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Case Report

Subarachnoid haemorrhage mimicking as myocardial infarction

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ABSTRACT

Electrocardiographic (ECG) changes due to subarachnoid haemorrhage (SAH) are seen frequently and mimic acute myocardial infarction. For appropriate therapeutic management it is very important to distinguish acute coronary syndrome from neurogenic myocardial injury, which is a reversible condition. A 35 year old male presented to us with history of acute chest pain, ECG suggestive of anterolateral myocardial infarction for which he underwent anticoagulant therapy. It was subsequently diagnosed to be a case of SAH due to ruptured anterior communicating artery aneurysm.

Keywords: ECG changes, Myocardial infarction, Subarchnoid haemorrhage

INTRODUCTION

Subarachnoid haemorrhage account for only 5% cases of stroke but at young age. Sudden onset severe headache (often described as worst headache of their life) is a cardinal feature.[1] Acute, severe headache in general practice indicated a serious neurological disorder in 37% (95% Cl 29-45%), and subarachnoid haemorrhage in 25% (18-32%). 12% (5-18%) of those with headache as the only symptom.[2] Nine studies of good quality, which reported the number of patients with aneurysmal subarachnoid haemorrhage with a history of sentinel headache, gave rates of 10% to 43%, true incidence may vary from near zero to about 40%.[3] Patients with acute vascular disorders of the CNS demonstrate an abundance of both rhythm and morphologic changes in their ECG. Of these a few will demonstrate myocardial dysfunction and or damage.[4] Approximately half of patients whose cardiac arrest is due to an intracranial haemorrhage may collapse without complaining of a headache.[5]

CASE REPORT

A 35 year old male with non-radiating acute chest pain was diagnosed initially as case of anterolateral myocardial infarction on basis of ECG changes (Figure 1a) in some another center.

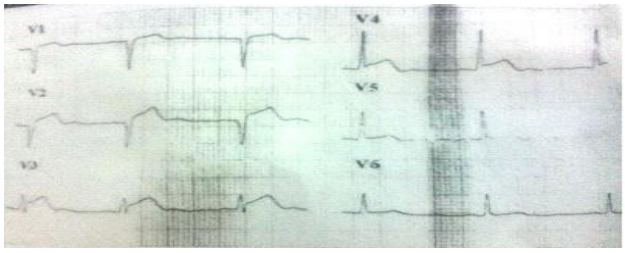

Figure 1(a): ECG showed anterior wall myocardial infraction (ST segment elevation in lead V2 to V4).

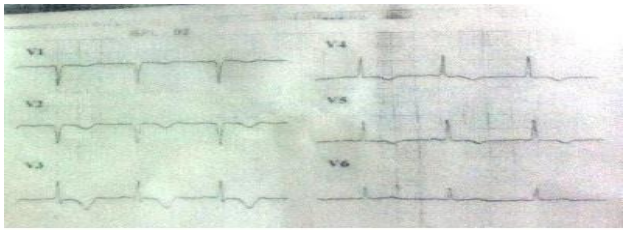

Figure 1(b): post thrombolytic ECG (ST segment return to base line with mild T- wave inversion.

Patient underwent thrombolytic therapy with injection Velix™ (tenecteplase) after which he had acute sudden severe headache and vomiting. ECG had done showed features of cerebrovascular accident (Figure 1b-1c).

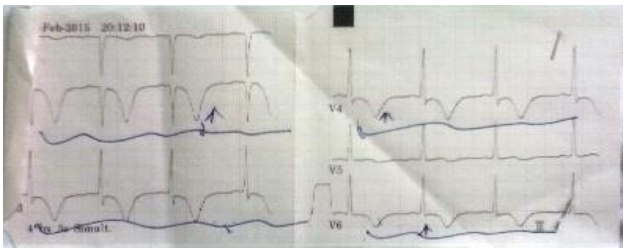

Figure 1(c): ECG showing features of cerebrovascular accident.

Non-contrast CT scan head done showing diffuse subarachnoid haemorrhage and anterior interhemispheric hematoma (Figure 2A). CT Angiography revealed anterior communicating artery aneurysm (Figure 3).

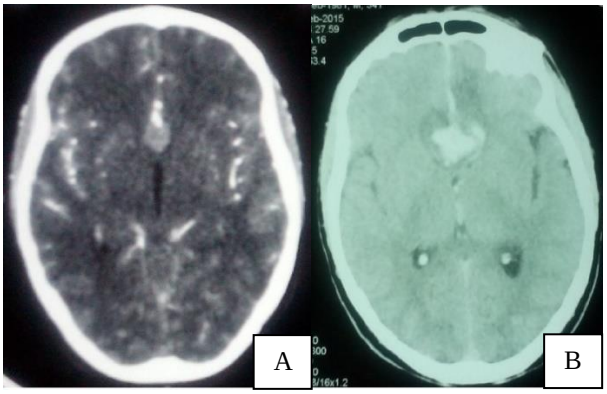

Figure 2(A): NCCT showing diffuse SAH done in another centre; (B) NCCT showing increase in size of hematoma.

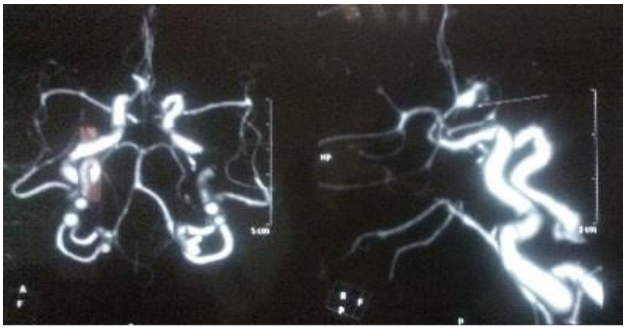

Figure 3: CTA brain showing Acom aneurysm.

Patient was referred and presented in WFNS grade 1 with complains of headache. NCCT head showed increase in size of haematoma probably re-bleed. (Figure 2B). 2D echocardiography was normal and troponin (Trop-T) test was negative. Patient underwent Right Pterional craniotomy and clipping of aneurysm. Post op was uneventful except for Right pneumothorax due to ruptured bulla (patient was a ganja addict) that was treated with a chest tube. Post-operative ECG was unremarkable (Figure 5). Patient discharged in stable condition, GCS-15/15 with no focal neurological deficits. Doing well at 1 year follow up.

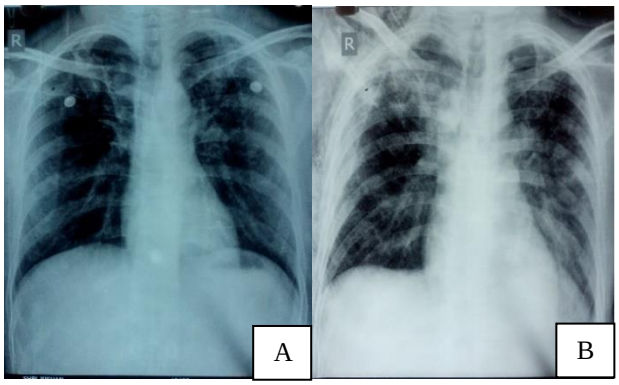

Figure 4(A): postop right side pneumothorax; (B) post ICD recovery no pneumothorax.

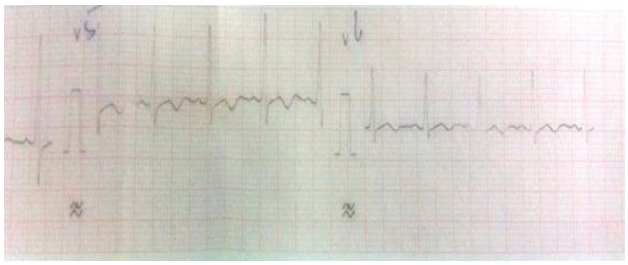

Figure 5: Post-operative ECG showing T-wave return to baseline.

DISCUSSION

In 1947 Byer et al firstly reported ECG changes in patients with cerebrovascular accidents.[6] Subarachnoid haemorrhage (SAH) is a neurological emergency with high mortality rates. It is mainly caused by an aneurysm (congenital/infectious/traumatic) or rupture of an arteriovenous malformation. It has been reported that abnormalities of electrocardiography, echocardiography and serum cardiac specific markers are associated with cerebrovascular disease.[7] The most common cause is subarachnoid haemorrhage but additional causes include head injury, meningitis and brain tumor.

Subarachnoid haemorrhage usually present with headache, only few cases presented with acute chest pain later they diagnosed to have spinal subarachnoid haemorrhage.[8] Alteration of rhythm and conduction have been detected in about 4% of patients with SAH, the most common arrhythmias are sinus bradycardia and atrial fibrillation/flutter (76% of arrhythmias observed by Frontera et al).[9] An electrocardiographic pattern was encountered in patients with cerebrovascular accidents which consisted primarily of T waves of large amplitude and duration. Large U waves were often present which

may fuse in part or entirely within the T wave. Because of the presence and fusion of the T and U waves, the prolonged Q-T interval associated with the pattern was most probably a Q-U interval.[10]

Anomalies of repolarisation are observed in about 25-75% of patients with SAH, especially in the first three days after admission. Because the repolarisation changes are often similar to those seen in myocardial ischemia and infarction, the interest to this subject has increased to avoid erroneous diagnosis of acute coronary syndrome that could interfere with a correct therapeutic management.

Moreover, approximately 40% of patients with SAH show a modest elevation of Troponin, CPK and CK-MB, although they do not rise to levels observed during acute myocardial infarction. Troponin elevation is correlated more to the degree of brain injury than to severity of cardiac dysfunction evidenced by echocardiography.

The most plausible pathologic theory remains an autonomic dysregulation caused by a lesion of cortical, hypothalamic and mesencephalic centers controlling the autonomic nervous system. An elevated concentration of catecholamines was observed in the hearts of animal models of intracranial haemorrhage.[11] Furthermore, catecholamine plasmatic levels are markedly elevated in patients with ECG changes than in patients without ECG variations.[12,13]

In about 3% of patients, cardiac arrest occurs at onset of the subarachnoid haemorrhage due to a fatal cardiac rhythm; resuscitation is essential, because half the survivors regain independent existence. Musuraca et all reported with a case that there are pitfalls for prehospital thrombolysis among the patients with intracranial haemorrhage due to these electrocardiographic changes.[14]

In patients with a subarachnoid haemorrhage troponin is elevated in approximately 20% and ECG changes without prognostic significance occur in 50 to 100%. The combination with an ST-elevation myocardial infarction is very rare. When ST-elevation myocardial infarction complicates a subarachnoid haemorrhage, a rapid decision about percutaneous coronary intervention and the use of antithrombotic drugs has to be made.[15] whenever feasible, immediate treatment of the aneurysm followed by percutaneous coronary intervention should be performed, followed by administration of antithrombotic agents.

In this case subarachnoid haemorrhage presented with acute chest pain and ECG showed ST segment elevation and anterolateral myocardial infraction. (ECG changes in SAH commonly reflect ischaemic heart disease and have been known to present with ST-segment elevation and T-wave inversion).[16] Patient was misdiagnosed and underwent thrombolytic therapy. Patient coming to emergency with acute chest pain may be a manifestation of subarachnoid haemorrhage so they should be properly investigated before starting thrombolytic therapy.

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