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Successful management of a case of left ventricular free wall rupture

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Abstract :
Cardiac rupture is a complication of myocardial infarction with an exceedingly high mortality rate. Imaging modalities, such as echocardiography, have facilitated premortem diagnosis, thus increasing chances of survival.
Keyword : Cardiac tamponade, Complications, Left ventricle, Myocardial infarction

Introduction:

Cardiac rupture is a complication of myocardial infarction with an exceedingly high mortality rate. We present a case report that details an episode of left ventricular free wall rupture and its successful outcome.

Casereport:

A 75-year-old woman presented to the emergency department complaining of severe retrosternal chest pain approximately 2 h in duration. The patient also reported three episodes of chest pain earlier that day, each of which lasted approximately 30 min before spontaneous resolution. The pain was described as ‘severe’ and ‘crushing’, radiated into his jaw and left arm, and was associated with nausea and diaphoresis. Her cardiac risk factors included history of hypertension and type 2 diabetes mellitus. Physical examination revealed an obvious distress with a blood pressure of 180/90 mmHg and a regular pulse of 86 beats/min. The electrocardiogram (ECG) demonstrated sinus rhythm, incomplete LBBB,ST elevation V1-V5 which prompted a diagnosis of acute myocardial infarction. Thrombolytic therapy was given, and symptom resolution and ECG normalization were achieved within 60 min of administration. Approximately 3 hours later, the patient suffered a recurrence of chest pain that was not relieved with nitroglycerin or morphine. A transient pericardial rub was noted on the next day following the myocardial infarction. On the morning of the third day, the patient developed severe chest pain, which she stated was distinctly different from that during previous episodes. She then underwent cardiovascular collapse and had an undetectable systolic blood pressure and distended jugular veins. An ECG revealed sinus tachycardia and electrical alternans. The patient was resuscitated with intravenous fluids and inotropes.

An urgent transthoracic echocardiogram was performed and demonstrated a diffusely hypokinetic left ventricle and a large quantity of circumferential pericardial fluid. Colour flow Doppler imaging failed to identify an area of rupture. The patient was subsequently investigated using left heart catheterization, which reinforced the transthoracic echocardiography findings and further demonstrated single-vessel coronary artery disease, a completely occluded left anterior descending. The patient was tentatively diagnosed with free wall rupture of the left ventricle with spontaneous seal.

The patient was brought to the operating room, where incision of the pericardial sac resulted in relief of cardiac tamponade and a marked improvement in blood pressure. The patient was placed on full cardiopulmonary bypass and the area of infarction was localized to the lateral wall of the left ventricle. No active bleeding was noted; however, a ventricular tear approximately 1 cm in length was identified immediately juxtaposed to the soft and necrotic area of the infarcted myocardium. A ventriculotomy was made through the ventricular tear and revealed that the area of infarction had extended to the posteromedial papillary muscle. An oversized Dacron patch was fashioned to include the area of infarct along with a surrounding 1 cm border of healthy myocardium. An interrupted, pledgeted, double-arm silk suture was passed circumferentially through the patch in an interrupted horizontal mattress fashion. The suture was then passed from the endocardial to the epicardial surface of the heart through noninfarcted tissue in an effort to provide a stable anchoring site. The Dacron patch was secured on the endocardial surface after tying the sutures on the outside of the heart. The ventriculotomy was subsequently closed with interrupted, double-arm, pledgeted silk sutures. Finally, the patient was weaned off cardiopulmonary bypass through the use of an intra-aortic balloon pump and moderate doses of inotropes. The patient had an uneventful postoperative course and was discharged 15 days following the operation.

DISCUSSION:

The most feared complication of myocardial infarction is that of cardiac rupture. Although it has an incidence of only 1% to 4% following a heart attack, it accounts for up to 20% of mortalities associated with infarction (1). The phenomenon was first reported by William Harvey in 1649; however, successful surgical repairs did not appear in literature until 1972 (2). Over 30 years later, reports involving patient survival following cardiac rupture still remain quite rare. The catastrophic consequences of rupture are the result of cardiac tamponade causing rapid hemodynamic collapse. Free wall rupture has been divided into two different forms on the basis of presentation, namely acute and subacute (3). The acute form (also referred to as blowout) involves cardiac arrest resulting from severe hypotension and electromechanical dissociation secondary to cardiac tamponade (4). The subacute form of free wall rupture (also referred to as oozing) does not involve cardiac arrest, but describes cases that consist of “moderate to severe pericardial effusion and hypotension of varied severity associated with sinus bradycardia or nodal rhythm” (5). In both acute and subacute forms of rupture, patients exhibit severe jugular venous distension associated with cyanosis in the neck and face. It is important to note that patients suffering from the subacute form of rupture may remain hemodynamically stable (4). The management of myocardial

infarction also influences the risk of cardiac rupture. A retrospective analysis found that rupture was more likely to occur following inadequate reperfusion of the infarcted area (5). This finding is supported by studies that have shown a reduced incidence of rupture with early thrombolytic therapy (6) and successful percutaneous coronary intervention (7). The reduced incidence of rupture associated with early reperfusion is attributed to its ability to halt the wave of necrosis extending through the myocardium, thus leaving an intact portion of epicardium (8). The structurally intact epicardium is thought to confer resistance, as evidenced by transmural infarction being a prerequisite for rupture (9). In contrast to early thrombolytic therapy, defined as less than 6 h following the onset of symptoms, early reports suggested that late thrombolytic therapy may actually increase the risk of rupture. The use of anti-inflammatory agents postinfarction, such as steroids and nonsteroidal anti-inflammatory drugs, has been suggested to increase the risk of rupture (10). The mechanism is thought to be related to an inhibition of fibroblast proliferation and connective tissue regeneration, resulting in impaired wound healing and the potential for infarct expansion. In contrast, the early use of angiotensin-converting enzyme (ACE) inhibitors has been suggested to be protective (11) – a concept supported by the ability of ACE inhibitors to lower the rate of postinfarction shock (12). Although successful salvage of cardiac rupture remains relatively rare, the increased use of imaging modalities, such as echocardiography, has led to earlier diagnosis, thus increasing the possibility of survival. This is especially true for the subacute form of rupture (13). However, even following prompt diagnosis, subsequent surgical management remains quite challenging for a variety of reasons. First, patients generally have marked hemodynamic instability and are therefore automatically considered to be within an extremely high -risk surgical population.

Conclusion:

Cardiac rupture continues to be a catastrophic complication of myocardial infarction. Transmural infarction secondary to acute total coronary occlusion appears to be a necessary substrate for rupture. Early revascularization through both thrombolysis and percutaneous coronary intervention has been shown to reduce its incidence. Once myocardial infarction is complicated by rupture of the left ventricular free wall, mortality remains exceedingly high, despite evolving diagnostic and surgical techniques. Therefore, the most effective therapy for cardiac rupture is likely its prevention through early revascularization.

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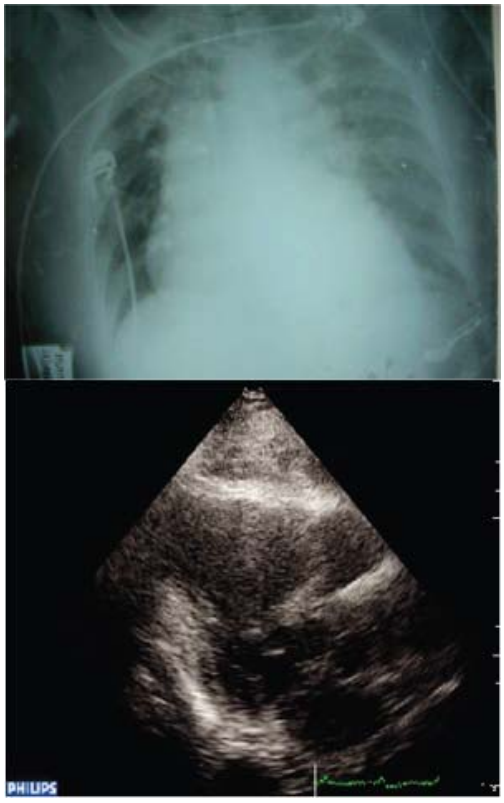
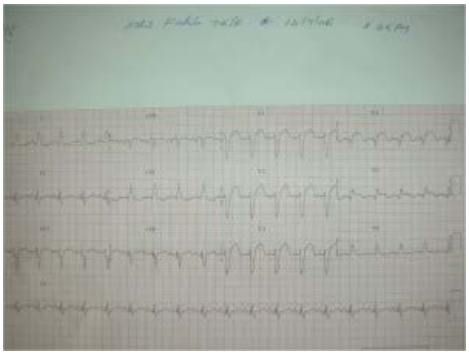
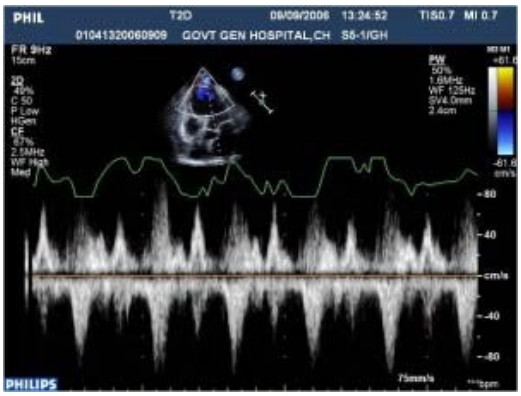
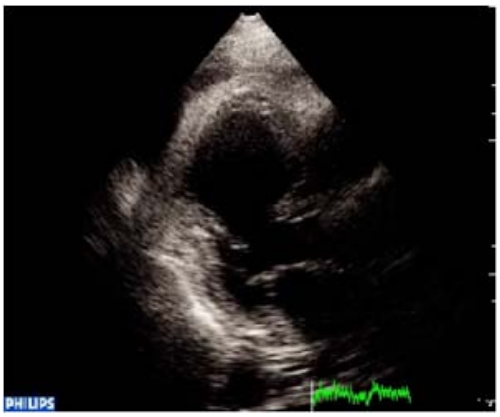


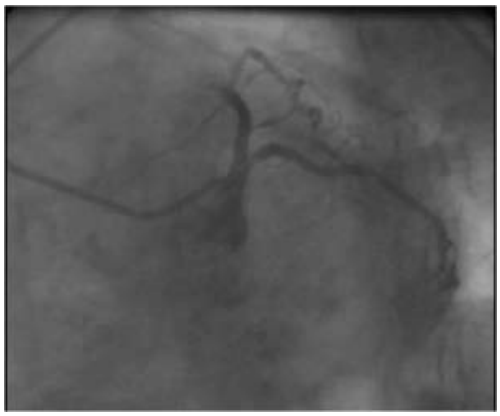
Free Wall rupture of left ventricle Doppler confirming the rupure



ECG







post operative picture
coronary angiogram showing 100% LAD occlusion