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Case Report

NSTEMI as Low Back Ache in a Patient with Type 2 Diabetes

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Abstract

An unusual case of NSTEMI presenting as low backache is described. NSTEMI is a complication of type 2 diabetes mellitus. It may have a variety of atypical presentations. We present here a case of a 54-year-old male presenting with low back pain but no chest pain, later diagnosed with NSTEMI based on serial troponin measurements.

Keywords: Low back ache; NSTEMI; Type 2 Diabetes Mellitus

Introduction

Acute Coronary Syndrome (ACS) is associated with signifi cant morbidity and mortality. While NSTEMI has been re ported to dominate in the Western population (70%); Indian studies show a preponderance of STEMI (70%) [1,2]. The median age at presentation also differs almost by a decade (68 years versus 56 years). Males outnumber females by a 3:2 ratio in their Western counterparts while an Indian report indicated nearly equal distribution [1,2]. The typical presentation of NSTEMI involves pressure-like substernal pain at rest or with negligible exertion radiating to the arm, neck, or jaw and lasting for more than 10 minutes [1,2]. Atypical symptoms include burning, stabbing or pleuritic pain stabbing, characteristic of indigestion [1-3]. Generally, patients presenting with ACS manifest typical symptoms. However, the probability of atypical presentations increases with advancing age, female gender, and in those with comorbidities like diabetes [4]. We present here a male patient with long-standing diabetes and atypical manifestations of NSTEMI.

Case Report

A 54-year-old medical superintendent with a14-year history of Type 2 Diabetes (T2D) presented to the emergency department with a sudden onset of severe low backache. It was the day of the earthquake and he was very tired at the end of the day after having being involved in the coordination of various departments in the aftermath of the catastrophe. The pain was mild in onset for a couple of hours initially, which suddenly aggravated. He appeared pale and weak with severe nausea, repeated vomiting, and profuse sweating. He was afebrile with no episodes of diarrhoea.

His physical examination on presentation showed a pulse rate of 110/minute, blood pressure of 110/80 mm Hg, respiratory rate of 27/min, and oxygen saturation of 97% in room air. The ECG was normal and Troponin I was negative (<0.03ng/ml).

His complete blood count, liver and kidney function tests, se rum amylase and lipase were within normal limits. Since ECG was normal and troponin I was negative, ACS was momen tarily ruled out.

Multi-dose insulin therapy was initiated along with an injec tion of tramadol for his backache. Nevertheless, the pain did not subside. An USG was performed which revealed a Grade 1 fatty liver. As per the 2016 ESC/GRACE, the target patient was suspected of ACS based on presentation and serial troponin measurements were planned. Though repeat serial ECGs did not reveal any abnormality, a repeat Troponin I around 6 hours of pain onset was positive (0.91ng/ml). His echocardiography demonstrated akinetic segments in the anterior cardiac wall with mildly reduced ejection fraction (48%). His chest radiograph was unremarkable.

The ischaemic risk was calculated based on the GRACE (Global Registry of Acute Coronary Events) score and the bleeding risk by the CRUSADE (Can Rapid Risk Stratifica tion of Unstable Angina Patients Suppress Adverse Outcomes with Early) Bleeding Score. The scores were found to be low [GRACE-105; (<140)] and therefore the patient was managed conservatively using vasodilator, antiplatelet and anticoagulant therapy. Statin and an ACE inhibitor were initiated. Throm bolysis was not required. The pain lulled and the patient was hemodynamically stable with no arrhythmia.

As a precautionary measure, the MRI was performed which re vealed disc protrusion and osteophytes at multiple levels along with nerve compression and radiculopathy with the absence of spinal stenosis. He was referred to a physiotherapist for further management.

Discussion

In the current case, the patient developed sudden aggravation

of initial mild onset low back pain without pain in the chest. Therefore, initial manifestations were suggestive of musculo-skeletal aetiology. Negative troponin and normal ECG also cor roborated non-ACS pathology. However, other characteristics such as profuse sweating, nausea frequent vomiting, and pain unyielding to tramadol led to suspicion towards an alternate diagnosis of ACS. Besides long-standing T2D was also a risk factor to be considered. Repeat evaluation revealed positive troponin with the absence of ST elevation. Hence a diagnosis of NSTEMI was made which guided subsequent management of the patient. Since evaluation revealed low risk, the patient was managed conservatively. NSTEMI usually results from severe coronary artery narrowing, transient occlusion, or mi croembolization of thrombus and/or atheromatous material [5]. The 54-year-old male patient in the current case was diabetic for 14 years. The atypical presentation is unique since litera ture has reports of upper and middle back pain however re ports of low back pain and ACS are lacking [6]. The evidence available regarding the association of low back pain with ACS is conflicting. Some authors have suggested that pain in some cases may be an early manifestation of atherosclerosis. Events causing or exacerbating local ischemia of the lumbar region may cause back pain. Non-specific back pain may have a vas cular basis; either underlying atherosclerosis or a defect caus ing transient ischemia [7]. Atherosclerosis limits blood supply and nutritional exchange to the lumbar intervertebral disc, pro moting the risk of disc degeneration [8].

It has been suggested that negative conventional cTn assay (value below the 99th percentile cut-off) at 0 and 3 hours of ED presentation, among low-risk chest pain patients, could un failingly rule out AMI [9]. However, in our patient troponin was positive at 6 hours. A recent prospective cohort study, us ing a large cohort of patients presenting to the ED with ACS symptoms, proposed that a small proportion of patients with intermediate cTnI values may need a third 6-hour cTnI mea surement for rule-in or rule-out of NSTEMI among patients with suspected ACS. Therefore, we believe Ottawa Troponin Pathway may be of significant clinical utility in the diagnosis of NSTEMI.

Conclusion

Long-standing T2D increases the risk of ACS as in our patient. Serial troponin assessments should be performed on those at risk of presenting with atypical manifestations even in the ab sence of chest pain.

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