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A Rare Presentation of Acute Coronary Syndrome with Posterior Fossa Intracranial Bleed

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Abstract Without significant coronary artery stenosis, ischemic ECG changes including ST segment elevation, segmental wall abnormality & elevated serum cardiac specific markers may develop after central nervous system injuries. Misdiagnosing these patients as acute MI may result in catastrophic outcomes. By reporting case of 56 year old male with posterior cranial fossa haemorrhage mimicking ST segment elevation MI, we hope to underline that careful attention to neurological abnormalities is critical in making correct diagnosis.

Keywords: *myocardial infarction, posterior cranial fossa haemorrhage*

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1. Introduction

ECG Changes are common in patients with cerebrovascular diseases, including subarachnoid haemorrhage, subdural haematoma & ischemic stroke [1,2,3], but typical ST segment elevation is rare. The exact phenomenon is not clear but the changes can be caused predominantly in the subendocardium of the left ventricle due to ischemia, which, in turn, can be caused by centrally mediated release of catecholamines induced by hypo-perfusion of posterior hypothalamus. Mild elevation in serum cardiac enzymes is often associated with ECG changes [4]. We would like to emphasize careful attention to abnormalities in neurological events, which can enable better evaluation and management.

2. Case Presentation

A 56-year old male patient presented to the emergency department with complaint of chest heaviness for several hours associated with vertigo and 6–8 episodes of vomiting. Clinical examination of the patient showed good general conditions with rhythmic pulse. The additional risk factor found along with the above mentioned complaint was hypertension. On arrival, he had no neurological symptoms and his blood pressure was 213/103, pluses 77 beats/minute, body temperature was 99F, respiratory rate was 18 breaths/minute, O2 saturation was 99% on room air and plasma glucose was 4.6619 mmol/l. On initial examination, we did not find any head trauma, abnormal respiratory sound or cardiac murmur. The ECG on presentation had ST segment elevation from leads V2–V4, & T-wave inversions in V6, II, AVL & AVF.

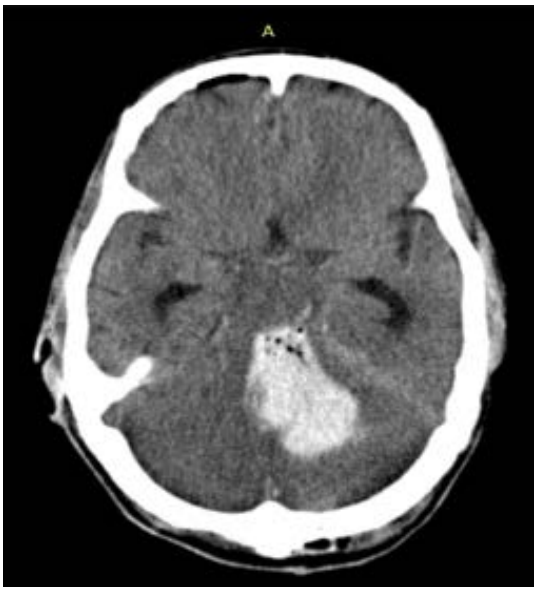

Figure 1. CT scans of the patient

Laboratory examination showed: LDH 281 (range: 24-195 u/l), CK-MB: 34 (< 25 u/l), SGOT (AST): 24 (upto 37 u/l). The echocardiogram showed symmetrically hypertrophied normal sized left ventricle with mild segmental left ventricular systolic dysfunction. Grade 1 diastolic dysfunction with ejection fraction: 50%. During patient's stay in emergency department, he vomited again and his blood pressure was continuously around 210/102. The CT scan brain (without contrast) was performed immediately, which showed posterior cranial fossa haemorrhage. After undergoing craniotomy and ventriculoperitoneal (VP) shunt placement, the patient's condition improved and he was discharge in 15 days without any neurological deficit.

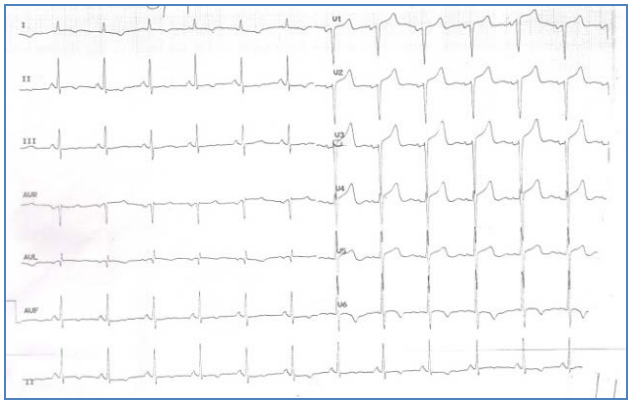

Figure 2. ECG of the patients

3. Discussion

Abnormalities of ECG, echocardiography and serum cardiac specific markers are related with cerebrovascular disease [5,6,7,8,9]. Hypertension is usual cause of primary haemorrhage into pons or cerebellum due to displacement of vessels specially in the absence of trauma. The incidence of hypertension is greater than one fifth of all spontaneous intra-cerebral haemorrhage (10). Some patients have abnormalities on ECG including a prolonged QT interval, depressed ST segment, rarely elevated ST segment, inverted or flat T waves, tall peaked T waves & U waves. These changes are most likely due to centrally mediated release of catecholamine induced by hypoperfusion of posterior hypothalamus. The autopsies of patients have revealed that patients with posterior fossa hemorrhage also have characteristics subendocardial myocardial lesion called contraction band necrosis in the heart [11,12,13]. In spite of all these studies the exact phenomena is still uncertain & future studies are needed.

When patient present in emergency department with no typical neurological symptoms but with initial reading of very high blood pressure and ECG show ST segment elevation, we may misdiagnose as acute MI and treat with multiple antiplatelet & anticoagulant agents, which may lead to harmful effects [14]. Moreover, by delaying the accurate diagnosis, it may result in catastrophic outcome. In this patient there is one reflecting point is his continuous raised blood pressures and we all are well known that stress or chronic hypertension induced cardiomyopathy may developed by cerebrovascular accidents. So even patient is present with acute ST elevation MI, we should consider a cerebral insult and act promptly to save the life of patient.

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