Chronic subdural hematoma associated with idiopathic thrombocytopenic purpura in an elderly female: A rare case report

Sachin Chhabra, Ashok Gupta

Department of Neurosurgery, SMS Medical College, Jaipur, Rajasthan, INDIA

Abstract: Intracranial hemorrhage is the most fatal complication of idiopathic thrombocytopenic purpura(ITP) and is very rare(<1%) mostly in young females and intraparenchymal or subarachnoid in most of the cases. We report a case of 63 years of female presented with acute SDH with petechiae in both lower limbs and headache. After ruling out all causes patient was diagnosed as having idiopathic thrombocytopenic purpura(ITP).Considering her haematological and neurological profile patient was kept conservatively. Platelets and steroids were given to the patient following which platelet counts improved but patient deteriorated clinically. Hence patient was operated and surgical evacuation of SDH was done through burr hole. Although some studies have shown disappearance of chronic SDH with medical management, but it failed in our case and so surgical evacuation was done.

Key words: subdural hematoma (SDH), idiopathic thrombocytopenic purpura (ITP)

Introduction

Intracranial hemorrhage is the most fatal complication of idiopathic thrombocytopenic purpura (ITP) and is very rare (<1%). In most of the cases it is either intraparenchymal or in subarachnoid space or at multiple sites (1, 2). Only a few cases of isolated subdural haematoma (SDH) with ITP have been reported in literature (1-7). ITP associated chronic SDH more often occurs in females and younger age was considered as a significant risk factor in its occurrence (7). In this report we describe the rare occurrence of ITP associated SDH in an elderly patient and its management.

Case report

A female patient, 63 years of age working as housewife, presented in outpatient department with chief complaints of generalized headache since 1 day associated with nausea with history of loss of consciousness 1 day back. She was not having any chronic illness and was not taking any medications. She did not give any history of trauma. On Examination, she was afebrile and was not having any neck stiffness. Patient had no neurological deficit. Patient pulse rate was 64/minute and blood pressure was 138/80 mm Hg. She was having multiple small painless petechiae present on both upper limbs. On

admission, patient was having Hb 11.5, TLC 8,000, platelet count 70,000. Patient random blood sugar and other biochemical investigations were in normal limit. On next day patient platelet count came down to 14000 which further reduced to 9000 on subsequent day. Patient prothrombin time (PT) was in normal limits (13.9 sec with INR 1.07). NCCT Head at the time of admission revealed hyperdensity in right frontotemporoparietal region with compressed ventricle on right side with midline shift towards left suggestive of subdural hematoma (Figure 1).

In view of patient condition and platelet count, patient was kept conservatively and platelets were transfused. Patient was further investigated for low platelet count and peripheral blood film of patient showed marked thrombocytopenia with few large platelets. RBC were normocytic normochromic and normal in count. No immature cells or atypical cells or parasite seen. Bone marrow aspiration cytology revealed marked thrombocytopenia, mild rouleaux formation with adequate megakaryocytes on aspirate smears and normal myeloid and erythroid series suggestive of megakaryocytic thrombocytopenia. Patient coagulation profile, total leucocyte count and differential leucocyte counts were in normal limits. Viral infection was ruled out. Dengue IgG and IgM antibodies were negative. Anti nuclear antibodies were negative. Hepatic viral markers and liver function tests were normal eliminating the hepatic cause. Considering patient history and clinical examination and ruling out all possible case of low platelet count, a diagnosis of ITP was made. As patient was having low platelet count, initially conservative treatment was done by transfusing 11 units of random donor platelets and steroids (prednisolone) were started under strict glucose monitoring and in tapering dose. After 9 days of conservative treatment patient platelet count improved but general condition deteriorated. Patient became drowsy and her GCS dropped from 15 to 12.Repeat CT scan showed right frontotemporoparietal chronic SDH with mass effect and midline shift towards left (Figure 2). Patient Hb was 11.2 and platelet count came out to be 1.37 lacs. Right frontal and parietal burr hole was made and drainage of haematoma done on urgent basis. Postoperatively patient was conscious and was obeying commands and was relieved of headache. She was discharged on fourth post operative day.

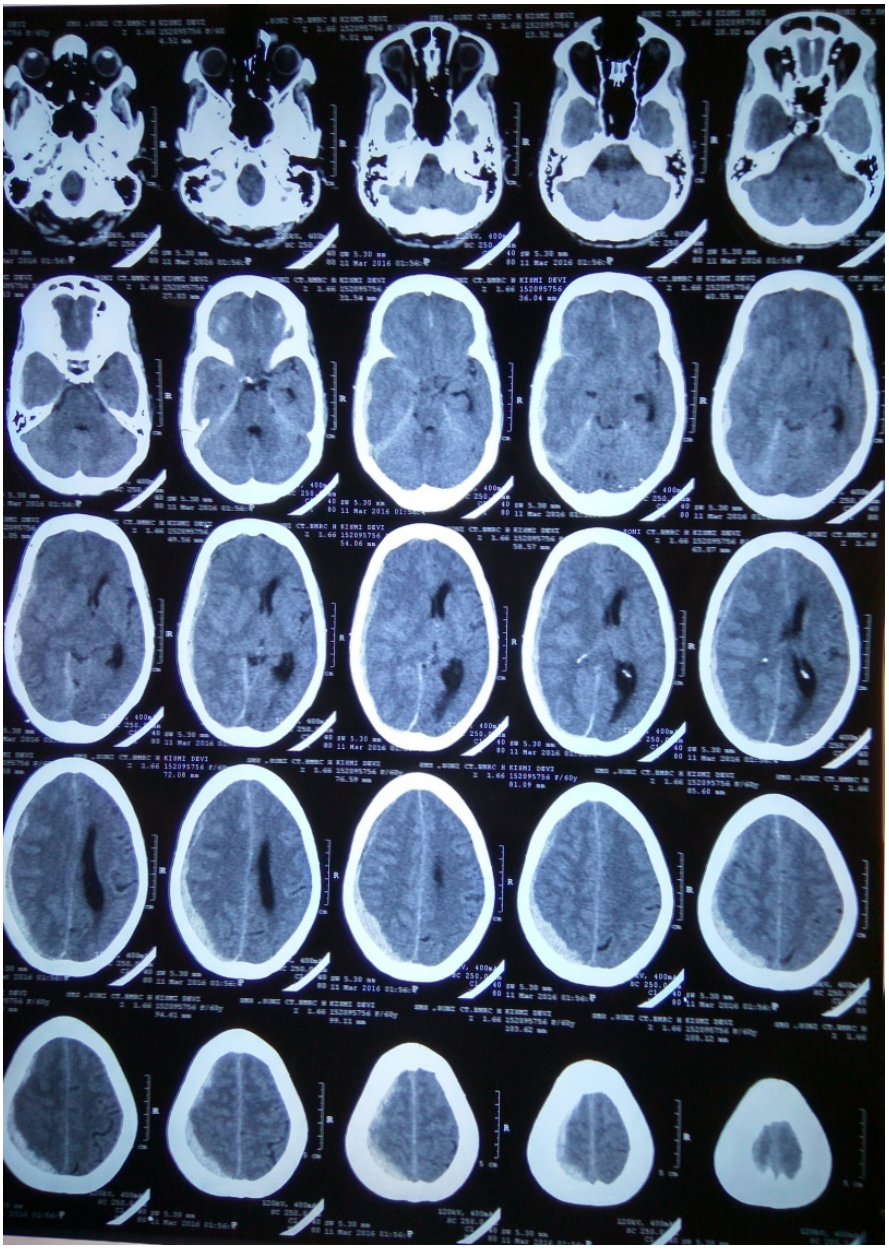

Figure 1 - Showing acute Frontotemporoparietal SDH at the time of admission

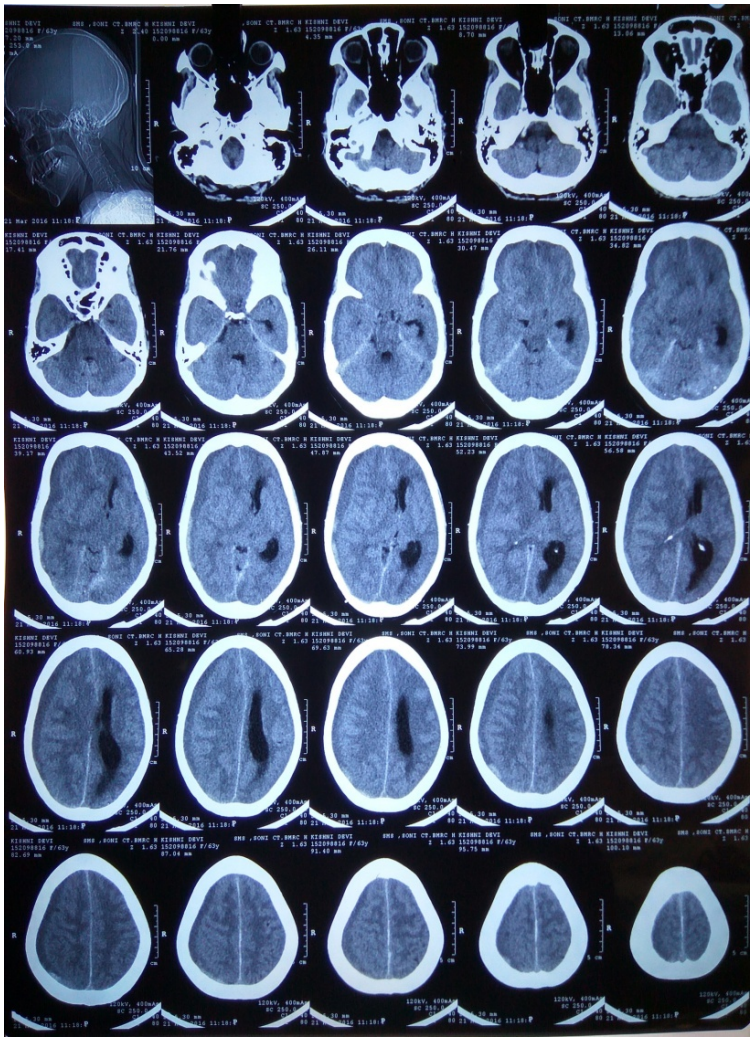

Figure 2 - Showing repeat scan of the patient having chronic SDH with mass effect

Discussion

ITP is a disorder characterized by antibody mediated destruction of platelets resulting in isolated low platelet count. Although it may cause bleeding but intracranial hemorrhage is very rare and most serious complication of ITP. Subdural hematoma is extremely rare in these patients of ITP developing intracranial hemorrhage. Chronic SDH is usually due to trauma but Seckin H et al in their review found that it may occur spontaneously in cases of. SDH associated with ITP (3). ITP most commonly occurs in females and H Takase et al showed that younger age was a significant risk factor for ITP associated SDH(7).In our case it occurred in 63 year old female with no past history of bleeding and trauma.

As it is a rare condition, no definite guidelines are given regarding management and management remains controversial. Previously surgery with evacuation of hematoma used to be the primary treatment. But in 1997, Gupta et al used medical management to treat an asymptomatic case successfully (4). Lee and Kim in their meta analysis of seven patients found that five patients improved non surgically (6). Later on Sreedharan et all in 1999 (2) and H Seckin et all 2006 (3) treated such patients conservatively. Conservative treatment involves intravenous administration of steroids or immunoglobulins or a combination of these two along with platelet transfusion. However any deterioration in patient clinical status demands an urgent repeat neuroimaging to decide about surgical evacuation. Considering our patient's neurological and haematological profile, patient was given medical management. Various studies have shown spontaneous resolution of SDH with transfusion of platelets and steroids. But in our case patient's platelet count improved but worsened neurologically after 9 days of conservative treatment. Therefore, surgical drainage of chronic SDH through burr hole was done. Thus, by our case report we emphasize that in such patients although a trial of conservative treatment can be given but it has to be weighed against patient haematological and neurological status.

Correspondence
Dr. Sachin Chhabra
Resident, Department of Neurosurgery,
SMS Medical College, Jaipur
Rajasthan (INDIA)
Mobile: 7877377683
E-mail: drsachinchhabra@gmail.com

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