Case Report

Non-traumatic Cardiac Tamponade : Two Autopsy Case Reports

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Cardiac tamponade due to free cardiac wall rupture is a medical emergency with almost 100% mortality. Sudden death can occur in a patient after myocardial infarction due to rupture of myocardium. But in certain cases, the patient dies suddenly without any specific cardiac symptoms. Only at the autopsy, the cause of death is found to be cardiac tamponade due to rupture of free wall, which has undergone ischemic necrosis. We are describing two cases of sudden death where death occurs due to free cardiac wall rupture after few non-specific symptoms. So the general physician should have high index of suspicion of cardiac diseases from nonspecific symptoms and refer the patient for further intervention.

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Cardiac tamponade is defined as collection of blood or fluid in the pericardium leading to compression of the heart caused by increased intra pericardial pressure[1]. The extra collection of fluid in pericardial space (Normally 15-50 ml)[2] is called pericardial effusion. When the collection is a chronic process, the pericardial sac can accommodate around 2000 ml of fluid due to adaptive stretching of pericardium but sudden accumulation of 200 ml of fluid can produce a critical state, called cardiac tamponade[2].

The causes of cardiac tamponade can be traumatic or non-traumatic. The traumatic causes may be either blunt or penetrating injuries to the heart, iatrogenic, post cardiac biopsy and transseptal rupture at cardiac catheterization. The non-traumatic causes include myocardial rupture after myocardial infarction, myocardial aneurysm, pericarditis, cancer, uremia and aortic dissection[3].

The myocardial wall usually fibroses & thinned out due to scarification after single or repeated episode of ischemia. At this weaker point, the myocardium ruptures leading to tamponade & sudden death which is many times diagnosed only at autopsy.

Case Report - 1

A 47 year old male school bus driver was found dead in the morning hour in his bathroom. The body was found in a prone position with his head leaned on the bathtub. He was healthy without known history of any disease except complain of intermittent epigastric discomfort, which was treated in line of gastritis but there was no history of hematemesis or melena. He was a chronic smoker but nonalcoholic. His wife said that he was apparently healthy last night. His elder brother and his father died at young age but she did not know the cause of death.

The deceased was well-nourished, muscular (168cm, 72kg). Postmortem lividity was fixed on back and rigor was well developed without evidence of any external injury.

The pericardium was intact containing about 600 ml of fresh blood & clot (Fig 1a). The heart (470 g) was hypertrophic with extensive transmural infarction of left ventricle. A perforation of 1.5 cm diameter was found at about 2 cm from the apex (Fig 1b). The left anterior descending artery show severe atherosclerotic change with focal area of thrombosis and complete luminal occlusion measuring about 0.5 cm in length and situated 2.5 cm from the coronary ostium. The left ventricular wall thickness was 18 mm. The infarcted area was ulcerated & necrosed measuring around 10mm in thickness. The left circumflex and right coronary artery also showed evidence of atherosclerosis with 50-60% occlusion. The aorta showed multiple atheromatous plaques that mainly affected the abdominal segment, which was mildly ulcerated. Stomach was intact and empty without any characteristic odour, mucosa congested.

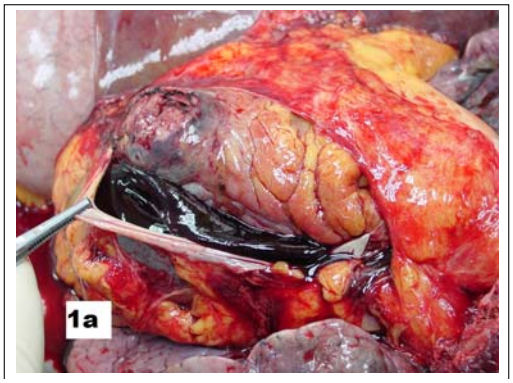

Fig 1a — Blood clot in the pericardial sac with perforated left ventricle

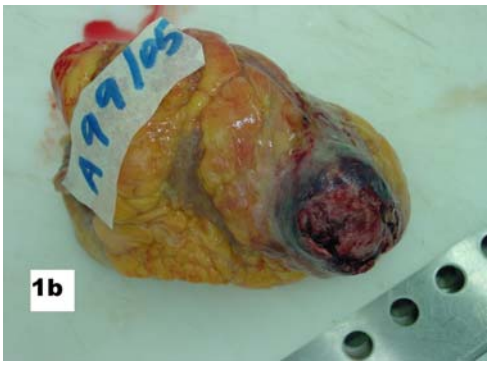

Fig 1b — Perforation of left ventricle

On histopathological study of heart, focal areas of hemorrhage, degeneration and necrosis of myocardium was found with mild infiltration by lymphocytes and neutrophils. Left anterior descending artery revealed severe atherosclerosis with complete occlusion and evidence of organized thrombus.

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Case Report - 2

A 43 year old unmarried female had history of fever for 2 days prior to her death. She also complained of chest pain and dizziness and sought treatment at a local GP clinic. At about 4 pm on the same day, she was woken up from sleep by her mother, but almost immediately collapsed upon getting up from the bed. As per the history narrated by her mother, the deceased had a similar episode 3 to 4 years ago, but cured after treatment. The deceased was unmarried and doing odd jobs. Due to financial issues, she was not able to continue the treatment. She was unable to tell either the kind of treatment given or produce any medical records.

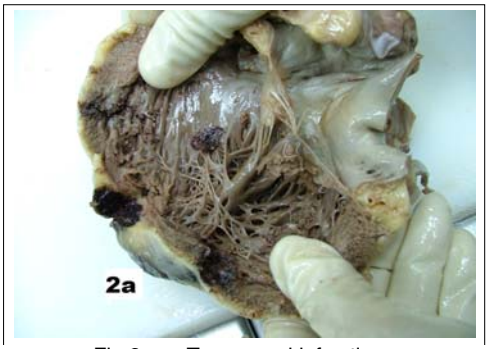

Fig 2a — Transmural infarction, haemorrhage in the myocardium

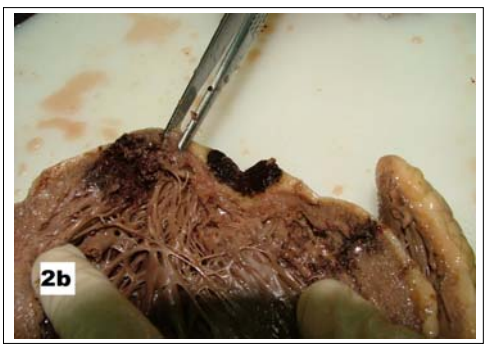

Fig 2b — Left ventricular hypertrophy

She was obese (80kg), measuring 157cm in length. Postmortem lividity was fixed on back and rigor mortis was well developed. No external injuries were detected.

The pericardium was intact and contained about 200ml of clotted blood. The heart (465 g) was hypertrophic. The pericardial fat and epicardium showed an area of hematoma measuring 4cm x 3cm at the apex. Cut section shows a transmural area of pallor and hemorrhage (infarction) of the left ventricular myocardium near apical region extending from the endocardium (Fig 2a). The left ventricular wall (Fig 2b) was slightly thickened (1.8cm thick). The left anterior descending artery showed moderate atherosclerosis with total occlusion of proximal 1/3rd by thrombosis. The right coronary artery showed mild atherosclerosis but was patent. Stomach was intact and empty.

On histopathological study of heart, focal areas of hemorrhage, degeneration and necrosis of myocardium was found with mild infiltration by lymphocytes and neutrophils. There was diffuse extravasation of blood into the pericardial fat. Left coronary artery showed atheromatous plaques comprising of histiocytes and lymphocytes with cholesterol clefts and micro calcifications on the intimal layer of the artery, with thrombosis causing significant to total occlusion of the artery.

In both cases, the cause of death was opined as cardiac tamponade due to myocardial rupture post myocardial infarction.

Discussion

Risk factors for free cardiac wall rupture include age >60 years, female gender, pre-existing hypertension and lack of left ventricular wall hypertrophy[7]. The lateral wall, at midventricular level, is the most common site for post-infarction free-wall rupture[7].

The most common cause of myocardial rupture is a recent myocardial infarction, with the rupture occurring three to five days after infarction[4]. Left ventricle free wall rupture occurs in 2% to 4% of patients following acute Myocardial Infarction (MI)[5]. In the modern era of early revascularization and intensive pharmacotherapy for MI, the incidence of myocardial rupture is about 1% of all MI[6].

The free wall rupture is associated with immediate hemodynamic collapse and death secondary to acute cardiac tamponade. The overall risk of death depends on the speed of diagnosis, treatment provided and the underlying cause of the tamponade. Though in one case series, it was described that the mortality rate was 100% if myocardial rupture involved the free wall of Left Ventricle (LV)[6].

But another study[8] describes, in some patients who survive LV free-wall rupture following Acute Myocardial Infarction (AMI), the rupture can be sealed by epicardium (visceral pericardium) or by a hematoma on the epicardial surface of the heart. This has been referred to as LV diverticulum or contained myocardial rupture. Pericardiocentesis and surgical drainage of hemopericardium may be indicated[9] or referred for urgent cardiac surgery (infarctectomy and Teflon patching)[10].

But sometimes, sudden death due to (LV free-wall rupture may be the first manifestation of Coronary Artery Disease (CAD) in a small percentage of patients with AMI[9]. The cases described here were presented with very nonspecific symptoms. The autopsy & histopathological findings indicate repeated ischemic episodes of myocardium. Though none of the deceased ever complained of severe chest pain, which could had led to diagnosis of acute myocardial infarction.

So the physicians should have to suspect for myocardial ischemia from nonspecific symptoms like recurrent epigastric distress in a hypertensive (first deceased) and chest discomfort with dizziness and history of syncope as in the second case.

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