Daclizumab: A Novel Therapeutic Option in Severe Bullous Pemphigoid

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Sir,

We describe the use of a novel immunosuppressive regimen with a specific humanized monoclonal antibody daclizumab, against CD25 (α-chain of the IL-2 receptor) for severe bullous pemphigoid (BP).

CASE REPORT

A 52-year-old woman with a body weight of 102 kg presented with widespread multiple blisters and bullae on the whole body before hospitalization. Clinical features and conventional histopathology confirmed the diagnosis of BP; immunohistology and immunoserological tests were performed and were compatible with a diagnosis of BP. The patient was subsequently treated with prednisolone 100 mg/day in combination with azathioprine 100 mg/day, cyclosporin A 200 mg and mycophenolate mofetil 2 g/day. However, disease control was not achieved and the blister formation progressed. During steroid therapy the patient gained 35 kg of body weight and developed diabetes mellitus with glucose levels around 180 mg/dl. Therefore, the dosage of prednisolone was reduced slowly to 5 mg/day, azathioprine was maintained and a bolus intravenous infusion of 100 mg (approximately 1 mg/kg body weight) daclizumab (Zenapax®) was given fortnightly. Azathioprine treatment with 50 mg/day was continued and steroids were subsequently reduced. In total, the patient received six infusions during hospitalization and as a patient in the day-care centre. The therapy with 5 mg prednisolone, daclizumab and azathioprine was well tolerated and no side effects were observed during or after treatment. Pathological glucose levels normalized and the patient slowly lost body weight. An improvement of the condition of the skin was observed within 2 weeks of treatment with daclizumab. The skin blisters completely resolved and a post-inflammatory hyperpigmentation remained as a residual effect. Clinical resolution persisted for approximately 3 months, before new blisters occurred despite the continued treatment with azathioprine and prednisolone. A second series of three daclizumab infusions (100 mg/day) was administered every 3–4 weeks. The patient responded rapidly and has remained free of skin lesions and blisters for more than 10 months so far.

DISCUSSION

Monoclonal antibodies against CD25 (daclizumab or basiliximab) have been developed as immunosuppressive drugs for patients after transplantion of kidney, pancreas islets, liver and heart (1–3). In addition, case reports about successful therapy with these novel agents have been published for the treatment of psoriasis vulgaris, erosive lichen planus and epidermolysis bullosa acquisita (4–8).

Anti-CD25 antibodies were also successfully used in combination with anti-CD20 antibodies in the treatment of BP in a patient with chronic graft-versus-host disease. In this case, anti-CD25 was required for the observed response, because it interrupted the T-helper cell fraction facilitating the secretion of antibodies against BPAG2 (9). As daclizumab also prevents T-cell activation in vivo (3), this agent was chosen to treat the severe form of BP in our patient.

Despite failure of conventional combination therapies, a dramatic response to daclizumab in combination with prednisolone and azathioprine was observed. Even under forced reduction of the steroid due to drug-induced diabetes and weight gain, the treatment with daclizumab had no side effects in our patient with severe BP.

The therapeutic application of a new monoclonal antibody such as daclizumab offers alternative treatment for severe or unresponsive BP and might help in the future to avoid and overcome severe side effects caused by high doses of steroids.

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