ORIGINAL RESEARCH PAPER
Volume-6 | Issue-11 | November-2017 | ISSN No 2277 - 8179 | IF : 4.176 | IC Value : 78.46
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

IMMUNE THROMBOCYTOPENIC PURPURA INDUCED INTRACRANIAL HEMORRHAGE IN A PATIENT WITH HEPATOPULMONARY SYNDROME.

Medicine

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ABSTRACT

Immune thrombocytopenic purpura (ITP) is an autoantibody-mediated thrombocytopenic disorder in which accelerated destruction of platelets occurs; platelet production may also be impaired by these antibodies. ITP is characterized by mucocutaneous bleeding. Rarely, more severe haemorrhages, such as intracranial haemorrhage, may occur. A female patient, she had the history of ITP and CLD produce intracranial haemorrhage and which was diagnosed from radiological examination. Treatment given to the patients includes cognitive enhancers, hemopoietic agents and hepatic protestants.

KEYWORDS

Mucocutaneous, Haemorrhage, Purpura, Thrombocytopenia

INTRODUCTION

Immune thrombocytopenic purpura (ITP) is a clinical syndrome in which a decreased number of circulating platelets (thrombocytopenia) manifests as a bleeding tendency, easy bruising (purpura), or extravasations of blood from capillaries into skin and mucous membranes (petechiae). Although most cases of acute ITP, particularly in children, are mild and self-limited, intracranial haemorrhage may occur when the platelet count drops below 10 × 109/L (<10 × 10^3/μL); [1] this occurs in 0.5-1% of children, and half of these cases are fatal. [1]

Intracranial haemorrhage (ICH) is the most devastating complication of immune thrombocytopenic purpura (ITP) in children, and prevention of ICH is the primary goal of ITP treatment. However, the great majority of patients with ITP, even those with very low platelet counts, do not experience severe bleeding, and ICH occurs in less than 1 in 100 children with ITP. [2] Existing case series are small, and reviews of published case reports may suffer from publication bias. The features that predispose patients to develop ICH in addition to severe thrombocytopenia remain poorly defined. Potential risk factors include platelet counts below 10 to 20 × 109/L, nonsteroidal anti-inflammatory drugs (NSAIDs), head trauma, vasculitis associated with systemic lupus erythematosis (SLE), and cerebral arteriovenous malformations (AVMs).[3]

CASE PRESENTATION

The 52 year old lady was admitted with complains of cough mucoid expectoration since 2 days, abdominal distension, loose stools, increased o2 requirement since 2 days. While asking to the patient it was found that she had k/c/o DCLD with portal hypertension with hepatomegaly, hepatopulmonary syndrome and hypertension. And she is on home treatment with O2 by nasal spray and her SpO2 was around 82% with oxygen. And she was admitted 2 months back because of urosepsis and she was on inj.etrapenem due to the presence of ESBL in her urine. And also have recurrent episodes of intracranial haemorrhage of due to idiopathic thrombocytopenic purpura and was on treatment with t.eltrombopag 50mg od. And she was not getting any antiplatelet drugs.

On the day of admission patient was conscious, oriented and have the compliance of fatigue along with seizures. And her clinical parameters which were in normal range. But on the next day patient produce fever spikes, 100.9F, headache and altered sensorium. But she had no history of dysurea and cold.

Haematology of the patient shows elevated ESR, neutrophilia (92%) and lymphopenia (4%). LFT shows elevated levels of serum bilirubin (4.2g/l), globulin (4.4g/l)and ammonia (1.1mcg/ml). But the serum albumin(2.8g/l) and calcium(7.6%) were low. However, RFT parameters were normal except urea, which is 59mg/dl. Clotting parameters was also checked to rule out the reason ICH and it only shows a mild spike raise in prothrombin time while the INR was in normal range of 1.86. Inhibitor study was done to detect the presence of an inhibitor, but it did not show its presence. Factor VII assay, which was also normal. Patient DIC profile was normal except the raise in D-Dimer level. Along with that platelet count was also abnormal of 7,000cell/μl on the day of admission and was on transfusion on first 2 days and later increased to 25,000. For proper diagnosis, we done a peripheral blood smear and it shows moderate thrombocytopenia with giant platelets and clumps. Vasculitis package was also done to confirm the diagnosis shows the presence of Antinuclear Antibody.

Radiologic studies of the brain shows the presence of clot on the left cerebral hemisphere followed by multiple calcifications on both cerebral hemispheres specifically in both capsule- ganglionic regions. And cortical based gliosis on left frontal region. Prominence of sulcal and cisternal spaces due to the age related cerebral atrophic changes. While the study of lower abdomen shows the presence of chronic hepatic parenchymal disease followed by peripancreatic and perispleenic collaterals along with acities and splenomegaly.

The patient was treated with t.eltrombopag 50mg and 25mg on alternative days, t.levitriacetam 500mg bd for seizures,t .urodeoxycholic acid 300mg bd, inj.citicoline 500mg bd, inj.Filgrastim 300mcg once weekly and supportive treatments. Because of very low platelet count, she was on platelet transfusion for two days.

DISSCUSSION

Idiopathic Thrombocytopenic Purpura (ITP) is defined as a hematologic disorder, characterized by isolated thrombocytopenia without a clinically apparent cause. The major causes of accelerated platelet consumption include immune thrombocytopenia, decreased bone marrow production and increases splenic sequestration. [4]

The incidence of ITP is 1.6/ 10,000 per year in the United States. Some reports show it is more common in females but others shown no difference in gender distribution. The bleeding in ITP is mucocutaneous, manifesting as petechiae, purpura, easy bruising, epistaxis, gingival bleeding, and menorrhagia. [3]

The treatment of ITP depends on disease presentation. If the patient is experiencing profound bleeding with very low platelet counts (10,000 to 20,000/UL), initial treatment with IV-Immunoglobulin (IV-Ig) or combined with IV methylprednisolone. It may be supplemented with either IV anti-D or with high dose dexamethasone until the platelet count exceeds 50,000/UL. Transfusion of platelet is warranted if life threatening haemorrhage occurs. The standard of practice for corticosteroids non-responders and platelet counts below 30,000/UL after 4 - 6 weeks of therapy is splenectomy, but it may be delayed for up to three years, particularly patients with insidious onset ITP.[2]

Patients who have chronic ITP, defined as persisting for more than 6 months, was treated with monoclonal antibodies such as Rituximab may also be used. It is postulated that mechanisms of macrophage blockade by opsonized B cells may account for the early responses. Immunosuppressant such as Danazole has been shown to improve platelet count in significant number of refractory ITP patients. It is believed to restore suppressor T-cell function and decrease antibodies

production by decreasing the number of available Fc receptors. Cyclosporine, Mycophenolate Mofetil and chemotherapeu-tic agents such as azathioprine, vinca-alkaloid, cyclophosphamide and interferon have been used with similar success.[4]

CONCLUSION

Intracranial haemorrhage is the most devastating complication of Immune Thrombocytopenic purpura. The female patient with age 52years have the episodes of recurrent intracranial haemorrhage produced due to ITP which was confirmed by Magnetic Resonance Imaging. The patient already had history of chronic liver disease and hepatopulmonary syndrome induced due to vasodilatation and angiogenesis in pulmonary vascular bed due to the release of endothelin 1 and nitric oxide from the injured liver. Haemorrhage that occurs due to low platelet count and was aggravated by CLD because most of the clotting factors are synthesised by liver that produce a raised prothrombin time, even though the INR was in normal range. After the treatment with cognitive enhances and hemopoietic growth factors, the patient was recovered but could not able to attain complete improvement. Even when the patient continues the medicines for a long time, the chances of reversal are high.

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