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RESEARCH ARTICLE

A CASE OF MULTIPLE NON-HAEMORRHAGIC INFARCTION IN A PATIENT ON STEROIDS FOR IMMUNE THROMBOCYTOPENIC PURPURA

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ABSTRACT

A 46-year-old male patient with a medical history of diabetes mellitus was diagnosed as ITP based on low platelet counts of 4 x 10^9/L. While being on oral prednisolone therapy, patient developed seizures and heavy speech. Imaging studies showed multiple small acute established non-haemorrhagic infarction in the left frontal and temporal regions. Patient was treated initially with aspirin 300 mg and subsequently continued on 80 mg aspirin daily for acute ischemic stroke. Further workup did not reveal any clear aetiology for the multiple non-haemorrhagic infarction. Patient had a short stay in ICU and was later transferred to the medical ward. Patient was thought to have had a complex partial seizure with secondary generalization secondary to hyperacute stroke. Etiology of stroke was thought to be most likely due to prothrombotic state.

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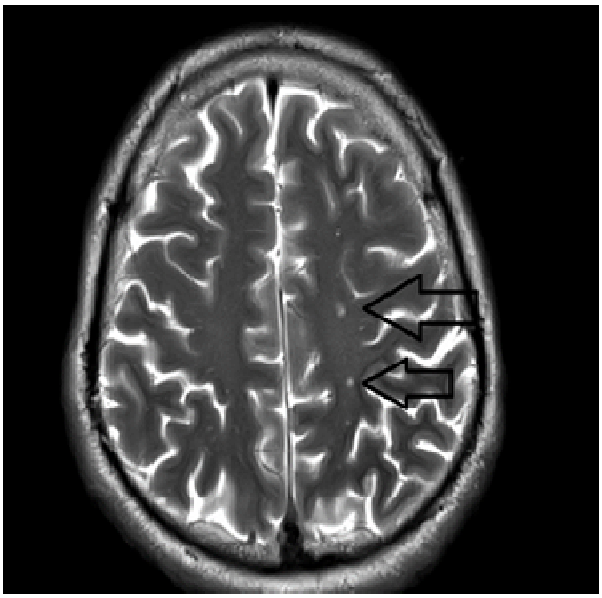
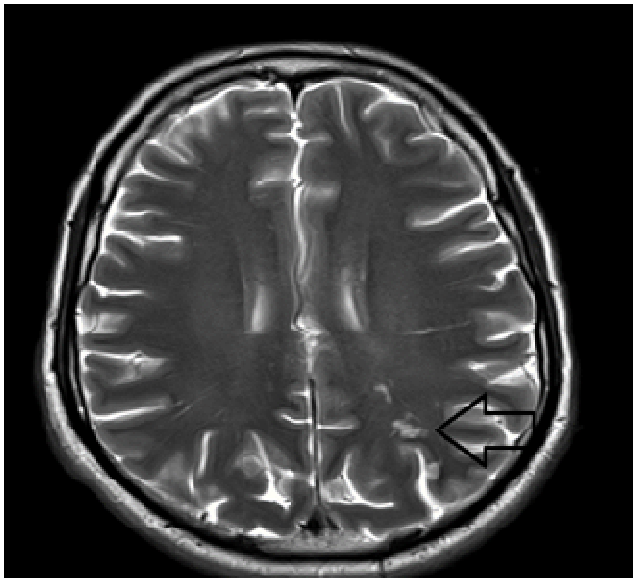
INTRODUCTION

A 46-year-old male presented to the Emergency Department with petechial rash and upper respiratory tract symptoms. Past medical history was only significant for diabetes mellitus for which the patient was taking regular oral hyperglycaemic medications. He had previously attended a primary health clinic one day ago where he was started on oral antibiotics for presumed respiratory tract infection. The petechial rash was localized in the lower limbs and the patient also had patches on tongue and was noted to have mucous membrane involvement. He had a small bullae on the ring finger of the right hand. There was also evidence of sub-conjunctival haemorrhage. An initial diagnosis of drug reaction secondary to antibiotic use or Idiopathic thrombocytopenic purpura was made. Before the blood results were processed by the pathology laboratory, patient left the Emergency Department against medical advice. The pathology laboratory later called informing that the platelet counts were only 4 x 10^9/L.

Patient re-presented next day with haematuria and worsening of rash. Upon re-examination patient still had evidence of sub-conjunctival haemorrhage, as well as presence of bilateral petechial rash in both lower limbs and truncal region. The previous small bullae on the right hand ring finger had now become swollen with evidence of ecchymosis. Repeat blood results showed platelet counts of only 4 x 10^9/L, however, haemoglobin, INR and APTT were normal. Patient's case was discussed with the haematology team and it was decided to admit the patient to the hospital. He was given glucocorticoids and intravenous immunoglobulin for treatment of immune thrombocytopenic purpura. Patient was subsequently discharged by the haematology team two days later on oral steroids with follow up in outpatient haematology team. Upon discharge, patient had platelet counts of 52 x 10^9/L.Patient was seen in outpatient haematology clinic one week later and was deemed to be progressing well with plan to taper off the oral prednisolone. Patient was seen in the Emergency Department the next day after his outpatient clinic visit with chief complaint of syncopal episode followed by seizure and weakness.

Patient was post-ictal following the episode of seizure. In the Emergency Department, patient was found to have a heavy speech. Stroke code was activated and patient had an urgent Computed Tomography (CT) of the brain which showed left parieto-occipital grey-white matter hypodensities. Patient subsequently had further episodes of tonic colonic seizures while in the Emergency Department for which he was intubated. Patient was initially given lorazepam 2 mg intravenous followed by Keppra 1,000 mg intravenously. Magnetic Resonance Imaging (MRI) was done which showed multiple small acute established non-haemorrhagic infarction in the left frontal and temporal regions. Patient was started on aspirin 300 mg orally for acute ischaemic stroke and later continued at 80 mg daily. Thrombolysis was not considered as patient had a NIHSS score of 0 and was having seizures which were contraindications for rTPA therapy. Patient was shifted to the intensive Care Unit (ICU) and started on Enoxaparin therapeutic dose. Patient was extubated two days after his ICU admission.Patient was thought to have had a complex partial seizure with secondary generalization secondary to hyperacute stroke. Aetiology of stroke was thought to be most likely due to prothrombotic state.Patient also had transesophageal echocardiography which was normal. Figure 1 and Figure 2 show the hypodensity areas on the MRI scan.





BACKGROUND

Acute ischaemic stroke (AIS) are not usually found in patients who have low platelet counts or a prothrombotic. Acute haemorrhagic stroke (AHS) are usually associated with patients having low platelets or who are prothrombotic as a result of haemorrhagic complications. Very low platelet counts are only occasionally noted to have resulted in acute ischemic strokes and immune thrombocytopenia is considered as a rare cause of ischemic stroke[1,2]. Idiopathic thrombocytopenic purpura (also known as immune thrombocytopenia or ITP) is manifested by decrease in platelet count which is usually below 100 x 10^9/L. Primary ITP is not associated with other conditions whereas secondary ITP can be associated with infections and autoimmune disaease[3]. Low levels of platelets are generally as a result of immune mediated platelet destruction along with suppression of platelet production[4]. The mechanism resulting in acute ischaemic stroke related to immune thrombocytopenia has not been fully understood. It has been proposed that ITP-induced platelet microparticles can contribute to stroke[5]. Majority of strokes are caused by ischemia which accounts to almost 80% of cases rather than haemorrhage which accounts for 20% of cases[6]. Cardioembolism, large vessel atherothromboembolism and small vessel occlusive disease are generally the cause of ischaemic strokes[7]. Essential thrombocytopenia has been suggested to be a risk factor for stroke mainly of ischemic small-vessel type[8]. Idiopathic thrombocytopenic purpura in the past has been known to have caused infarction of the middle cerebral artery[9].Ischemic stroke is considered as a complication of Immune thrombocytopenia[10]. There has only been a few case reports suggesting ischemic stroke in patients with Immune thrombocytopenia[11]. Initial treatment in acute stroke involves thrombolytic therapy and early initiation of supportive care[12]. In cases where thrombolytic therapy is contraindicated, treatment with antiplatelet therapy has been advocated. However, use of antiplatelet therapy and anticoagulants in patients with stroke and ITP need to be adjusted depending on each patient's risk factors[13]. One of the contraindications to the thrombolytic therapy is low platelet counts of less than 100 x 10^9/L[14]. Avoidance of thrombolytic therapy in context of low platelet count is due to the high risk of bleeding in particular intracranial haemorrhage. Elevated plasma levels of procoagulant factor VIII (FVIII) have commonly been found in patients with acute ischaemic stroke (AIS)[15,16]. Higher mortality has been reported in patients with acute ischemic stroke who had thrombocytopenia and thrombocytosis on initial admission[17].

DISCUSSION

On initial presentation of the patient to the hospital, petechial rashes in the lower extremities and lesions on the tongue and mucous membrane were attributed to immune thrombocytopenia. Patient was appropriately started on immunoglobulin and steroid therapy. A week later, when the patient was seen in the outpatient clinic by haematology team, the platelets count were improving on steroid therapy but patient ended up having multiple non-haemorrhagic infarctions the next day. It is postulated that the infarctions developed while patients had low platelet levels and only manifested clinically when patient started having seizures and returned to the hospital.

CONCLUSION

Immune thrombocytopenia has been related to ischaemic strokes but on rare occasions. Even when the patient is on steroid therapy for ITP, risk of ischaemic stroke cannot be underestimated.

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