Scientific Research Publishing

Case Reports in Clinical Medicine, 2017, 6, 36-58
http://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

"Swiss-Cheese" Left Ventricle in Acute Myocardial Infarction—A Case Report

Ramachandran Muthiah

Thoothukudi Medical College Hospital, Thoothukudi, India
Email: cardioramachandran@yahoo.co.uk

How to cite this paper: Muthiah, R. (2017) "Swiss-Cheese" Left Ventricle in Acute Myocardial Infarction—A Case Report. *Case Reports in Clinical Medicine*, **6**, 36-58.
https://doi.org/10.4236/crcm.2017.62005

Received: January 4, 2017
Accepted: February 18, 2017
Published: February 21, 2017

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 within 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% died within 5 days and 82% died 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

1. 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.

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 begins. Collagenase 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 occurs 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**.

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

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 compli-

Table 1. Becker and van Mantgem classification of myocardial rupture.

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

cations 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 **Figure 1** to **Figure 6**.

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

Table 2. Morphological types of myocardial rupture.

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)

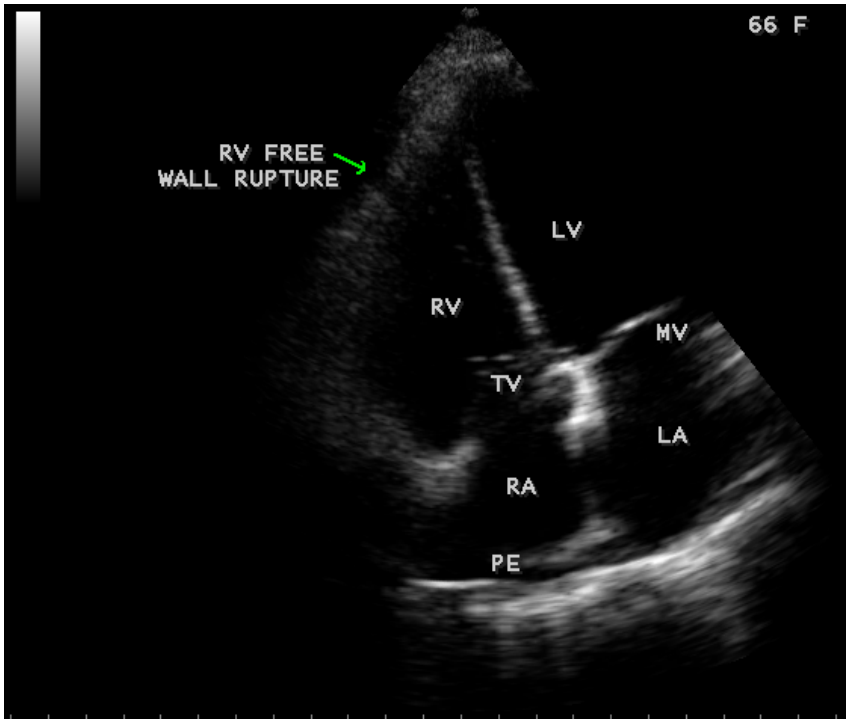

Figure 1. 2D echocardiographic imaging showing the small RV (right ventricular) free wall rupture in a 66-year-old female with acute inferior wall infarction in apical four chamber view.

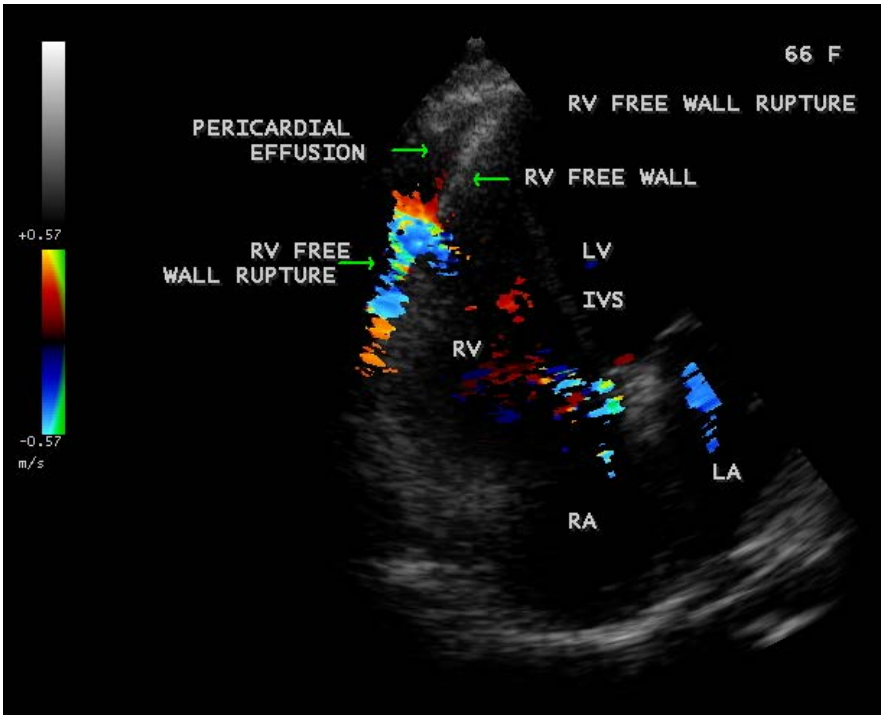

Figure 2. Color doppler imaging showing the RV free wall rupture into the pericardial space with effusion in a 66-year-old female with acute inferior wall infarction.

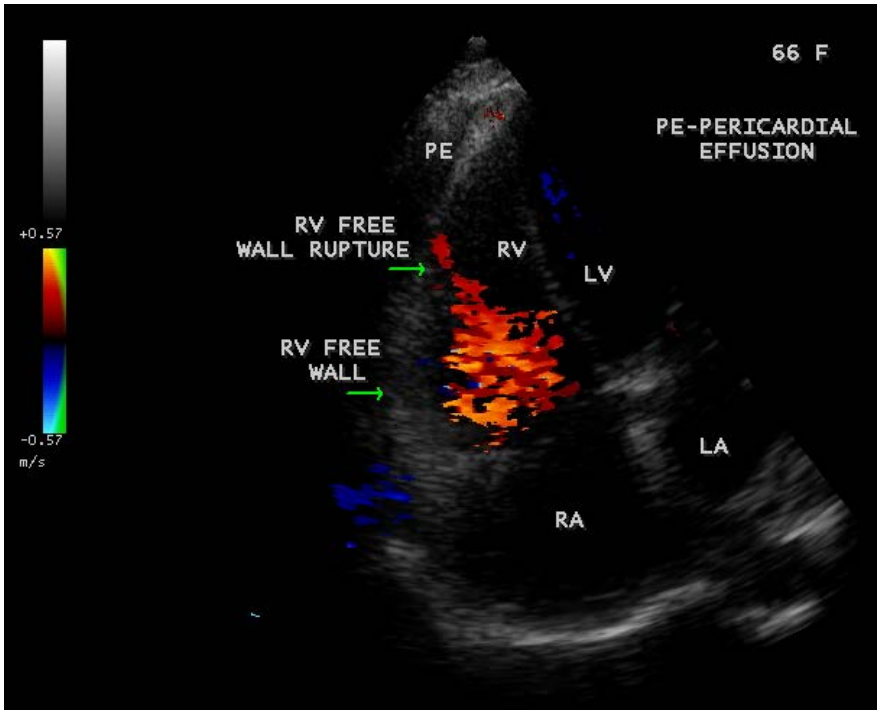

Figure 3. Color doppler imaging showing the RV free wall rupture with a shunt into RV cavity in a 66-year-old female with acute inferior wall infarction.

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.

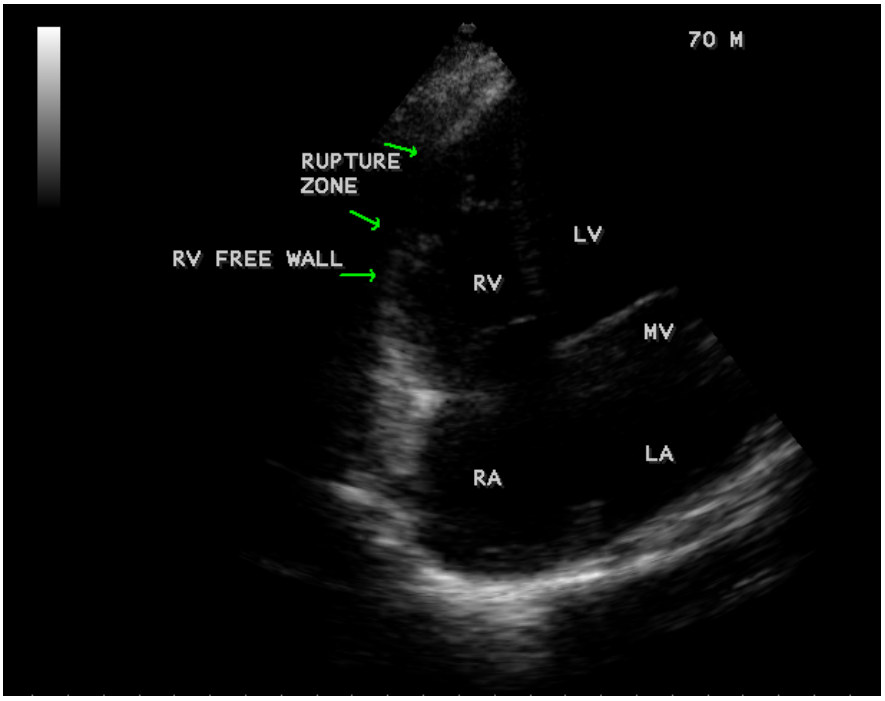

Figure 4. 2D echocardiographic imaging showing the large RV free wall rupture in a 70-year-old male with right ventricular infarction in apical four chamber view.

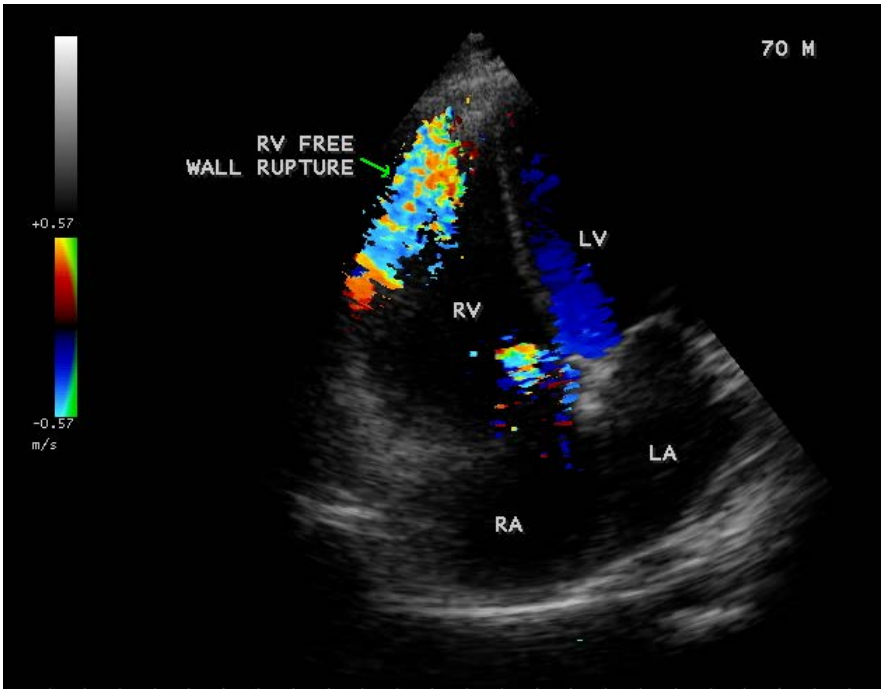

Figure 5. Color doppler imaging showing the RV free wall rupture in a 70-year-old male with right ventricular infarction.

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

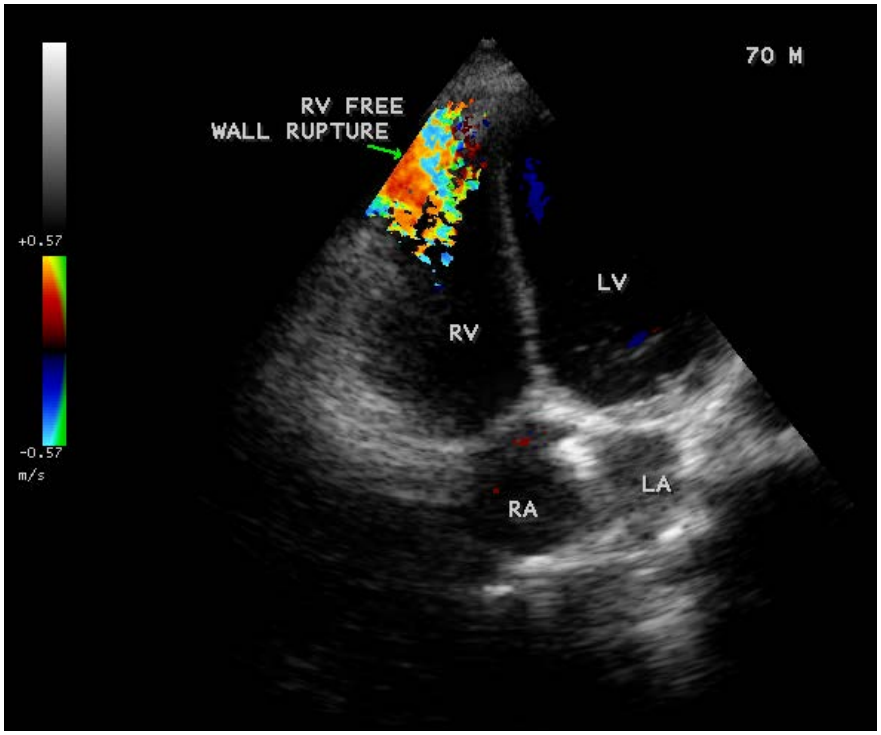

Figure 6. Color doppler imaging showing the RV free wall rupture with a dense doppler signal in a 70-year-old male with right ventricular infarction.

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 are 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 7.** He was in cardiogenic shock and supported with intravenous fluids and inotropic agents. He subse-

quently 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 8 and it was not responded to intravenous amiodarone and cardioversion, the

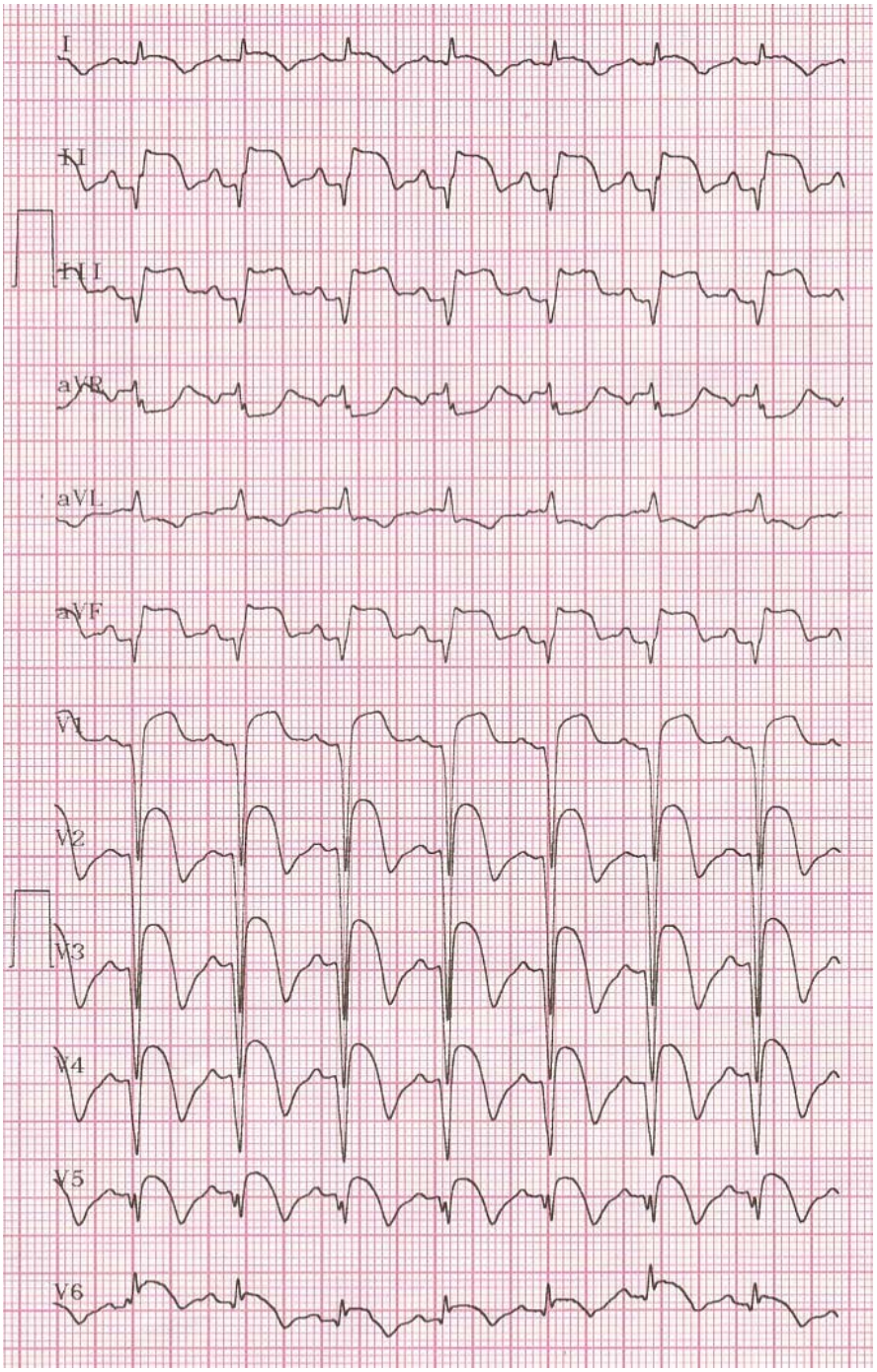

Figure 7. ECG (2 days after thrombolysis) showing persistent ST segment elevation in anterior and inferior leads (wraparound LAD (left anterior descending coronary artery)) occlusion.

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 Figure 9 to Figure 20 with contractile dysfunction.

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, and 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.

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 [19] [20] and 2.2% - 10% in various series [21]. 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.

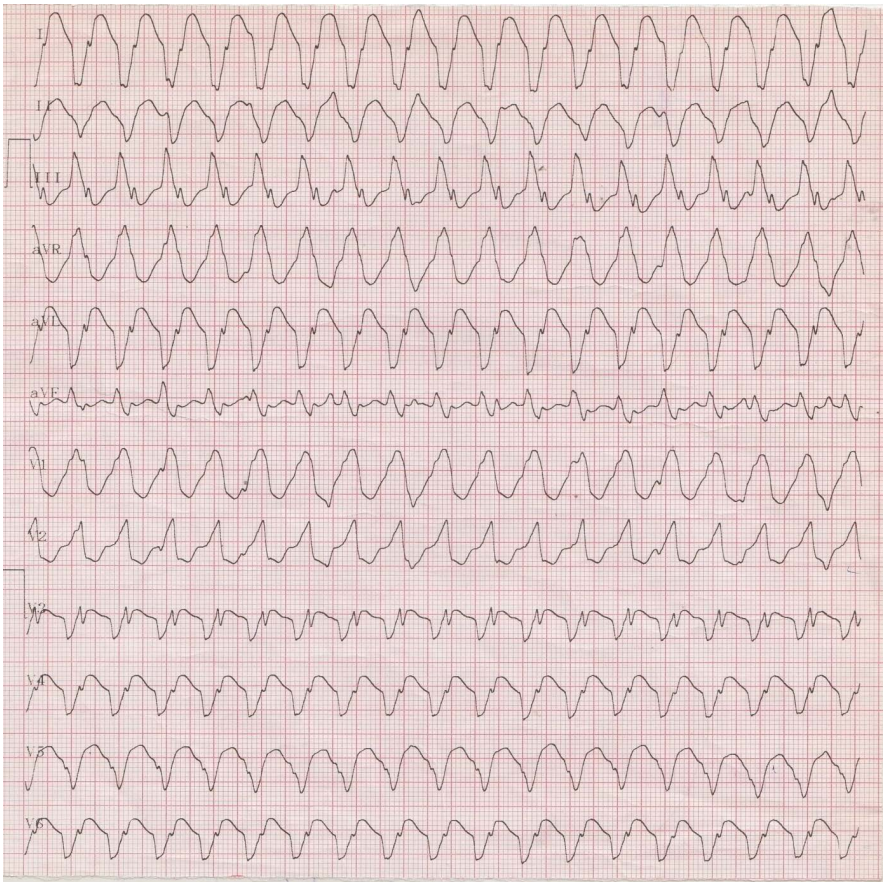

Figure 8. ECG showing ventricular tachycardia of left posterior septal origin.

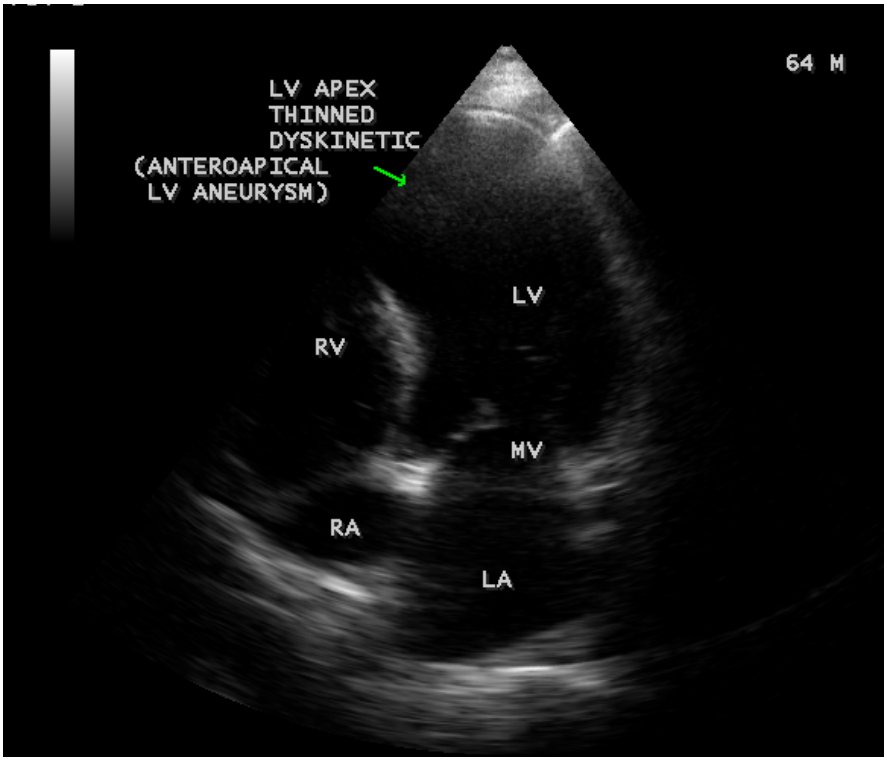

Figure 9. Apical four chamber view showing the thinned, dyskinetic anteroapical septum with aneurysm [22].

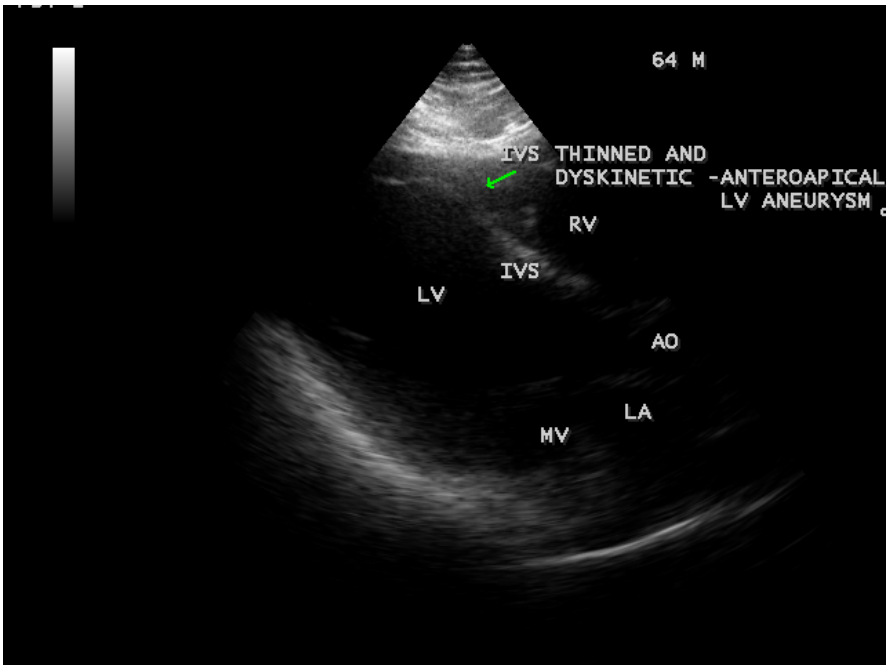

Figure 10. Parasternal long axis view showing the thinned out anteroapical septum.

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) develop within first 48 hours and represents 40% - 60% of cases and the

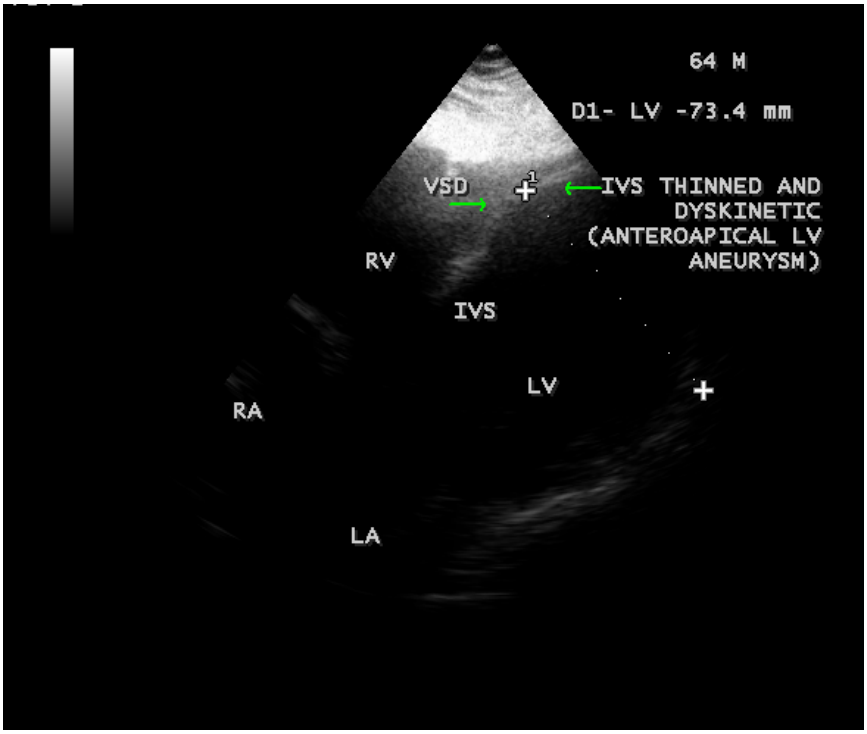

Figure 11. Tilted apical view showing the dilated, hypokinetic left ventricle.

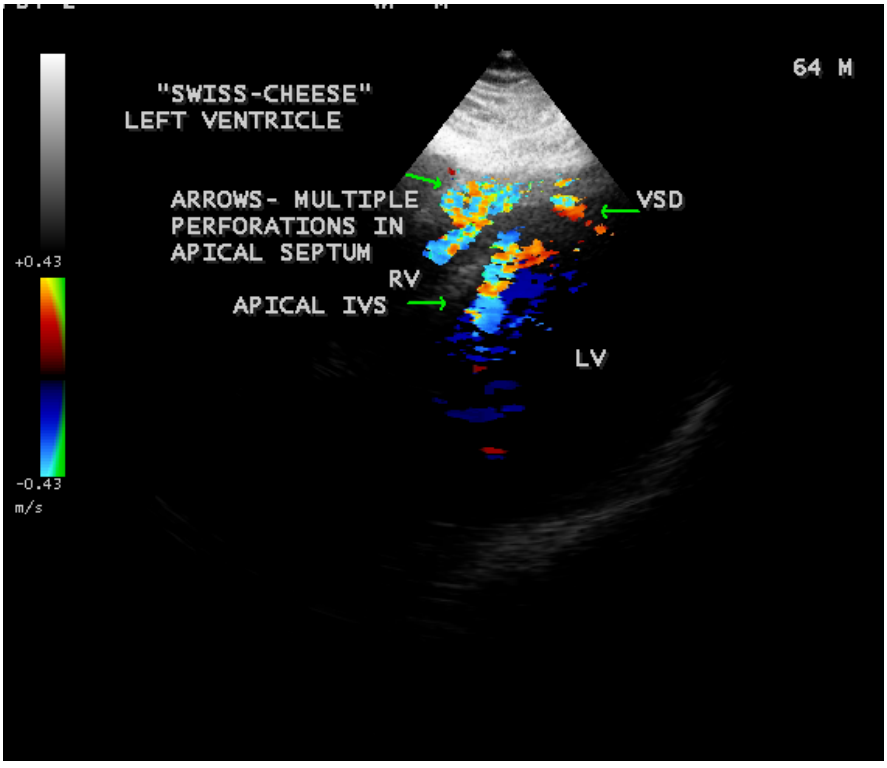

Figure 12. Tilted apical view showing the multiple perforations of the apical septum.

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

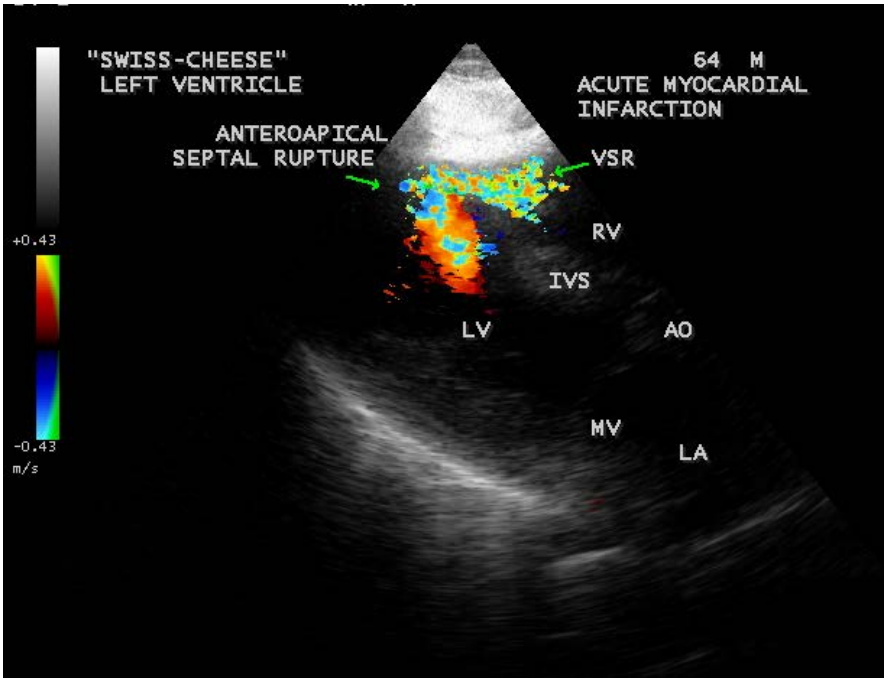

Figure 13. Parasternal long axis view showing the anteroapical ventricular septal rupture.

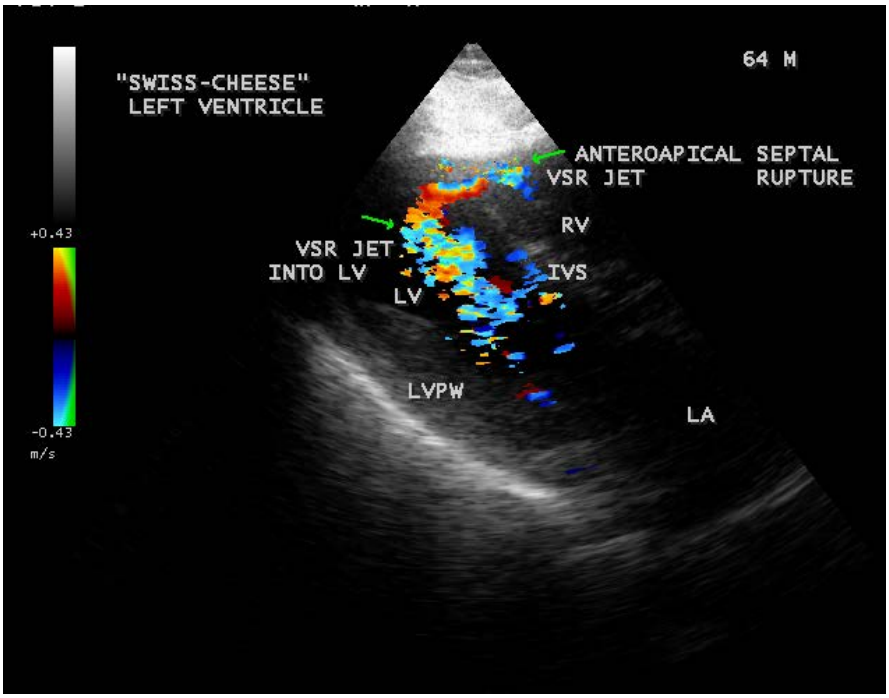

Figure 14. Parasternal long axis view showing the flow into the left ventricle through the septal rupture.

(systolic blood pressure > 150 mm Hg) 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 coughs. It is less severe, more compatible with survival with

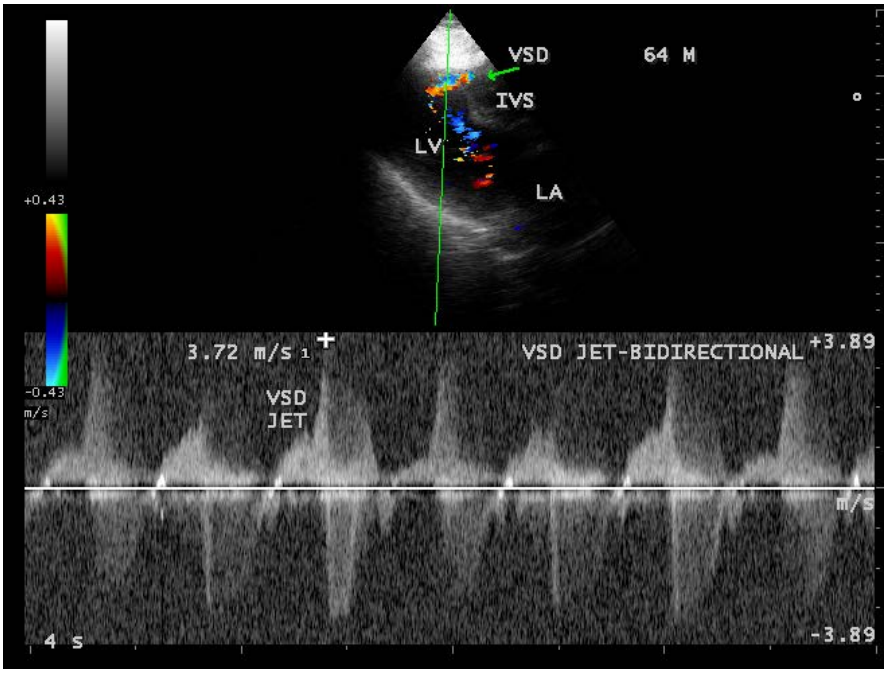

Figure 15. CW (Continuous Wave Doppler) showing the bidirectional jet of ventricular septal rupture (VSR).

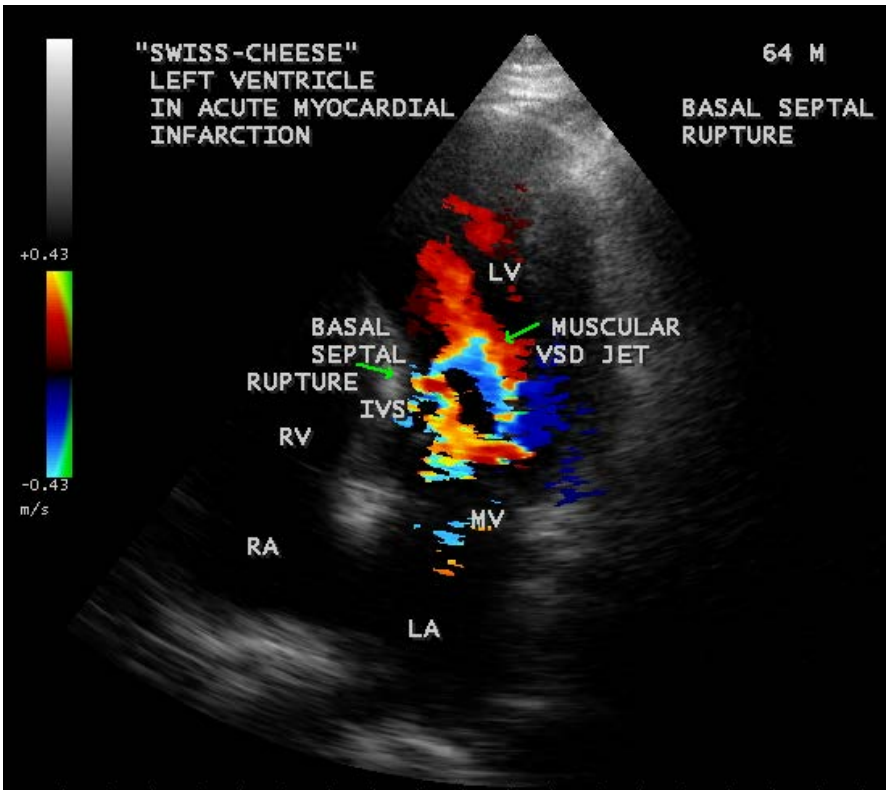

Figure 16. Apical four chamber view showing the basal septal rupture.

an anfractuous tract between the layers of the myocardium and may account for 30% of all cases of in-hospital free wall rupture.

Even though most of the ventricular ruptures occur in the free wall of the left

ventricle [23], the rupture occurs in the interventricular septum in approximately 15% to 20% of cases [24] and it complicates 1% - 2% of acute myocardial infarction presentations in pre-thrombolytic era [25]. The incidence has declined to about 0.2% in thrombolytic era and most contemporary series shown that it is

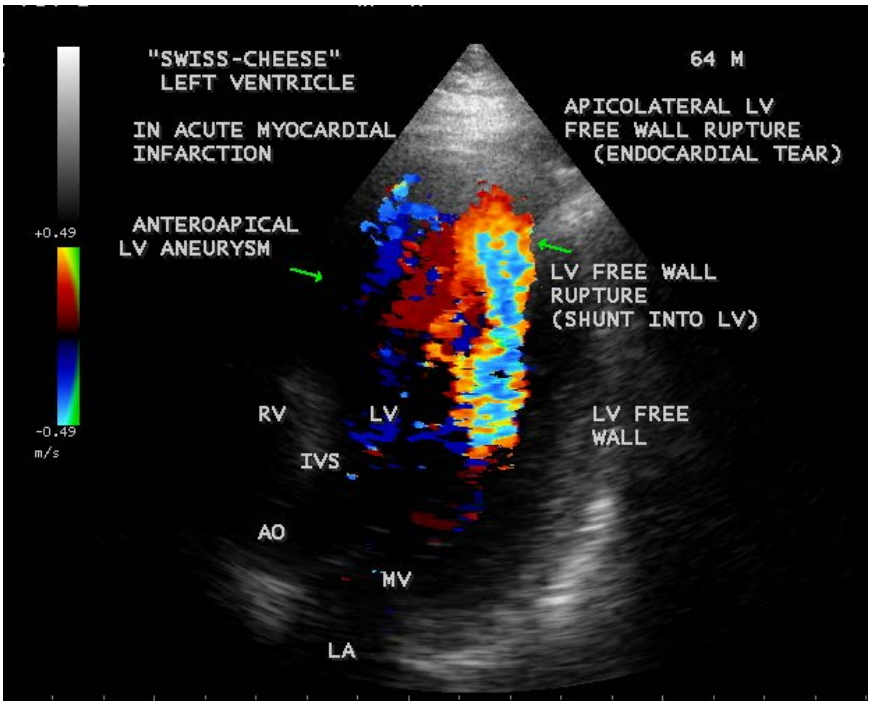

Figure 17. Apical 3 chamber view showing the apicolateral LV (left ventricular) free wall rupture.

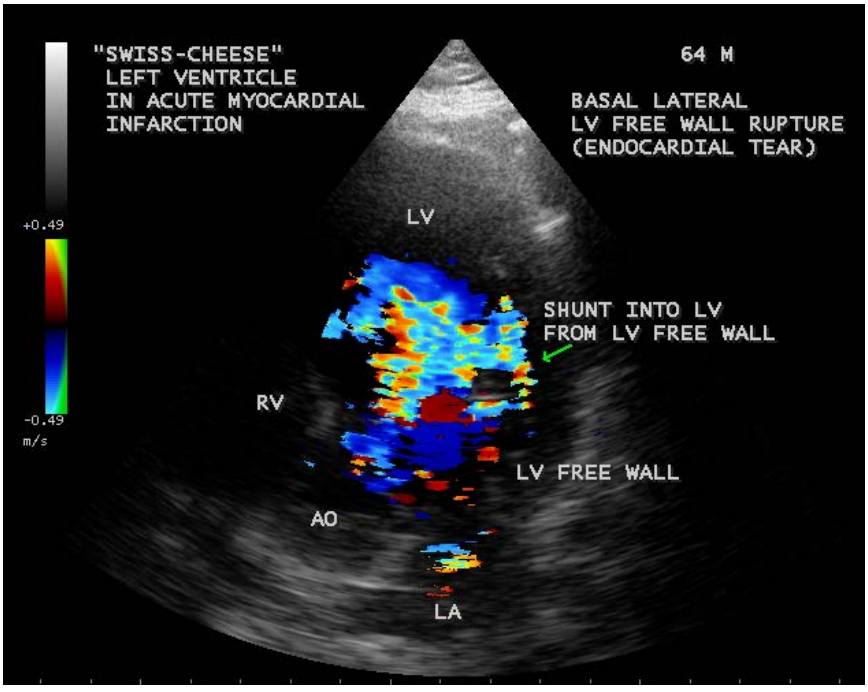

Figure 18. Apical 3 chamber view showing the basal lateral LV (left ventricular) free wall rupture.

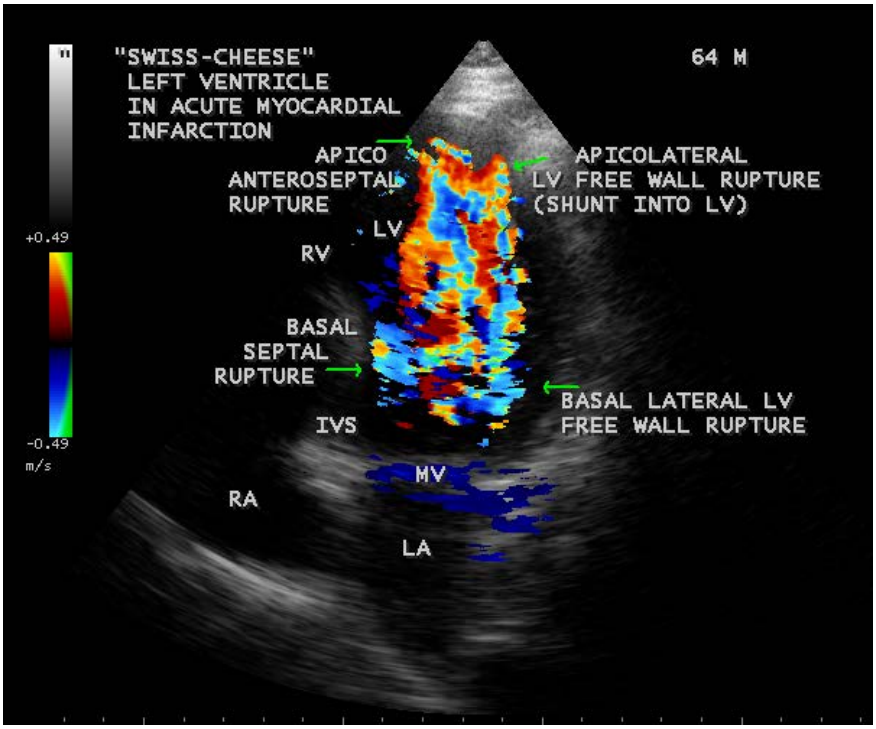

Figure 19. Apical 4 chamber view showing the "Swiss-cheese" left ventricle with multiple sites of ruptures.

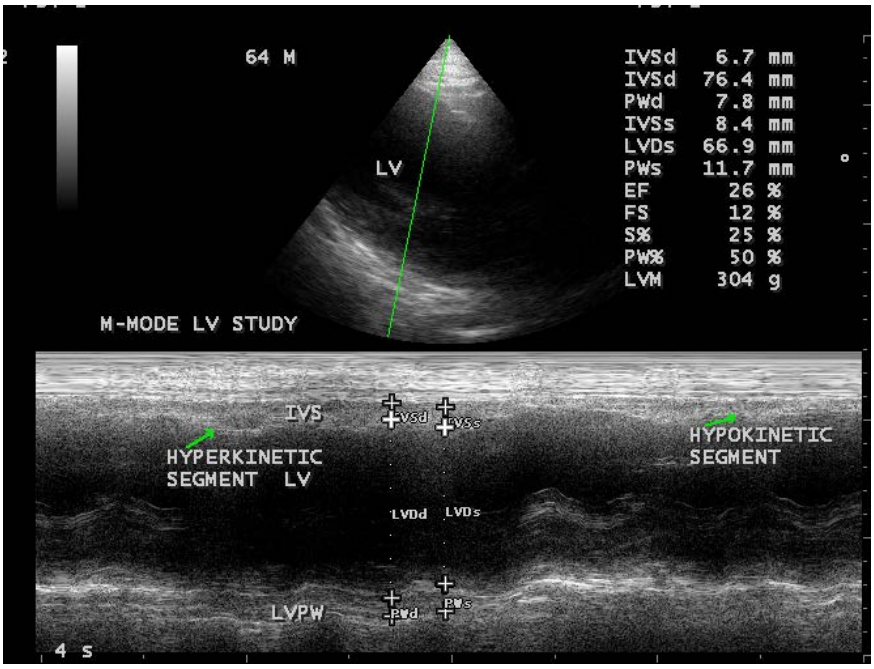

Figure 20. M-Mode LV study showing the contractile dysfunction of the left ventricle with an ejection fraction of 26%.

increasingly rare, complicating between 0.17% - 0.31% of patients presenting with acute myocardial infarction [26]. Two pathological types of ventricular septal rupture were described by Edwards *et al.* [27] in an autopsy report as simple ruptures defined as direct through-and-through defects and complex

Table 3. Risk factors for myocardial rupture.

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 [28] [29] Multi-vessel disease in inferior infarct rupture [30] Steroid use and late thrombolysis (>12 - 24 hours) [31] [32] [33]

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 12** 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 7** 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 9-11** [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.

3.1. 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. Postinfarction ventricular septal defects may be small or multiple or consists of linear tears as shown in **Figure 12**, **Figure 13**, **Figure 16** and coexisting with left free wall rupture as shown in **Figures 17**, **Figure 18** and it resembles the Emmantel Cheese, a yellow, medium-hard cheese, riddled with holes that originate in the area around Emmantel in Switzerland and so described as "Swiss-Cheese" left ventricle as in **Figure 19**. The shunt across the ventricular septal rupture is bidirectional as shown in **Figure 15**. The majority of shunt is towards the dilated, dyskinetic low pressure chamber of left ventricle as shown in **Figure 14** 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 19**, leading to contractile dysfunction with an ejection fraction of 26% as in **Figure 20** in this patient.

3.2. Management

3.2.1. 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 decrease 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 need 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 a prolonged attempt 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 uses of ACE inhibitors but not beta blockers were found to reduce the risk of VFWR (ventricular free wall rupture) [46].

3.2.2. 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 30 day 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 edges of the septal rupture fibrose sufficiently to support a percutaneous implantable occlusive device.

3.2.3. 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 ap-

proach 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.

3.3. 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 1-3** and a large RV free wall rupture in a 70-year-old male with right ventricular infarction was shown in

Figures 4-6.

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 performed in 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].

References

[1] Sanchez-Quintana, D., Garcia-Martinez, V., Climent, V. and Hurle, J.M. (1995) Morphological Changes in the Normal Pattern of Ventricular Myoarchitecture in the Developing Human Heart. *The Anatomical Record*, **243**, 483-495. https://doi.org/10.1002/ar.1092430411

[2] Figueras, J., Alcalde, O., Barrabes, J.A., Serra, V., Alguersuari, J., Cortadellas, J., *et al.* (2008) Changes in Hospital Mortality Rates in 425 Patients with Acute ST-Elevation Myocardial Infarction and Cardiac Rupture over a 30-Year Period. *Circulation*, **118**, 2783-2789. https://doi.org/10.1161/CIRCULATIONAHA.108.776690

[3] Sutherland, F.W.H., Guell, F.J., Pathi, V.L. and Naik, S.K. (1996) Postinfarction Ventricular Free Wall Rupture Strategies for Diagnosis and Treatment. *Annals of Thoracic Sur*gery, **61**, 1281-1285. https://doi.org/10.1016/0003-4975(95)01160-9

[4] Batts, K.P., Ackermann, D.M. and Edwards, W.D. (1990) Postinfarction Rupture of the Left Ventricular Free Wall: Clinicopathologic Correlates in 100 Consecutive Autopsy Cases. *Human Pathology*, **21**, 530-535. https://doi.org/10.1016/0046-8177(90)90010-3

[5] Becker, A.E. and Van Mantgem, J.P. (1975) Cardiac Tamponade. A Study of 50 Hearts. *European Journal of Cardiology*, **3**, 349-358.

[6] Perdigao, C., Andrade, A. and Ribeiro, C. (1987) Cardiac Rupture in Acute Myocardial Infarction. Various Clinico-Anatomical Types in 42 Recent Cases Observed over a Period of 30 Months. *Archives des Maladies du Coeur et des Vaisseaux Journal*, **80**, 336-344.

[7] Kumar, S., Kaushik, S., Nautiyal, A., Choudhary, S.K., Kayastha, B.L., Mostow, N., *et al.* (2011) Cardiac Rupture in Takotsubo Cardiomyopathy: A Systematic Review. *Clinical Cardiology*, **34**, 672-676. https://doi.org/10.1002/clc.20957

[8] Schneider, B., Athanasiadis, A., Schwab, J., Pistner, W., Gottwald, U., Schoeller, R., *et al.* (2014) Complications in the Clinical Course of Tako-Tsubo Cardiomyopathy. *International Journal of Cardiology*, **176**, 199-205. https://doi.org/10.1016/j.ijcard.2014.07.002

[9] Rodrigues, A.C., Guimaraes, L., Lira, E., Oliveira, W., Monaco, C., Cordovil, A., *et al.* (2013) Right Ventricular Abnormalities in Takotsubo Cardiomyopathy. *Echocardiography*, **30**, 1015-1021. https://doi.org/10.1111/echo.12215

[10] Finocchiaro, G., Kobayashi, Y., Magavern, E., Zhou, J.Q., Ashley, E., Sinagra, G., *et al.* (2015) Prevalence and Prognostic Role of Right Ventricular Involvement in Stress-Induced Cardiomyopathy. *Journal of Cardiac Failure*, **21**, 419-425. https://doi.org/10.1016/j.cardfail.2015.02.001

[11] Sung, J.-M., Hong, S.-J., Chung, I.-H., Lee, H.Y., Lee, J.-H., Kim, H.-J., Byun, Y.S., Kim, B.O. and Rhee, K.J. (2017) Rupture of Right Ventricular Free Wall Following Ventricular Septal Rupture in Takotsubo Cardiomyopathy with Right Ventricular Involement. *Yonsei Medical Journal*, **58**, 248-251. https://doi.org/10.3349/ymj.2017.58.1.248

[12] Akcay, M., Senkaya, E.B., Bilge, M., Yeter, E., Kurt, M. and Davotoglu, V. (2011) Rare Mechanical Complication of Myocardial Infarction: Isolated Right Ventricular Free Wall Rupture. *Singapore Medical Journal*, **52**, e7-e9.

[13] Willins, F.A. and Dry, T.J. (1948) A History of the Heart and Circulation. WB Saunders, Philadelphia.

[14] London, R.E. and London, S.B. (1965) Rupture of the Heart. A Critical Analysis of 47 Consecutive Autopsy Cases. *Circulation*, **31**, 202. https://doi.org/10.1161/01.CIR.31.2.202

[15] Krumbhaar, E.B. and Crowell, C. (1925) Spontaneous Rupture of the Heart. *American Journal of Medical Sciences*, **170**, 828. https://doi.org/10.1097/00000441-192512010-00005

[16] Van Torsel, R.A. and Edwards, J.E. (1972) Rupture of Heart Complicating Myocardial Infarction: Analysis of 40 Cases Including Nine Examples of Left Ventricular False Aneurysm. *Chest*, **61**, 104.

[17] Lautsch, E.V. and Lanks, K.W. (1967) Pathogenesis of Cardiac Rupture. *Archives of Pathology*, **84**, 264.

[18] Lee, H.M., Lee, Y.T., Kim, W.S., *et al.* (2013) Surgical Treatment of Post-Infarction Left Ventricular Free Wall Rupture. Three Cases Review. *The Korean Journal of Thoracic and Cardiovascular Surgery*, **46**, 357. https://doi.org/10.5090/kjtcs.2013.46.5.357

[19] Moreno, R., Lopez de Sa, E., Lopez-Sendon, J.L., Garcia, E., Soriano, J., Abeytua, M., *et al.* (2000) Frequency of Left Ventricular Free Wall Rupture in Patients with Acute Myocardial Infarction Treated with Primary Angioplasty. *American Journal of Cardiology*, **85**, 757-760. https://doi.org/10.1016/S0002-9149(99)00855-3

[20] Reddy, S.G. and Roberts, W.C. (1989) Frequency of Rupture of the Left Ventricular Free Wall and Ventricular Septum among Necropsy Cases of Fatal Acute Myocardial Infarction Since Introduction of Coronary Care Units. *American Journal of Cardiology*, **63**, 906-911. https://doi.org/10.1016/0002-9149(89)90137-9

[21] Hidlay, D. and Ward, R. (2016) Left Ventricular Free Wall Rupture. *Images in Clinical Medicine*, *Rhode Island Medical Journal*, **99**, 39.

[22] Feigenbaum, H., Armstrong, W.F. and Ryan, T. (2005) Coronary Artery Disease. In: Feigenbaum, H., Armstrong, W.F. and Ryan, T., Eds., *Feigenbaum's Echocardiography*, 6th Edition, Chapter 15, Figure 15.50, Lippincott Williams and Wilkins, Philadelphia, 470.

[23] Fencley, M., Chang, V.P.O. and Rourke, M.F. (1983) Myocardial Rupture after Acute Myocardial Infarction: Ten Year Review. *British Heart Journal*, **49**, 550-556. https://doi.org/10.1136/hrt.49.6.550

[24] Vlodacer, Z. and Edwards, J.E. (1977) Rupture of Ventricular Septum and Papillary Muscle Complicating Myocardial Infarction, *Circulation*, **55**, 815-822. https://doi.org/10.1161/01.CIR.55.5.815

[25] Birnbaum, Y., Fishbein, M.C., Blanche, C. and Siegel, R.J. (2002) Ventricular Septal Rupture after Acute Myocardial Infarction, *New England Journal of Medicine*, **347**, 1426-1432. https://doi.org/10.1056/NEJMra020228

[26] Moreyra, A.E., Huang, M.S., Wilson, A.C., Deng, Y., Cosgrove, N.M. and Kostis, J.B. (2010) Trends in Incidence and Mortality Rates of Ventricular Septal Rupture during Acute Myocardial Infarction. *American Journal of Cardiology*, **106**, 1095-1100. https://doi.org/10.1016/j.amjcard.2010.06.013

[27] Edwards, B.S., Edwards, W.D. and Edwards, J.R. (1984) Ventricular Septal Rupture Complicating Acute Myocardial Infarction. Identification of Simple and Complex Types in 53 Autopsied Hearts. *American Journal of Cardiology*, **54**, 1201-1204. https://doi.org/10.1016/S0002-9149(84)80067-3

[28] Skehan, J.D., Carey, C., Norrell, M.S., *et al.* (1989) Patterns of Coronary Artery Disease in Post-Infarction Ventricular Septal Rupture, *British Heart Journal*, **62**, 268-272. https://doi.org/10.1136/hrt.62.4.268

[29] Mann, J.M. and Roberts, W.C. (1988) Acquired Ventricular Septal Defect during Acute Myocardial Infarction: Analysis of 38 Unoperated Necropsy Patients and Comparison with 50 Unoperated Necropsy Patients without Rupture. *American Journal of Cardiology*, **62**, 8-19. https://doi.org/10.1016/0002-9149(88)91357-4

[30] Parry, G., Goudevenos, J., Adams, P.C., *et al.* (1992) Septal Rupture after Myocardial Infarction: Is Very Early Surgery Really Worthwhile? *European Heart Journal*, **13**, 373-382.

[31] Giugliano, G.R., Giugliano, R.P., Gibson, C.M. and Kuntz, R.E. (2003) Meta-Analysis of Corticosteroid Treatment in Acute Myocardial Infarction. *American Journal of Cardiology*, **91**, 1055-1059. https://doi.org/10.1016/S0002-9149(03)00148-6

[32] Becker, R.C., Charlesworth, A., Wilcox, R.G., Hampton, J., Skene, A., Gore, J.M. and Topol, E.J. (1995) Cardiac Rupture Associated with Thrombolytic Therapy: Impact of Time to Treatment in the Late Assessment of Thrombolytic Efficacy (LATE) Study. *Journal of American College of Cardiology*, **25**, 1063-1068. https://doi.org/10.1016/0735-1097(94)00524-T

[33] Honan, M.B., Harrell, F.E.J., Reimer, K.A., *et al.* (1990) Cardiac Rupture: Mortality and Timing of Thrombolytic Therapy, a Meta-Analysis. *Journal of American College of Cardiology*, **16**, 359-367. https://doi.org/10.1016/0735-1097(90)90586-E

[34] Rhydwen, G.R., Charman, S. and Schofield, P.M. (2002) Influence of Thrombolytic Therapy on the Patterns of Ventricular Septal Rupture after Acute Myocardial Infarction. *Postgraduate Medical Journal*, **78**, 408-412. https://doi.org/10.1136/pmj.78.921.408

[35] Honda, S., Asaumi, Y., Yamane, T., *et al.* (2014) Trends in the Clinical and Pathological Characteristics of Cardiac Rupture in Patients with Acute Myocardial Infarction over 35 Years. *Journal of American Heart Association*, **3**, e000984.

[36] Che, J., Li, G., Chen, K. and Liu, T. (2016) Post-MI (Myocardial Infarction) Free Wall Rupture Syndrome. Case Report, Literature Review and New Terminology. *Clinical Case Reports*, **4**, 576-583. https://doi.org/10.1002/ccr3.565

[37] Hayashi, T., Hirano, Y., Takai, H., *et al.* (2005) Usefulness of ST Segment Elevation in the Inferior Leads in Predicting Ventricular Septal Rupture in Patients with Anterior Wall Acute Myocardial Infarction. *American Journal of Cardiology*, **96**, 1037-1041. https://doi.org/10.1016/j.amjcard.2005.06.032

[38] Pollak, H., Diez, W., Spiel, R., Enenkel, W. and Mlczoch, J. (1993) Early Diagnosis of Subacute Free Wall Rupture Complicating Acute Myocardial Infarction. *European Heart Journal*, **14**, 640-648. https://doi.org/10.1093/eurheartj/14.5.640

[39] Seizer, A., Gerbode, F. and Kerth, W.J. (1969) Clinical, Hemodynamic and Surgical Considerations of the Rupture of the Ventricular Septum after Myocardial Infarction. *American Heart Journal*, **78**, 598-607. https://doi.org/10.1016/0002-8703(69)90511-0

[40] Schlesinger, Z., Lieberman, Y., Landesberg, A. and Neufeld, H.N. (1971) Repair of Ventricular Septal Defect and Left Ventricular Aneurysm Following Myocardial Infarction. *Thorax*, **26**, 615-618. https://doi.org/10.1136/thx.26.5.615

[41] Hill, J.D., Lary, D., Kerth, W.J. and Gerbode, F. (1975) Acquired Ventricular Septal Defects. Evolution of an Operation, Surgical Technique, and Results. *Journal of Thoracic and Cardiovascular Surgery*, **70**, 440-450.

[42] Lazopoulos, G., Manns-Kantartzis, M. and Kantartzis, M. (2009) Giant Left Ventricular Aneurysm and Intraventricular Septal Defect after Silent Myocardial Infarction. *Hellenic Journal of Cardiology*, **50**, 142-143.

[43] Richards, K.L., Hoekenger, D.E., Leach, J.K. and Blaustein, J.C. (1979) Doppler Cardiographic Diagnosis of Interventricular Septal Rupture. *Chest*, **76**, 101-103. https://doi.org/10.1378/chest.76.1.101

[44] Keren, G., Sherez, J., Roth, A., Miller, H. and Laniado, S. (1984) Diagnosis of Ventricular Septal Rupture from Acute Myocardial Infarction by Combined Two-Dimensional and Pulsed Doppler Echocardiography. *American Journal of Cardiology*, **53**, 1202-1203. https://doi.org/10.1016/0002-9149(84)90665-9

[45] Deja, M.A., Szostek, J., Widenka, K., *et al.* (2000) Post Infarction Ventricular Septal Defect—Can We Do Better? *European Journal of Cardiothoracic Surgery*, **18**, 194-201. https://doi.org/10.1016/S1010-7940(00)00482-6

[46] Chang, R.Y., Tsai, H.-L., Hsiano, P.-G., *et al.* (2016) Comparison of the Risk of Left Ventricular Free Wall Rupture in Taiwanese Patients with ST-Elevation Acute Myocardial Infarction Undergoing Different Reperfusion Strategies. A Medical Record Review Study. *Medicine*, **95**, e5308.

[47] Szkutnik, M., Bialkowski, J., Kusa, J., *et al.* (2003) Postinfarction Ventricular Septal Defect Closure with Amplatzer Occluders. *European Journal of Cardiothoracic Surgery*, **23**, 323-327. https://doi.org/10.1016/s1010-7940(02)00812-6

[48] Benton, J.P. and Barker, K.S. (1992) Transcatheter Closure of Ventricular Septal Defect: A Nonsurgical Approach to the Care of the Patient with Acute Ventricular Septal Rupture. *Heart and Lung*, **21**, 356-364.

[49] Landzberg, M.J. and Lock, J.E. (1998) Transcatheter Management of Ventricular Septal Rupture after Myocardial Infarction. *Seminars in Thoracic and Cardiovascular Surgery*, **10**, 128-132. https://doi.org/10.1016/S1043-0679(98)70006-1

[50] Holzer, R., Balzer, D., Amin, Z., *et al.* (2004) Transcatheter Closure of Postinfarction Ventricular Septal Defects Using the New Amplatzer Muscular Ventricular Septal Defect Occluder: Results of a U.S.(United States) Registry. *Catheterization and Cardiovascular Interventions*, **61**, 196-201. https://doi.org/10.1002/ccd.10784

[51] Fitz-Gibbon, G.M., Hooper, G.D. and Heggtveit, H.A. (1972) Successful Surgical Treatment of Postinfarction External Cardiac Rupture. *Journal of Thoracic and Cardiovascular Surgery*, **63**, 622.

[52] Montegut, F.J. (1872) Left Ventricular Rupture Secondary to Myocardial Infarction. *The Annals of Thoracic Surgery*, **14**, 75. https://doi.org/10.1016/S0003-4975(10)65202-2

[53] Nasir, A., Gouda, M., Khan, A. and Bose, A. (2014) Is It Ever Possible to Treat Left Ventricular Free Wall Rupture Conservatively. *Interactive Cardiovascular and Thoracic Surgery*, **19**, 488-493. https://doi.org/10.1093/icvts/ivu140

[54] Liebelt, J.J., Yang, Y., DeRose, J.J. and Taub, C.C. (2016) Ventricular Septal Rupture Complicating Acute Myocardial Infarction in the Modern Era with Mechanical Circulatory Support, a Single Center Observational Study. *American Journal of Cardiovascular Disease*, **6**, 10-16.

[55] Heitmiller, R., Jacobs, M.I. and Daggett, W.M. (1986) Surgical Management of Postinfarction Ventricular Septal Rupture. *The Annals of Thoracic Surgery*, **41**, 683-691. https://doi.org/10.1016/S0003-4975(10)63093-7

[56] Padro, J.M., Mesa, J.M., Silvestre, J., *et al.* (1993) Subacute Cardiac Rupture Repair with a Sutureless Technique. *The Annals of Thoracic Surgery*, **55**, 20-24. https://doi.org/10.1016/0003-4975(93)90468-W

[57] Cobbs, B.W., Hatcher, C.R. and Robinson, P.H. (1973) Cardiac Rupture: Two Long-Term Survivals. *JAMA* (*Journal of American Medical Association*), **223**, 532-535. https://doi.org/10.1001/jama.1973.03220050034008

[58] David, T.E. and Armstrong, S. (1998) Surgical Repair of Postinfarction Ventricular Septal Defect by Infarct Exclusion. *Seminars in Thoracic and Cardiovascular Surgery*, **10**, 105-110. https://doi.org/10.1016/S1043-0679(98)70003-6

[59] David, T.E., Dale, L. and Sun, Z. (1995) Postinfarction Ventricular Septal Rupture: Repair by Endocardial Patch with Infarct Exclusion. *The Journal of Thoracic and Cardiovascular Surgery*, **110**, 1315-1322. https://doi.org/10.1016/S0022-5223(95)70054-4

[60] Kawashima, D., Maeba, S., Saito, M. and Ono, M. (2016) Postinfarction Ventricular Septal Rupture Closure without Ventriculotomy. *Asian Cardiovascular and Thoracic Annals*. https://doi.org/10.1177/0218492316683758

[61] Piatek, J., *et al.* (2016) Non-Fatal Outcome after Left Ventricular Free Wall Rupture. *Annals of Surgery International*, **2**, 31.

[62] Marella, P., Hussein, H., Rajpurohit, N. and Garg, R. (2013) Leaking Heart: Ticking Time Bomb. *North American Journal of Medical Sciences*, **5**, 620-622. https://doi.org/10.4103/1947-2714.120802

[63] Moreno, R., Lopez Sendon, J., Garcia, E., Perez de Isla, L., Lopez de Sa, E., Ortega, A., Moreno, M., Rubio, R., Soriano, J., Abeytua, M. and Garcia-Fernandez, M.A. (2002) Primary Angioplasty Reduces the Risk of Left Ventricular Free Wall Rupture Compared with Thrombolysis in Patients with Acute Myocardial Infarction. *Journal of American College of Cardiology*, **39**, 598-603. https://doi.org/10.1016/S0735-1097(01)01796-X

[64] Bates, E.K. (2014) Reperfusion Therapy Reduces the Risk of Myocardial Rupture Complicating ST-Elevation Myocardial Infarction. *Journal of American Heart Association*, **3**, e001368.

[65] Gaudiani, V.A., Miller, D.G., Stinson, E.B., *et al.* (1981) Postinfarction Ventricular Septal Defect: An Argument for Early Operation. *Surgery*, **89**, 48-55.

[66] Serpytis, P., Karvelyte, N., Serpytis, R., Kalinauskas, G., Rucinskas, K., Samalavicius, R., *et al.* (2015) Postinfarction Ventricular Septal Defect: Risk Factors and Early Outcomes. *Hellenic Journal of Cardiology*, **56**, 66-71.