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Left Ventricular Rupture in Transmural Myocardial Infarction Without Enzymatic or Electric Disturbances: Medico-legal Implications

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Abstract

Background

The early and accurate diagnosis of acute myocardial infarction may be a challenging task for doctors in the emergency room. The patient's history, electrocardiographic and cardiac markers data are sometimes non diagnostic and misleading which may lead to death. In this case, physician's responsibility may be engaged.

Case report

A 48-year-old man was admitted in the Emergency Department because of chest pain. Exploration (Percritical ECG, enzymatic levels and transthoracic echocardiogram) did not show any abnormality. He received a symptomatic treatment and was allowed to return home. The patient was confined to bed during his stay at home because of persisting chest pain. Two days later, he presented syncope and arrived dead to hospital. At autopsy, an abundant hemopericardium made by coagulated blood was noted, associated with an obstruction of the circumflex of about 90% of its light. A rupture of the myocardium was repaired in the lateral wall of left ventricle, surrounded by a semi recent infarction.

Discussion

The peculiarity of this case is the difficulty of the diagnosis of the myocardial infarction because of the normality of the enzymatic assessment and the absence of electric modifications during the crisis and two hours later. Misdiagnosed cases can lead health professionals to legal suits. The claims about diagnostic error can be usually subject to trials in both of penal judgment and compensation trial.

Keywords: Myocardial infarction; Electrocardiography; Creatine kinase; Liability; Legal

Introduction

Left ventricular free wall rupture (LVFWR) is a rare and fatal complication of acute myocardial infarction (AMI). The early and accurate identification of myocardial necrosis in patients experiencing features suggestive of acute coronary syndrome is an important challenge. It depends upon the interpretation of the patient's history, electrocardiographic and cardiac markers data [1]. However, these parameters are known to be unspecific and sometimes non-diagnostic or unequivocal [2]. Thus, the diagnosis of myocardial infarction may be misleadingly unnoticed which may harm patients and result in increased morbidity or death. In this case, Doctor's liability may be engaged for missed diagnosis.

We aim to report an autopsy case of transmural myocardial infarction complicated with Left ventricular free wall rupture in a patient without enzymatic or electric disturbances and we discuss the physician's responsibility.

Case Presentation

A 48-year-old man, without past medical history, was admitted into the Emergency Department of a Peripheral Health Center because of chest tightness of two days duration.

Physical examination did not reveal any abnormalities, despite an elevated blood pressure (170/110 mmHg). Per critical electrocardiogram was unremarkable (Figure 1). Specially, it did not reveal ST segment and T wave abnormalities. No electrocardiographic changes evolved 2 hours later (Figure 2). Results of laboratory tests were within normal limits: enzymatic cardiac levels (total Creatine Phosphokinase) were at 140 UI (normal 0-185 U/L), CK-MB to CK ratio was <5%, and the dosage of troponin I was technically impossible (lack of reagent). Transthoracic echocardiogram did not highlight cardiac failure or kinetic trouble (left ventricular function at 63%). The pericardial sac was free of effusion. Thus, the patient was discharged with the diagnosis of parietal pain. He was confined to bed during his stay at home because of persisting chest pain. Two days later, he presented syncope and arrived dead to hospital.
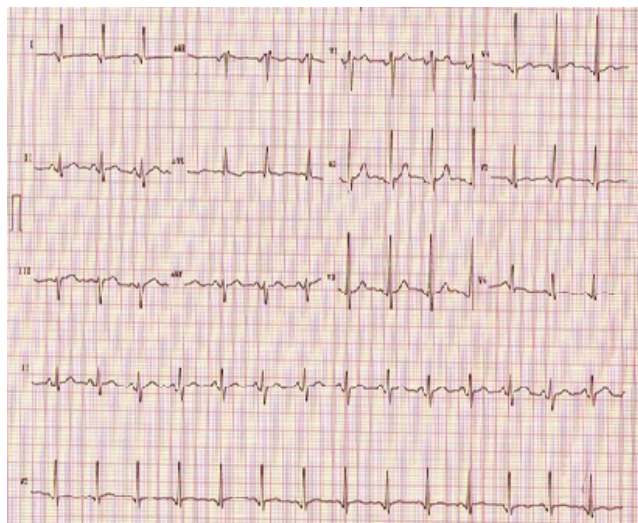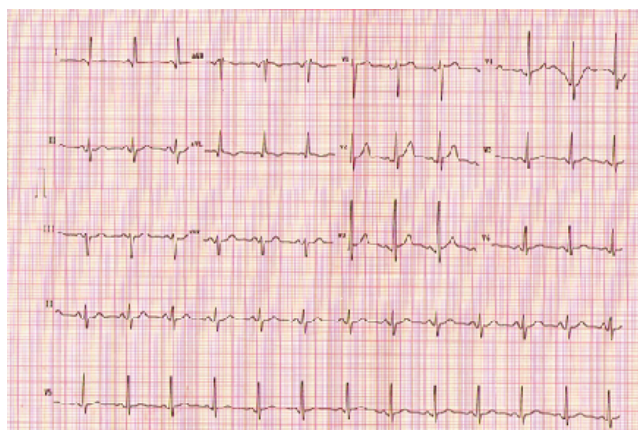
Figure 1: Percritical ECG

Figure 2: ECG performed 2 hours later

Figure 3: Hemopericardium

Figure 4: Left ventricular free wall myocardial rupture complicating an acute myocardial infacrtion.

Figure 5: Left ventricular free wall myocardial rupture.

A Forensic autopsy was requested. Internal examination noted an abundant hemopericardium (Figure 3) made by coagulated blood. The heart weighted 350 g. The wall of the left ventricle being 13 mm thick. The valvular apparatus was macroscopically normal. The dissection of coronary arteries showed an obstruction of the circumflex of about 90% of its light. A rupture of the myocardium (Figures 4 and 5) was repaired in the lateral wall of left ventricle, surrounded by a semi recent infarction. The liver was congestive and weighted 1800 g. Toxicological screening was negative. Post mortem histological tests confirmed the diagnosis of myocardial infarction (coagulative necrosis, wavy fibers and pink cytoplasm).
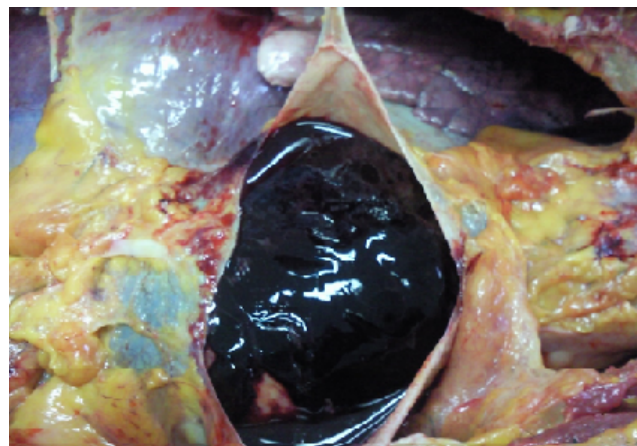
Discussion

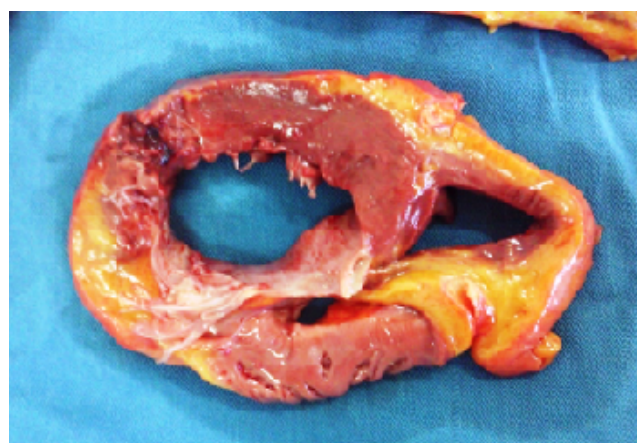The diagnosis of AMI can be a challenging task for many physicians in an emergency department. It was previously based on the criteria set by the World Health Organization (WHO). A patient is diagnosed with myocardial infarction if he presents two of the following 1: typical history of ischaemic type chest pain; 2: changes on serial ECG tracing; 3: typical rise and fall of serum cardiac enzymes [3]. However, each of these variables is known to lack precision.

In fact, a history of prolonged chest pain is often absent in patients with infarction, and when present, it may be due to other events than myocardial ischemia [4]. The ECG is also an important tool for detecting AMI. When typical changes of AMI are present on the admission ECG (ST elevation and new Q-wave), they are quite specific and have a very high positive predictive value for the diagnosis, but their absence should not rules it out, as many as 30-50% of patients may initially present with normal or non-diagnostic ECG [5]. Johnson WJ [5] reported that new Q wave changes on electrocardiograms in patients with myocardial infarction are absent in approximately 30% of autopsy-proven cases. Wagner et al. [2] identified 29 of 84 patients with definite infarction but falsely negative ECG.

Because of the above, the WHO criteria were refined to give more prominence to cardiac biomarkers [3]. The joint European Society of Cardiology/ American College of Cardiology Committee, redefines myocardial infarction according to cardiac markers as an increase in cardiac troponin cTnI, cTnT or an increase in creatine Kinase muscle and brain (CK-MB) [6]. Nowadays, most physicians rely heavily on serum level changes of cardiac enzymes to retain or exclude the diagnosis of acute myocardial infarction. Cardiac markers play an important role in this field when the patient's history and ECG are non-diagnostic or misleading [7]. Creatine kinase (CK) is one of the oldest biochemical markers of myocardial damage. It has a clinical sensitivity of 90% for the diagnosis of AMI. Unfortunately, this is not matched by high specificity. It is released within 12 hours after symptom onset of AMI, peaks in serum at 24-36 hours, and returns to normal in 48-72 hours. As a result of these release kinetics, measurement of total CK is not suitable for the early diagnosis (within 6 hours) of AMI [8] and serial sampling is the most effective method for that aim [9]. In this case report, the exclusion of the diagnosis of AMI was based only on one single value of CK at presentation which appears to be unreliable. CK as a marker is also unsuitable for the detection of myocardial damage that may occur in patients presenting with Non-ST elevation myocardial infarction like in this case report.

A marker that is suitable for the early diagnosis of AMI and the detection of small injuries to the heart should be abundant in the myocardium and not present in other tissues, have a total cardiac specificity with undetectable plasma concentration and finally it should be released completely and quickly when myocardial damage occurs [10]. CK does not fulfill these criteria since it is widely distributed in the body and have a high range up to 200 IU/L [9]. In the other hand, there are cases of AMI without elevation of total CK concentration. This situation leads to the failure to accurately diagnose the myocardial ischaemic damage and delays in the initiation of appropriate treatments [11]. As a result and to improve on the cardiac specificity of CK for the diagnosis of AMI, it was recommended to measure both total CK and CK-MB (the cardiac specific isoenzyme of CK). A CK-MB to CK ratio up to 6% is reported to be specific for myocardial injury [10]. In this case report, the ratio was <5%. This is may be explained by the delay from the onset of the symptoms which is >48 h. Thus, the diagnosis value of CK-MB will be applicable only to patients who are seen early after the onset of symptoms. Wagner et al [2] reported that the absence of CK-MB in patients who are seen more than 24h remote from the onset of their acute episode may not be used as evidence for exclusion of the diagnosis AMI. However, physicians should keep in mind that there are various non-cardiac causes for elevated CK-MB like severe skeletal muscle damage. In these situations, cardiac troponins can be used to differentiate cardiac and non-cardiac pathologies. Elevated troponins concentrations have been reported to be a specific marker for the diagnosis of AMI. Their clinical sensitivity approaches 100% at about 12h after symptom onset [12].

Myocardial rupture is a catastrophic complication of AMI that directly causes death in 8% of cases [13]. It occurs within 7 days, in 1-4% of patients with AMI [14]. It may occur in patients with their first transmural AMI like in our case. Prodromal manifestations reported in the literature are intractable vomiting, restlessness, persistent chest pain, cardiovascular collapse… In the case reported here, death occurred 2 days after the onset of the symptoms, which suggest a subacute or stuttering ruptures. This implies a gradual or incomplete ruptures of the infarcted area with slow bleeding in the pericardial sac causing progressive cardiac tamponade [14].

The above reported case showed how much paraclinical features of AMI can be misleading. Previous studies have shown that between 4% and 8% of patients with missed AMI are sent home [15]. This finding confirms the relatively low rate of missed AMI patients reported by Lee et al (3.8% of missed diagnosis) [16] and McCarthy et al (2%) [17]. diagnosed cases can lead health professionals to legal suits. Accordingly, misdiagnosis of AMI is a common cause for malpractice claims. In the United States, diagnostic errors become the most prevalent type of malpractice claim [18]. Unrecognized AMI frequently involved multiple breakdowns. The leading contributing factors related to the misdiagnosis of acute chest pain in the emergency department are failure to perform an adequate history or physical examination, failure to identify atypical presentation, failure to order or misinterpret an appropriate diagnostic test and failure to order an appropriate specialized consultation [19]. The claims about diagnostic error can be usually subject to trials in both of penal judgment and compensation trial. According to the Tunisian Criminal Code (TCC), doctors can be prosecuted under section 225(involuntary assault by negligence or inattention) which is punishable with imprisonment for a term that may extend to 1 year. They are also being punished for involuntary homicide, if death occurs, under section 217 of the TCC. Also, the doctor can be made liable in civil law for paying compensation and damages.

Thus, for quality assurance and to avoid the failure to recognise AMI, physicians should be more aware and consider atypical presentations, document more detailed histories, do not accurately rely on paraclinical tests (ECG and cardiac markers) and readily admit patients with vague or suspicious symptoms. McCarthy et al. [17] have reported that 25% of missed AMIs might have been prevented by not sending home patients with symptoms believed to be due to ischemic heart disease. This reinforces the idea that unequivocal symptoms should provoke discussion among emergency physicians to avoid erroneous discharge.

References

1. Charles R Roe (1977) Diagnosis of myocardial infarction by serum isoenzyme analysis. Ann. Clin. Lab. Sci 7:201-209.
2. Galen SW, Charles RR, Lee EL, Robert ARi, Andrew GWet al. (1973) The importance of identification of the Myocardial-Specific Isoenzyme of

Creatine Phosphokinase (MB Form) in the diagnosis of acute myocardial infarction. Circulation 47:263-269.

3. Nomenclature and criteria for diagnosis of ischemic heart disease. Report of the Joint International Society and Federation of Cardiology/World Health Organization Task Force on Standardization of Clinical Nomenclature. Circulation 1979; 59:607-609.

4. Kannel WB, Feinleib M (1972) Natural history of angina pectoris in the Framingham study. Am J Cardiol 29:154-163.

5. Johnson WJ, Achor RWP, Burchell HB, Edwards JE. (1959) Unrecognized myocardial infarction. Arch Intern MED 103: 253-261.

6. Alpert JS, Thygesen K (2000) Myocardial Infarction Redefined - A consensus document of the joint European Society of Cardiology/ American College of Cardiology Committee for the redefinition of Myocardial Infarction. Eur Heart J 21:1502-1513.

7. Hedges JR, Rouan GW, Toltzis R, Goldstein WB, Stein EA (1987) Use of cardiac enzymes identifies patients with acute myocardial infarction otherwise unrecognized in the emergency department. Ann Emerg Med 16:248-252.

8. Gibler WB, Lewis LM, Erb RE, Makens PK, Kaplan BC, et al. (1990) Early detection of acute myocardial infarction in patients presenting with chest pain and nondiagnostic ECG's: serial CK-MB sampling in the emergency department. Ann Emerg Med 19:1359-1366.

9. Duma RJ, Seigel AL(1965) Serum creatine phosphokinase in acute myocardial infarction. Arch Intern Med 115:443-451.

10. Hafidh AH, Keith AF (2009) Cardiac Markers in the Early Diagnosis and Management of Patients with Acute Coronary Syndrome. SQU Med J 9: 231-246

11. Van BM, Maes V, Huyghens L, Derde MP, Meert R, Gorus FK (1992) Analytical and clinical evaluation of creatine kinase MB mass assay by IMx: comparison with MB isoenzyme activity and serum myoglobin for early diagnosis of myocardial infarction. Clin Chem 38: 2380-2386.

12. Wu AH, Valdes RJ, Apple FS, Gornet T, Stone MA, Mayfield SS, et al. (1994) Cardiac troponin-T immunoassay for diagnosis of acute myocardial infarction. ClinChem 40:900-907.

13. Offer A, Ronald S, Akaira N, Igor DG, Frank WS (2005) Left Ventricular free wall rupture in acute myocardial infarction. Tex Heart Inst J 32:424-426.

14. Lateef F, Nimbkar N (2003) Ventricular free wall rupture after myocardial infarction. Hong Kong J Emerg Med 10:238-246.

15. Michael JS, Marian JV, Therese AS (2006) The risk of missed diagnosis of acute myocardial infarction associated with emergency department volume. Ann. Emerg. Med 48:647-655.

16. Lee TH, Rouan GW, Weisberg MC (1987) Clinical characteristics and natural history of patients sent home from the emergency room. Am J Cardiol 60:220-224.

17. McCarthy BD, Beshansky JR, D'agostino RB, Selker HP (1993) Missed diagnoses of acute myocardial infarction in the emergency department: results from a multicenter study. Ann Emerg Med 22:579-582.

18. Pope JH, Aufderheide TP, Ruthaze R, Woolard RH, Fieldman JA, et al. (2000) Missed diagnoses of acute cardiac ischemia in the emergency department. N Engl J Med 342:1163-1170.

19. Allen K, Tejal KG, Ann LP, Catherine Y, Eric JT, et al. (2007) Missed and delayed diagnoses in the emergency department: a study of closed malpractice claims from 4 liability insurers. Ann Emerg Med 49:196-205.