CASE REPORT

Successful surgical repair of an ischemic myocardial rupture

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CASE REPORT

A 57-year-old male patient who had a past medical history of hypertension and diabetes mellitus, had collapsed on the ground. He had mild chest pain and autonomic symptoms about two hours before the incident but had not sought medical advice.

He was immediately admitted to a nearby hospital within 30 minuts and found to have low blood pressures of 90/60 mmHg.

As there was a history of dengue fever among family members at that time the medical team suspected him to be in dengue shock and transferred him to National Hospital Colombo for further care. In the ETU it was found that there were some ischemic changes in ECG. The bedside USS scan of the plural, pericardial and peritoneal cavity was done to exclude effusions. It revealed a significant pericardial effusion with tamponade. An urgent 2D echocardiography by cardiology team (Figure 1) was done to further evaluate the effusion and drain it.

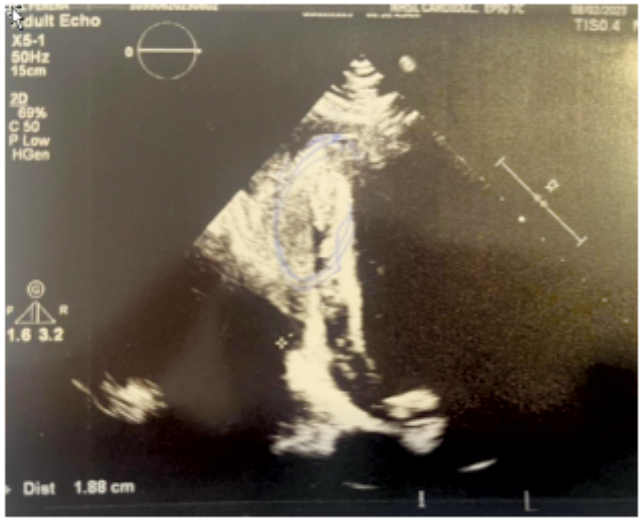

Figure 1. The image of the 2D transthoracic echocardiogram done on the patient. The rupture of the cardiac wall is clearly seen.

The cardiologist noticed the effusion with impending tamponade and echo findings further confirmed an inferior wall myocardial rupture. There was concomitant mild LVH and aortic stenosis.

Immediate surgery was planned and following hurried assessment and resuscitation, the patient was taken to the operating theatre.

An emergency sternotomy and release of the pericardial tamponade was done. A serpiginous rupture of the myocardium of the inferior wall of the left ventricle was noted. The left ventricle had mild hypertrophy with visible area of acute infraction. The ruptured myocardium was repaired under cardiopulmonary bypass. A reverse saphenous vein was grafted to the posterior descending artery (PDA), which is supplying that territory of the infraction. The left coronary arteries were disease free on intraoperative assessment.

The serpiginous myocardial rupture was repaired with 40 prolene sutures and reinforced with 40 mattress sutures with Teflon strips to distribute the tension to prevent cutting through the weakened myocardium (Figure 2). Tissue glue was applied externally to reinforce the repair.

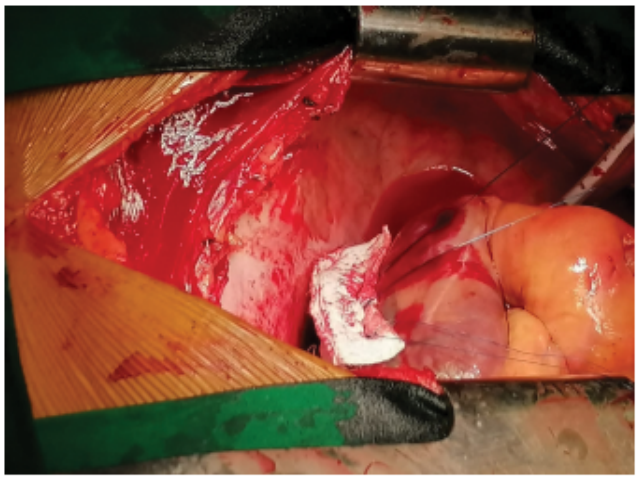

Figure 2. The apprreance of the repaird myocardial rupture area.

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After successful surgery, the patient was observed at ICU for 7 days and observed in HDU facility for up to 18 Days. A 2D echo cardiogram was done in post OP day 2 revealed no further pericardial effusion, no significant wall motion hypokinesia A CT coronary angiogram was done after 21 days of the surgery. The left coronary artery was normal, the right side RGSV graft was functioning well. The patient was discharged from hospital after 25 days of surgery.

DISCUSSION

Ischemic myocardial rupture is a rare complication of acute myocardial ischemia and infraction (MI). As per available data, myocardial rupture may play a significant part in early sudden deaths following acute MI[1]. In some instances, the prevalence may be more frequent than expected. The causes for sudden death following MI include infarction of a larger area of cardiac muscle leading to cardiac failure, intractable cardiac arrhythmias, conduction block, acute mitral regurgitation, pulmonary hypertension and pulmonary embolism. A sudden cardiac death can be easily attributed to one of these reasons. An acute ischemic rupture can go undetected as it needs to have postmortem findings, biopsy and imaging evidence.

There are multiple figures for the incidence and prevalence of myocardial rupture in the literature. The current overall incidence is 4%[2]. As per Rencuzogullari et al[5], about 1.98% of patients with STEMI had a complication such as ischemic myocardial rupture.

The VALIANT (Valsartan in Acute Myocardial Infarction) trial[3,4] and some other studies showed out of the sudden cardiac death occurred, 40% resulted from fatal MI or myocardial rupture.

In another study, ischemic myocardial rupture was presented in 30.7% (n-47) patients out of which 35 patients had no prior medical issues as per a retrospective study done (sample size n=147, 112 were male) by Hutchings et al[1].

According to this study males had only 20% chance to have a myocardial rupture following an ischemic event whereas females had 60%. Age was seen as an important depended factor for a rupture. In our case scenario the patient is a middle-aged male. The rupture was in the inferior wall. As per the same study, the frequency of the posteroinferior wall rupture is 38%. The anterior wall is the commonest site of myocardial rupture (45%). Lateral wall (9%), apex (6%). It is rare for right ventricle to undergo rupture (2%).

A single center study done by Becker et al[2] revealed the incidence of free wall rupture is decreasing over last few decades. The current overall incidence is 4%. When the data were stratified into 5-year blocks between 1977 and 2006, the incidence of rupture declined from >4% during 1977-1982 to <2% during 2001-2006.

The reason for this decline is due to progressive increase of reperfusion therapies, better control of blood pressure, using of beta blockers, ACEI and antiplatelet such as aspirin etc.[2].

Another possible reason is developing of better transport facilities to transfer patients to cardiac catheterization laboratories and cardiac surgery centers and utilization of imaging techniques over decades had offered the patient new opportunities to get diagnosed and intervene before a frank rupture and death occur.

The prognosis of a myocardial rupture is alarming. A 2020 multicenter retrospective study[9] (2001-2018) that reviewed surgical management of postinfarction left ventricular (LV) free-wall rupture and its early outcomes in 140 patients found a 36.4% operative mortality, with the main cause of death being low cardiac output syndrome. Postoperative myocardial re-rupture occurred in 10 patients (7.1%).

Independent predictors for early operative mortality were pre-procedure LV ejection fraction, cardiac arrest at presentation, female sex, and the need for pre-procedure extracorporeal life support (including extracorporeal membrane oxygenation [ECMO])[9].

A myocardial ischemia can ultimately be fibrosed and form a cardiac aneurysm. Sometimes a contained rupture can cause a pseudoaneurysm. Pseudoaneurysms are associated with high probability of re-rupture and sudden death. But some survivors have also being reported.

Ischemic myocardial rupture occurs acutely with a MI or even later after the infraction. The probability of recurrent MI or ischemic myocardial rupture tended to occur in the early post MI period most commonly within 1st month, and declined with the time[3,4].

1.98% of the STEMI patients of the study done by Rencuzogullari et al.[5] had ischemic myocardial rupture. They evaluated the relationship between myocardial rupture and patient's SYNTAX Score (SS) and SYNTAX Score 11 (SS11). The found SYNTAX score and SYNTAX Score 11 of myocardial rupture patients were significantly higher than those of patients without myocardial rupture.

Therefore, STEMI and patients of high SYNTAX scores are at high risk of having an ischemic myocardial rupture.

When concern the clinical presentation it is worth to notice that there is no specific symptom which can learn in contrast of a usual features of an acute MI.

They complain of moderate to severe chest pain. ECG changes of ischemia. They also present with symptoms similar to shock and tamponade. Most of the patient ultimately leads to a cardiac arrest[6]. Our patient also experienced giddiness and vertigo and collapsed on ground before he was hospitalized. As in our case a myocardial rupture case may miss initially as its presenting features overlap with other differential diagnosis and are of nonspecific to rupture itself.

In our case the initial contact medical team thought the patient was having dengue shock syndrome when he initially presented after collapsing at home as the entire family had dengue fever around that time.

On the other hand, myocardial rupture is likely not as rare as once believed. It is probably actually being remains undiagnosed as postmortems are not frequently done to evaluate these cases.

Diagnosis is a challenge. A rapid pattern recognition, early diagnosis and arrangement of immediate interventions and medical therapy can decide the patient's survival rate. One should have to have a high index of suspicion to not to miss an ischemic myocardial rupture. One of the best ways of diagnosing is left ventriculography during cardiac catheterization. This can be performed if the patient is stable hemodynamically will help visualization of contrast blush into the myocardium and pericardium.

Standard transthoracic echocardiography which we used in our case can occasionally demonstrate an intramyocardial hematoma and signs of a cardiac wall rupture. This technique in conjunction with an effusion and characteristics consistent with tamponade (including chamber collapse, dilated inferior vena cava and respiratory variations of valvular inflows by doppler analysis) increasingly suggests myocardial rupture.

The use of contrast echocardiography[7] can also aid in the diagnosis. A contrast enhanced CT study and cardiac magnetic resonance CMR has also being used in diagnosing cardiac rupture and further evaluate the possible mode of repair when the patients being stable.

Management of myocardial rupture

Even if the patient is stable and sometimes asymptomatic as in self-contained rupture, surgical repair is indicated. It's recommended to perform the emergency surgery at the best earliest slot with ongoing resuscitation.

As per some studies the interval between diagnosis of left ventricular free wall rupture (LVFWR) and surgery was 2.9 +/- 1.1 hours[10]. In our case it took 2 hours to start the surgery.

Patients can be supported with mechanically and medically. IV fluids, inotropes and vasopressors to maintain the patient stable at permissive hypotension. Extracorporeal membrane oxygenation ECMO, placement of intra aortic balloon pump IABP can provide initial mechanical support until the surgery is performed.

Surgical management is the preferred way. This includes strategies to provide stability to the myocardium. This is achieved by placement of biological glue-soaked prosthetic meshes, pericardial patches or applying sutures. In addition to the reinforcement and repair the surgeon may decide to remove the already infracted area of the myocardium(infractectomy). The defect is then closed with a patch placement and ventricular wall reconstruction.

Techniques of surgical repair

The repair can be done with CPB or without CPB. Several successful repairs of rupture on beating heart have been reported. Free wall rupture is usually repaired surgically by removing the infracted area and approximating the zone by Teflon or Dacron patches or by using biological glues.

When surgically treating an ischemic infraction, a CABG is often needed. This is more so when the rupture associated with ischemic VSD and MR. Over decades several different techniques have been developed to repair the rupture of ventricular wall[11]. Some of them are sutured techniques (ST), while some are sutureless techniques (STL). In our case we used a sutured technique to repair the rupture.

The older sutured technique carries the disadvantage of placing tying sutures through an already infracted, friable necrotic muscle. As these sutures can give away, a re-rupture can occur. But the availability of tissue adhesive and surgical glues have allowed repairing ischemic ruptures without tying sutures. In this method, a collagen mesh / sponge ex gelfoam or a prosthetic mesh soaked in tissue glue is placed over the ruptured area without using tying stiches[12,13].

The best surgical method to tackle this serious complication of post-acute MI is still in controversy. When considering the risk of re-rupture and post operative bleeding both methods are not superior to the other.

As per a recent review,[14] there is no statistically significant demarcation between the outcomes of the two surgical methods. Even the in-hospital mortality for both SL and STL techniques were comparable. Nevertheless, in the ST group a higher rate of operative mortality and post operative bleeding requiring re opening were seen. The incidence of myocardial re-rupture was seen high in STL group.

The differences in the mortality rates observed in between the two techniques are dependent on other variables too. The hemodynamic status of the patient at the time of presentation is one of them.

For some surgeon's sutures give some false reassurance than adhesive tissue glue on the cardiac wall rupture. This is evident as only a small number of patients with blowout ruptures underwent STL repair (14.8%) [14].

But as per some studies blowout ruptures of the cardiac wall is best approached in ST[12]. This is what we practiced in our case. When there is direct communication between the ventricular cavity and pericardial cavity, it is wise to believe that a mere glue patch may not withstand intraventricular pressure. Further studies are required to state which technique is superior.

The mortality rate of the surgical LVFWR at early stage have been reported to vary between 17.1% to 34.3% as per literature[14,15,16].

There is a place to CABG but there is no answer to the debate concerning the effect and usefulness of concomitant CABG to the culprit vessel in the setting of surgical repair of ischemic LVFWR.

Though survival benefits are unclear, Mantovani et al[11] found out that performing of a CABG has positive impact on survival and patients are free of angina. But many surgeons do not revascularize the infracted region as it is already dead. But other suspected coronary vessels are often grafted. In our case we grafted the culprit vessel. The CT coronary angiogram done after few weeks showed a well-functioning saphenous vein graft.

In an emergency situation similar to the index case, execution of a coronary angiogram is not possible most of the time. It is customary to perform a CABG to nearby suspected vessels. Therefore, the best way to tackle the issue is to perform a coronary angiogram before the surgery repair of the ischemic rupture if the patient is stable[11].The current recommendations by European Society of Cardiology and AHA American Heart Association guidelines are to achieve a temporary circulatory stabilization until the surgery by the mean of mechanical circulatory supporters such as IABP or ECMO[18,19].

The use of IABP providing mechanical support to reduce the afterload can prevent transition of an oozing area to a blowout rupture during the pre-operative period. Also, it has benefits of reducing the risk of low cardiac output syndrome after the surgery which is one of the commonest causes of death following myocardial rupture repair. We used IABP in our patient until the 3rd post operative day.

Though there is no clear evidence to support any benefit of MCS on survival, observations are there to that ECMO support represented an independent predictor of operative death.

CONCLUSIONS OF THE CASE DISCUSSION

Ischemic left ventricular free wall rupture is a serious complication of acute myocardial infraction. It is a challenging surgical situation. But surgical repair is possible with an early post operative mortality rate of 36.4%. Poor prognostic factors are female sex, low preoperative left ventricular ejection fraction, need of IABP, preoperative ECMO and cardiac arrest at presentation.

A CABG performing at the time of repair does not confer significant survival advantage.

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