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Case Report

Acute Myocardial Infarction with Multiple Perforations in a "Swiss-Cheese" Pattern

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Abstract

Introduction: To present a rare occurrence of "Swiss-cheese" defects of left ventricle in acute myocardial infarction. **Case Report:** A 64-year-old male with persistent ST segment elevation in anterior and inferior leads developed sudden deterioration 2 days after thrombolysis. Echocardiography revealed ventricular septal and LV (left ventricular) free wall ruptures at multiple sites with contractile dysfunction and the patient died suddenly followed by an episode of ventricular tachycardia. Discussion: Myocardial rupture may complicate in 10% of acute myocardial infarctions and it is the second most common cause of in-hospital mortality next to pump failure. It is responsible for 15% of in-hospital deaths and 50% die within 5 days and 82% die within two weeks of index infarction. Conclusion: Aggressive early diagnosis and surgery may confer a survival rate as high as 75%. The prognosis is grave in patients presented with cardiogenic shock and multiorgan dysfunction, surgery is best avoided and supportive medical therapy may be adequate in such cases.

Keywords: "swiss-cheese" left ventricle; ventricular septal rupture (VSR); LV free wall rupture; ventricular tachycardia; cardiogenic shock

Introduction

Left ventricular wall comprises three layers, superficial (subepicardial), middle, and deep (subendocardial) and the longitudinal alignment of myocardial strands of one layer interconnect with strands of next layer in continuum, not separated by cleavage planes or sheets of fibrous tissue. The superficial layer occupies approximately 25% of wall thickness and these oblique strands are in continuity with longitudinal strands of deeper layer at the base and apex of the ventricle, which constitutes < 20% of wall thickness. The middle layer consists of circumferential strands and comprises 53-59% of ventricular wall thickness [1]. It is thickest around the base, encircling the inlet and outlet portions and thinning out towards the cardiac apex. The middle layer of the left ventricle occupies the major portion of the myoarchitecture of the ventricular septum which forms the parietal wall of both right and left ventricle and it is lacking at the apical portion of the septum. The bulk of the myocardium is formed by the contractile cardiac myocytes. The endomysium, a network of fibrocollagenous interstitial connective tissue surrounds each myocyte and provide a supportive framework. The perimysium, a network of thicker interstitial connective tissue surrounds group of myocytes and prevent malalignment between bundles. The ends of each myocyte branches and adjoin adjacent myocytes to produce a complex three-dimensional network, the "syncytium". After acute myocardial infarction, the myocytes develop a typical wavy appearance and exhibit cytoplasmic hypereosinophilia and nuclear pyknosis. 8 hours later, interstitial edema and neutrophilic infiltration occur and after 24 hours, cross-striations are lost and focal hyalinization appear. Collagenae activity appears in the 2nd day and peaks at 7th day of infarction, leading to collagen degradation. New collagen fibers (type III early and type I later) appear on the day of 14. Removal of necrotic debris starts within 4 days of infarction, complete after 4-6 weeks and replaced by scar tissue. Myocardial rupture is a laceration or tearing of the ventricular wall and most commonly occurs in the setting of acute myocardial infarction. Ischemic myocardial rupture occurs between the time of collagen degradation and the laying down of new fibrous tissue (2-14 days) and typically seen between 3 to 5 days after infarction [2]. During rupture, increased number of leukocytes, collagenases (matrix metalloproteinases) and intramyocardial hemorrhage occur at the site rupture. Myocardium regains its normal biomechanical strength after 7 days [3] and the incidence of rupture is highest in the first 7 days following myocardial infarction [4]. Myocardial rupture can be classified into 3 types [5] by Becker and van Mantgem as given in Table 1.

Type I	Abrupt slit-like tear that generally occurs within 24 hours of an acute myocardial infarction (without thinning)
Type II	Erosion of the infarcted myocardium, which is suggestive of a slow tear of the dead myocardium and typically occurs more than 24 hours after the infarction. The infarcted myocardium erodes before rupture and is covered by a thrombus
Type III	Early aneurysm formation and subsequent rupture. I.e., perforation of a previously formed aneurysm.

Table 1: (Becker and van Mantgem classification of myocardial rupture).

Morphologically, four types of myocardial rupture have been described [6] as given in Table 2.

Type I	Little dissection or infiltration of the myocardium (direct rupture)
Type II	Multicanalicular trajectory with extensive myocardial dissection
Type III	Rupture is protected either by a thrombus at the orifice on the ventricular side or by a pericardial adhesion (pericardial symphysis)
Type IV	Incomplete as the trajectory does not traverse through all layers (epicardial, endocardial, or intramyocardial rupture)

Table 2: (Morphological types of myocardial rupture).

Kumar, et al [7] reported cardiac rupture in Takotsubo cardiomyopathy, a reversible disease often triggered by acute emotional or physical stress, characterized by ECG changes mimicking acute myocardial infarction and acute complications such as cardiogenic shock occurs in 50 % of cases [8]. This can lead to left ventricular rupture and recent studies reported right ventricular involvement in 28-50% [9],[10], the friable right ventricle susceptible to rupture due to mechanical wall stress in biventricular Takotsubo cardiomyopathy [11]. Isolated RV free wall rupture as a complication of inferior wall and right ventricular myocardial infarction is an uncommon finding in transthoracic echocardiographic examination [12] and it was reported in Figures 15 to 20.

Review of literature: Historically, the first clinical reference to post-infarction left ventricular wall rupture was reported by William Harvey in 1647 [13]. London and London in an analysis of 1000 cases of fatal myocardial infarction found that 50% of ruptures occurred within 3 days and 89% within 14 days [14]. They reported that repeated and prolonged chest pain occurring in 55% of patients with cardiac rupture due to slow leakage of blood into the pericardial space prior to complete rupture and only in 10% of cases without rupture. An accumulation of 75 ml of blood is sufficient to produce cardiac tamponade and death in acute ruptures. The anterior wall of left ventricle is involved more commonly than the posterior wall [15]. Von Torsel and Edwards suggest that symptomatology was consistent with a gradual evolution of cardiac rupture in reviewing 40 cases [16]. Leutsch and Lanks demonstrated that the rupture was progressive rather than abrupt by finding an organizing thrombus at the site of rupture [17]. Common sites of myocardial rupture are anterior or lateral walls and a mid ventricular position along the apex to base axis is most frequent (66%). Blow-out and ooze ruptures are the other pathological variations sometimes used. Blow-out ruptures present as macroscopic tear in the epicardium and the communication between the LV cavity and pericardial space is observed. In oozing ruptures, no macroscopic defects are visible [18] Multiple sites of rupture in the left ventricular myocardium including the interventricular septum following an acute myocardial infarction is uncommon and so this case had been reported.

2.Case Report: A 64-year old hypertensive, non-smoker male was brought to the emergency room with shortness of breath and hypotension. He was drowsy and extremities were cold and clammy. He had a history of out of hospital thrombolysis with streptokinase 2 days before for a sudden onset of chest pain and elevated cardiac enzymes (Troponins and CK-MB). His pulse was feeble and blood pressure not recordable. Auscultation revealed a grade 4/6 loud, harsh systolic murmur with a palpable thrill over the left sternal border and apex, not conducted to axilla and back. Basal crackles were present over the lung fields. Renal and liver parameters were normal. ECG revealed a persistent ST segment elevation simultaneously seen in anterior and inferior leads as shown in Figure 1. He was in cardiogenic shock and supported with intravenous fluids and inotropic agents. He subsequently developed an episode of ventricular tachycardia, triggered by inotropic agents and hypotension (Left posterior septal origin as evidenced by RBBB (right bundle branch block) morphology in V1 with right axis and changing contour of QRS complexes as RBBB morphology in V1, V2 and LBBB (left bundle branch block) morphology V4, V5 and negative deflection in V6 as shown in Figure 2) and it was not responded to intravenous amiodarone and cardioversion, the patient's condition deteriorated and died suddenly despite resuscitative measures. Emergency Transthoracic 2D echocardiography revealed an anteroapical LV (left ventricular) aneurysm with multiple perforations of the interventricular septum and LV free wall as shown in Figures 3 to 14 with contractile dysfunction.

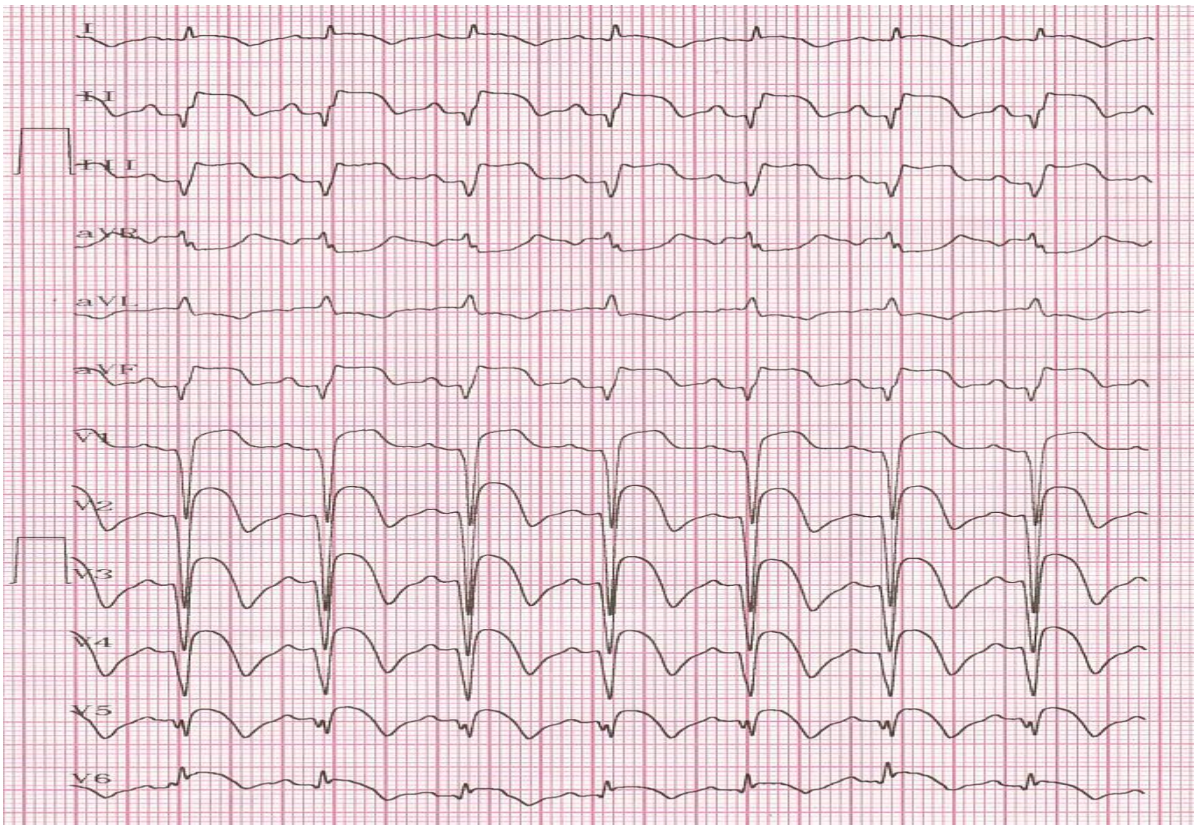

Figure 1: ECG (2 days after thrombolysis) showing persistent ST segment elevation in anterior and inferior leads (wraparound LAD (left anterior descending coronary artery) occlusion.

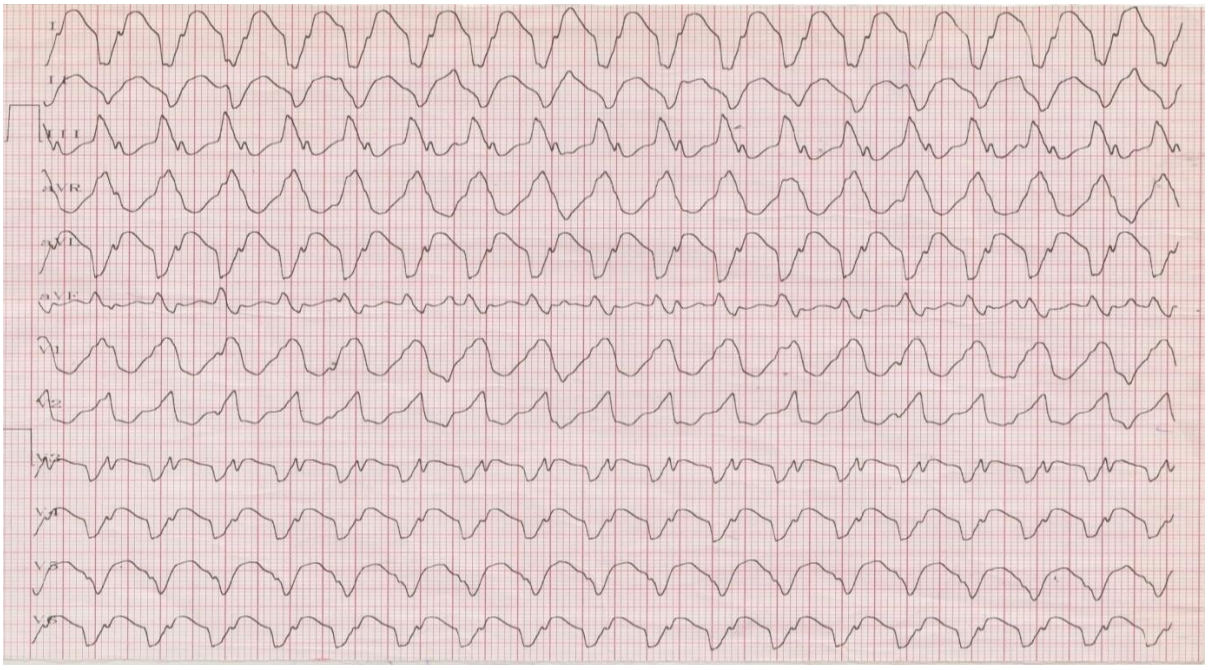

Figure 2: ECG showing ventricular tachycardia of left posterior septal origin.

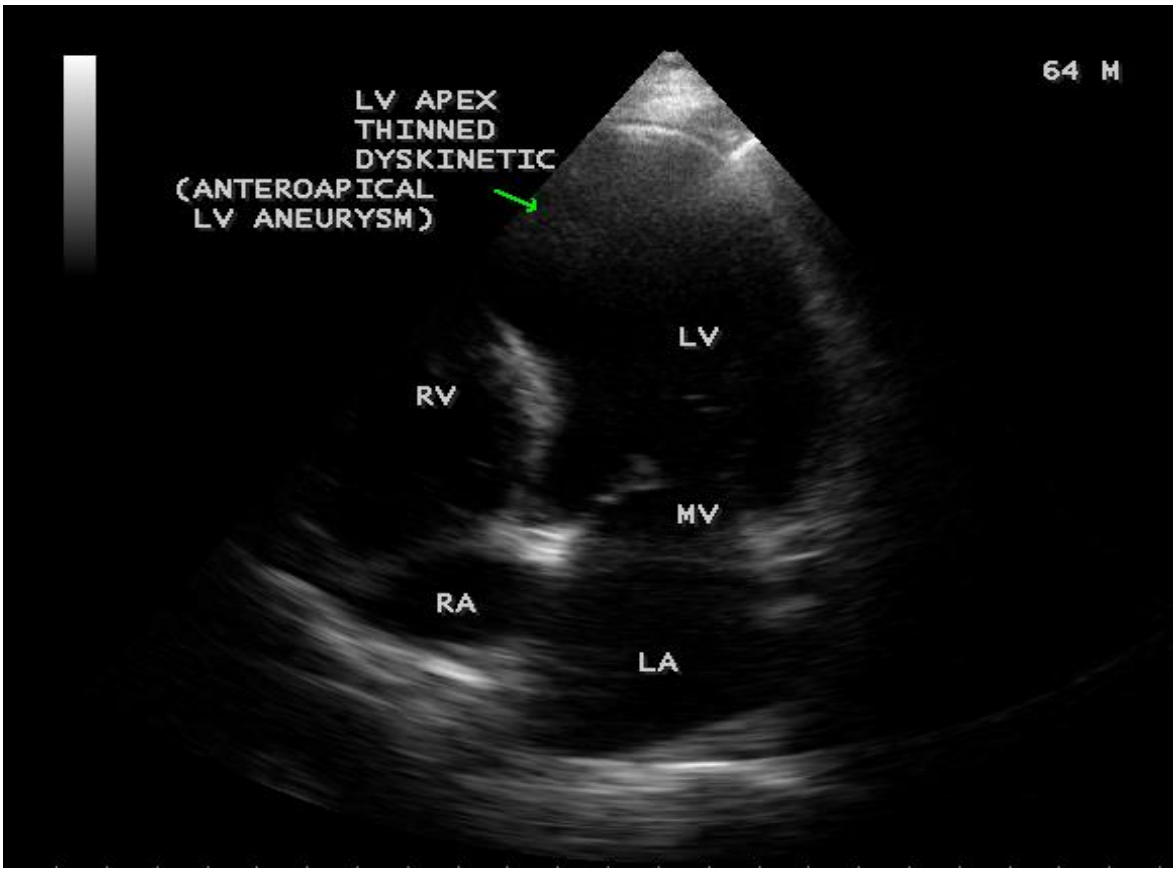

Figure 3: Apical Four chamber view showing the thinned, dyskinetic anteroapical septum with aneurysm [19].

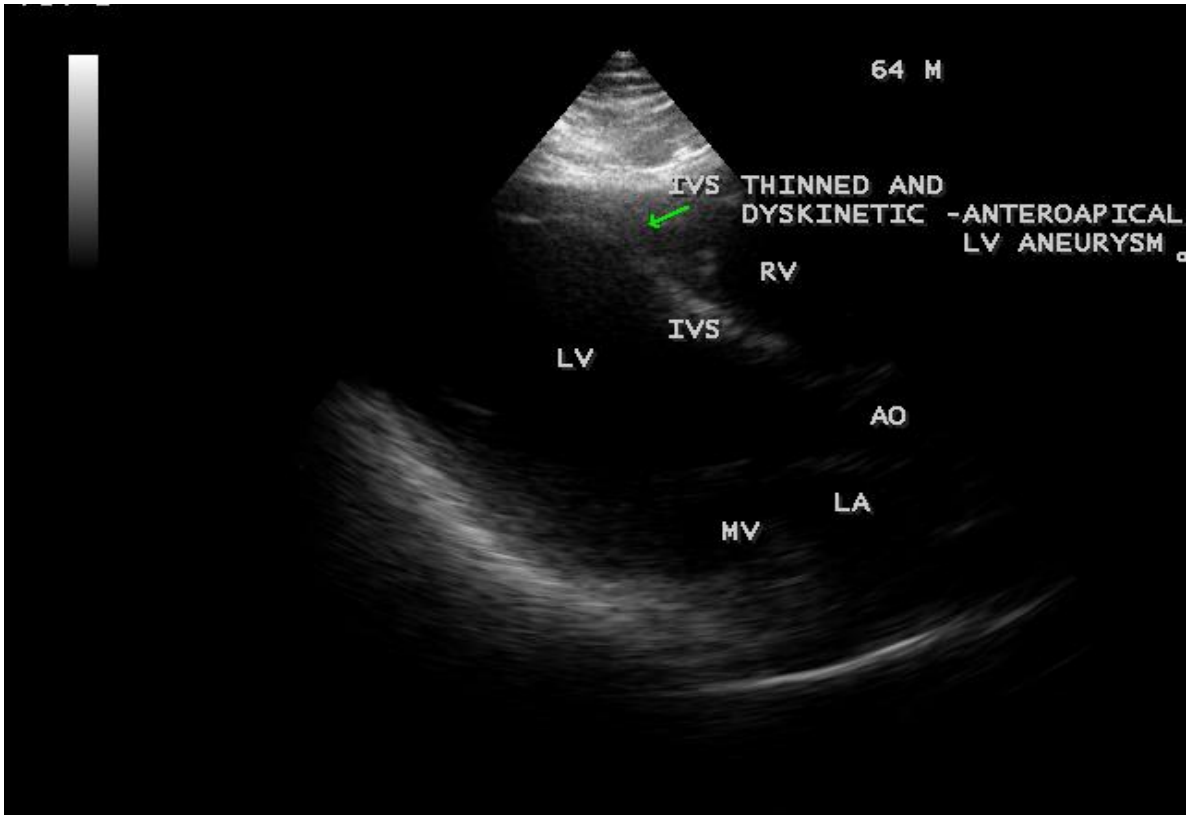

Figure 4: Parasternal long axis view showing the thinned out anteroapical septum.

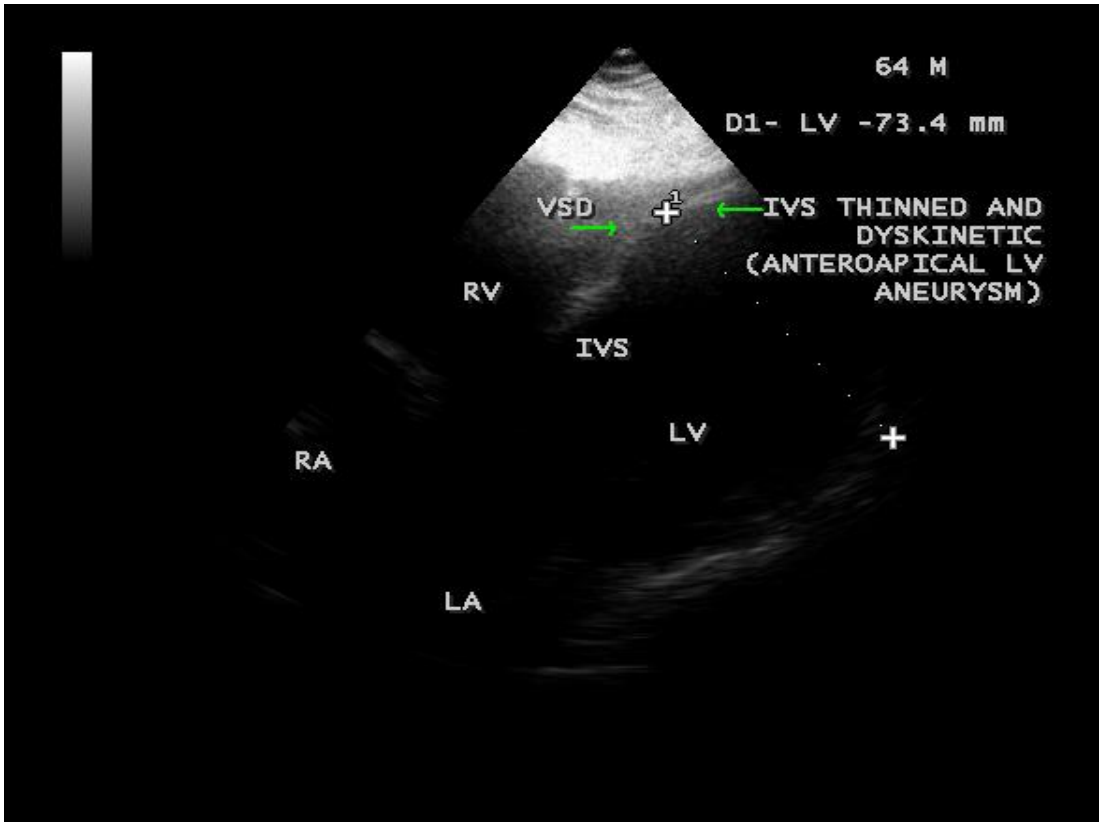

Figure 5: Tilted apical view showing the dilated, hypokinetic left ventricle.

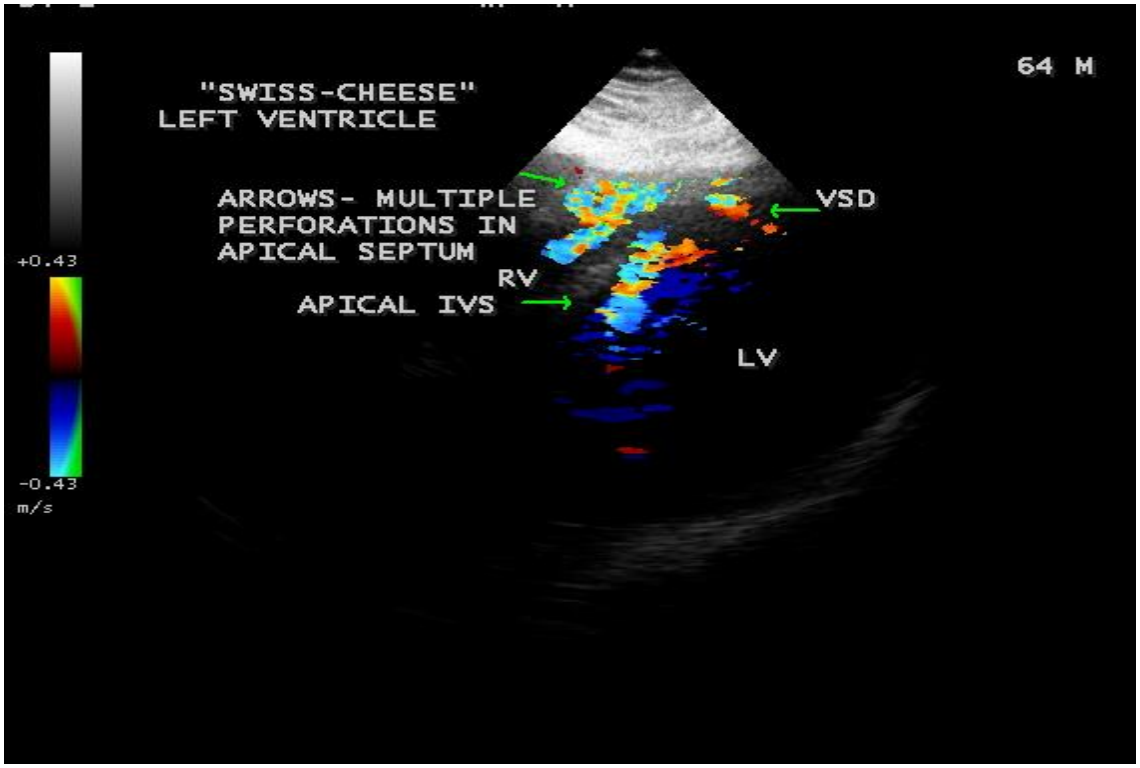

Figure 6: Tilted apical view showing the multiple perforations of the apical septum.

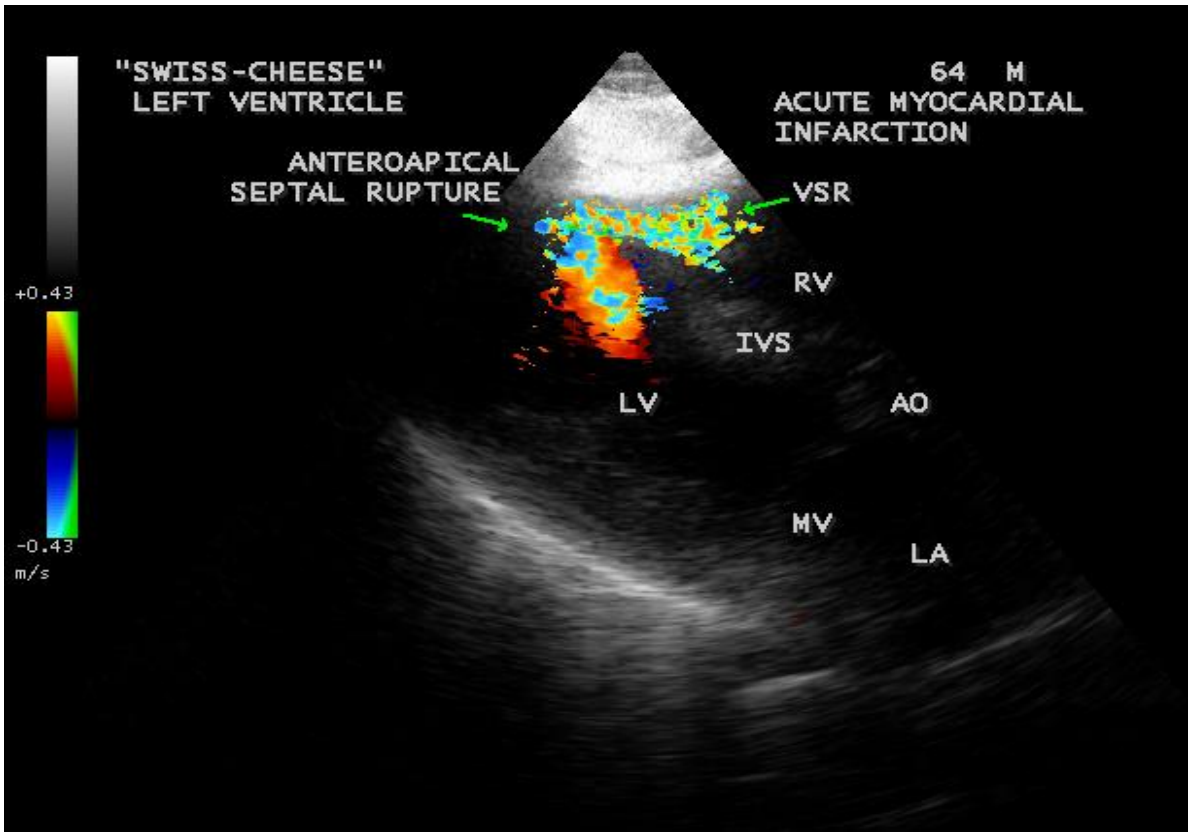

Figure 7: Parasternal long axis view showing the anteroapical ventricular septal rupture.

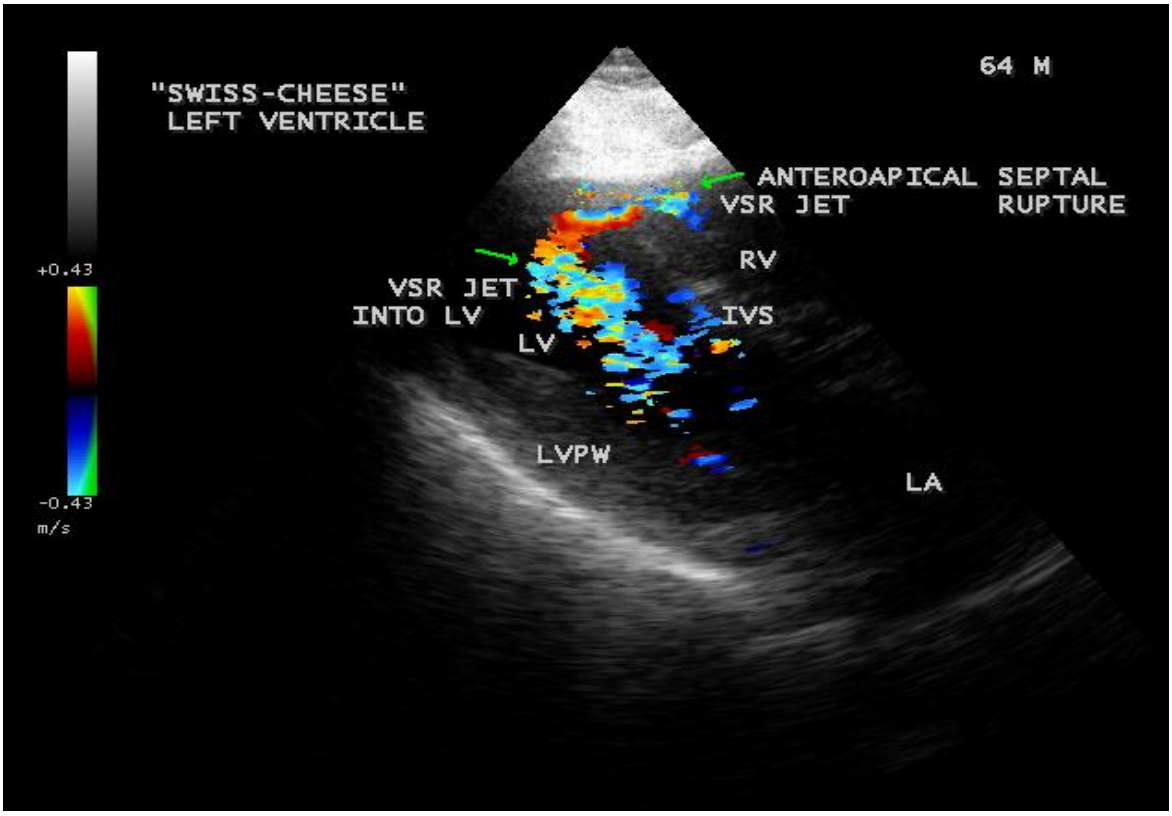

Figure 8: Parasternal long axis view showing the flow into the left ventricle through the septal rupture.

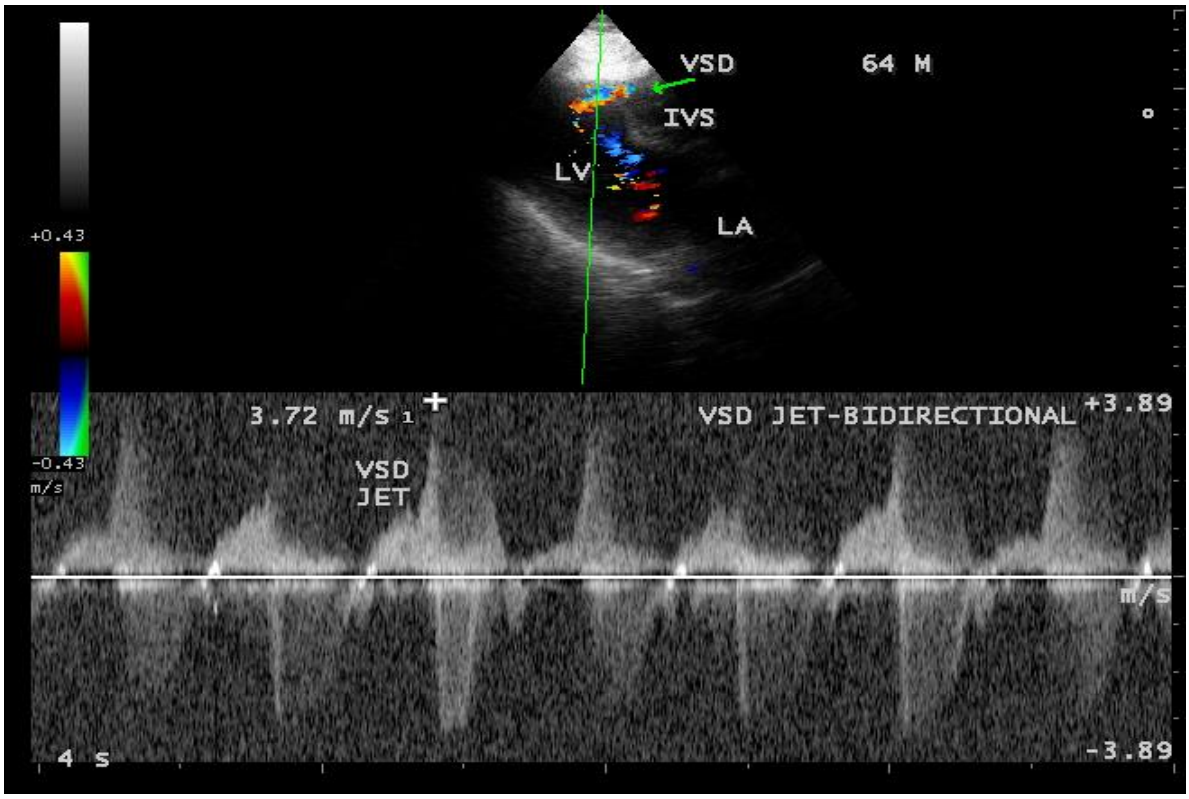

Figure 9: CW (Continuous Wave Doppler) showing the bidirectional jet of ventricular septal rupture (VSR).

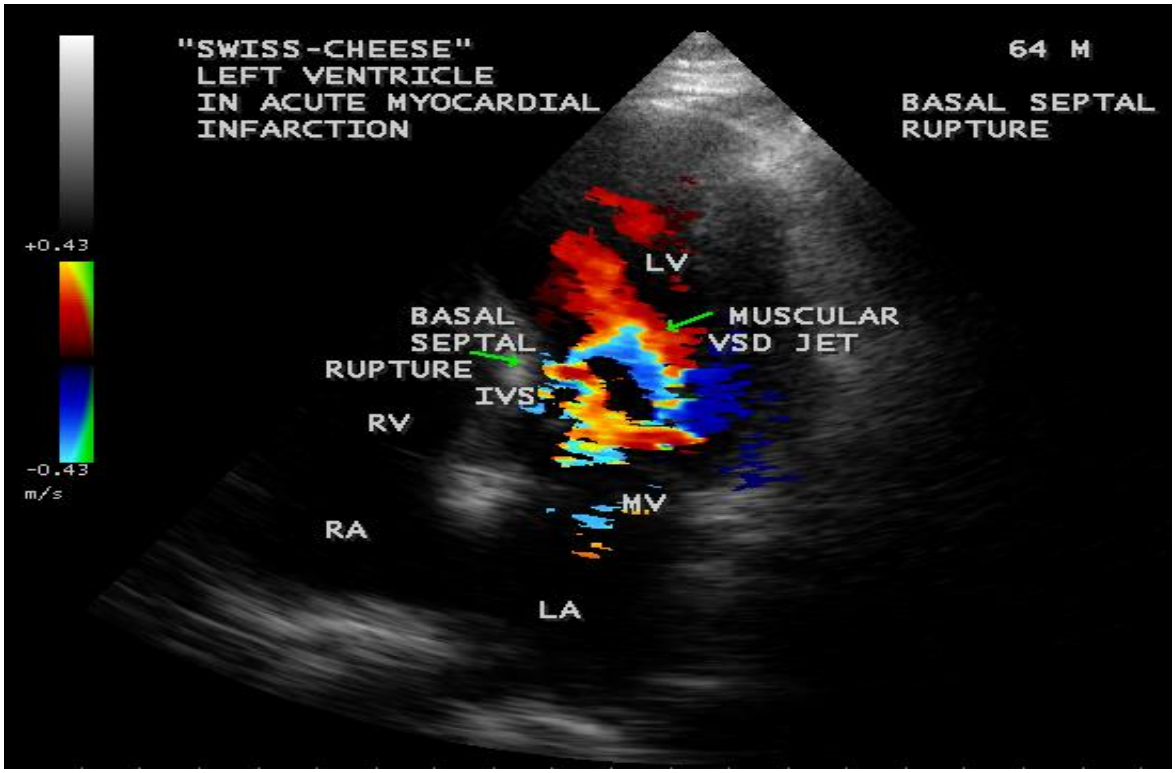

Figure 10: Apical four chamber view showing the basal septal rupture

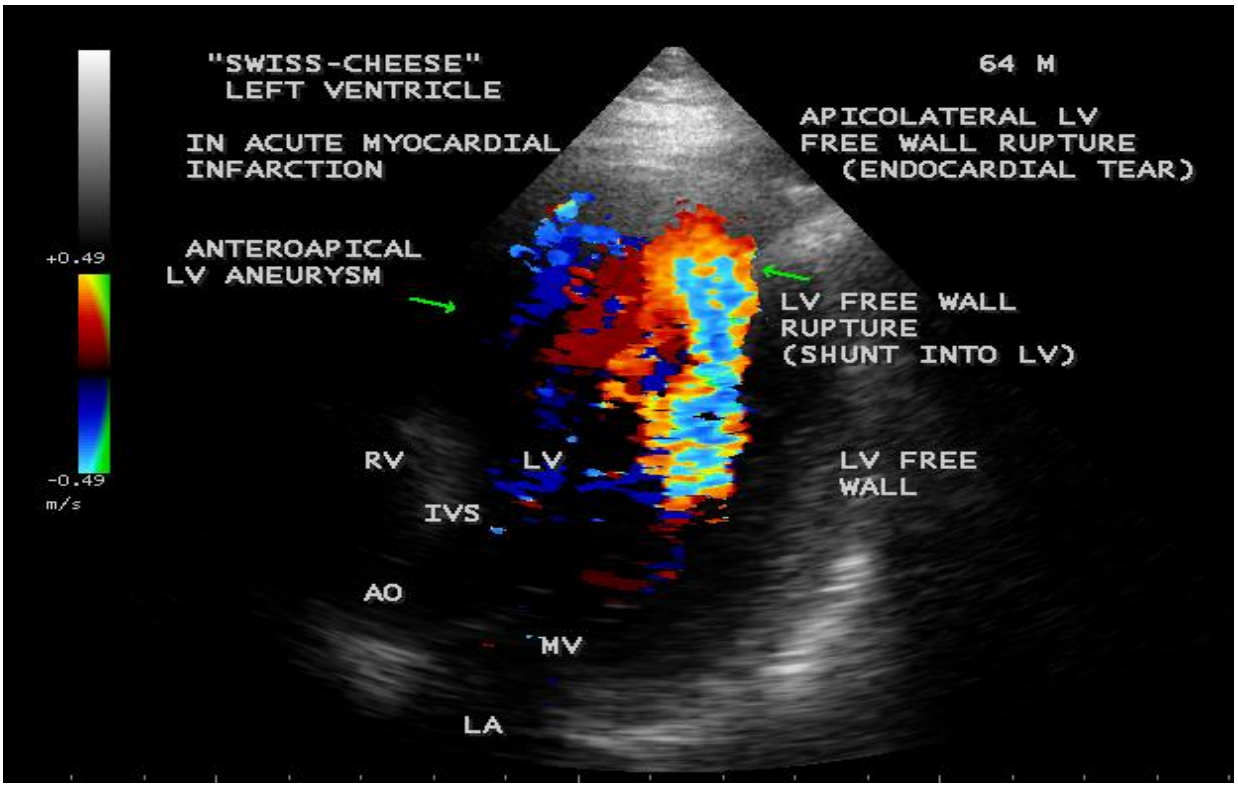

Figure 11: Apical 3 chamber view showing the apicolateral LV (left ventricular) free wall rupture.

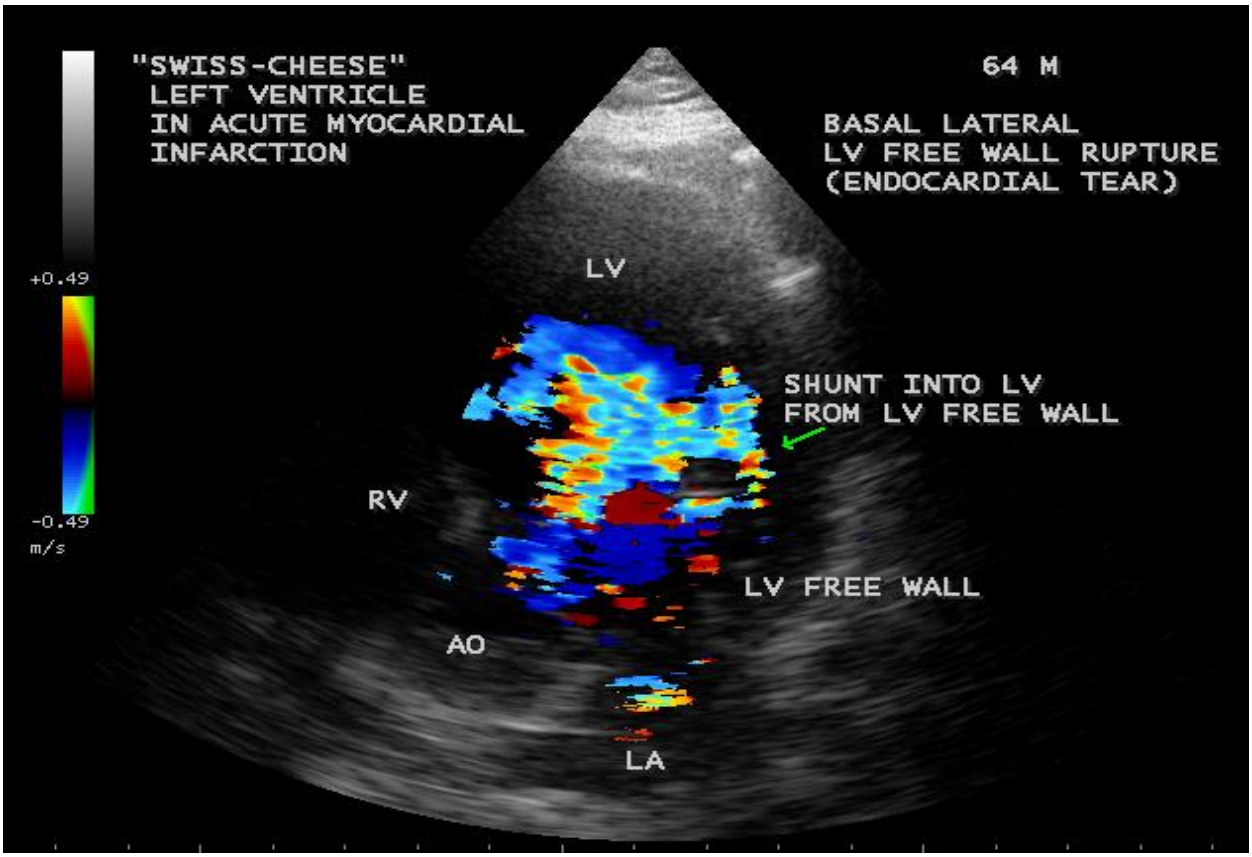

Figure 12: Apical 3 chamber view showing the basal lateral LV (left ventricular) free wall rupture.

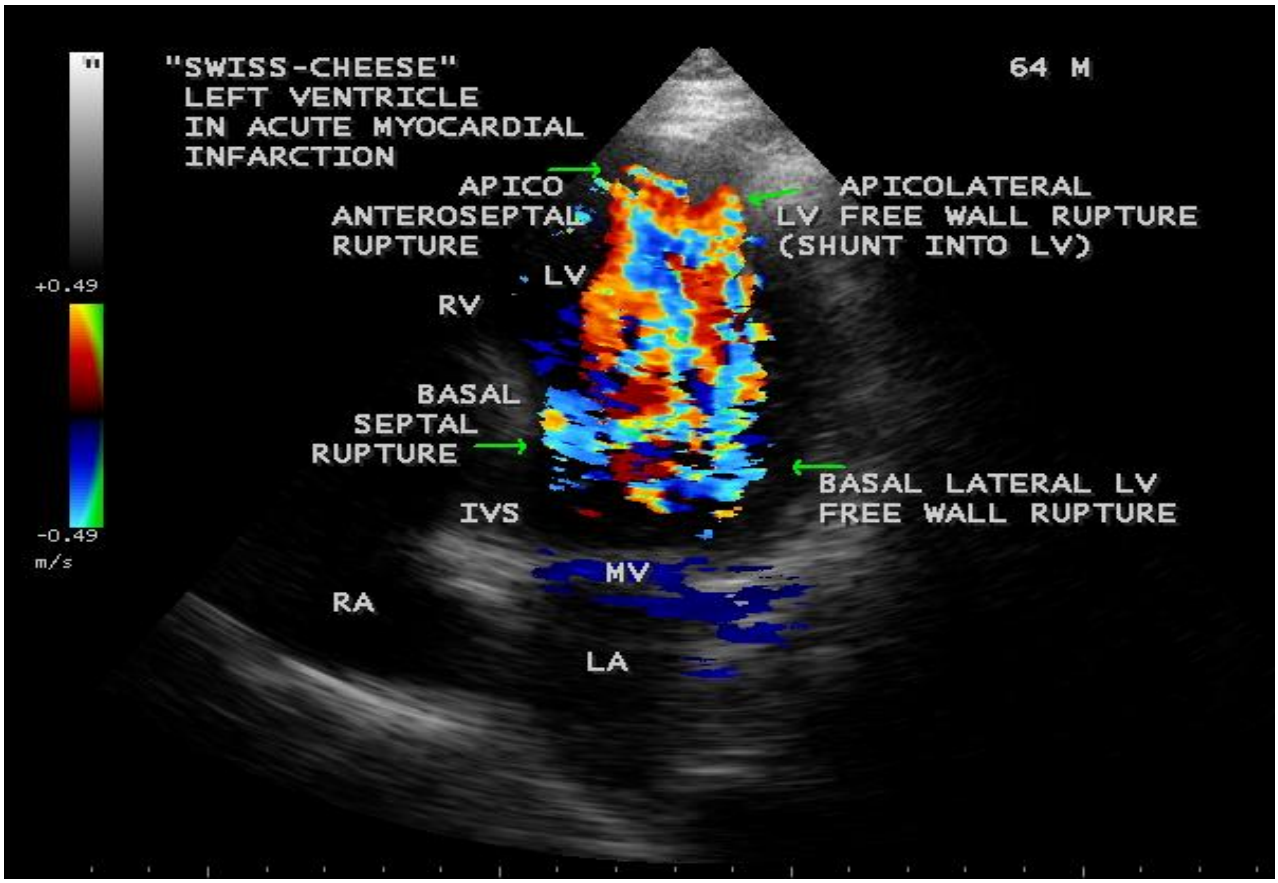

Figure 13: Apical 4 chamber view showing the "Swiss-cheese" left ventricle with multiple sites of ruptures.

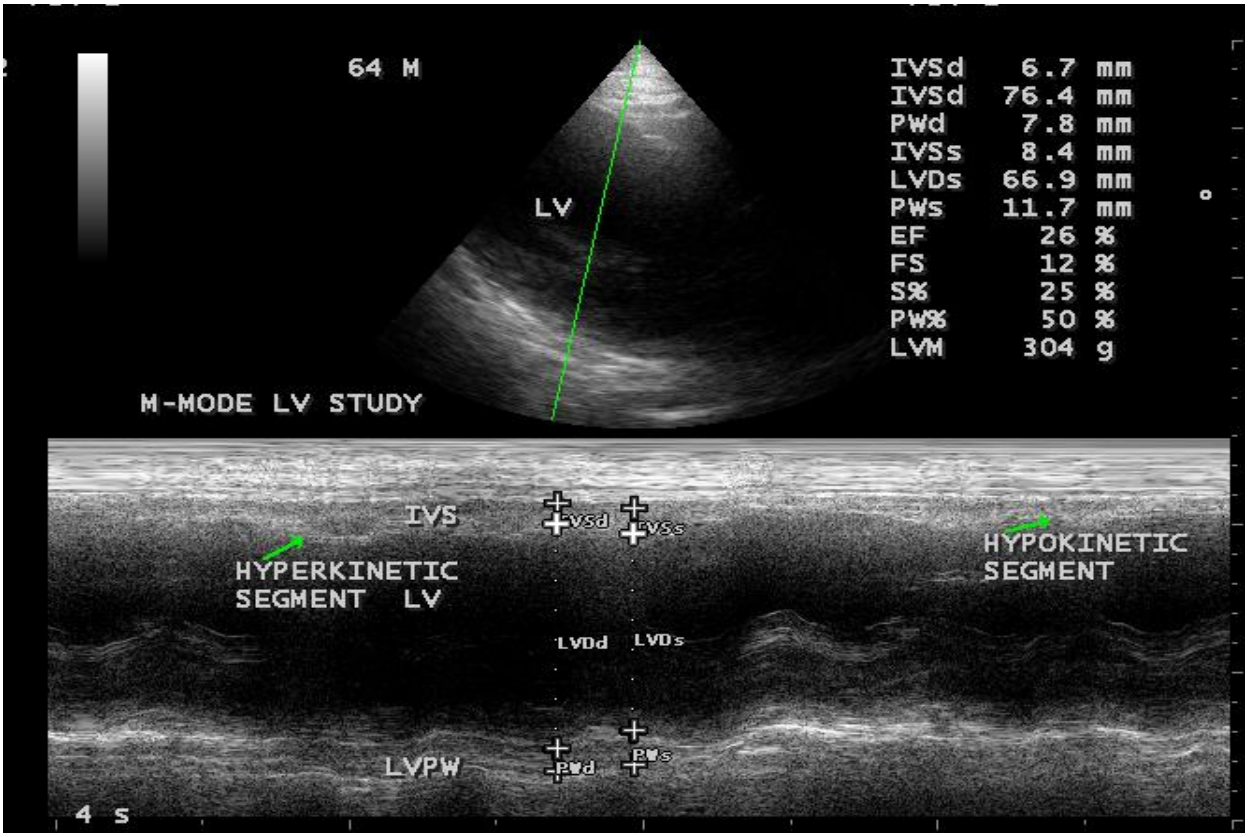

Figure 14: M-Mode LV study showing the contractile dysfunction of the left ventricle with an ejection fraction of 26%.

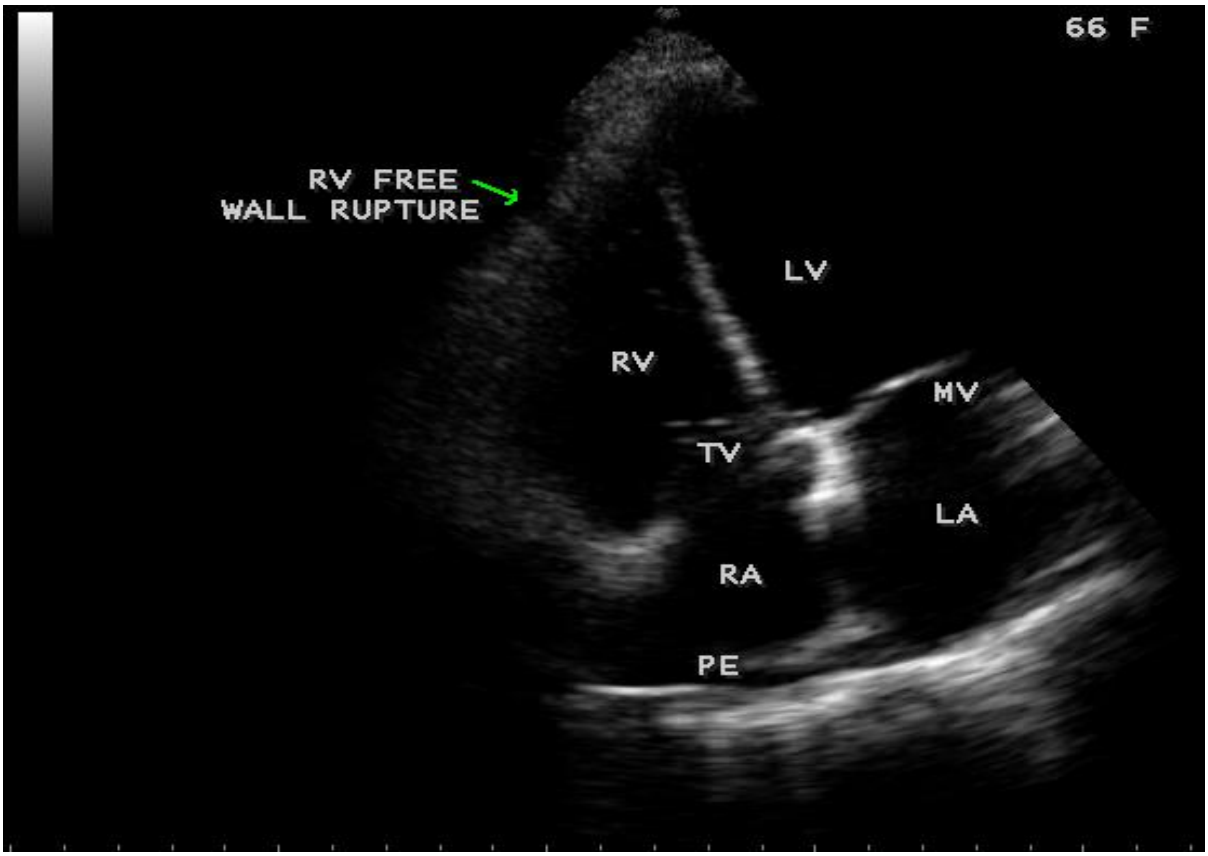

Figure 15: 2D echocardiographic imaging showing the small RV (right ventricular) free wall rupture in a 66-year old female with acute inferior wall infarction in apical four chamber view.

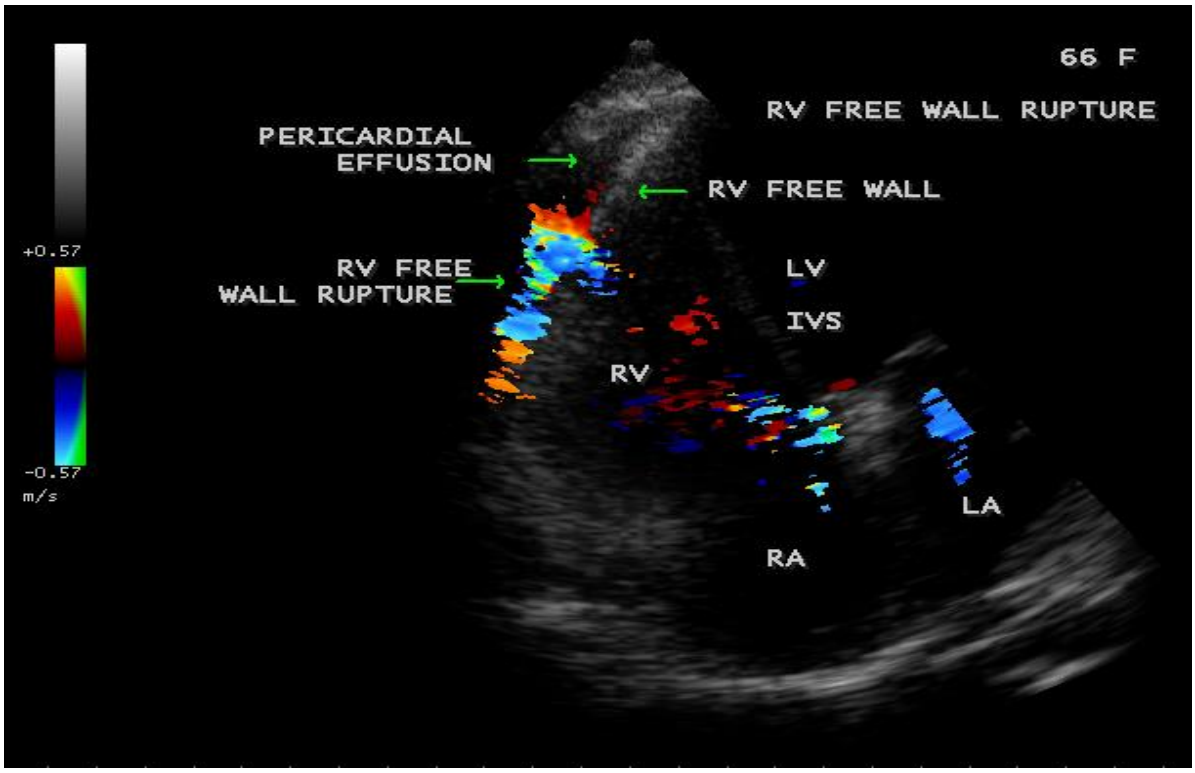

Figure 16: Color Doppler imaging showing the RV free wall rupture into the pericardial space with effusion in a 66-year old female with acute inferior wall infarction.

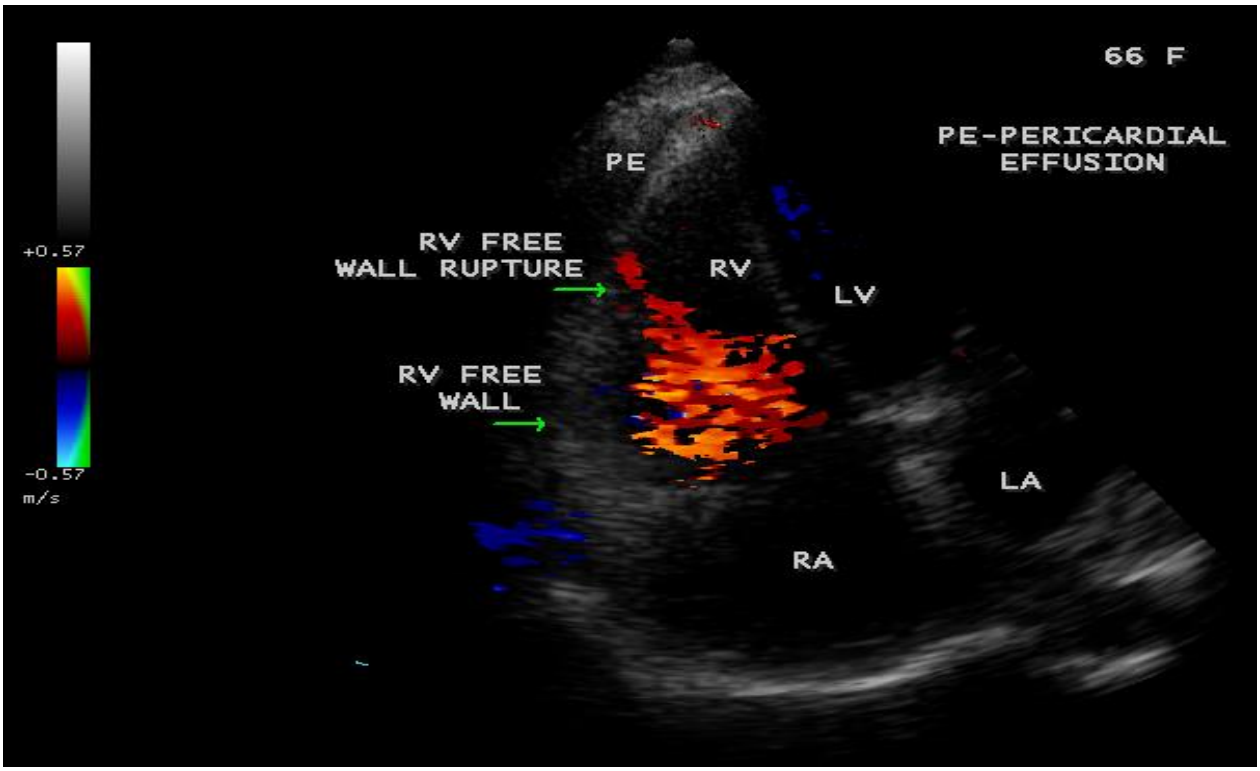

Figure 17: Color Doppler imaging showing the RV free wall rupture with a shunt into RV cavity in a 66-year old female with acute inferior wall infarction.

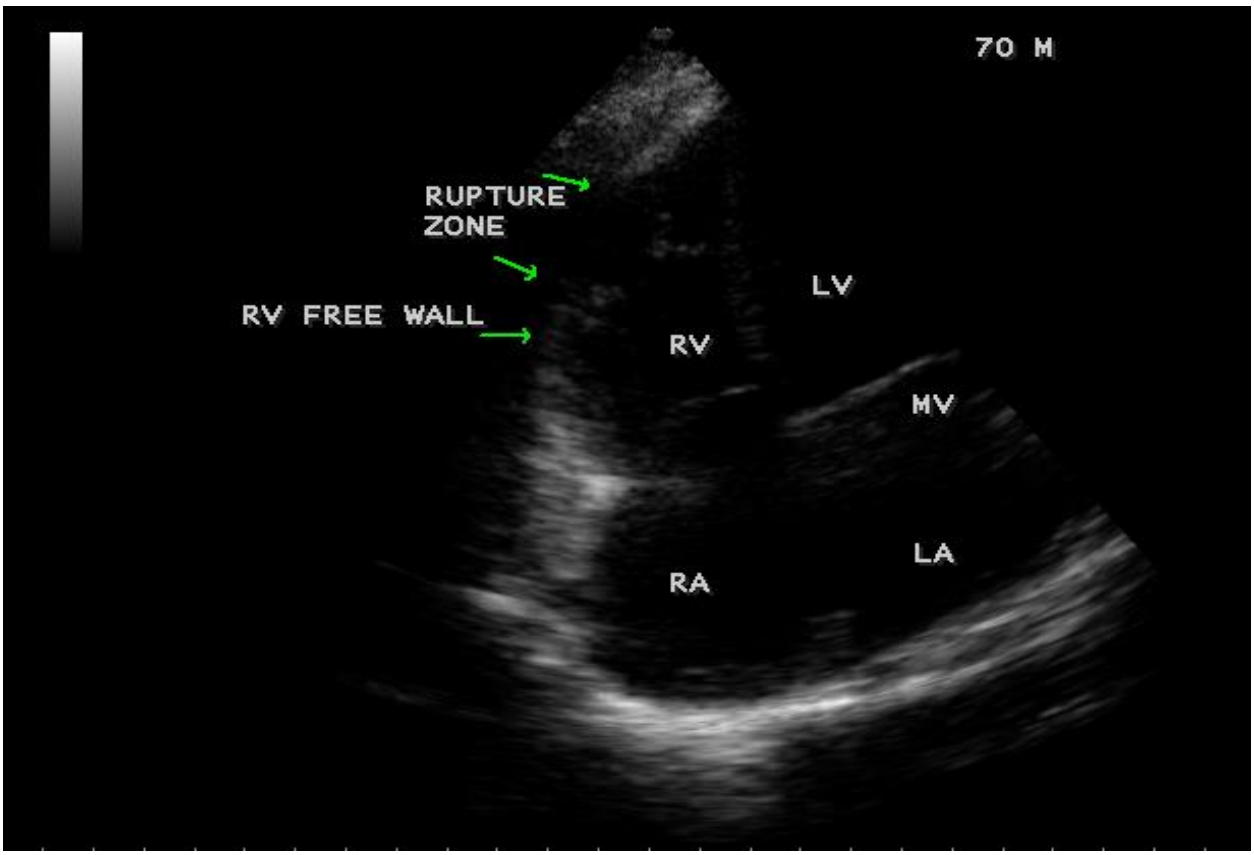

Figure 18: 2D echocardiographic imaging showing the large RV free wall rupture in a 70-year old male with right ventricular infarction in apical four chamber view.

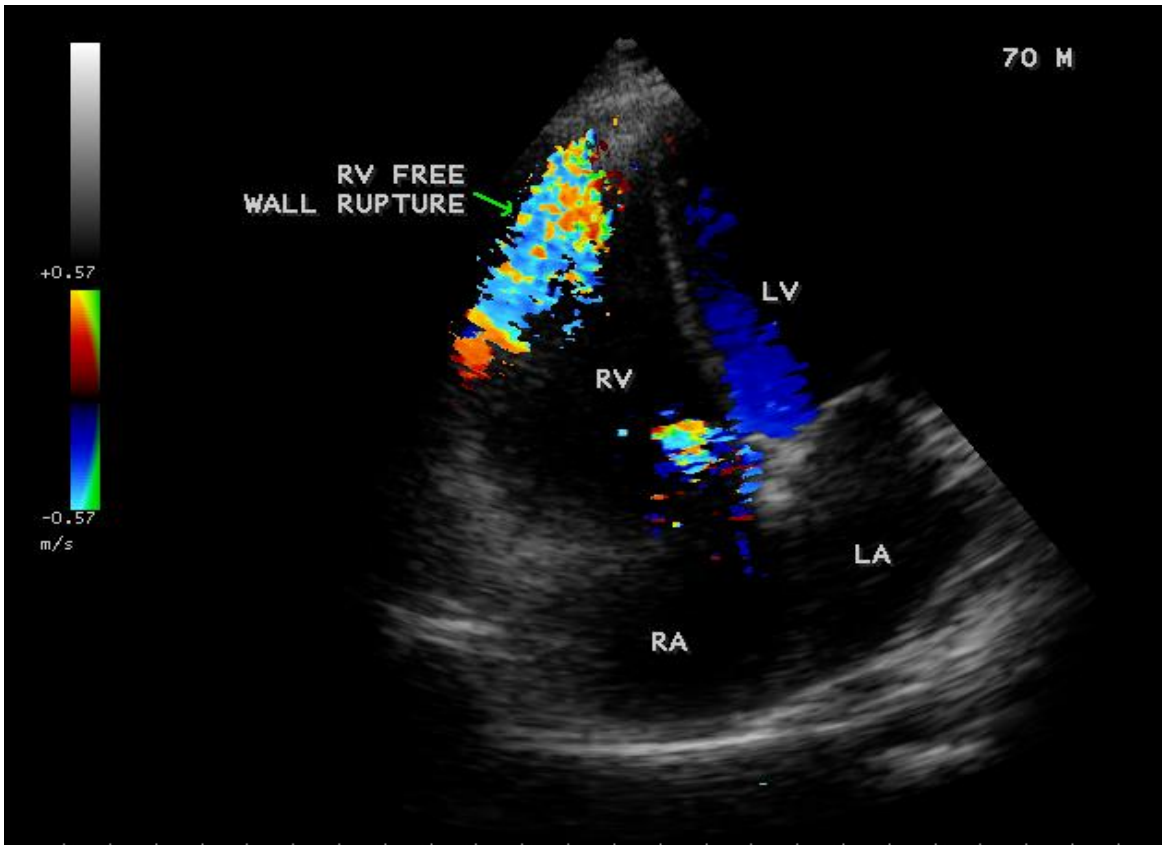

Figure 19: Color Doppler imaging showing the RV free wall rupture in a 70-year old male with right ventricular infarction.

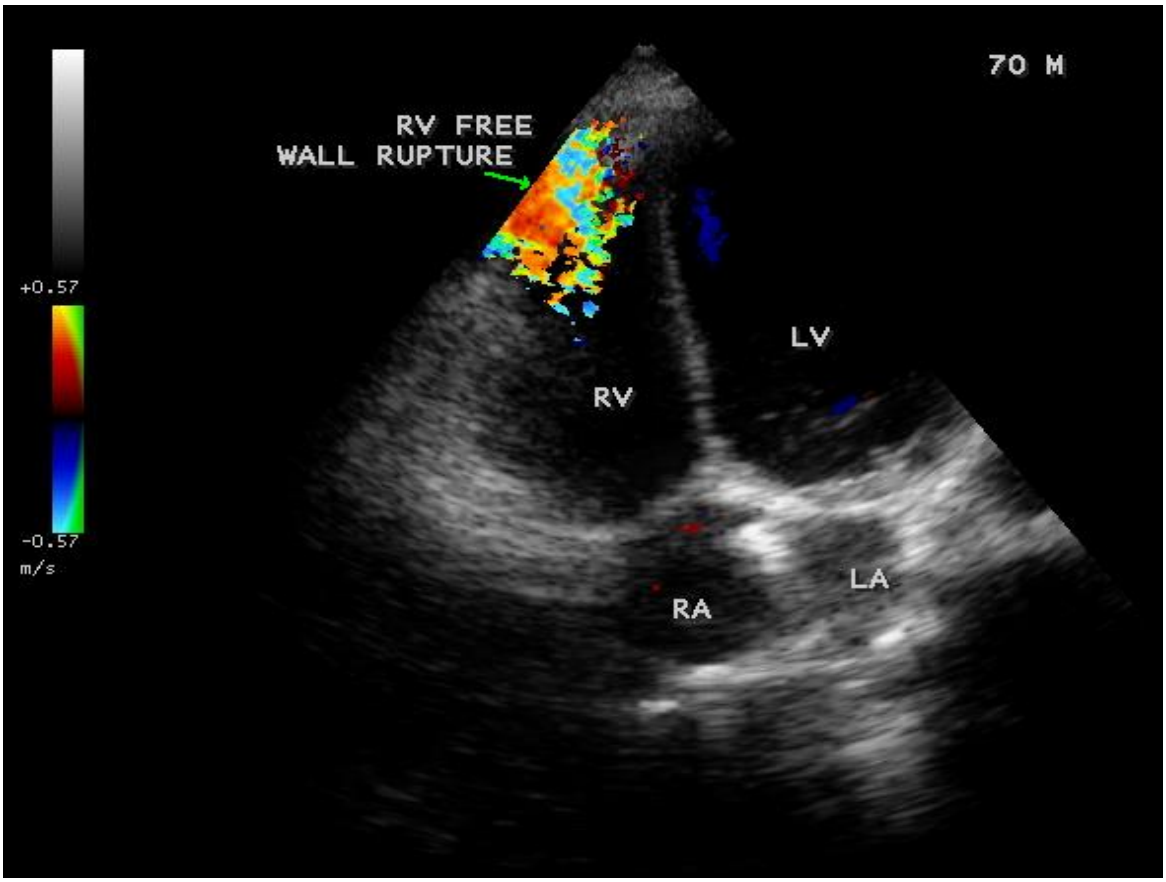

Figure 20: Color Doppler imaging showing the RV free wall rupture with a dense doppler signal in a 70-year old male with right ventricular infarction.

3.Discussion

Myocardial rupture is a catastrophic complication of acute myocardial infarction and most often occurs near the edge of the necrotic myocardium where it abuts the hyperemic healthy zone having the greatest inflammatory activity and high shear stress. It involves the free wall of the ventricles, interventricular septum, papillary muscles and rarely involves the atrial walls. It is more common in patients aged > 60 years and usually seen in women (1.4:1). Contributing risk factors for myocardial rupture are listed in Table 3.

Hemodynamic	Increased intraventricular pressure
Structural	Myocyte necrosis Collagen matrix resolution Intense inflammation
Traditional	Older age (> 60 years) Female sex (non-smoking woman) Previous hypertension First lateral or anterior wall myocardial infarction with RBBB (right bundle branch block) No history of angina Single vessel disease and less evidence of collateral circulation [20],[21] Multivessel disease in inferior infarct rupture [22] Steroid Use And Late Thrombolysis (> 12 – 24 hours)? [23],[24],[25]

Table 3: (Risk factors for myocardial rupture).

Left ventricular free wall rupture is 4-10 times more often than the rupture of the interventricular septum or papillary muscle with an incidence of 2-4% of myocardial infarction [26],[27] and 2.2-10% in various series [28]. It was localized to anterolateral, anteroapical, inferolateral and posterior walls. Higher incidence of rupture (44%) in the lateral wall of the ventricles is probably due to the increased stress resulting from the contraction of papillary muscles. Postinfarction pericarditis manifested as pleuritic chest pain and friction rub may be present in some cases before the onset of left ventricular free wall rupture and indicates the transmural extension of the infarct. Early rupture (acute form) develops within first 48 hours and represents 40-60% of cases and the patients may die suddenly before reaching the hospital due to severe hypotension or electromechanical dissociation secondary to acute pericardial tamponade as a result of strain in the infarcted zone caused by sustained arterial hypertension (systolic blood pressure >150 mmHg) and ambulatory activity. In early rupture, there is hardly any thinning of the infarcted zone. Late rupture (subacute form) develops beyond the second day in an already expanded infarcted region and less affected by hypertension, but often triggered by undue physical efforts such as persistent vomiting and cough. It is less severe, more compatible with survival with an anfractuous tract between the layers of the myocardium and may account for 30% of all cases of in-hospital free wall rupture. Eventhough most of the ventricular ruptures occur in the free wall of the left ventricle [29], the rupture occurs in the interventricular septum in approximately 15 to 20% of cases [30] and it complicates 1-2% of acute myocardial infarction presentations in pre-thrombolytic era [31]. The incidence has declined to about 0.2% in thrombolytic era and most contemporary series shown that it is increasingly rare, complicating between 0.17-0.31% of patients presenting with acute myocardial infarction [32]. Two pathological types of ventricular septal rupture were described by Edwards et al [33] in an autopsy report as simple ruptures defined as direct through-and-through defects and complex ruptures characterized by serpigenous, hemorrhagic tracts with myocardial disruption and necrosis extending beyond the primary site with a convoluted course. Simple ruptures are more common after anterior myocardial infarction and complex ruptures are more frequent in inferior myocardial infarction. Of interest, 80% of all complex ruptures occurring in patients with inferior infarction and 21% of ruptures are complex in anterior myocardial infarction. The nature of presentation has changed as the average time interval between infarction and rupture is closer to 24 hours [34]. Ventricular free wall rupture (VFWR) in patients with reperfusion therapy (either primary PCI or thrombolytic therapy) occurs in 24-48 hours and is characterized by slit-like myocardial tear or erosion with hemorrhage due to activation of plasmin by thrombolytic agents [35] when the reperfusion time is delayed. About 60% of ventricular ruptures occur with infarction of the anterior wall, 40% with posterior or inferior wall and men are affected more commonly than women. Sometimes multiple septal perforations may occur simultaneously as shown in Figure 6 or within several days of each other. Acute onset of shortness of breath, chest pain, diaphoresis, unexplained emesis, cool and clammy skin and syncope may herald the onset of ventricular septal rupture in acute myocardial infarction. A triad of recurrent or persistent chest pain, recurrent or persistent ST segment elevation and unexplainable hypotension is termed as “subacute free wall rupture syndrome” (post-myocardial infarction free wall rupture syndrome) [36]. A loud, harsh systolic murmur, associated with palpable thrill in 50% of cases audible over left sternal border and apical areas is the most consistent physical finding of postinfarction ventricular septal rupture and it indicates the onset of sudden deterioration in a previously stable patient with the development of heart failure or cardiogenic shock. Persistent ST segment elevation after acute myocardial infarction is associated with higher incidence of myocardial rupture. In the setting of acute myocardial infarction, ST elevation in inferior and anterior leads as shown in Figure 1 as a result of occlusion of a large wraparound left anterior descending coronary artery (Left anterior descending coronary artery (LAD) reaching the apex, wraps around the left ventricular apex and travels some distance in the posterior inter-ventricular groove and supplying the apical inferior aspect of the heart – type III LAD) is associated with an increased risk of ventricular septal rupture [37]. New ST segment changes (“saddle-shaped ST-segment elevation”) or persistent non-inversion of T-waves in the affected leads may suggest a less noisy ‘stuttering” type of rupture [38]. Postinfarction septal defects are localized in the muscular part of the septum and are associated with a high incidence of left ventricular aneurysm as shown in Figures 3,4,5 [39] as 30% according to Schlesinger et al [40] and 68% by Hill et al [41]. Without ventricular septal rupture, the incidence of aneurysm is considerably low with an incidence of 12.4%. Silent myocardial infarction may result as an asymptomatic ventricular septal rupture or chronic heart failure [42]. Anterior myocardial infarction is associated with rupture of the apical septum and in inferior myocardial infarction, it often occurs at the base of the heart with high mortality mainly due to severe right ventricular dysfunction. Complete spontaneous closure of such an acquired defect is extremely rare.

Echocardiographic Features: Emergency bedside transthoracic echocardiography is the diagnostic modality of choice in all types of myocardial rupture. Two- dimensional echocardiography can directly

visualize the entire length of interventricular septum and its defects. Richards et al [43] found the unusual flow signals near the right ventricular side of the ruptured septum by using Doppler and M-mode echocardiography and Keren et al [44] by using Doppler and two-dimensional echocardiography. across the ventricular septal rupture is bidirectional as shown in Figure 9. The majority of shunt is towards the dilated, dyskinetic low pressure chamber of left ventricle as shown in Figure 8 in this patient. Non survivors may be a reflection of more extensive myocardial disruption with hemorrhage into the neighbouring tissue from complex ruptures as shown in Figure 13, leading to contractile dysfunction with an ejection fraction of 26% as in Figure 14 in this patient.

Management

Medical Therapy: Medical therapy is initiated in an attempt to stabilize the patient hemodynamically by reducing the afterload with vasodilators and by increasing the cardiac output with inotropic agents. Vasodilators also reduces the left-to-right shunt associated with the mechanical defect and thereby increases the cardiac output. Intravenous nitropglycerin is the preferred agent in ischemic heart disease. Profound cardiogenic shock precludes the use of vasodilator therapy and often necessitate the vasopressor support. Rapid fluid administration to increase the preload and to improve cardiac output in cases of free wall rupture is advisable. IABCP (intraaortic balloon counterpulsation) may be helpful to reduce the left ventricular afterload and thus increasing the cardiac output. It reduces the left-to-right shunt and also causes diastolic augmentation with an increase in coronary blood flow and myocardial oxygen consumption. It is not a substitute for urgent intervention and patients with cardiogenic shock, it should be followed by immediate intervention. The role of IABCP in left ventricular free wall rupture is less clear. This aggressive approach often results in temporary hemodynamic stability and patients may deteriorate rapidly. Thus, achieving hemodynamic stability before surgery is beneficial, but prolonged attempts to improve the patient's hemodynamic status is hazardous [45]. In animal studies, beta blockers and angiotensin converting enzyme (ACE) inhibirors were found to reduce the activation of matrix metalloproteinase and synthesis of collagen. In a recent study, the use of ACE inhibitors but not beta blockers were found to reduce the risk of VFWR (ventricular free wall rupture) [46].

Catheter therapy: Device closure is difficult while the margins of the septal rupture are soft [47]. It may provide a temporary hemodynamic relief and allows surgical closure when the infarcted myocardium has had time to fibrose [48]. The outcome of most patients with postinfarction ventricular septal rupture associated with cardiogenic shock, inferior myocardial infarction, complex defects and right ventricular dysfunction is unfavourable with device closure. Landzberg and Lock [49] performed percutaneous closure of postinfarction ventricular septal rupture by using Clamshell double umbrella and cardioSEAL devices. Better clinical results were obtained by Holzer et al [50] with a 30day mortality of 28% in 16 patients treated with initial device closure. Transcatheter closure of ventricular septal rupture by using Amplatzer septal occluder may be the treatment of choice in patients with subacute ventricular septal rupture. Understanding of the size, shape and borders of the defects are important before attempting device closure. The defects <15 mm in size is optimal. Inferior/posterior defects are unfavourable as they lack an adequate tissue rim to secure the device. Serpigenous defects may complicate significant leakage around the defect and freshly infarcted myocardium may exhibit ongoing necrosis and making device instability, peri-device leak and even device embolization. Patients who undergo percutaneous closure should receive dual antiplatelet therapy for 6 months followed by baby aspirin (75 mg/day) thereafter. Ventricular assist devices, a temporary device will maintain the patient's hemodynamics while the edge of the septal rupture fibrose sufficiently to support a percutaneous implantable occlusive device.

Surgical therapy: In 1972, Fitz Gibbon and Montegut performed the first successful surgery for the correction of left ventricular free wall rupture due to ischemic heart disease [51],[52]. Nasir, et al [53] concluded that surgery is superior to conservative management for patients presenting with free wall rupture. A delayed surgical approach with the use of circulatory support is the preferred method for ventricular septal rupture (VSR) repair and the stable hemodynamics is a significant predictor of survival [54]. The ideal time for surgical repair is 2-3 weeks after the rupture when the edges of the defect become firmer and fibrotic. The repair is more secure, easily accomplished and more receptive to sutures during this time. Immediate surgery is usually indicated [55] since postinfarction rupture rapidly leads to hemodynamic deterioration and cardiogenic shock. In patients presented with cardiac tamponade and hypotension, catheter pericardiocentesis is indicated and 10-50 ml of pericardial fluid is aspirated to maintain hemodynamic stability. A second pericardiocentesis is advised if tamponade reccurs and unstable. If failed, emergency thorocotomy and application of a Teflon or pericardial patch to the epicardial surface of the ruptured site with cyanoacrylate biologic glue [56]. If active bleeding is present, infarctectomy of ruptured tissue followed by a Teflon buttressed suture [57] is preferred when the use of patch is unsuccessful. The anteroapical defects are closed by buttressing the defect with viable muscle from the adjacent anterior left ventricular wall. Smaller defects located high in the ventricular septum are closed with a Dacron patch. For the high posterior or inferior defects, use of a synthetic patch closure to prevent tension is preferred. When multiple defects are present, they are concealed by the trabeculae of the ventricle and there is difficulty in defining the borders of the defect and so poor surgical results may occur. In such cases, a technique of endocardial patch with infarct exclusion by avoiding the damage to the dysfunctional right ventricle and to restore the geometry of left ventricular myocardium to preserve the left ventricular function in patients with transmural infarction having infarct expansion with ventricular aneurysm and rupture had been described [58]. The technique consists of by performing left ventriculotomy through the infarcted muscle and securing a glutaraldehyde-fixed bovine pericardial patch to the endocardium of the left ventricle (all around the infarcted myocardium), excluding rather than excising the infarcted septum and ventricular walls and the ventriculotomy is simply closed over the pericardial patch [59]. This technique was preferred in this patient. For basal septal rupture, closure of the defect with bovine patches by incising the tricuspid septal leaflet through right atrial approach without ventriculotomy was preferred [60]. Gore-Tex patches or strips are most commonly used in ongoing squirting rupture and sutureless management is preferable in the oozing type [61]. Concomitant coronary artery bypass grafting (CABG) may be required and helpful to improve long-term survival.

Screening of population: A small RV (right ventricular) free wall rupture in a 66-year-old female with acute inferior wall infarction was shown in Figures 15 to 17 and a large RV free wall rupture in a 70-year-old male with right ventricular infarction was shown in Figures 18 to 20.

4.Conclusion

The free wall rupture in acute ST-elevation myocardial infarction is under-recognized [62] and sometimes it is subacute, may not be typical of an acute blow-out rupture and leading to death within minutes. Instant diagnosis is crucial to detect free wall ruptures by transthoracic echocardiography. The incidence of myocardial rupture was decreased if primary percutaneous intervention was performedin acute myocardial infarction [63] and it is about 2-3% [64]. If there is extensive myocardial damage with hemodynamic compromise, early intervention is urgently needed. Most studies show that overall mortality rate of early surgical approach is < 25%, it was lowest in apical septal ruptures, lower in anterior septal rupture (10 to 15%) and high for posterior defects (30 to 35%). Therefore, surgical treatment must be carried out on an emergency basis even if the patient was stable [65] and

90% die without surgery. The time between the onset of acute myocardial infarction and surgical intervention is the most important factor determining the outcome and in-hospital survival [66].

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