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Case Report

Aortic Dissection presenting as inferior ST Elevation Myocardial infarction and Importance of early echocardiography

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Abstract

We describe a case of a 70 years old female patient in whom an initial diagnosis of acute coronary syndrome (ACS) revealed to be finally an acute aortic dissection. This case report emphasizes the importance to maintain a high grade of suspicion of aortic dissection as a possible alternative in presence of electrocardiographic myocardial ischemic changes. In many medical centers where thrombolytic therapy, antiplatelet receptor blockers, heparin or percutaneous coronary angioplasty is the first line therapy for ACS the outcome may be catastrophic in situation such as in aortic dissection.

Keywords

Aortic dissection; Myocardial infarction

Introduction

Acute aortic dissection, caused by arterial hypertension, constitutes a hypertensive emergency. It is characterized by the sudden separation of the medial layer of the vessel. This creates a false lumen and the formation of a hematoma evolving with high cardiovascular morbidity and mortality. We describe a case of a 70 years old female patient in whom an initial diagnosis of acute coronary syndrome (ACS) revealed to be finally an acute aortic dissection. We emphasize the importance of differential diagnosis between myocardial infarction and aortic dissection, two critical situations which require precise diagnosis and the correct therapeutic conduct.

Case presentation

A 70 year old female patient with a past history of uncontrolled hypertension, diabetic mellitus, dyslipidemia and unstable angina presented with difficulty in breathing and retrosternal chest pain for two day duration. Chest pain was tightening in nature and severe, lasted for six hours with no response to sublingual glyceryl nitrate. She also had associated autonomic symptoms. Subsequently chest pain was relieved.

Her initial examination findings on admission were, absence of fever, absence of pallor, blood pressure in both arms (100/60mmhg) without discrepancies, heart rate 88/min and regular low volume pulses. Pedal oedema was not observed. Jugular venous pulse was not elevated and precordium was normal. During inward stay she complained of intermittent pleuritic type chest pain which was not attributed to heart failure as there were no clinical signs suggesting heart failure. But, subsequently two days later a pericardial rub was noted. Urgent electrocardiogram revealed ST segment elevation in inferior leads II, III, aVF in both ECGs (Image 1). Thrombolysis was not done in private sector for inferior ST elevation myocardial infarction (STEMI), because of late presentation. This ECG finding persisted in the following days despite enoxaparin administration. All investigation findings were within normal limits except for the troponin. Troponin was positive with the values of 1.8 ng/ml. But, it was reduced to normal levels in the following days. Chest X-ray was done as patient had dyspnoea and widened superior mediastinum with enlarged aortic knuckle was evidenced in CXR.

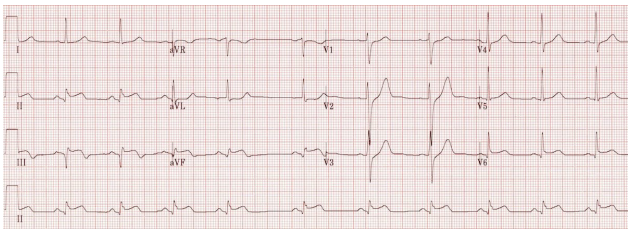

Image 1 .Electrocardiogram performed demonstrating inferior STEMI consistent with right coronary artery infarction.

Her transthoracic echocardiogram (TTE) findings suggested as moderate pericardial effusion due to an

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aortic dissection (*Image 2*). Computed tomography aortic angiogram showed an aortic dissection starting from the root of the aorta 1cm distal to aortic valve, extending along the aortic arch proximally, all the way down the descending aorta in to left common iliac artery and left common femoral artery suggesting Standard type A Aortic dissection. Her coronary angiogram was normal. Anti-platelet agents and anticoagulants (enoxaparin) were discontinued. Patient was advised bed rest and her target systolic blood pressure was tightly maintained below 110 mmHg as advised by the cardiothoracic surgery team. Patient underwent cardiothoracic surgery following day. Surgically correction done with a Teflon conduit. On day one postoperatively she was operated again due to a high intercostal drainage. She became hypotensive and ventilated requiring multiple inotropes. On day four post operatively she succumbed due to multi organ failure.

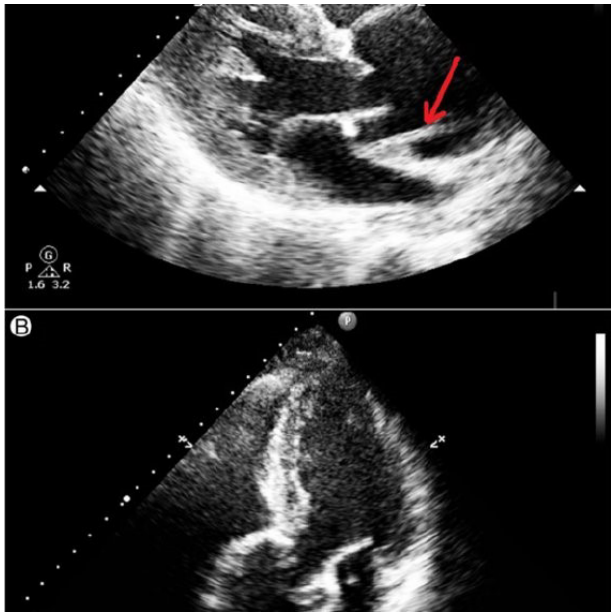

Image 2. Transthoracic echocardiogram images of aortic dissection.

Discussion

Myocardial infarction due to aortic dissection is uncommon. Stanford type A Aortic dissection (TAAD) can cause myocardial hypo perfusion in 8-10% patients due to retrograde dissection causing pressure effect in coronary ostium(1). TAAD is usually involved with right coronary artery (RCA) and causes inferior STEMI in 3% of patients(1). Of the patients presenting with both condition, only 1.5% patients will receive primary PCI(2). Imaging modalities are usually bypassed to proceed primary PCI to shorten the door to balloon me (DTB) (2). TAAD is associated high mortality of 1-2% per hour since the onset of symptoms which are diagnosed delay or left untreated(3). Atypical presentations of TAAD is challenging in clinical practice to diagnose and treat when initial ECG of patient has ST elevation(3). Inferior STEMI is one of the atypical presentation of TAAD(4). However, starting antiplatelet drugs or fibrinolytic agent in STEMI due to TAAD can cause catastrophic bleeding(2).

International registry for aortic dissection (IRAD) have established the factors involving delayed diagnosis TAAD (5). Atypical presentations of TAAD is one of the factor contributing to delayed diagnosis (6). Tearing chest pain, back radiation of pain and discrepancies in blood pressure or pulse volume may point TAAD (7). However, when clinical symptoms and signs are very subtle, TAAD may be easily missed. Having high suspicion for TAAD in inferior STEMI is essential to avoid the fatal consequences. Bedside transthoracic echo (TTE) could be easy and cost effective way to evaluate TAAD in the emergency setting(8). If a dissection flap present in TTE, the diagnosis of TAAD is straightforward. However dissection flap cannot be found in most of the cases(8). But presence of other finding like aortic root dilation, aortic regurgitation and pericardial effusion may points towards diagnosis of TAAD. CT aortography remains the gold standard method for diagnosis of TAAD(8).

Resection and replacement of thoracic aorta is the standard treatment for TAAD(2). So far, there is no consensus regarding the optimal management of STEMI due to TAAD. Early surgery is needed for the correction of AD(8). However when a unstable patient is opted for surgery correction, it may be beneficial to stent the coronary arteries and proceed to surgerical as evidenced by previous studies(8). Despite the modern surgical techniques, surgical mortality remains high(2).

Conclusion

STEMI is and uncommon complication of TAAD. Delayed diagnosis of TAAD may lead to lethal consequences as the damage is two folded because of bleeding into the false lumen with malperfused coronaries. Timely thrombolysis may result in catastrophic bleeding. TAAD can be easily missed unless high suspicion is maintained to diagnose. Early bedside TEE will help to diagnose without any delay. Surgical mortality due to AD remains high despite advancement in surgical technique. Coronary stenting

could be useful before surgery as a bridging tool for unstable patients.

Conflict of interest

None

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