CASE REPORT

Inferior wall Myocardial Infarction Masquerading like Peptic Ulcer Disease

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Abstract

In acute myocardial infarction, time equals muscle. Diagnosis and intervention within one to two hours of pain onset decreases mortality significantly.[1] An event of acute coronary syndrome (ACS) is usually symptomatic and commonly seen in elderly population. Hence, ACS event in younger population, especially if asymptomatic, makes it a formidable diagnostic challenge. The following case is of a 32-year old woman who presented to the emergency room with severe abdominal pain; without any associated symptoms of chest pain, shortness of breath, palpitations, dizzinessor sweating and was diagnosed with an inferior wall myocardial infarction (MI).

Keywords

Bed side ultrasonography, atypical myocardial infarction, regional wall motion abnormality

Case report

A 32-yr old patient complaints of severe abdominal pain of 6/10 in intensity and burning located in the epigastric region without any radiation; associated with nausea and without any aggravating or alleviating factors. She has a history of peptic ulcer disease and is on daily regimen of proton pump inhibitors (pantaprazole). She has no other significant medical history like diabetes mellitus, hypertension, or coronary artery disease (CAD). She has no known drug allergies and denies history of alcohol abuse, illicit drug use and smoking. She has no significant family history. In addition, all vital signs are within normal limits. Provisionally, she is diagnosed with peptic ulcer disease and was given intravenous pantaprozole 40 milligram; approximately half an hour later, she showed significant improvement in symptoms with decrease in pain. Electrocardiography (EKG) showed sinus rhythm with non specific ST segment changes noted in II, III and AVF leads. Troponin-T levels were normal. Bedside 2-D Echocardiography showed inferior region wall motion abnormality. Serial EKG's were ordered. Within an hour, ST segment elevation is noted in II, III and AVF leads.

A conclusive diagnosis of Inferior wall MI was made from the EKG changes and new onset of regional wall motion abnormality on echocardiography.

Discussion

An EKG is usually performed when the patient presents with complaints such as chest pain, palpitations, rapid pulse, shortness of breath, dizziness, lightheadedness and severe fatigue. Currently, no guidelines have been recommended by the American Heart Association (AHA) for doing an EKG in asymptomatic and low cardiac risk patients.[2] Cardiac risk factors like diabetes mellitus, hypertension, hypercholesterolemia, smoking, advanced age and family history of heart disease helps in provisional diagnosis. In a separate study conducted for analyzing the association of risk factors with the occurrence of MI, 21.3% of patients with four or five cardiac risk factors had acute myocardial infarction(AMI). On the other hand, 12.2% of patients with no cardiac risk factors had AMI.[3]

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AMI is diagnosed by detection of a rise of cardiac tropon in with at least one value above the 99th percentile with integration of at least one of the findings; symptoms of acute myocardial is chemia; new ischemic electrocardiographic (ECG) changes; development of pathological Q waves; Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology.[4–6] Type 1 myocardial infarction is an acute athero-thrombotic coronary event while type 2 myocardial infarction is due to an imbalance between oxygen demand and supply other than CAD.

In a separate study reviewing the missed diagnosis, out of a cohort of 112,000 patients with MI, diagnosis was missed in 993 patients (0.9%). Patients with the missed diagnosis had presented to the hospital with chest pain or other cardiac conditions, were subsequently discharged, and later got re-admitted with acute MI within 7 days.[7] Another study demonstrated that out of a cohort of 41,000 patients, 11% initially had a non-diagnostic EKG. One third of these patients with non diagnostic EKG had STEMI by 30 minutes and 50% of the remaining patients developed STEMI by 45 minutes. [8,9]

A high incidence of unrecognized myocardial infarction may also be due to the absence of the classic presentation, which is without risk factors, classical angina and/or without significant EKG changes. This leads to an increased mortality rate in patients with atypical presentation in comparison with the classic presentation of AMI.[10–13] The most common atypical presentation mimics peptic ulcer disease; around 7% of these patients get completely relieved with antacids, like in this presented case. There is no standard scoring system to grade these presentations or assist in reducing the number of missed diagnosis. Regional wall motion abnormalities (RWMAs) develop within seconds in severe cardiac is chemia and can be visualized with echocardiography. These wall motion changes occur prior to the onset of changes in EKG or the development of clinical symptoms which usually take a few minutes. Hence, evaluation of wall motion with echocardiography can be useful in such cases where in the EKG is non diagnostic but has a high suspicion of ischemic heart disease.[14]

Conclusion

Cardiac risk factors help to diagnose ACS but absence of cardiac risk factors does not completely rule out ACS. Even if antacids alleviate the patient's pain, the risk for AMI remains. Young patients can have atypical presentation of AMI mimicking as acute abdominal pain. As initial EKG and troponins can be normal in patients with acute abdominal pain with underlying MI, a single normal EKG or normal troponins is not enough to discharge the patient; an EKG should be repeated before discharge. In addition, bedside echocardiography should be done to assess wall motion before high risk patients even with non-cardiac complaints are discharged. New guidelines for diagnostic bed side echocardiography are needed to avoid missing of AMI with atypical symptoms.

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