*Case Report*

# Death due to RTA injury or Cardiac Pathology? - A case report

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**Abstract**

An elderly male following a road traffic accident (cyclist vs motorbike) presents with a history of loss of consciousness for the variable period and weakness of all limbs, sustained injury to the head lower back hip, and neck region, and diagnosed as traumatic quadriparesis and ? cervical cord injury. With no apparent injury to the chest. Initial CT findings show signs of cervical spondylosis and paraspinal cervical spasm. The patient responded for initial treatment but the patient got discharged against medical advice. After 15 days he came back to the hospital with a history of breathlessness and chest pain and succumbed on the same day. The post-mortem examination revealed a rupture of the left ventricle with cardiac tamponade. After the post-mortem report investing officer raised the question of can the death may have been caused due to RTA?

***Key Words*:** RTA, Rupture of heart, Cardiac tamponade, Myocardial infarction.



## Introduction:

Hemo-pericardium due to ventricular free wall rupture as an immediate complication of myocardial infarction is a rare cause of sudden death. Cardiac tamponade due to blunt chest trauma is a rare entity and an unusual consequence. Myocardial rupture is a complication of acute myocardial infarction that directly causes death in 8% of patients[1]. Sudden cardiac death in different circumstances gives different views/pictures leading to the construction of a false manner of death. Here is a case in which the cause of death was allegedly attributed to a recent road traffic accident but turns out to be a natural death.

## Case report:

A 70-year-old man came to the casualty dept with a history of chest pain and breathlessness for 3 hours and a history of a road traffic accident 15 days back, in which he said to be sustained injuries to the head, neck, lower back, hip, bleeding from mouth and weakness of all limbs. On examination, there were no injuries to the chest and ECG reveals acute coronary insufficiency, extensive myocardial infarction, monomorphic ventricular tachycardia, cardiogenic shock with no recordable blood pressure, and feeble pulse. The patient succumbed in less than 30 mins. An autopsy was conducted later.

The relatives and police attributed his cause of death to the road traffic accident and the case was booked under unnatural death. Accordingly, the body was subjected to autopsy. The postmortem examination revealed he was an elderly male, moderately built and nourished. An abrasion was noted over the right great toe which was covered with scab fallen at places. A surgically sutured wound measuring 2 cm length was noted over the left side of the frontal region with an abrasion surrounding it. On internal dissection, the cranium, spinal cord, and

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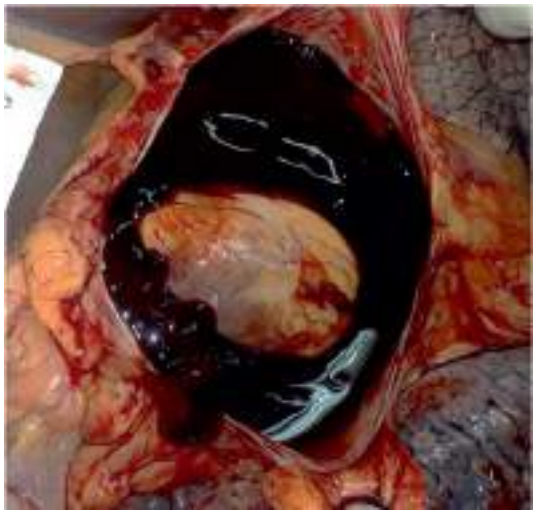
abdominal cavities were intact. The distended pericardium was seen in the thoracic cavity. On opening, the pericardium 350 ml of blood and clots (Figure 1) were retrieved from the pericardial sac. The heart was removed and examined, which was enlarged weighing 500 g and white fibrotic patches were seen on the surface of the heart over the anterior aspect of the right ventricle and posterior aspect of the left ventricle. There was a rupture measuring 0.5 cm x 0.5 cm x cavity deep present over the posterior wall of the left ventricle (Figure 2). The left ventricular wall was 3 cm in thickness and coronaries were thickened, calcified, and narrowed by 85-90% by atheromatous plaques.

**Figure 1: Showing pericardium filled with blood and clots**



The dissected heart, pieces of both lungs, and kidneys were subjected for histopathological examination. The histopathological examination of the heart revealed hypertrophied myocytes (Figure 3) with the intracellular cytoplasmic accumulation of lipofuscin (Figure 4), with disarray in arrangement and focal area showing waviness of myocytes (Figure 5) with necrosis and hemorrhage. Microscopy

**Figure 2: showing rupture measuring 0.5 cm x 0.5 cm x cavity deep present over the posterior wall of the left ventricle**

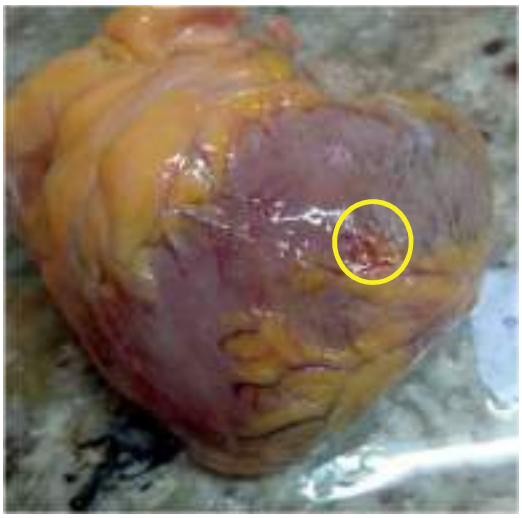


**Figure 3: Showing myocytes which are hypertrophied**

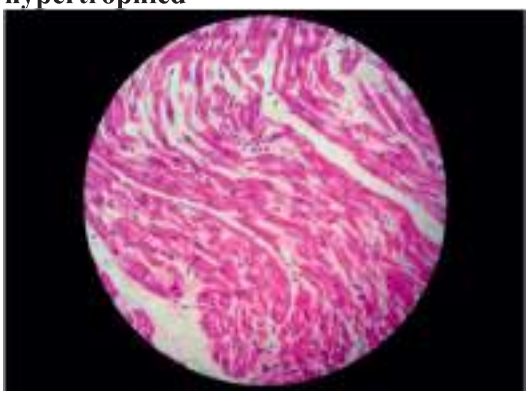


**Fig.4 showing intracellular cytoplasmic accumulation of lipofuscin**

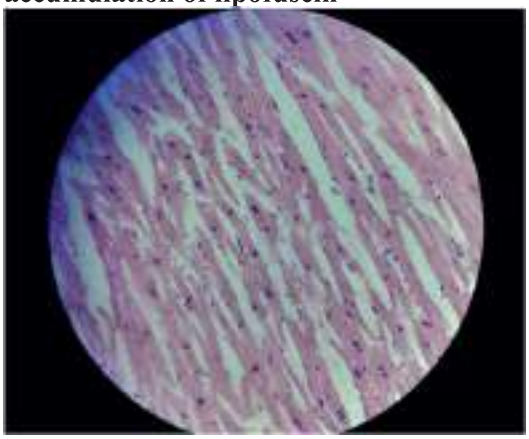
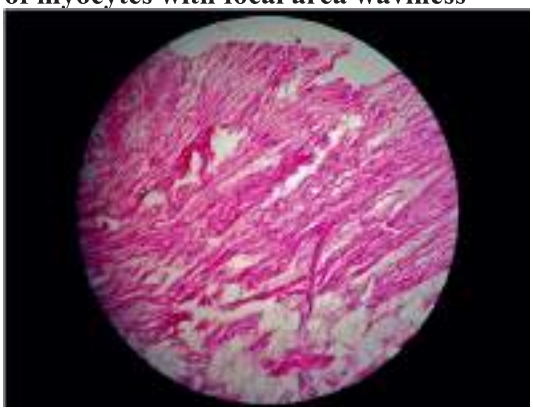


**Fig. 5 showing a disarrayed arrangement of myocytes with focal area waviness**



of lung and kidney revealed pulmonary edema and one kidney showed congestion and another showed features of chronic pyelonephritis with a simple cyst. Impression of left ventricular hypertrophy with myocardial infarction and rupture of the left ventricular wall was made from histopathology. Accordingly, the cause of death was opined as "death is due to cardiac tamponade due to rupture of heart wall as a consequence of myocardial infarction".

But investigating officer raised a query as to the manner of death, questioning, is this rupture of the heart is a consequence of road traffic accident injuries, which were sustained by the deceased 15 days back?

To analyze the case, the case history and records of the incident of road traffic accident was referred. The patient was admitted with an alleged history of a road traffic accident with a history of injuries to the head, lower back, hip, and neck region 15 days back. There was a history of loss of consciousness for a variable period, weakness in all limbs, neck pain, and bleeding from the mouth. At the time of admission, the initial examination of the patient reveals patient was conscious, oriented, his vital parameters were normal, oxygen saturation was 98% and GCS (Glasgow Coma Scale) was E4V5M3 with motor power of 3/5 in all the limbs. Swelling over the right foot with abrasion and small abrasions noted over the left side of the cheek and lower lip. There were two puncture wounds over the centre of the forehead and loosening of lower incisor teeth were also found. He gave no history of trauma to the chest and abdomen or complaints related to chest and abdomen. He was non-hypertensive and not a diabetic. But he was a smoker for the last 40 years. There was no history of chest pain, ischemic attacks, etc. CT scan of head and neck revealed the presence of scalp hematoma over the frontal region, straightening of the cervical spine, growing osteophyte, and reduced disc space between cervical 6th and 7th vertebrae suggestive of paraspinal cervical spasm and cervical spondylosis. ECGs done during his first stay in the hospital were within normal limits. Accordingly, the patient was diagnosed to be suffering from traumatic quadriparesis and was treated conservatively. His general condition was improving during the stay in the hospital, however, the patient got discharged against medical advice after 7 days of treatment.

As per the history from relatives, he was recovering in the home after discharge from the hospital. He was regaining strength in all limbs. On the day of admission to the hospital the second time, that is after 14 days of road traffic accident, he suddenly developed chest pain and breathlessness.

**Discussion:**

Myocardial rupture is an early complication of acute myocardial infarction with a bimodal peak of incidence (within 24 hours and 3-5 days) range being 1-14 days. It may present as ventricular free wall rupture, papillary muscle rupture, or ventricular septal rupture[2].

The ruptured heart is the most common cause of hemopericardium and cardiac tamponade, the rupture always occurring through an infarct. Left ventricular rupture is the 2nd leading cause of death among patients with acute myocardial infarction[3]. Rupture of the free wall of the heart results in hemopericardium, abrupt hemodynamic deterioration due to cardiac tamponade, and

usually death within a very short time following rupture[4].

The traditional risk factors of left ventricular free wall rupture are older age, female sex, previous hypertension, and the first lateral of anterior wall acute myocardial infarction[5].

Ischemia and myocardial infarction due to acute coronary insufficiency are well-known complications of atherosclerosis of coronary arteries, a chronic disease of blood vessels. Such massive myocardial infarction involving a large area of the heart is likely to end up in rupture of the affected area. Because of this, the blood gets collected in the pericardial sac, which causes impedance to cardiac filling and is fatal on most occasions. Atherosclerosis is a long-standing and gradually progressive disease of coronary arteries that is asymptomatic in many of the patients.

Delayed cardiac rupture often occurs after blunt trauma to the chest. The possible causes of delayed cardiac rupture include ischemic changes after cardiac contusion and fracture of the ribs. A diagnosis of delayed cardiac rupture should be considered if patients have a history and evidence of chest trauma. Arrhythmias and conduction defects are the most common complications of cardiac contusions. Myocardial contusion is usually associated with transient right bundle branch block and rarely with left bundle branch block.

Getz in their case report gives signs to identify cardiac rupture in traumatic chest injury cases. According to him, out of proportion or unresponsive hypotension and hypovolemia, unremitting haemothorax, persistent metabolic acidosis, elevated CVP or distended neck veins with hypotension[6] suggest cardiac wall rupture.

Williams and others in their case report correlate signs with the incidences of signs. In which 100% hypotension, 90% elevated CVP or distended neck veins, 92% confused or coma, 70% systolic BP <80mm Hg, 78% CVP >20cm H2O7. Beck's triad: hypotension, neck distended or elevated CVP, muffled heart sound[7].

In a case report by Fenton et al, a 38 year-old-man involved in a motor vehicle accident was intubated. His 7th rib had fractured with bilateral pleural fluid. During posterolateral thoracotomy, pericardial laceration was noted. It was diagnosed as bichamber rupture (right atrium and ventricle)[8].

In a case report by M.F. Dunsire et al, a 14 year-old-girl with a history of blunt trauma to the abdomen presented with epigastric pain, tachycardia, arterial pressure of 95/65 mm Hg. Later she developed hypotension and increased tachycardia and increased CVP. It was diagnosed as ruptured right ventricle and cardiac tamponade[9].

In a case report by Roth et al, a 33-year-old male suffered a blunt chest trauma following which he suffered multiple rib fractures along with perforation of the left ventricle[10].

In a case report by Mehta et al, a 25-year-old male presented to the emergency ward with an alleged history of fall from a running train, following which he sustained blunt chest and abdomen trauma along with head injury. He had fractures of the 2nd, 3rd and 4th ribs with pneumothorax on the right side with 6-8 cm vertical rent over the anterior inferior wall of the right ventricle[11].

In a case report by Yun-Ju-yang et al, a 27-year-old female had attempted suicide with a knife. A single stab wound to her anterior chest wall and some cuts to her left wrist were noted. Chest x-ray showed no pneumothorax. Toxicological screening analysis showed the presence of benzodiazepines. Cardiac enzyme analysis showed elevated troponin I and CKMB and ECG revealed sinus tachycardia with left anterior hemiblock and right bundle branch block. Non-contrast CT showed soft tissue injury and the presence of air in the anterior chest wall left of sternum associated with pneumo-mediastinum and pneumo-pericardium. Neither pneumothorax nor

any obvious pericardium effusion. On the 18th day after the injury, she returned to ER with complaints of sudden onset of left chest pain radiating to the back. ECG showed left anterior hemiblock and right bundle branch block and CT showed fluid collection in mediastinum and pericardium. Intra-operative trans-esophageal echocardiography showed pericardial effusion with a single ventricular septal rupture (delayed cardiac rupture)[12].

In the present case, there was no history of injuries to the chest and abdomen and no obvious injuries were noted on examination during the first admission. The ECG taken at first stay in the hospital was normal and vital were stable. There was no evidence of clinical signs of impending rupture in the present case which were noted by other authors in their case reports/studies mentioned above. The injuries to the spinal cord are more frequent and are likely to be precipitated in patients with a pre-existing disease like spondylosis noted in this case. Such spinal cord injuries cause weakness/paralysis of limbs. The recovery is usually delayed in such cases. The injuries to the spinal cord and myocardial infarction are not related to each other and can occur independently. Hence the rupture of the heart in the present case is not related to injuries sustained in the said road traffic accident and it is due to complication of atherosclerosis- a chronic disease.

**Conclusion:**

The myocardial infarction and its rupture are complications of atherosclerosis of coronary vessels which is a chronic disease of blood vessels. It is possible to have paralysis or paraesthesis in limbs due to injuries to the spinal cord which are likely to be accentuated due to underlying pre-existing disease of vertebrae (spondylosis). Hence the myocardial infarction leading to rupture of the left ventricular free wall in the present case is unlikely to be caused due to said road traffic accident as none of the injuries sustained by the deceased are suggestive of injuries to the chest and heart. Injuries to the spinal cord and rupture of the heart as a complication of myocardial infarction are unrelated to each other in the present case.

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