AUTOIMMUNE BULLOUS DISEASES

REFRACTORY BULLOUS PEMPHIGOID IMPROVED BY DISCONTINUATION OF PHENYTOIN AS AN CYP3A4 INDUCER

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Background: Systemic corticosteroids are main treatment of bullous pemphigoid (BP). CYP3A4 hepatic enzyme, has an important role in steroid catabolism and metabolism of foreign compounds, with the majority of pharmaceutical compounds being substrates for CYP3A4. Several antiepileptic drugs e.g. phenytoin and carbamazepine, are known to induce CYP3A4 strongly and attenuate effect of steroids.
Here, we experienced a case of refractory bullous pemphigoid improved by discontinuation of phenytoin.

Observation: A 72-year-old man presented with a 7-month history of pruritus and generalized annular edematous erythema with blister. He had a past history of infantile cerebral palsy and epilepsy, treated by phenytoin and primidone. Based on the physical and histopathologic examination findings, BP was diagnosed and treatment with 50mg/day prednisolone (PSL) commenced. Since his skin eruptions worsened in spite of initial treatment, he was referred to our hospital. Although corticosteroid pulse therapy (500mg/day for 3 day) , 150mg daily mizoribine, intravenous immunoglobulin therapy dose of 400mg/kg/day for 5days and double filtration plasmapheresis (2 times/week for 3weeks) were administered, erythema and tense bullae was still spread. As relationship of BP and antiepileptic drugs had been reported, especially phenytoin, CYP3A4 inducer. After we changed phenytoin to levetiracetam, his skin symptoms improved rapidly. His skin symptoms had kept good after gradually decreases of PSL.

Key message: In refractory BP treated with corticosteroid, we need attention to other medicine which associated with metabolism of CYP3A4.