ST elevation - An indication of reversible neurogenic myocardial dysfunction in patients with head injury

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ABSTRACT

This report describes a patient who presented with signs of meningitis four days after head injury. The patient had ST elevation on electrocardiography along with hypotension and positive troponin T test, mimicking inferior wall infarction. The ST changes resolved within 48 hours of intensive care management. Subsequent investigations failed to document any myocardial infarction.

Key words: Head injury, ST elevation, reversible neurogenic myocardial dysfunction

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INTRODUCTION

Different electrocardiographic (ECG) abnormalities following head injury have been cited in literature, including the pediatric age group.[1] ST segment elevations in the ECG along with hypotension and elevated levels of troponin T as a sequela of head injury has not been reported. This report describes features of inferior wall myocardial infarction in a patient who had sustained head injury in the recent past and developed meningitis. The ECG changes and hypotension persisted for 48 hours. Cardiac angiography and Tc-99m pyrophosphate (PYP) myocardial infarct scan proved the non cardiac origin of the ST elevation. Intensive care management may improve the outcome of patients who have neurological impairment along with ST elevation.

CASE REPORT

A 31-year-old male patient presented to our emergency ward with headache, fever and irritability. He had sustained a road traffic accident four days back, associated with bleeding from right ear. There was no significant past medical history including any cardiac illness. On examination, the heart rate was 120/minute and the arterial pressure was 120/90 mm Hg. He was febrile with axillary temperature of 102°F. The score on Glasgow coma scale was 14 (E 4V4M6), as the patient had a confused speech with spontaneous eye opening and a dull response to verbal command. ECG monitoring showed sinus tachycardia. Subsequently, he had a generalized convulsion followed by loss of consciousness. The trachea was intubated and ventilation was mechanically controlled. A computed tomographic (CT) scan showed fracture of petrous part of the temporal bone along with small specks of air in the left frontal and right occipital region. The patient was shifted to the neurosurgical intensive care unit (ICU) for ventilatory support. He had hypotension, not responding to boluses of intravenous fluids. A central venous catheter was inserted which recorded a central venous pressure of 10-12 cm H2O. Hypotension was accompanied with significant elevation of ST...
segment in Leads II and V5 of the ECG monitor. A 12 lead ECG showed ST segment elevation of the inferolateral leads [Figure 1]. A bedside Troponin T test suggested myocardial ischemia. The patient was diagnosed to have an acute inferolateral wall myocardial infarction. Dopamine infusion was commenced at a dose of 10μg/kg/minute to stabilize the blood pressure. Cardiac medications were initiated, consisting of oral aspirin 100mg, metoprolol 12.5mg, isosorbide-5-mononitrate 30mg, trimetazidine 35mg and Atorvastatin 20mg. He localized only to painful stimuli. Cerebrospinal fluid cytology was suggestive of pyogenic meningitis for which appropriate antibiotics were started. Continuous monitoring and serial ECGs were done. Over next 36 hours the patient became afebrile, hemodynamically stable and showed marked neurological improvement. Dopamine infusion was tapered off. Mechanical ventilation was discontinued and the tracheal tube was removed. Within 48 hours of admission to ICU there was improvement of the ST segment elevation in the ECG [Figure 2]. An echocardiography carried out two days later, suggested mild global reduction of cardiac contractility with left ventricular ejection fraction of 45%. Normal coronaries were seen on angiography performed on 9th day and a Tc-99m pyrophosphate (PYP) myocardial infarct scan on 10th day revealed a normal radiotracer uptake by the myocardium. All the cardiac medications were stopped. The patient was discharged from the hospital on 10th day with full Glasgow Coma Score (E4V5M6) and no neurological deficit.

DISCUSSION

There can be varied causes of ST segment elevations in the ECG of a patient with head injury. It can occur as the earliest sign of an acute myocardial infarction. However, they are typically followed by evolving T wave inversions and often by Q waves in the same lead.[2] In our patient the ST elevation was noted initially, which normalized subsequently. Additionally, normal coronaries and radionuclide scan ruled out the possibility of myocardial infarction. In acute pericarditis the ST segment elevations persist for several days.[3] The other possibility that could have led to such ECG changes following road traffic accident is cardiac contusion.[4] There was no evidence of chest injury. Hemodynamic instability with cardiac contusion is suggestive of severe myocardial injury. However there was no such evidence on radionuclide imaging in our patient.

ST segment elevations have also been seen in patients with neurogenic stunned myocardium.[5,6] We suspect myocardial dysfunction in our patient following the neurological sequelae, which recovered within 48 hours following admission to ICU. This is similar to an observation in an animal model whereby depressed cardiac function following temporary coronary occlusion improved by 48 hours.[7] Our patient had post-traumatic meningitis. It is probable that the convulsive seizures would have led to an increase in the intracranial pressure and consequently a decrease in cerebral perfusion pressure. The resultant sympathetic activation would have led to coronary vasospasm along with myocardial dysfunction.[8] However myocardial dysfunction has the ability to improve its contractile performance under inotropic stimulation,[9] which was observed in our patient. There were ST segment elevations in the ECG and hypotension which returned to normal corresponding to improvement in neurological status. We believe that ST segment elevations in the ECG of our patient following the neurological sequelae as a case of reversible neurogenic myocardial dysfunction.

The phenomenon of reversible neurogenic myocardial dysfunction following ST elevation in the ECG has not been described in patients with head injury. There are only animal models and one case report where in ST elevation has been known to occur following head injury.[10,11] We ruled out cardiac contusion and myocardial injury in our patient using a Tc-99m pyrophosphate (PYP) scan unlike Wittebole et al.[11]
Their patient did not manifest hemodynamic instability, unlike ours. The occurrence of ST elevation in the ECG of patients with subarachnoid hemorrhage has been well documented.\textsuperscript{[5,6,8,12]} The incidence of ECG changes in the patients with subarachnoid hemorrhage has been shown to correlate with the amount of intracranial blood.\textsuperscript{[13]} However, in a report, there was a closed head injury with no evidence of intracranial blood.\textsuperscript{[11]} This is suggestive of the fact that ST elevations in the ECG can occur in neurosurgical patients with raised intracranial pressure due to causes other than intracranial bleeding.

It is relevant for the anesthesiologists in the practice of neurotrauma to understand the phenomenon of reversible neurogenic myocardial dysfunction. ST elevation may result in a dilemma during the management of such a case. In a neurosurgical setting it is possible that seeking a cardiology opinion and going through a battery of tests for diagnosing the cause of ST elevation may take time and delay may result in adverse outcome.

ST segment elevations in the ECG of a neurosurgical patient should be viewed seriously. It may not indicate myocardial infarction in a patient with neurological injury. The management and probable outcome of patients with head injury along with such ECG changes is unknown.

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