Clinical picture

Rituximab-induced toxicity

A 80-year-old woman with relapsing, idiopathic thrombocytopenic purpura was admitted because of diffuse cutaneous and mucosal petechiae and ecchimoses; no sign of visceral bleeding was noted. A marrow aspirate confirmed the absence of morphologic abnormalities in the blood cell precursor lineages. Blood counts revealed Hb 14.5 mg/dl and platelets 1000 x 10^9/l. She was treated with two courses of rituximab, 375 mg/mq/week. Purpura disappeared soon, platelet counts rose to 70 600 x 10^9/l, and she was then discharged. However, after 1 week, she was admitted again because of general malaise, diarrhea and vesicular rash of the face (Figure 1) consistent with herpes simplex virus (HSV) infection. We also noted a high C-reactive protein level and a very low CD4 count (11%; normal values, 38–46%; absolute count 99 x 10^9/l); IgM anti-HSV Type 1 antibodies were detected; IgG and IgM anti-HSV Type 2 were absent; and serum immunoglobulins were within normal ranges. The next morning left hemiparesis was recorded; subsequent magnetic resonance imaging of the brain was consistent with ischemic stroke. The patient developed progressive clinical deterioration with coma and deep vein thrombosis and finally died.

Monoclonal antibodies have dramatically improved the prognosis of many patients with lymphoma. Rituximab, a chimeric antibody directed against the CD20 molecule on lymphocytes membrane, is known to provoke profound and persistent B lymphocyte depletion, to an extent that has an impaction on the T-cell subsets too.1 Although these effects stand at the basis of its therapeutic effect in a wide range of autoimmune disorders,2 cases and small series of grave and sometimes fatal consequences of immunosuppression are increasingly reported.

Figure 1. Rapidly progressive, widespread, crusted papules, vesicles and erosions around the mouth.

Conflict of interest: None declared.

References