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 significant morbidity and mortality. While NSTEMI has been reported 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 a 14-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 been 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).

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 corroborated 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 microembolization 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 literature has reports of upper and middle back pain however reports 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 vascular basis; either underlying atherosclerosis or a defect causing transient ischemia [7]. Atherosclerosis limits blood supply and nutritional exchange to the lumbar intervertebral disc, promoting 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 unfailingly rule out AMI [9]. However, in our patient troponin was positive at 6 hours. A recent prospective cohort study, using a large cohort of patients presenting to the ED with ACS symptoms, proposed that a small proportion of patients with intermediate cTnl values may need a third 6-hour Ctnl measurement 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 absence of chest pain.

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