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CLINICAL CASE

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RECOGNITION OF MYOCARDIAL RUPTURE IN EMERGENCY: CASE REPORT

RECONOCIMIENTO DE ROTURA MIOCÁRDICA EN EMERGENCIA: REPORTE DE CASO

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ABSTRACT

A 86-year old female patient with hypertension was admitted to emergency due to chest pain, dyspnea, hypotension and bradycardia with confused symptoms. The electrocardiogram and the laboratory are compatible with non-Q wave myocardial infarction, with a left ventricular ejection fraction of 20% in the ultrasound, with pericardial effusion and hyperechoic mass compatible with a clot. The diagnosis was a low cardiac output due to an evolutive non-Q-wave myocardial infarction with myocardial rupture. The patient required endotracheal intubation and mechanical ventilation, evolved with cardiac arrest and was resuscitated without success. The diagnosis and timely management cardiac rupture is explained as a mechanical complication of acute myocardial infarction.

Key words: Echocardiography; Myocardial infarction; Myocardial rupture. (source: MeSH NLM)

RESUMEN

Paciente mujer de 86 años, hipertensa que ingresa a emergencia por dolor torácico, disnea, hipotensión y bradicardia con cuadro confusional. El electrocardiograma y el laboratorio es compatible con infarto agudo de miocardio no Q, encontrándose en la ecovisión una fracción de eyección ventricular izquierda de 20% con derrame pericárdico y masa hiperecogénica compatible con coágulo. El diagnóstico planteado fue estado de bajo gasto cardiaco porinfarto de miocardio no Q evolutivo con rotura miocárdica. La paciente requirió intubación endotraqueal y ventilación mecánica, evolucionó con paro cardiaco y fue reanimada sin éxito. Se discute el diagnóstico y manejo oportunos de la rotura cardiaca como una complicación mecánica del infarto agudo miocárdico.

Palabras clave: Ecocardiografía; Infarto miocárdico; Rotura miocárdica. (fuente: DeCS BIREME)

INTRODUCTION

The left ventricular free wall rupture is, after cardiogenic shock, the second most frequent cause of death in the hospital in acute myocardial infarction. Its incidence has decreased in recent years due to rapid reperfusion of the ischemic territory, the early use of beta-blockers, and angiotensin-converting enzyme inhibitors. It went from 6% in the pre-intervention era to less than 1% with the appearance of primary angioplasty[1,2]. We report the case of an 86-year-old patient with a low cardiac output due to a non-evolving myocardial infarction with myocardial rupture.

CASE REPORT

A 86 years old female patient, mixed race with a history of high blood pressure and bradycardia diagnosed a year ago. She received as treatment amlodipine, enalapril, aspirin, and simvastatin. She entered an emergency room due to a loss of consciousness. Her relatives said that she being low of energy for a few days and one hour before her admission she started to fade and show dyspnea.

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She showed at a clinical examination blood pressure of 60/40 mmHg, heart rate at 78 beats per minute, respiratory rate of 30 per minute, saturation of oxygen in 80% and a hemoglucotest of 149mg/dL. The patient physical examination showed poor general condition, sleepy, with distal coldness and capillary refill greater than 2 seconds, no cyanosis was evident. low intensity rhythmic cardiac sounds. Systolic blow II / VI, spontaneous ventilation with a reservoir mask, and bad respiratory pattern. Abdomen soft depressible, no palpable visceromegaly. The neurological examination without signs of focalization. ScaleGlasglow: 10 points (AO 4, RV 2, RM 4).

The electrocardiogram showed sinus rhythm, a frequency of 78 beats per minute, with signs of hypertrophy of left ventricle, without ST-segment elevation, or presence of Q waves (Fig.1). The arterial gas analysis(AGA) evidenced decompensated metabolic acidosis. Normal anion gap with lactate in 7.9 mmol/L. Ph7.29,pCO2 21.1mmHg, pO 225.4mmHg, cHCO3 9.9 mmol / L,SO2 99.6%. In the blood count, leukocytes 9.11 x103mm3, hemoglobin 11.3 gr / dL, platelets 181 000 xmm3, glucose 245 mg/dL, urea 24.4 mg/dL, creatinine0.76 mg/dL, sodium149.3 mmol / L, potassium 3.46 mmol / L.TGO 378 U/L, TGP 105 U/L, total bilirubin 0.25 mg /dL, direct bilirubin 0.17 mg/ dL, alkaline phosphatase55 U/L, albumin 2.84 g / dL, amylase 206 U/L, lipase 45U/L, prothrombin time 16.44 sec. Total CPK 31U/L, troponin I 7.271 ng/dL. Chest x-ray did not show a greater finding (Fig. 2) and eco-vision reveals LVEF 20% with pericardial effusion and a mass hyperechogenic compatible with clot adhered to the wall (Fig. 3). The diagnosis was made of low cardiac output due to non-Q-wave myocardial infarction evolutive with myocardial rupture.

Enter mechanical ventilation, the control AGA showed mixed acidosis with lactate at 6.3 mmol / L, increased the minute volume and remained levels of positive pressure at the end of expiration (PEEP)less than 5 cmH2O. The patient presented with episodes of sinus bradycardia with pauses and escape beat Nadal on a sinus rhythm and heart rate in 62 beats per minute. In transthoracic ultrasound control was found pericardial effusion of 250ml with the collapse of right cavities, shunts are not appreciated, no significant valve changes. Proceed to perform pericardiocentesis, obtaining 150 cc of Serohemic fluid. Forty minutes later the patient enters cardiorespiratory arrest, asystole twice without success in the maneuvers of revival.

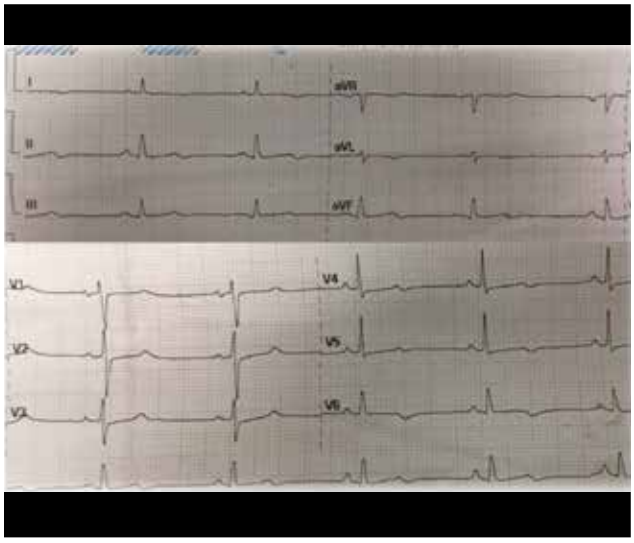

Figure 1. Electrocardiogram. Rhythm Sinus, FC: 78x ', 40 ° axis, non ST elevation , no Q wave present.

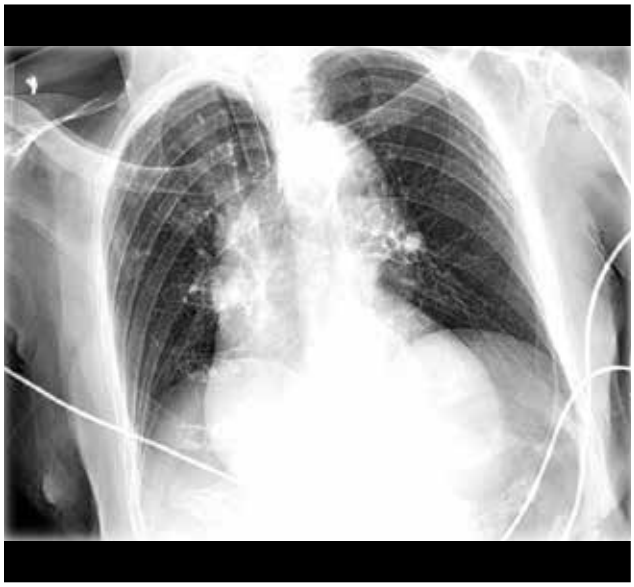

Figure 2. Chest x-ray (Rx-T): The frontal Rx-T (17.XII.17) shows lung parenchyma without nodules or masses, no interstitial thickening or bronchiectasis or honeycombing is evidenced. No pleural effusion.

DISCUSSION

LMyocardial rupture is a mechanical fatal complication of an acute myocardial infarction (AMI) it is directly responsible for mortality of 8% of infarcted patients. The breaking of the free ventricle left wall occurs in approximately 2% of cases, but today, in the era of percutaneous coronary intervention (PCI), is less frequent[3]. 40% of the cases occur in the first 24 hours of the acute ischemic event and 85% within the first week[1,2,4]. The rupture does not usually happen after 10 days when the healing has taken place[1].

No specific physical finding has been reported for

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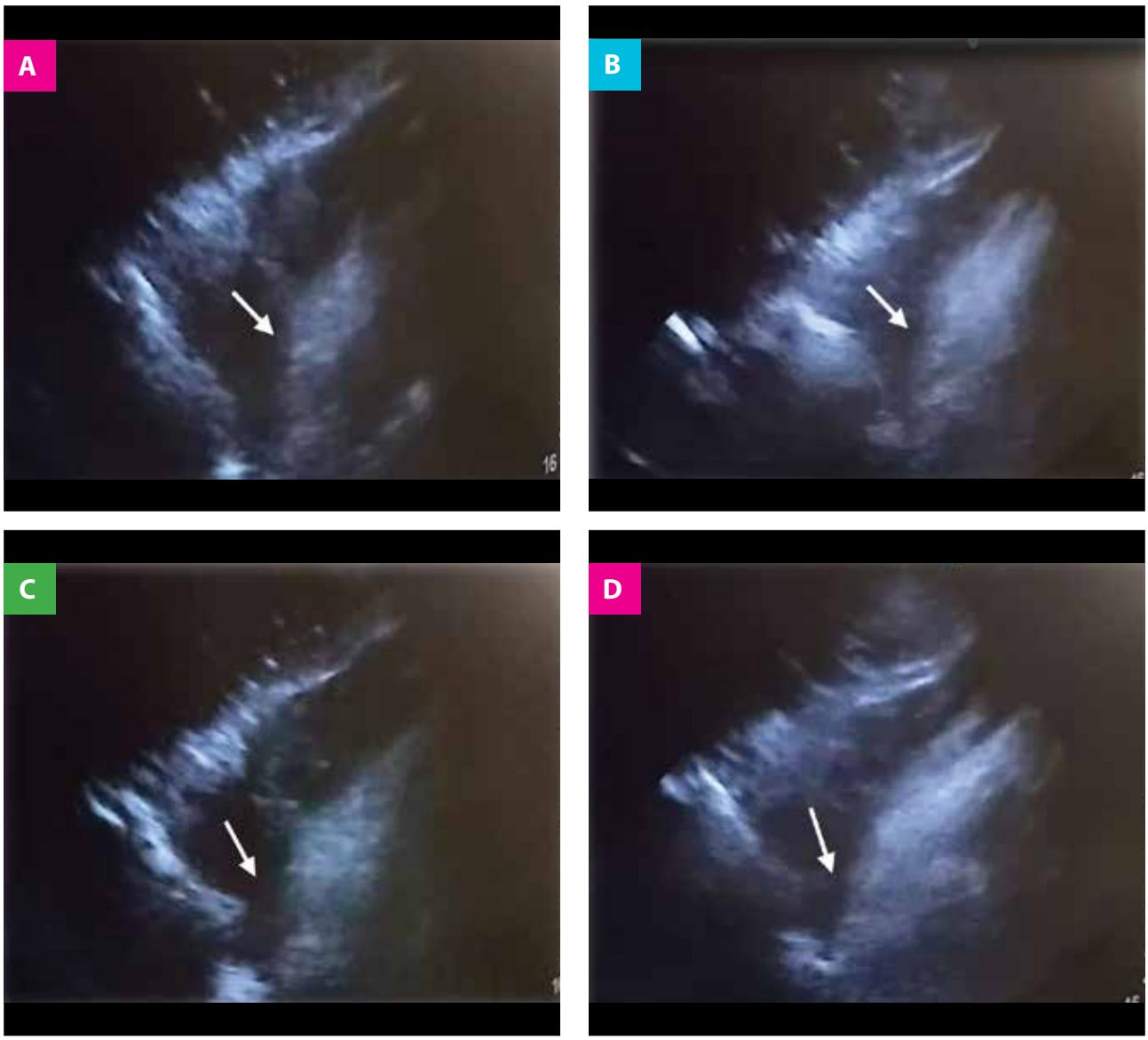

Figure 3. Echocardiogram (17.XII.17). Image A, B, CYD: Pericardial effusion of approximately 250cc (prepericardio centesis) with hemodynamic compromise, dilated left ventricle with right ventricle collapse (white arrow). Shunts are not appreciated. No significant valvular rations.

cardiac rupture and a reason could a rapid deterioration of the clinical condition as in the present case. The bedside echocardiography is the fastest diagnostic method[5].

A high index of suspicion and early diagnosis is of vital importance for the survival of the patients They are considered predisposing factors; as the patient presented; old age, female, high blood pressure without previous angina attacks and persistent or recurrent chest pain. He also considers as risk factors a first anterior or lateral transmural infarction without symptoms of manifest heart failure and persistent elevation ST[2,3] segment. The patient entered with a clinical picture compatible with coronary acute syndrome. Since her entry, she was in a poor general condition, hypotensive, confused and with signs of shock. You should also consider in these cases the possibility of cardiac rupture. In that environment, the break of the free wall is prone in patients with transmural myocardial infarction involving the descending anterior artery and circumflex. The administration of thrombolytic agents can promote heart breakage presumably because of a hemorrhagic trend[2]. The time of the free wall break has decreased from 3 to 5 days in the pre-thrombolytic era to 12 to 24 hours in the thrombolytic era. On the contrary, ICP reduces the incidence of myocardial rupture and improves mortality and morbidity due to IMA[6].

There are three clinical forms of ventricular wall free breakage. The most frequent is acute rupture (70% of cases) followed by massive intrapericardial hemorrhage and fatal evolution of tamponade in

few minutes without enough time for intervention[4,6]. Second, the subacute break which is characterized because the tamponade is established with less hardness (due to an incomplete tear) and the patient can survive several hours. It is estimated that more than half of patients with sub-acute rupture die during the first 6 hours as happened in the present case. They have described exceptional cases of subacute rupture with long-term survival without surgical treatment. Third, the break can go unnoticed in the acute phase of the infarction, probably being contained by pre-existing pericardial adhesions, leading to the formation of a false aneurysmchronic[2,4,6]. The clinical picture is characterized by cardiovascular collapse with electromechanical dissociation, with a fatal outcome in a few minutes[2,3]. However, in 25% of the cases, the presentation is subacute (due to thrombosis or adhesions that the seal) which facilitates the intervention[3]. The patient may or may not have premonitory symptoms, but if they are present, they present persistent chest pain, agitation, and recurrent emesis[6]. The subacute break of the left ventricle free wall can simulate reinfarction by the recurrence of pain and new elevations of the ST segment, although more frequently it appears as a hemodynamic sudden deterioration with cardiac tamponade, persistent hypotension, and low expenditure state[2,7,8]. It is form has a very serious prognosis, with need of urgent surgical intervention, and is associated with high mortality, up to 80% if there is breakage of free wall[2].

Changes in the electrocardiogram in patients with cardiac rupture may vary from normal to elevated anteroseptal ST with deviation axis to the left, previous ST elevation, typical changes of lower IMA or later IMA, pseudonormalization of T wave, ST-segment elevation in AVL, new Q-waves in at least two derivations and changes not specific in the ST-T. Electrocardiogram (EKG) did not show important changes and the patient had an important troponin elevation. The persistent ST elevation or positive deviation of the T wave that persists for more than 72 hours, is not sensitive or specific subacute rupture[6]. The last state of the break corresponds to bradycardia and electromechanical dissociation, diagnostic method always important but not decisive in case of cardiac rupture[1].

Echocardiography is a sensitive technique for myocardial rupture diagnose, although the presence of the spill alone is not enough to diagnose it, because it can be present in 28% of patients after coronary acute syndrome and subacute rupture[5,6]. Ultrasound for ventricular rupture diagnosis has 70% or more of sensitivity and 90% of specificity. The findings include echogenic pericardial fluid of more than 5mm of thickness and findings of a right ventricle diastolic collapse, dilated inferior vena cava and marked respiratory variation in the flow of the entrance of the tricuspid and mitral valves[4,7,8]. The presence of echogenic masses increases sensitivity(97%) as specificity (93%). Also, the moderate to severe pericardial effusion (> 10 mm) in the patient with high IMA ST is associated with 43% of 30-day mortality[6]. Thus; The spill does not increase morbidity and mortality in heart attack of the myocardium, but it serves as another indicator of infarction in a large area. Major spills or echocardiograms dense spills that represent hemorrhage should always make doctors consider a breakage of the free wall[9].

The use of emergency pericardiocentesis is controversial. Thought as a contributor to the patient's state of shock the procedure was practiced. The indication in patients that undergo cardiac tamponade constitutes a measure of rescue for patients awaiting immediate surgical management. However, it can cause an increased tension of the damaged myocardium and potential extension of the area of cardiac rupture[1,7,8]. The initial treatment is aimed at stabilizing the patient and includes intravenous oxygenation/ventilation fluids with small volumes and inotropics[6].

Echocardiography is a valuable tool for non-invasive diagnosis that can provide information about a cardiac function and valvular abnormalities and alternative explanations for causes of chest pain and shortness of breath. It is widely recognized that echocardiography of emergency is the most versatile imaging technique and cost-effective to evaluate patients with unstable cardiovascular diseases[9,10]. It is the most commonly available and can be done by an emergency doctor in the resuscitation room.

Despite the high surgical risk, the surgical repair remains the gold standard for control of this rare condition[11]. The current guides of the American College of Cardiology and the American Association of the heart ACCF / AHA (American College of Cardiology Foundation and American Heart Association) recommend surgical repair of an emergency regardless of hemodynamic stability at the time of diagnosis. Despite the general agreement among experts on the need for surgical repair, the time of repair and perioperative therapeutic management are controversial[7,8]. In the case presented, the patient did not enter the operating room due to the time it took to develop her results. However,

there is some controversial point whether all cases should implement surgery, revascularization may be recommended, no surgery, or to delay the surgery.

CONCLUSION

This case exceptionally highlights the role of non-invasive diagnostic techniques, especially the use of echocardiography in all patients with acute coronary syndrome[1,9] and also to take into account this pathology for timely diagnosis and treatment.

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