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Acute Ischemic Stroke in a young patient with Immune Thrombocytopenic Purpura: A Case Report

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Abstract: Immune thrombocytopenic purpura occurs when platelets undergo premature destruction as a result of autoantibody or immune complex deposition on their membranes. It is commonly associated with bleeding complications but thrombotic events are rare. We describe a 25 year old female patient who presented with immune thrombocytopenic purpura associated with acute ischemic stroke due to thrombosis in the right middle cerebral artery. Our case report highlights that ischemic stroke can be seen in immune thrombocytopenic purpura cases and probable mechanism underlying it.
Keywords: Immune thrombocytopenic purpura, acute ischemic stroke.

INTRODUCTION

Immune thrombocytopenic purpura (ITP) is caused by platelet specific autoantibodies that bind to autologous platelets, which are then rapidly cleared from the circulation by the mononuclear phagocyte system via Fc-gamma receptors in the spleen and liver. ITP has two types acute and chronic [1].

ITP mainly leads to thrombocytopenia and bleeding manifestations like menorrhagia, purpura, epistaxis, gingival bleeding and intracranial hemorrhage. Although rare but ITP may also lead to thrombotic complications like ischemic stroke [2, 3]. We describe a 25 year old patient with ITP who developed acute ischemic stroke.

CASE REPORT

A 25 year old female patient presented to the emergency department with complaints of weakness of the left side of the body since 6 hours. Her past medical history revealed ITP since 3 years. She also complained of menorrhagia since one year. She was treated with corticosteroids and blood transfusion initially, but treatment was stopped by the patient as she became asymptomatic and she did not follow up regularly. The informed consent was taken from the patient husband.

On physical examination blood pressure was 120/80 mmHg, pulse was 88 beats/min and pallor was present. Her pelvic examination was normal. Her neurological examination showed weakness of left side of the body and aphasia without any cranial nerve involvement.

Laboratory findings were as follows; hemoglobin 2.7 gm%, white blood cell count 10170 cells/cmm, hematocrit 10.8%, platelets 8000/mm^3, serum creatinine 0.6 mg/dl, blood urea 15 mg/dl, sodium 135 mmol/l, potassium 4.7 mmol/l and liver function test was within normal limits. Prothrobin time, partial thromboplastin time and international normalized ratio were normal. Peripheral smear showed microcytic hypochromic anemia with thrombocytopenia. Electrocardiogram was in normal sinus rhythm and echocardiography was normal. She had a negative direct and indirect coombs test. She was negative for human immunodeficiency virus, hepatitis B and hepatitis C virus. Her abdomen ultrasound was also normal. She was negative for antinuclear antibody, anti-ds DNA, anticardiolipn antibody, antiphospholipid antibody and lupus anticoagulant. Her mri brain showed acute ischemic infarct in right middle cerebral artery. These findings confirmed that patient was having ITP. We started treatment with iron, folic acid and cyanocobalamin preparations, blood transfusions and corticosteroids which included injectable methylprednisolone 1gm/day for 3 days followed by 1mg/day prednisolone orally in divided doses. After 3 whole blood transfusions and corticosteroids treatment for one week patient hemoglobin increased to 9.1gm% and platelet count increased to 90,000/mm^3. Then we initiated treatment with anticoagulants and antiplatelets

with watch on her platelet counts every 48 hours. Platelet count remained in range between 80,000 to 1, 10,000/mm^3 during subsequent week. Patient condition improved and she was discharged.

DISCUSSION

ITP is a diagnosis of exclusion. Although ITP is associated with intracranial hemorrhage but in some cases it may also present with ischemic stroke. Thrombosis is rare in ITP and only some cases have been reported [4, 5].

Thrombotic complications in ITP may occur if the patient is not taking treatment as in our case. The mechanism of thrombosis in ITP is complex. Anti-platelet antibodies, which are present in most cases of ITP, cause complement mediated fragmentation of platelets and the release of platelet microparticles (PMP). Although PMP protect against bleeding in cases of ITP, they may also promote intra vascular thrombus formation by causing platelets activation [6, 7]. The risk of thrombotic complications is also increased by the endothelial damage induced by auto-antibodies directed against antigens present on both platelets and endothelial cells [8].

Treatment of ITP in acute ischemic stroke is a matter of concern. We in our case started treatment with anticoagulants and antiplatelets as platelet counts reached above 50,000/mm^3. During the treatment patient platelet counts remained in range between 80,000 to 1, 10,000/mm^3. As patient general condition improved we discharged the patient on prednisolone 5 mg/day with clopidogrel 75 mg/day. After 15 days of discharge patient blood counts were repeated and it showed hemoglobin of 10.2gm% and platelet count of 94,000/mm^3. She was advised to continue with same treatment as she was advised during discharge.

CONCLUSION

Although rare but ITP can lead to ischemic stroke mainly in untreated cases. The role of fibrinolytics, anticoagulants and antiplatelets is controversial and it should be individualized on the basis of risk factors and platelet counts.

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