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CASE REPORT

"Acute Cardiac Tamponade attributable to strenuous physical work resulting in Sudden Natural Death: Review of Literature and Original Autopsy case series"

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Abstract
Sudden or unexpected Deaths especially when a person not known to have been suffering from any dangerous disease, injury or poisoning and dies within 24 hours after onset of terminal illness are often subjected to postmortem examination to rule out foul play. A similar case of unexpected natural instantaneous death is being reported with acute cardiac tamponade as cause of death.
Key words: Acute Cardiac Tamponade, Sternous physical work, sudden Natural death.

Introduction

Incidence of Sudden unexpected natural deaths is approximately 10% of all deaths. Diseases of cardiovascular system accounts for 45-50% of such deaths as compare to respiratory system involvement (15-30%) , CNS System (10-18%), alimentary system (6-8%), GUT system (3-5%) and miscellaneous (5-10%).

Heart is surrounded by a membrane covering called "Pericardial sac". The Pericardial sac consists of two layers, the outer parietal layer and inner visceral layer. It normally contains about 20ml to 50ml of fluid to cushion and lubricate the heart so as to facilitate its contraction and expansion. Cardiac temponade is a life threatening complication caused by excessive accumulation of fluid in pericardial sac leading to impaired cardiac filling and compromised cardiac output resulting in cardiac emergency. Reduction in stroke volume and epicardial coronary artery compression results in myocardial ischemia. The compressing force can be due to accumulation of blood, pus or other fluids in the pericardial sac (as a result of trauma, infection or surgical procedure), which accumulates fast enough and in sufficient quantity to compress the heart so as to restrict flow of blood in and out of the cardiac chambers.

Risk Factors for Cardiac Temponade

1. Malignancy (end stage of Lung tumor)
2. Infectious Pericarditis in case of HIV positive/Viral/Tuberculosis/ Fungal infections.
3. Cardiovascular surgery (Open Heart Surgery/ CABG)
4. Post Coronary Intervention (Coronary dissection and perforation)
5. Post Myocardial Infarction (after MI or Heart Attack)

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6. Connective Tissue Disorders like SLE, Rheumatoid arthritis.
7. Iatrogenic e.g. after sterna biopsy, pericardiocentasis, central IV line insertion, and transvenous pace maker lead implantation and radiation therapy to chest,
8. Drugs and Medications such as Anti Arrhythmic Drugs, Antihypertensive Drugs e.g. Minoxidil, Hydralazine, Procainamide.
9. Dissecting Aortic Aneurysm.

The most common causes of tamponade are pericarditis (infection and non-infection), iatrogenic (cardiac invasive procedures and post-surgery), and malignancy [1] For all patients, infectious diseases are still the most common cause of pericardial tamponade but, due to an increasing number of cardiac interventional procedures (cardiac ablation, device lead implantation and percutaneous coronary intervention), the incidence of haemopericardium seems to be increasing. Rare causes are collagen diseases (systemic lupus erythematosus, rheumatoid arthritis, scleroderma), radiation, aortic dissection, uraemia, post-myocardial infarction and bacterial infection [2].

Acute cardiac tamponade is usually caused by bleeding due to trauma, aortic dissection or is iatrogenic. Chronic fluid accumulation or subacute cardiac tamponade is characterised by the patients being more asymptomatic in the early phase but, when the pressure rises above the pericardial stretch point, they complain of dyspnoea, chest discomfort, peripheral oedema, fatigue, or tiredness, all symptoms attributable to increased pericardial pressure and limited cardiac output.

Etiopathophysiology

The rate of fluid accumulation in the pericardial sac is important. If fluid accumulates slowly or gradually then it may not restrict the blood flow inside heart chambers, until unless massive fluid accumulation occurs. When there is rapid massive collection of Blood or any fluid in pericardial cavity, compression on heart impairs the pumping action of vascular system leading to decreased stroke volume and cardiac output. Decreased filling of heart results in decreased supply of oxygenated blood to various tissues. These results in faster heart beat (Pulse), rapid fall in blood pressure, faster breathing and swollen neck veins. Ultimately consciousness lost and results in sudden death.

cardiac tamponade comprises a continuum from an effusion causing minimal effects to one causing circulatory collapse. The stiffness of the pericardium determines fluid increments precipitating tamponade [3, 4]. Rapid accumulation of as little as 150 mL of fluid can result in a marked increase in pericardial pressure and can severely impede cardiac output. In contrast, 1,000 mL of fluid may accumulate over a longer period without any significant effect on diastolic filling of the heart. This is due to adaptive stretching of the pericardium over time. A compliant pericardium can allow considerable fluid accumulation over time without haemodynamic compromise.

Majority of Hemo-pericardium (HP) related Cardiac tamponade (CT) are attributable to either intrapericardial rupture of dissecting ascending Aortic aneurysms (RD3A) or acute Rupture of Myocardial infarctions (RAMI). HP has been described as a complication of AMI in 5-10% cases **[5].** It is Commoner in Male <70years and more frequent in women after 70years of age. Two thirds of RAMI found to be associated with coronary artery thrombosis. Anterior wall rupture was commoner in female. A number of other albeit rare etiologies reported includes vascular lesions (angiosarcoma, hemangioma) anticoagulation therapy, acute bacterial pericarditis, Takayasu disease, Rhematoid Arthirits and iatrogenic interventions. Post Traumatic causes are infrequent.

The volume of intrapericardial blood varies between 150 to 1000ml.

Cardiac tamponade is a clinical syndrome caused by an increase in intra-pericardial pressure due to accumulation of blood, pus or any other fluid. Cardiac tamponade is prevalent in 25%

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to 30% of large pericardial effusions, which can be exhudative or rarely transudative. Primary Mesothelioma of pericardium comprises less than 1% of cases which results in heart failure due to cardiac tamponade either by a serous effusion or by direct tumorous constriction of the Heart. Common presenting complaint is substernal chest pain of duration varying from 30 minutes to 6 hours **[6].**

Pathophysiology

It is related to an increase in intrapericardial fluid pressure that exceeds atrial venous pressures, thereby impeding venous return to the heart[7]. The normal volume of pericardial fluid is 30 to 50ml. Rapidly evolving HP with 200-300ml Blood is more likely to cause death from CT than slowly evolving pericardial fluid accumulation to the extent of 500ml to 2000ml due to gradual distension of the pericardial sac **[7].**

During fluid accumulation, left- and right-sided atrial and ventricular diastolic pressures rise, and equalise the pressure similar to the pericardial sac (20-25 mmHg). The equalisation is closest during inspiration. Thus, pericardial pressure dictates intracavitary pressure leading to a progressive decline in cardiac volumes. The decreased preload accounts for the reduced stroke volume and compensatory increased contractility, and tachycardia is not enough to maintain stroke volume, thus leading to reduced cardiac output. Since the filling pressure in the right side of the heart is lower than in the left side of the heart, filling pressure increases more rapidly in the right than in the left side of the heart. Pulsus paradoxus is an abnormal decline (>10 mmHg) in systemic arterial pressure during inspiration. Normally, the intrathoracic pressure decreases during inspiration which allows blood to flow easily into the right heart. Conversely, the left heart filling decreases during inspiration, as the intrapericardial volume is fixed. During expiration, the intrathoracic pressure increases which leads to less right heart filling and augments filling of the left heart chambers. When fluid accumulates in the pericardial space, the intrapericardial pressure increases. This leads to a compression of the right heart, increasing the right heart pressure. Thus, the right heart filling is now relying more heavily on the decreased intrathoracic pressures during inspiration to fill, exaggerating the blood pressure change. The interventricular septum shifts to the left during inspiration and encroaches on the left ventricle, leading to a further reduction in stroke volume of the left ventricle **[8].**

Case No.1

A 46 year old married male in military service well built and well nourished brought in the month of July during early morning hours to the Emergency ward of Military Hospital with history of sudden unconsciousness and fall outside his barrack while going to bathroom. On examination his Blood Pressure and Pulse found to be unrecordable, Pupils were dilated and non reactive to external light stimuli. Cardio Pulmonary resuscitation attempted but he could not be revived and declared dead. On Autopsy No External as well as internal injury mark found to be present. No abnormality could be detected on Gross Internal examination of intra cranial and Abdominal contents. Stomach contained 200ml of semi digested food content with no peculiar smell. On Dissection of Pericardium 1000ml of clotted and fluid blood found to be present. A tear measuring 1.5cm x 0.2cm detected in the cardiac wall corresponding to right ventricular area. Whole Heart along with other Viscera was subjected to Histoptahological examination. Weight of Heart was 290gms and measured 12.0cm x 10.0cm x 4.0cm. On Microscopic examination focal areas of Infarct seen in congested myocardium. Left Ventricular wall thickness was reported to be 0.9 to 1.2cm and of right ventricular wall was 0.3cm to 0.4cm. Lumen of Right and Left Coronary arteries found to be patent. Sign suggestive of focal steatosis with congestion were seen on examination of Liver and cellular swelling of tubular epithelial cells with congestion observed in kidneys.

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Congestion observed on microscopic examination of Lungs and Spleen. Cause of Death declared to be Cardiac Temponade consequent upon Myocardial Infarction.

Case No.2

70 years old moderately built and moderately nourished North Indian Punjabi male was brought dead to the emergency of GGS Medical college, Faridkot in extreme winter weather, with history of chest pain, sweating and restlessness and without any known medical history. At Autopsy, face was found to be livid and there was no injury mark was present. On internal examination pericardial sac found to be distended and on dissecting it, dark fluid blood drools out with interspersed blood clots in between weighting about 500gms, diffuse infiltration of blood was present in the atrial wall surrounding the coronary Ostia. The Remainder of the postmortem examination was negative grossly. No Abdominal, Pelvic and Cranial pathology could be identified. Cause of death opined to be Cardiac tamponade as a result of natural disease process. Gross Photographs taken during Autopsy.

Case No.3

27 year aged young male admitted in emergency ward of tertiary care center after being referred by Medical officer from Periphery District Hospital for complaint of chest injuries in the form of two Lacerated Puncture wounds measuring 0.5cm x 0.4cm, with history of assault using a Blunt pointed object. His ECG found to be within normal limits. X ray chest was suggestive of widening of mediastinum with cardiomegaly with lucency along the cardiac margin in left hemithorax and no bony injury. CT scan chest was suggestive of moderate pericardial effusion with hyperdense soft tissue thickening in perivascular region. After about 4 days stay and conservative management, Patient discharged by treating surgeon in satisfactory condition. Patient suffered sudden collapse while playing volleyball in his hometown after 9 days of discharge from hospital and declared brought dead at nearby Private Hospital. Subsequently subjected to Postmortem examination by Board of Doctors of Forensic Medicine Department at GGS Medical College, Faridkot. On Autopsy Cyanosis of Lips and Nails found to be present. On internal examination, after dissecting pericardial sac, through and through tear found to be present over the Aorta with Clots of Blood in the pericardium. Margins of the tear observed to be everted. whole Heart with adjoining appendages subjected to histopathological examination and cause of death declared as cardiac temponade.

Figure 1 Case no. 2 Showing Hemopericardium in the form of Dark Fluid and Clotted Blood

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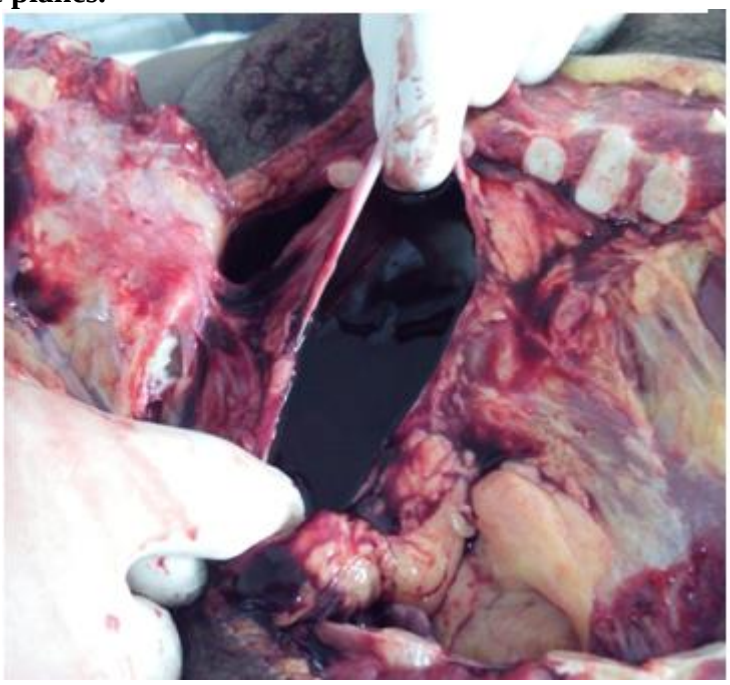
Figure 2: Case No.1 showing tear in Anterior Left Ventricular free wall with infiltraion of blood in soft tissue planes.



Discussion

Left ventricular rupture is the 2nd leading cause of in hospital death among patients with acute myocardial infarction. It a increase in wall rupture frequency noted since the widespread availability of coronary care facilities. The complication is almost uniformly fatal and the diagnosis is seldom made before death. Hence a high index of suspicion is required to detect this potentially devastating complication. 80% ruptures occur within the 1st week and 40% within the first 24 hours after Myocardial infarction.

Altun, et al., encountered such natural demises only in two cases with no specific causes of death **[9].** Factors influencing rupture of cardiac wall:

1. Age >55years
2. Pre existing Hypertension
3. Lack of left ventricular wall hypertrophy
4. History of Myocardial infarction
5. Coronary artery thrombosis.
6. Killip class I or II
7. Persistent ST Segment elevation
8. Sudden electromechanical dissociation

Diagnostic characteristics

The haemodynamic impact of an effusion ranges from none or mild to cardiogenic shock which leads to a clinical presentation ranging from acute to subacute. Acute or rapid cardiac tamponade is a form of cardiogenic shock and occurs within minutes. The symptoms are sudden onset of cardiovascular collapse and may be associated with chest pain, tachypnoea, and dyspnoea. The decline in cardiac output leads to hypotension and cool extremities. The jugular venous pressure rises which may show as venous distension at the neck and head. Clinically Acute Cardiac Tamponade found to be associated with three medical signs, known as **Beck's Triad**:

1. Distant Heart Sounds,
2. Distended Jugular Veins and

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3. Decreased arterial pressure (Hypotension).

Other signs are presence of **cyanosis of lips and nails**, Continuous ECG monitoring may suggest findings of dysrhythmia formation with **decreased QRS voltage** caused by swinging of the heart which is specific, but not sensitive for tamponade, restlessness and anxiety. The quickest and most sensitive imaging test to confirm cardiac rupture is a transthoracic **Echo**cardiogram (Ultrasound Test) showing fluid with echogenic masses in pericardial fluid and wall defects(10). **Pericardiocentesis** also called pericardial Tap, is a surgical invasive procedure to detect or remove abnormal excessive fluid from the pericardial sac, hence it is of diagnostic as well as therapeutic value. Removal of 5 to 10ml of Blood or fluid from sac dramatically increase stroke volume and cardiac output by 25 to 50%. It can be repeated when found necessary after reassessment.

The **chest X-ray** reveals a normal cardiac silhouette until the effusions are at least moderate in size (~200 mL). In general, an **enlarged cardiac silhouette** is neither sensitive nor specific for the diagnosis of cardiac tamponade. Small effusions are seen posteriorly and are typically less than 10 mm in thickness (pericardial pressure <10 mmHg). Moderate effusions tend to be posterior and may be circumferential and are usually 10-20 mm in thickness (pericardial pressure <10 mmHg), whereas **large effusions** tend to be circumferential and **greater than 20 mm in thickness** (pericardial pressure >15 mmHg).

To save life of the patient intravenous fluid given to maintain normal Blood pressure, to reduce the workload on heart supplementary oxygen given along with antibiotics etc. Prompt surgical repair is the definitive treatment; however operative mortality rates are high with immediate mortality rate of 24% and hospital mortality rate of 52%. Survival rate with this rarely reported complication can be increased with greater awareness, high index of suspicion for quick diagnosis and availability of Echocardiography at peripheral health centers so as to enable earlier attempts at surgical repair (11)**.**

Conclusion

In cases with alleged history of Trauma Pericardial effusion means Hematoma collection which need to be supported by clinical signs suggestive of temponade that can result in cardiopulmonary collapse. Rapid Focused Assessment with Sonography for Trauma (FAST) can establish presence of pericardial effusion. There is little evidence in the literature reflecting on the causes or demographics or Hemo Pericardium in routine postmortem practice. With the exception of individual case reports, most of evidence available in standard textbooks is non referenced. Only 1-3% of all deaths were attributable to Cardiac temponade. Autopsy Diagnosis is solely based on Gross inspection. Microscopic histo-pathological examination may be requested to rule out neoplastic process.

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