University Journal of Medicine and Medical Specialities

ISSN 2455- 2852 2017, Vol. 3(5)

A rare combination of Idiopathic thrombocytopenic purpura (ITP) with Guillian barre syndrome(GBS) - A case report

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Abstract : Idiopathic thrombocytopenic purpura (ITP) is a disorder caused by the interaction of IgG and other antibodies with the antigen on platelet surface. ITP associated with Guillain-Barre syndrome (GBS) has been reported rarely to occur in the same patient simultaneously. We present the case report of a young male patient who presented with acute ITP and Guillain-Barre syndrome at the same time. This case is interesting because IgG might play a prominent role in the development of both ITP and Guillain-Barre syndrome.
Keyword : Guillian-Barre syndrome, Immune thrombocytopenic purpura,auto-immunity,demyelination

A 22 years old unmarried male working as an iron rod fixer in building construction was referred to RGGGH chennai from GMKMCH,Salem for further evaluation and management of bleeding diathesis and weakness. Patient was apparently normal 2 months back, and then he developed minor recurrent episodes of bleeding gums and epistaxis over a period of 1 month. He initially ignored his symptoms. After that he started having pain in the right calf, thigh & back which was progressive over the next two weeks. Then he noticed weakness of right lower limb in the form of difficulty in walking which was progressive, ascending and flabby. He went to GMKMCH Salem, where initial basic investigations and x-ray LS spine were normal. Gradually he developed left lower limb weakness also and became immobile 4 days later.He went against medical advice and took native treatment (oil-massage and bandage) for 5 days. During that period he developed weakness of both upper limbs with decreased sensations of all modalities below the hip. He got readmitted in GMKMCH Salem, where his baseline investigations including platelet count (1.6 lakhs) were normal. During the next 3 days, he developed bleeding gums, epistaxis, hematemesis, hematochezia& malena. The platelet count decreased to 10,000 per cubic millimeter and Hb was 7.3 g/dl.Patient was treated with blood & platelet transfusions, anti-ibrinolytics, nasal packing and then referred to RGGGH, chennai.

On Examination he had severe pallor. There was sub conjuctival hemorrhage and gum bleeding.He also had purpuric spots and ecchymotic patches in his limbs. His vitals were stable.CNS examination showed that his higher mental functions were normal. He had bilateral LMN type of facial palsy with labial dysarthria

other cranial nerves were clinically normal. He had muscle wasting in all his 4 limbs symmetrically. There were no fasciculations.Tone, power and reflexes were decreased in all four limbs. Plantar was bilaterally flexor. All modalities of sensation were decreased in both lower limbs upto knees and in both hands.Cerebellum examination andexamination of autonomic nervous system were normal.Fundus was normal.Other systems were normal.

ECCHYMOTIC PATCHES IN THE PATIENT

Investigations

CBC showed a total count of 17000 with a differential count - P67, L19 E-14.HB was 8.1gms,ESR-20/46 mm. The platelet count was 3000 cells per cubic millimeter.Peripheral smear showed microcytic hypochromic RBCs, platelets were decreased in number but normal in morphology, WBCs were normal in number, distribution and morphology. Reticulocyte count was 2.5%.Direct coombs test was negative. PT,APTT,INR were within normal limits. Platelet count after transfusion became 1.5lakhs per cubic millimeter RFT ,LFT ,ECG, chest xray were within normal limits.USG abdomen was normal. HIV ELISA,ANA,HBsAG,IgM ANTIHCv were negative Bone marrow aspiration was Normocellular, megakaryocytes were adequate. Erythropoiesis and myelopoiesis were normal with few atypical cells.

MRI spine was normal

CSF ANALYSIS showed Sugar- 55mgs%,protein-700mgs/dl,cell count- acellular

NERVE CONDUCTION STUDY revealed acute motor sensory axonal neuropathy

The above history and investigations satisfied the criteria for both ITP and GBS (7) individually.. Hence we made a diagnosis of ITP with GBS. Patient was treated with inj.vitamin K, fresh frozen plasma, platelet and packed red cell transfusions.

Inj. Methyl prednisolone was given in a dose of 500 mg/day for 5 days with which patient's platelet count showed good improvement.

DISCUSSION

This case is a rarity because of association of ITP and GBS. So far only 5 cases have been reported all over the world. ITP is an isolated decrease in the platelet count caused by immune-mediated destruction of platelets and inhibition of their production. Similarly, GBS is characterized by inammatory demyelinating neurological process that clinically manifests as progressive muscle weakness and areexia. In the pathogenesis of GBS, both humoral factors and cell-mediated immune phenomena damage myelin and the myelin-producing Schwann cells [1,2] It is likely that both cellular and humoral immune mechanisms contribute to tissue damage in GBS. T cell activation is suggested by the finding that elevated levels of cytokines and cytokine receptors are present in serum and in cerebrospinal fluid (CSF).It was initially thought that GBS was likely to be primarily a T cell–mediated disorder; However, abundant data now suggest that autoantibodies directed against nonprotein determinants may be central to many cases. Majority of antiplatelet antibodies in patients with ITP are directed against GPIIb/IIIa (~80%), and the remainder against the GPIb/IX complex and other platelet glycoprotein such as GPIV and GPIa/IIa.The mechanism involves presentation of platelet antigens by activated antigen-presenting cells, which thereby activate both CD4+ T-cell clones and antigen-specific T-cell clones. These T-cell clones, having different antigen specificities, induce different B-cell clones to produce antibodies against distinct platelet antigens(8).Intravenous immunoglobulinsplay important role in treatment of both ITP and GBS. Auto immune diseases have a common mechanism of disrupted self-tolerance.That is why a person with one auto-immune disease becomes susceptible to another. Following the line GBS has been reported in association with SLE and also with ITP,either alone or in combination with other diseases like Hashimoto's thyroiditis.(2,4,5,6)

The simultaneous development of two autoimmune disorders in a patient points to a possible common genetic factor among these two diseases. Further investigations into similar cases are needed to clarify this association [1–6].

The patient is now under regular follow up and his weakness has improved dramatically. He has no bleeding manifestations. He is planning to resume his work shortly.

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