

MANAGEMENT OF A REFRACTORY CASE OF IMMUNE THROMBOCYTOPENIC PURPURA.

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Introduction. The immune thrombocytopenic purpura (ITP) is defined as a secondary pathological condition of a peripheral thrombocyte hyperdestruction occurring through an immunologic mechanism exceeding the ability of normal compensatory thrombocyte formation and in a clinical translation through a purpura syndrome and at hematology level, through thrombocytopenia with megakariocytosis.

Clinic case. We submit here the case of a patient of 52 years old with the diagnosis of ITP identified in January 2012 and having Morb Pott antecedents since 1993 ; she underwent surgery and treated with tuberculostatics. At the time of the diagnosis determination, the patient showed epistaxis , petechiae ecchymosis at the level of the lower and upper limbs, headache, acute thrombocytopenia and mild microcytic hypochrome anemia. The patient underwent investigations to exclude a secondary thrombocytopenia. There have been done several investigations detecting an infection with *Helicobacter Pylori* and positive HBs antigen and for these reasons she followed a medical treatment. Initially, she followed pulse therapy with Solu-Medrol, but no therapeutic response was obtained. A new line of corticosteroid therapy -Dexamethasone was tested, but no response either. As the corticosteroid therapy provided no response, there was started the treatment with Revolade obtaining a favorable response, on short term. The patient temporarily responded at the increase of the Revolade dosage associated to Dexamethasone. In July 2012, she came back because of the disease relapse and she was administrated immunoglobulins, followed by splenectomy with good response, but, 3 weeks since the surgery the severe thrombocytopenia was present again. Between September 2012 and July 2013 she was periodically hospitalized for substitutive treatment, showing severe persistent thrombocytopenia. The treatment with Vinblastin was tested but no response was obtained. In October 2013 there occurred a hemorrhagiparous syndrome predominantly at the lower limb level accompanied by hemorrhagic bullae at the level of the oral mucous membrane and the treatment with Mabthera was started, but no therapeutic response was obtained. Between January and November 2014 she was periodically hospitalized for substitutive treatment, and in December 2014 the treatment with Romiplostim with weekly dosages started and no adverse effects were noticed. After one month of treatment the dosage increase was required, and the level of thrombocytes was maintained within the normal range.

Conclusions. The evolution is characterized by a refractory feature, the lack of response at the corticosteroid therapy, rapid relapses after therapy and splenectomy and requiring multiple therapeutic lines, but with favorable sustained response to treatment with Romiplostim.