

Papillary Muscles Rupture as Delayed Sequelae of Trauma

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ABSTRACT

Background: Spontaneous rupture of papillary muscle from sepsis is extremely rare. Most cases of papillary muscle ruptures are due to myocardial infarction or trauma. We describe a case of spontaneous acute papillary muscle rupture in absence of history of ischemic cardiomyopathy or blunt chest trauma.

Case Report: A 42 year-old man was transferred to intensive care unit with signs and symptoms of cardiogenic shock. Past history revealed that he had met with a road traffic accident 19 months ago with fracture mid shaft of left femur. He was treated for the same and failed to have regular follow up visits. Physical examination revealed osteomyelitis at the fractured site with sinus formation and pyrexia. His general condition declined thereafter and expired due to cardiac failure few hours later. At autopsy there was rupture of antero-lateral papillary muscle of mitral valve. Microscopic section of the ruptured papillary muscle revealed formation of focal abscess with neutrophil infiltration.

Conclusion: Papillary muscle ruptures are associated with significantly increased morbidity and mortality. Therefore early diagnosis is very important in providing appropriate treatment. This case emphasized the fact that delay in diagnosis and institution of appropriate antimicrobial therapy for sepsis may result in life threatening complications. Although this occurrence is uncommon, papillary muscle necrosis and subsequent rupture must be considered as cause of delayed deaths in cases of road traffic accidents with septicemia.

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► *Implication for health policy/practice/research/medical education:* Papillary Muscles Rupture as Delayed Sequelae of Trauma

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1. Introduction:

Death after injury can be divided into three distinct time periods. Immediate deaths,

death during first few hours of hospitalization and delayed deaths. Immediate deaths (45%) are at accident scene from severe injuries, in first hours of hospitalization death is due to head injuries, hemorrhage, hypoxia etc., delayed deaths are due to head injury, ARDS, multiple system organ failure etc. Multiple system organ failure after injury is due to systemic inflammatory response to initial injuries. For the severely injured patient, it is postulated that there is an initial state of inflammation caused by trauma and that within this state of inflammation, there exists a critical balance between beneficial effects of inflammation and the potential for the immune system to cause tissue damage. Secondary stimuli like hypoxia, sepsis, hypovolemia, blood transfusion, surgery could disrupt the balance of immune system and provoke onset of multiple system organ failure (1-3). Systemic infection may respond to appropriate antibiotics, however fatal complications may ensue as a result of residual myocardial abscess.⁴ Myocardial abscess have been reported in 0.2–1.5 % of deaths due to septicemia (4, 5). Its presence is clinically obscured by the accompanying picture of severe sepsis. Most cases have occurred as a complication of generalized sepsis, with primary septi foci such as urinary tract infection, osteomyelitis, diverticular abscess and cellulitis or endocarditis (6). In rare cases papillary muscle abscess and subsequent rupture of papillary muscle may develop as a complication (4). This complication has been diagnosed by transthoracic and transesophageal echocardiography (7). We report a case of patient in whom papillary muscle rupture of mitral valve is caused by a papillary muscle abscess which was diagnosed at autopsy and histopathological examination.

2. Case report:

A 42-year-old man was admitted to Intensive Care Unit (ICU) with signs and symptoms of cardiogenic shock. Past

history revealed that the patient had met with a road traffic accident nineteen months ago with fracture midshaft of left femur. He was treated for the same with internal nail fixation and failed to have regular follow-up visits. There was no history of ischemic cardiomyopathy or trauma to the chest. On examination, respiration was shallow with inadequate respiratory attempts and oxygen saturation. Heart rate was 120 beats /minute; blood pressure was 150/110 mm Hg, electrocardiograph revealed sinus tachycardia.

There was no other significant abnormality on systemic examination. Local examination revealed sinus tract opening with edematous skin, increased warmth, tenderness and redness in the left thigh. Clinical diagnosis of osteomyelitis at the fractured site (non-union) with sinus formation was made and the blood culture was positive for *Staphylococcus aureus*. When he was observed in ICU his heart rate was 120 beats/ min, pulse was feeble, BP was not recordable and later he developed bradycardia and cardiac arrest. Cardiopulmonary resuscitation (CPR) was started. After 30 minutes of resuscitation he could not be revived. Clinically, cause of death was thought to be multiple organ failure due to septicaemia. Autopsy was done to establish the cause of death. At autopsy, on external examination, blood stained purulent discharge was seen through three tracking sinus indicative of osteomyelitis. On dissection of Heart, left atrial thickness was 3mm, right ventricular thickness was 8mm, left ventricular thickness was 2cm, mitral valve circumference was 7cm, tricuspid valve circumference was 8cm. Rupture of the anterolateral papillary muscle of mitral valve was noted (fig. 1). Coronary arteries were patent, other organs were unremarkable. Provisional diagnosis of death due to congestive cardiac failure due to papillary muscle rupture secondary to septicemia as a result of road traffic accident was made. However organs were sent for histopathological examination for

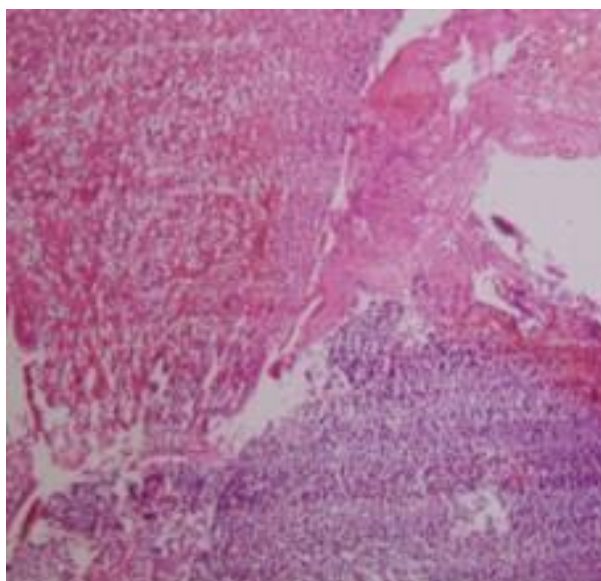


Fig. 1. Microscopic Examination of Papillary Muscle.

confirmation. On microscopic examination, papillary muscle showed evidence of degeneration and necrosis with deposits of fibrin and inflammatory exudates suggestive of papillary muscle necrosis (fig. 1). Cerebral cortex showed evidence of acute infarctive change with vacuolization and pyknosis of nuclei suggestive of infarction of cerebral cortex (Temporal area). Pons shows evidence of neuronal degeneration, nuclear pyknosis, intense eosinophilia, vacuolar degeneration, deposits of hemosiderin suggestive of hypertensive encephalopathy. Kidney, liver, gall bladder, lung was unremarkable. Final histopathological diagnosis of “Anterolateral mitral valve-Papillary muscle necrosis with hypertensive encephalopathy and acute infarction of cerebral cortex” was made.

3. Discussion:

Isolated rupture of papillary muscle is usually secondary to a blunt chest trauma, myocardial infarction, infective endocarditis or sepsis (4, 8). In the present case blunt chest trauma was ruled out based on history and autopsy examination. Ischemic rupture could not be the cause for the illness as there was no history of chest pain, and no ECG abnormalities, but histopathological examination revealed

focal abscess and necrosis of papillary muscle. Thus we concluded that papillary muscle rupture was due to abscess secondary to septicemia. In the literature a rare case of ruptured papillary muscle as a consequence of sepsis due to *Clostridium* has been described (7). Because of the low incidence of the disease exact pathogenesis is unclear (8). In cases of papillary muscle rupture due to infarcts the postero lateral papillary muscle is affected more often than the antero lateral papillary muscle because the latter receives blood both from the diagonal branches of the descending left anterior artery and the marginal branches of the curve. The postero medial muscle owes its blood supply only to the posterior inter ventricular artery (8). Whereas in the present case due to septicemia the anterolateral muscle is more often involved for the same reason of dual blood supply. There is a predilection for the valve ring area at atrio-ventricular junction to be a site for such abscess. This could be due to direct extension from the valve or this area may be more susceptible, as the fibrous skeleton is relatively avascular (4).

4. Conclusion:

Myocardial abscess is a disease with significant morbidity and mortality. Clinical history and laboratory findings are

often nonspecific and non-diagnostic. Definitive diagnosis is relied upon direct evidence from histology and/or microbiology at surgery or autopsy. In cases of delayed death due to trauma with fractures, septicemia and its complications like systemic abscesses and infarcts should be anticipated. Trauma with septicemia can lead to infective endocarditis. Early detection by frequent blood cultures can prevent these complications.

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