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CARDIAC TAMPONADE AND TUBERCULOSIS IN BLUNT TRAUMA: A FORENSIC ANALYSIS

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Abstract:

Life is incredibly valuable, and the unexpected death of an individual raises significant concerns for both family members and investigating officers. This case study focuses on a 71-year-old man who was admitted to two different hospitals due to chest discomfort and a reported history of traumatic chest injuries sustained 10 days prior to his death. Determining the presence and extent of severe chest trauma in this case posed a considerable challenge, alongside evaluating the manner and cause of death. The post-mortem investigation ultimately ruled out the likelihood of severe physical chest trauma. It identified the cause of death as "cardiac tamponade due to rupture of the infarcted myocardial wall," with the manner of death determined to be "natural." Additionally, the histopathological investigation revealed an unusual finding of "miliary tuberculosis of the heart." This case underscores the crucial role of thorough post-mortem examinations in clarifying the cause of death and addressing allegations of trauma.

Introduction:

Left ventricular wall rupture is a potentially fatal complication that can develop following a myocardial infarction (MI). Left ventricular wall rupture is most likely to develop 1 to 4 days after acute myocardial injury, and is one of the more fatal sequelae of MI [1]. Blunt cardiac rupture is characterized as full thickness laceration of the myocardium [2]. Although severe injuries that cause cardiac tamponade are typically devastating, these are not often associated with low-impact chest trauma [3]. Tuberculosis is a major public health concern globally, with pulmonary and extrapulmonary manifestations predominating. In affluent nations, tuberculosis is the least common cause of tuberculous pericarditis [4,5]. Herein, we present a unique case: ten days prior to the deceased's blunt chest trauma, the individual was kept at home by the family. On the eleventh day, he was admitted to hospital with chest discomfort and subsequently died during treatment. A meticulous post-mortem examination revealed that cardiac tamponade was the cause of death. Histopathological analysis demonstrated that the cardiac tamponade resulted from the rupture of an acutely infarcted myocardial wall, which is an exceedingly rare manifestation of miliary tuberculosis of the heart.

Case history:

We received the deceased corpse of a 71-year-old male for post-mortem examination. The investigating officer reported that ten days earlier, the deceased had suffered blunt trauma to the chest. The family kept him at home, and on the eleventh day, he was admitted to hospital with chest discomfort and died during treatment. The inquest report, conducted according to CrPC 174 of Indian law and witnessed by two government servants, indicated no visible injuries on the corpse. The investigating officer provided the necessary legal documents and treatment records from two private hospitals where the deceased had been treated for approximately 10 hours before his death.

Initially, the deceased was admitted to a multi-specialty hospital for chest pain. The discharge statement mentioned a history of blunt injuries to the chest and abdomen caused by unknown individuals, as relayed by relatives. However, the physician's examination did not find any evidence of fresh or old blunt trauma. ECG results showed ST wave changes in lead I, AVL, V5, V6, total leucocyte count of 26.1 x 10^3/mm^3, neutrophils at 79%, creatinine at 2.73 mg/dl, SGOT at 562.3 IU/L, SGPT at 276.2 IU/L, CK-MB at 55.7 ng/lit, Troponin-I at 9.54 ng/lit, and BNP at 117 pg/ml. A chest X-ray revealed a widened mediastinum without rib fractures. The preliminary diagnosis was pericardial tamponade due to anterior wall MI or suspected contusion. The patient was transferred to a more advanced facility for further care.

At the higher centre cum Medical College, the patient was managed for pericardial tamponade. A cardiologist performed a 2D echo-guided pericardiocentesis, removing a blood clot and 300 to 350 cc of blood. Despite resuscitation efforts, the patient died. The prognosis was cardiac tamponade caused by anterior wall MI or suspected contusion. Due to the history of blunt chest trauma, the case was treated as medico-legal. Despite the absence of trauma findings reported by two specialised doctors, a team of autopsy surgeons and forensic medicine professionals conducted a thorough post-mortem assessment in a government setting.

Before the post-mortem examination, the team meticulously reviewed the inquest findings and hospital treatment records. The external inspection of the body showed no signs of recent or old injuries. An I-shaped incision revealed no recent or old muscle haematomas in the chest or abdominal wall. Opening the thoracic cavity exposed a tense, purplish pericardium with 350 millilitres of blood and clots. The heart was flabby, and the anterolateral wall of the left ventricle had a vertical tear with haemorrhagic infiltration. The coronary arteries showed partial atherosclerosis, and haemorrhagic patches were noted on the anterolateral wall of the left ventricle. All visceral organs appeared congested. Tissue samples were preserved for histopathological examination. The findings indicated that cardiac tamponade caused by the rupture of an acutely infarcted myocardial wall was the cause of death, classifying the death as "Natural."

The deceased's relatives disagreed with this assessment, intending to file a legal complaint under Section 302 of the Indian Penal Code

against the alleged attacker.

The pathologist's role in this case was crucial, conducting a histopathological examination (HPE) of the visceral organs, particularly the heart. HPE findings confirmed the gross observations of cardiac tamponade (Figure 1 & 2) linked to myocardial wall rupture due to acute myocardial infarction and miliary tuberculosis of the heart. Significant atherosclerotic changes were observed in the ascending aorta, with 60% blockage of the right coronary artery, 30% blockage of the left anterior descending artery, and 60% blockage of the left coronary artery. The heart showed haemorrhage, necrotic myocardial fibres, and dispersed inflammatory infiltrates, along with caseating granulomas composed of lymphocytes, Langhans giant cells, and epithelioid cells. The lungs displayed pulmonary oedema, intra-alveolar haemorrhage, and haemosiderin-rich macrophages, indicating interstitial pneumonia (Figure 3 to 5).

This case is exceptional due to the extensive range of examinations conducted, including assessments of physical trauma, cardiac tamponade, rare cardiac conditions (miliary tuberculosis of the heart), and histopathology. The HPE not only validated the findings of the autopsy surgeons but also served as a defence for the autopsy surgeons and investigating officers against potential legal actions by the deceased's kin.

Discussion:

Cardiac tamponade is one of the most dangerous conditions that can result in a person's unexpected death [6]. Normally, 50 millilitres of pericardial fluid in the pericardial space lubricates the heart's activity. Cardiac tamponade occurs when there is a sudden accumulation of excessive fluid in the pericardial area (greater than 250 ml), inhibiting the heart's function [7]. Sudden death often raises numerous questions among the deceased's loved ones and the investigating officer. In some cases, hospital treatment records provide autopsy surgeons with critical information about the deceased's condition, possible assault, or allegations [8].

In this case, no evidence of injuries was found either during the post-mortem examination or in the hospital treatment records. Cardiac tamponade can result from various situations, including severe trauma and rupture of the myocardial infarct wall. Automobile accidents [9, 10] are the most common cause of fatal chest trauma, often leading to multiple chest injuries and either blunt or penetrating cardiac trauma.

Ewing's postulates stipulate that to establish a connection between the deceased and an injury, there must be proof of an injury, indisputable trauma, injury within an acceptable time frame, and evidence of disease at the site of the injury [11]. Various investigations, such as ECG changes and elevated cardiac enzymes, notably CK-MB and Troponin-I, can be used to identify cardiac damage [12, 13, 14]. In this case, changes were evident in all these investigations, and Ewing's postulates help to disprove the claim of blunt trauma, suggesting natural rather than unnatural causes of death.

Although cardiac tamponade is a major health concern, it is a rare manifestation of tuberculosis in affluent nations [15, 16]. Histopathological analysis in this instance revealed not only the rare involvement of the heart by miliary tuberculosis but also indicated a haemorrhagic myocardial infarction. This suggests that the rupture of the myocardial infarction wall, rather than cardiac tuberculosis, primarily caused the cardiac tamponade.

This case is exceptional because the potential claim of blunt chest trauma was successfully disproved, and the manner of death was determined to be "natural." The cause of death was identified as "cardiac tamponade due to rupture of myocardial infarction, associated with the extremely rare presentation of miliary tuberculosis of the heart."

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List of Abbreviations: Myocardial infarction – MI, Tuberculosis - TB, electrocardiogram – ECG, Histopathological examination - HPE

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