide, and limited intravenous fluid normal saline and was transferred to intensive care unit (ICU). Relevant laboratory investigations were requested as indicated in (Table 1 and 2).

Table 1: Showing relevant laboratory results at early hospitalization.

Laboratory test	**Result**	**Reference range**
White blood cell count (WBC)	13.33 x 10^9/L	4-10 x 10^9/L
Neutrophils	10. x 10^9/L	2-7 x 10^9/L
Lymphocytes	2. 37 x 10^9/µL	0.8-4 x10^9/L
MXD	0.96 x 10^9/µL	0.1-1 x 10^9/L
Hemoglobin	10. g/dL	11-17 g/dL
Platelets count	183 x 10^9/µL	100-300 x 10^9/L
Sodium	129 mmol/L	134-149 mmol/L
Potassium	4.5 mmol/L	3.5-5.2 mmol/L
Chloride	87.0 mmol/L	96-106 mmol/L
Urea	18.5 mmol/L	2.5-6.5 mmol/L
Creatinine	2.1 mg/dL	0.6-1.3 mg/dL

Table 2: Showing further relevant laboratory results.

Laboratory test	Result	Reference range
D-dimer	0.8 mg/L	<0.5 mg/L
Cardiac Troponin T	7.221 ng/mL	0.00-0.30 ng/mL
NT Pro-BNP	10524.2 pg/mL	<300 pg/mL
Random blood sugar	Unrecordably high	mmol/L
PTTK	33sec	24-38sec
PT	13sec	10-15sec
Total cholesterol	97 mg/dL	<200 mg/dL
Triglyceride	170 mg/dL	<150 mg/dL
HDL-Cholesterol	6 mg/dL	35-55 mg/dL
LDL-Cholesterol	57 mg/dL	<150 mg/dL

Urinalysis revealed protein + and hematuria ++ and calculated admitting plasma osmolality was 317.5mosmol in keeping with HHS. While relevant requested imaging investigations include: Electrocardiogram (ECG) revealed sinus tachycardia with heart rate of 137 beat per minute, significant ST elevation lead II, III, aVF, prolong QTc, lateral leads ST-T abnormality (Figure 1).

24 hours after admission examination further review revealed patient tachypneic RR= 30/m, SPO2 85% at room air, flapping tremor, PR=70/min small volume, BP: 79/49mmHg, Heart sound was soft S1, S2, loud P2, pansystolic murmur loudest at left sternal edge grade 3/6 non-radiating, bi-basal fine crackles. A, provisional diagnosis of inferior wall STEMI complicated by HF (Killip class IV), post-MI VSD, CS, CRS and co-occurring HHS in MetS was entertained with differential diagnosis of post-MI papillary muscle rupture (PMR). Vasopressor (dobutamine) support commence, and other line of management sustained along with conservative renal therapy.

Transthoracic echocardiography (TTE): Revealed thickened interventricular septal thickness in diastole (IVSTd) of 12.2 mm, left ventricular posterior wall thickness in diastole of LVPWTd of 13 .0 mm, dilated left atrium with internal diameter of 41.9 mm, normal left ventricular (LV) internal diameter of 51.4 mm, concentric LV wall hypertrophy, normal LV systolic function with EF of 74.75%, heart valves appears normal in motion/morphology, normal right atrial and ventricular internal diameter, normal night ventricular (RV) systolic function with tricuspid annular plane systolic excursion (TAPSE) of 19.0 mm. 2-Dimension (2-D) echocardiogram parasternal short axis view demonstrated echo drop out along interventricular septum measured 18.6.mm in size in keeping with VSD (Figure 2 and 3) and color Doppler demonstrated left to right shunt across interventricular septum in apical 4-chamber view in keeping with VSD (Figure 4), and 2-Dimension (2-D) video (Video 1 and 2) demonstrating VSD in parasternal short axis view, while video (Video 3) Color Doppler demonstrating left to right shunt across VSD in apical 4-chamber view.

Mild mitral regurgitation (MR), pulmonary regurgitation (PR) and moderate tricuspid regurgitation (TR), moderate pulmonary arterial hypertension [pulmonary arterial systolic pressure (PASP) of 44 mmHg at rest], Grade 1 LV diastolic dysfunction.

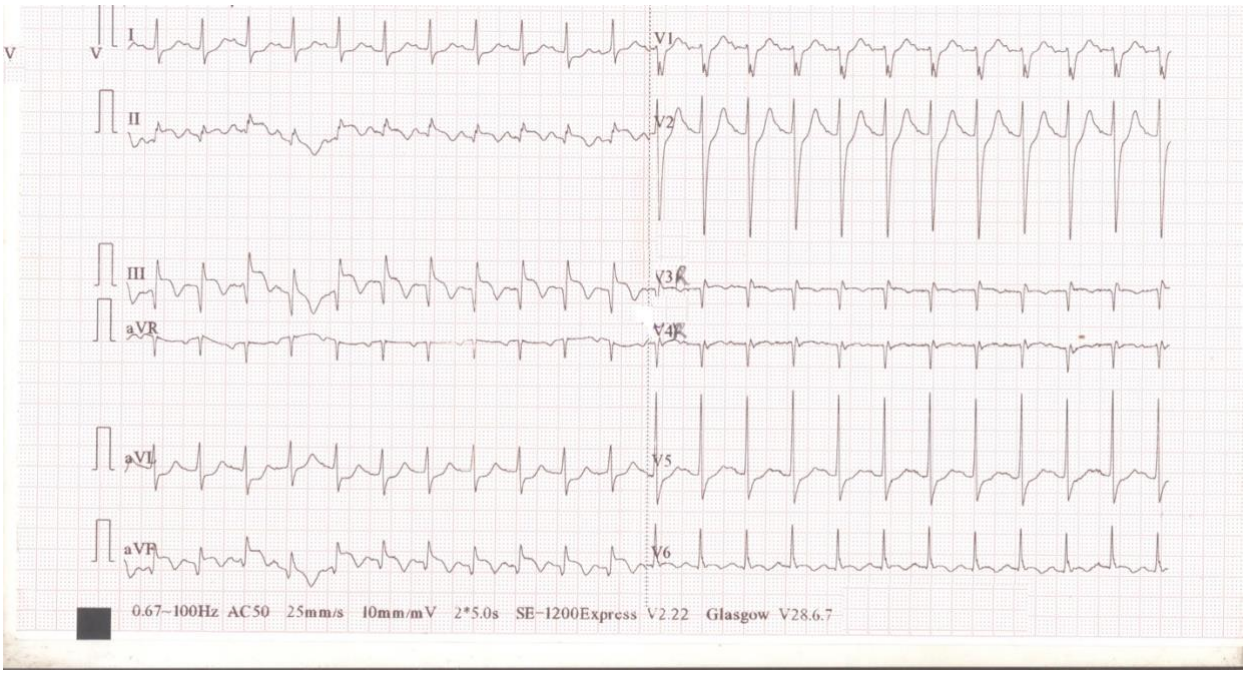

Figure 1: ECG showing sinus tachycardia, significant ST elevation in leads II, III, aVF and lateral ST-T abnormality, prolonged QTc.

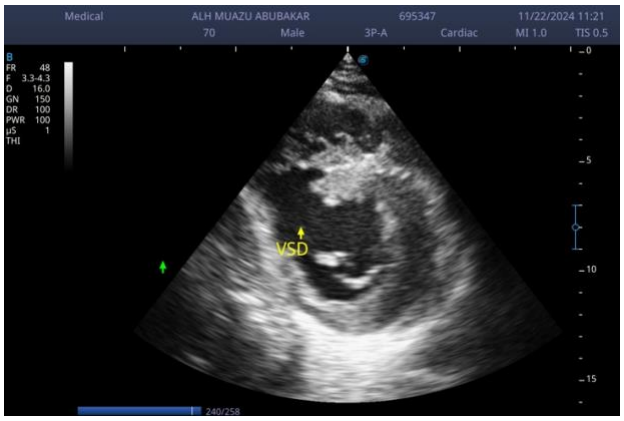

Figure 2: Parasternal short axis view showed echo drop out along interventricular septum in keeping with acquired ventricular septal defect (VSD).

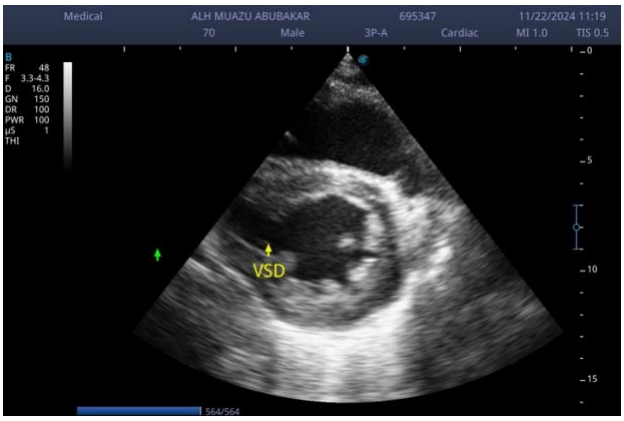

Figure 3: Second parasternal short axis view also showed post-MI ventricular septal defect (VSD) along the interventricular septum.

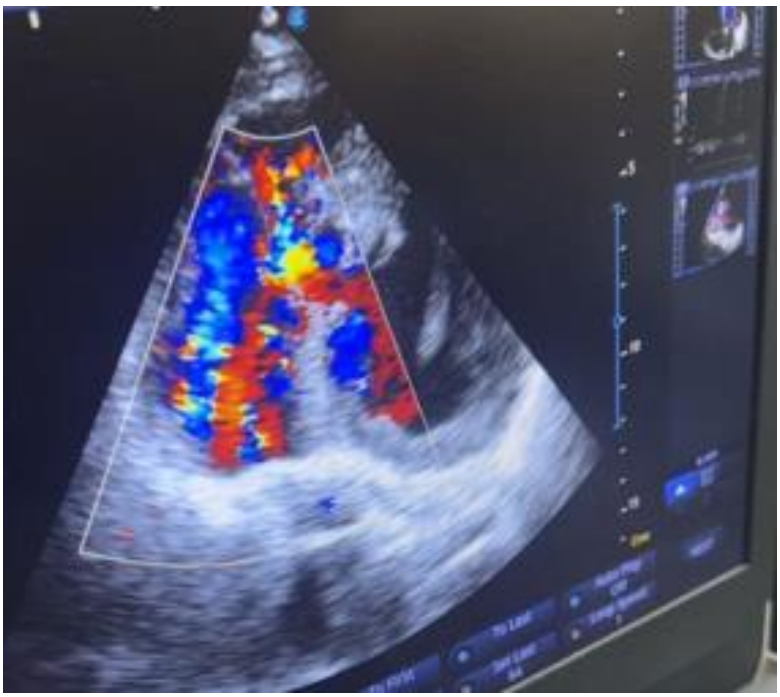

Figure 4: Showed color Doppler showed left to right shunt across post-myocardial infarction ventricular septal defect.

Video Legends

Video 1: Showing echo drop out across interventricular septum in keeping with post-MI ventricular septal defect in parasternal short axis view.

Video 2: Second video also showing echo drop out across interventricular septum in keeping with post-MI ventricular septal defect in parasternal short axis view.

Video 3: Color Doppler demonstrating left to right shunt across ventricular septal defect.

Note: Videos related to this article can be found online at: https://www.literaturepublishers.org/archive/A-Perfect-Storm:-Navigating-the-Complexities-of-Post-myocardial-Infarction-Ventricular-Septal-Defect-in-Metabolic-Syndrome.html

Further clinical evaluation revealed weight 74kg, height 164cm, body mass index (BMI) of 27.6 and waist circumference of 109 cm. A definitive diagnosis of inferior wall STEMI complicated by HF, post-MI VSD, CS, CRS type 1 and co-occurring HHS was established. He was stabilized with appropriate medical management with improve renal and glycemic control and referred to heart surgery capable hospital. After 6 days of hospital stay.

Discussion

The combination of acute HF, post-MI VSD, CS, and CRS occurs rarely in metabolic syndrome. This complex clinical scenario presents significant diagnostic and treatment challenges, particularly in resource-limited settings. While there are a few reported cases of post-MI VSD with severe complications from other regions of the world, such cases are virtually absent in sub-Saharan Africa, despite the increasing prevalence of MI [2,3]. This scarcity may be due to inadequate specialist care and diagnostic tools, leading to missed diagnoses or early mortality. Additionally, underreporting of such cases may contribute to this lack of data.
In sub-Saharan Africa Yonga et al. [14], reported an interesting case study of post infarction VSD in Nairobi Kenya which is probably the first published case study. Yonga et al. [14]. This case study differs from our index case in several ways (1) Our index case was elderly male while Yonga et al. case was middle-aged female; (2) Unlike Yonga et al. [14] case this index case presented with catastrophic complications (HF, CS, CRS), and co-occurring HHS. Yonga et al. case presented with stable hemodynamics 3weeks after MI unlike our index case that presented to hospital 3 days after symptoms onset and develop hemodynamic instability 24hours after admission; (3) Our index case has established MetS unlike case by Yonga et al. [14], that has no established MetS.

In literature several risk factors have been linked to post- MI VSD. In our case, the following risk factors were identified: advanced age [6], a history of hypertension [6], absence of prior MI [6], delayed treatment intervention beyond 24 hours [6], and a severe clinical presentation (Killip class III and IV) [5]. Diagnosing post-MI VSD in sub-Saharan Africa is challenging due to limited resources and expertise. Despite these limitations, our case highlights several key points. The patient's underlying metabolic syndrome, initial misdiagnosis as acid peptic disease, inferior wall MI, co-occurrence of catastrophic complications and delayed presentation underscores the need for heightened clinical suspicion. The pansystolic murmur loudest in left sternal edge, pulmonary edema, and cardiogenic shock were crucial physical findings pointing to post-MI VSD. Clinicians in resource-limited settings should maintain a high index of suspicion for post-MI VSD in patients with risk factors for MI and post-MI VSD, acute MI symptoms, and suggestive physical examination findings. Early recognition and timely referral to cardiac surgery capable centers are crucial for optimal patient outcomes.

While pulmonary embolism was considered a strong differential diagnosis due to the patient's presenting symptoms and underlying risk factors, several findings were against this diagnosis. These included a pansystolic murmur loudest at left sternal edge, ECG findings consistent with inferior wall MI, echocardiographic evidence of post-MI VSD, identifiable risk factors for MI and post-MI VSD, elevated cardiac markers, and NT Pro BNP with a near normal D-dimer.

Post-myocardial infarction papillary muscle rupture (PMR) was also considered a strong differential diagnosis due to the patient's presentation, including classic symptoms suggestive of acute MI, symptoms suggestive of acute heart failure at presentation and a timeframe consistent with PMR development [15]. The physical examination findings in keeping with acute HF (cardiogenic pulmonary edema), systolic murmur and blood pressure in keeping with shock, all were observed 3-4 days after onset of classic symptoms suggestive of MI in this index case. However, several factors argued against this diagnosis. The presence of a pansystolic murmur loudest at the left sternal edge, in contrast to post-MI PMR systolic murmur that usually present as early systolic murmur or pansystolic murmur loudest at apex or inaudible murmur in severe LV systolic dysfunction [15]. Additionally, the development of cardiogenic shock a day after admission this is usually in contrast to post-MI PMR that present with flash pulmonary edema with concurrent hypotension or cardiogenic shock usually observed 3-7 days after the onset of MI symptoms [15], and the echocardiographic findings of VSD with mild mitral regurgitation were inconsistent with the typical presentation of PMR, which often present with severe mitral regurgitation and immediate or early cardiogenic shock [15]. The case faced one limitation in it management that include (1) Patient could not have coronary angiography due to non-availability.

This case is unique due to its rare presentation of inferior wall STEMI with catastrophic complications (acute HF, post-MI VSD, HHS, CS, and CRS). These complications often occur in resource-limited settings, where misdiagnosis is common due to limited specialist, diagnostic capabilities, and healthcare costs. Despite these challenges, our patient achieved a favorable outcome. To the best of our knowledge, there is no reported case of post-MI VSD with such severe complications in sub-Saharan Africa. To sum up, this case highlights the critical role of a high index of suspicion, thorough history-taking, meticulous physical examination, and focused investigations (ECG, cardiac markers, and echocardiography) in promptly diagnosing post-MI VSD in resource-limited settings. Patients presenting with symptoms suggestive of MI and examination finding of new onset pansystolic murmur loudest at the left sternal edge should be carefully evaluated for post-MI VSD and make timely referral to a cardiac surgery capable center, can significantly improve patient outcomes.

Conclusions

This case report highlights the rare and severe complications of inferior wall STEMI (acute HF, post-MI VSD, CS, and CRS) with co-occurring HHS in metabolic syndrome. These complications can pose significant diagnostic and treatment challenges, particularly in resource-limited settings. The high risk of MI with catastrophic complications in MetS can be attributed to hypercoagulable, prothrombotic, procoagulant, and hypofibrinolytic states associated with MetS, combined with traditional cardiometabolic and post-MI VSD risk factors. To improve outcomes in post-MI VSD and its associated catastrophic complications in resource constrained settings. Timely diagnosis, medical management and prompt referral to a cardiac surgery centre are essentials. This can only be achieved when clinicians have a high index of suspicion, conduct thorough history-taking, perform meticulous physical examinations and clinical reasoning, and utilize focused investigations on any patient presenting with classic symptoms suggestive of MI with examination findings of new onset systolic murmur.

Learning Points

(1) To understand the pathophysiology of post-MI VSD in MetS.

(2) To understand the spectrum of symptoms, physical examination, hemodynamic, ECG and echocardiographic and laboratory findings of post-MI VSD.

(3) The need for clinician of resource constrained setting to have high index of suspicion for post-MI VSD in any patient with metabolic syndrome who presented with classic symptoms suggestive of MI with examination finding of new onset systolic murmur.

(4) To understand the importance of thorough focused history taking, meticulous physical examinations, focused investigations, clinical and reasoning in the unravelling post-MI VSD.

(5) To understand the need for prompt recognition and timely referral of post-MI VSD to heart surgery capable hospital in resource constrained settings.

Authors Contributions

(I) Conception and design: HU, OOI, NMI, RC, AH, AM, AJB; (II) Administrative support: HU, AM, AJB; (III) Provision of study material or patient: HU, OOI, RC, NMI, AJB, AM, HA; (IV) Collection and assembly of data: All the authors (V) Data analysis and interpretation: All the authors U, (VI) Manuscript writing: All the authors (VII) Final approval of manuscript: All the authors.

Abbreviations

MI: Myocardial infarction; VSD: Ventricular septal defect; STEMI: ST-elevation myocardial infarction; MetS: Metabolic syndrome; PMR: Papillary muscle rupture; HF: Heart failure; CS: Cardiogenic shock; CRS: Cardiorenal syndrome; HHS: Hyperosmolar hyperglycemic state; PE: Pulmonary embolism

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