International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Case Report

Acute Anterior Wall Myocardial Infarction: An Unexpected Cause

Kiron Sukulal[1], Ajit Deshmukh[2]

[1]Assistant Professor, Department of Cardiology.
[2]Assistant Professor, Department of Radiodiagnosis and Imaging,
Sree Mookambika Institute of Medical Sciences, Kanyakumari District, Tamil Nadu, India.

Corresponding Author: Kiron Sukulal

Received: 26/03/2015 *Revised: 28/04/2015* *Accepted: 01/06/2015*

ABSTRACT

A chronic alcoholic presented with ECG changes suggestive of acute anterior wall myocardial infarction. He was in cardiogenic shock and renal failure. Patient improved symptomatically after thrombolysis and inotropic medications. His ECG and echocardiogram normalised 2 days after admission. His coronary angiogram was normal. After recovering from alcohol withdrawal symptoms he revealed a history of fall on the day prior to admission. Magnetic resonance imaging revealed acute cervical spinal cord injury. The severe hypotension due to spinal cord injury was the culprit responsible for his cardiogenic shock and renal failure.

Keywords: myocardial infarction, spinal cord injury.

INTRODUCTION

A 50 year old chronic alcoholic admitted with ECG changes suggestive of acute anterior wall myocardial infarction and in cardiogenic shock was initially managed with thrombolytics and inotropes. The coronary angiogram was normal. On detailed evaluation he was detected to have odontoid fracture in cervical spine and kinking of spinal cord due to a fall on previous day of admission. The acute spinal shock after the fall had produced severe hypotension and coronary ischemia. Spinal cord injury in young men is most commonly due to fall. [1]

CASE REPORT

A fifty year old chronic alcoholic male was admitted with ECG suggestive of acute anterior wall myocardial infarction (right bundle branch block with ST segment elevation in V1 and V2- Figure 1). He was drowsy and in cardiogenic shock and renal failure. Echocardiographic evaluation showed decreased contractility of anterior left ventricular (LV) wall. Since his renal parameters were high, primary angioplasty was not considered. He was thrombolysed with streptokinase and started on inotropic support. Next day he had delirious symptoms due to alcohol withdrawal. His haemodynamic status and renal function improved gradually. His ECG taken 2 days after admission was normal (Figure 2).

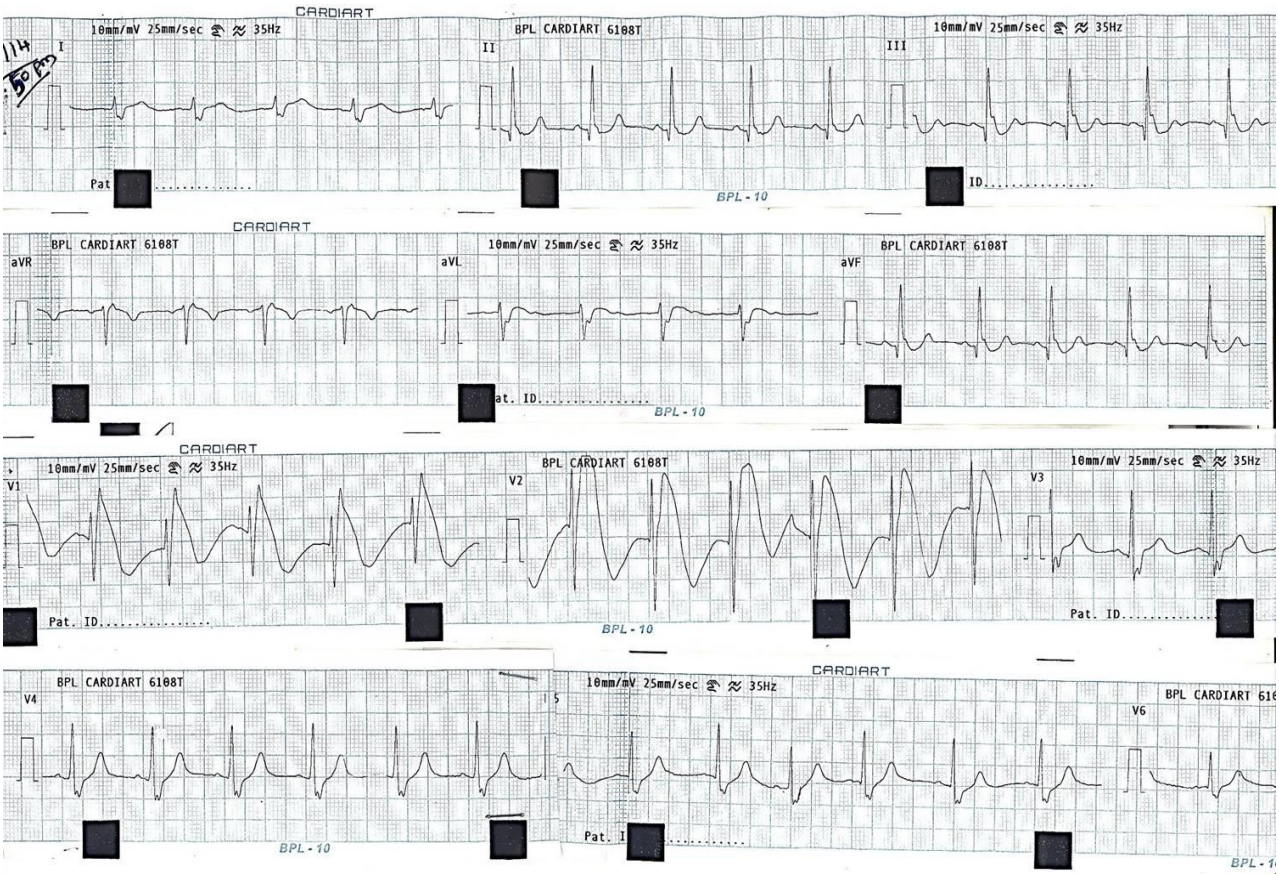

Figure-1

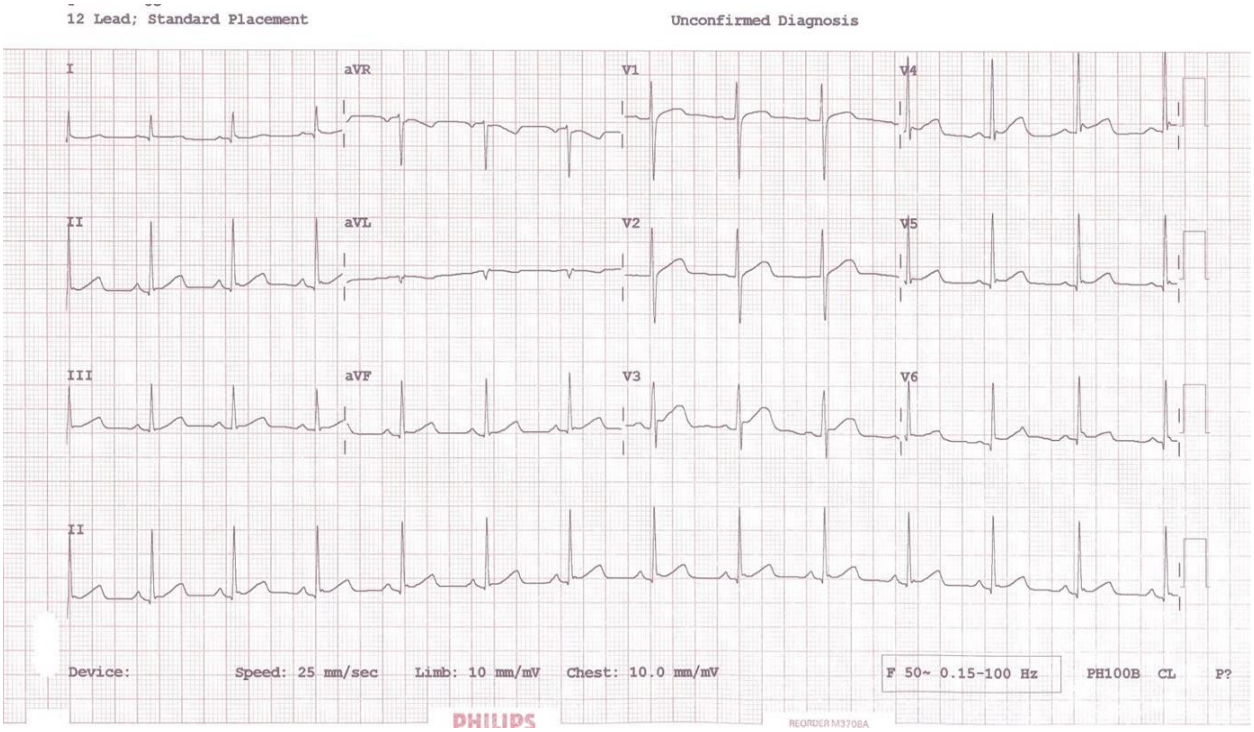

Figure-2

Repeat echocardiogram showed improved left ventricular function and normal LV contractility. A coronary angiogram was plannedas his renal function had improved. His coronary angiogram showed normal coronaries (Figure 3).

Once his sensorium improved, he recollected an episode of syncope and fall on the previous day of admission. Clinically he had neck stiffness and quadriparesis. Magnetic resonance imaging of the cervical spine showed type 3 odontoid fracture(thin arrow) and posteriorly displaced fractured segment causing kinking of spinal cordat C2 level (thick arrow) (Figure 4). The acute spinal shock due to spinal cord injury (SCI) had triggered the above mentioned cascade of events. The severe hypotension produced by spinal shock had reduced coronary and renal perfusion. The coronary perfusion was reduced to the extent of producing ECG changes suggestive of myocardial infarction.

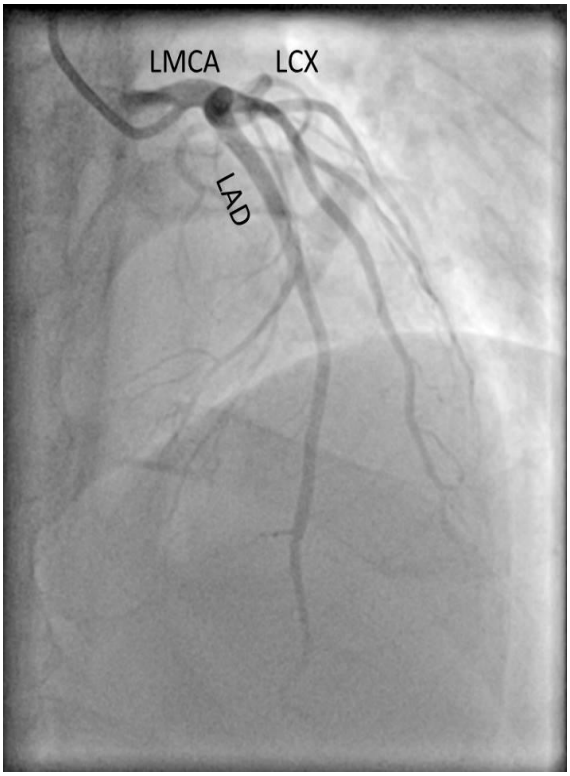

Figure-3

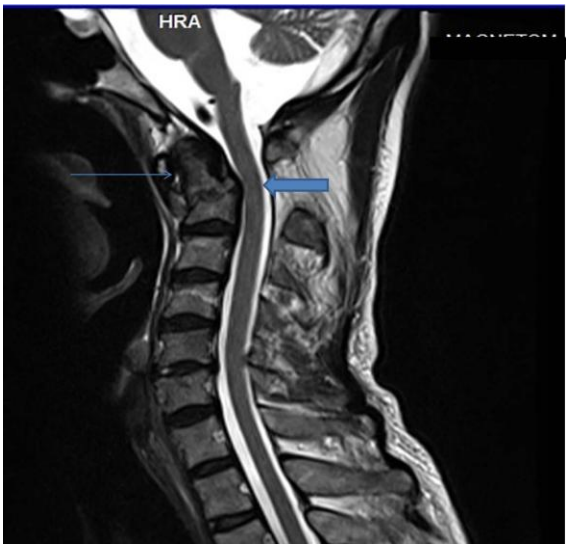

Figure-4

DISCUSSION

Spinal shock occurs due to acute spinal cord injury which can result in complete and incomplete transection of spinal cord. SCI mainly occurs in young men and falls account for a major aetiology. The commonest mechanism in trauma is subluxation of the vertebral elements causing a pincer like direct damage to the cord. A four-phase classification to spinal shock has been postulated: areflexia (Days 0–1), initial reflex return (Days 1–3), early hyperreflexia (Days 4–28), and late hyperreflexia (1–12 months). [2]

From a cardiovascular point of view, initially a massive release in catecholamines occurs, particularly after high cervical injury producing dramatic hypertension and tachycardia. Following this phase, disruption of descendent pathways results in sympathetic hypoactivity and hypotension. Hypotension is attributable to a combination of vasodilatation, decreased inotropism, and bradycardia (if above T5 and cardiac sympathetics are involved). Bradycardia occurs because of unopposed vagal tone. Cardiovascular disturbances are the leading causes of morbidity and mortality in both acute and chronic stages of SCI. [3]

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How to cite this article: Sukulal K, Deshmukh A. Acute anterior wall myocardial infarction: an unexpected cause. Int J Health Sci Res. 2015; 5(7):452-455.

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