Letters to the Editor

Dihydroergotamine raises the level of serum growth hormone

To the editor: A patient with a high serum growth hormone (GH) level which seemed to be induced by dihydroergotamine (DHE) is described. DHE acts as a partial alpha-adrenergic agonist as well as a central alpha-adrenergic antagonist (1). It has been widely used to treat migraine and orthostatic hypotension. A central alpha-adrenergic agonist is known as a factor of increasing GH secretion. However, it has not been reported that DHE raises the level of GH.

Report of a case - A 30-year-old male was referred to our hospital because of orthostatic hypotension. The DHE had been administered for about 3 yr. Because he was tall on examination, we measured serum GH level, which showed high serum level (15.0 ng/ml; N< 5.0 ng/ml). However, brain computed tomographic scan could not detect the pituitary tumor or other brain disease. In addition, the level of GH decreased from 17 ng/ml to 0.3 ng/ml after administration of 75 g glucose, which is known as one of the screening tests for acromegaly (2). The serum GH level returned to normal level within 2 days after administration of DHE was discontinued. Therefore, we suspected that DHE raised the serum GH level. To confirm this GH raising effect, we made a DHE loading test. The serum was taken before and after taking 1 mg of DHE. The GH level increased like this: 0' 0.9 ng/ml, 30' 0.6 ng/ml, 60' 1.9 ng/ml, 90' 17.0 ng/ml, 120' 14.0 ng/ml, 150' 11 ng/ml, 180' 3.5 ng/ml. Serum levels of glucose and fatty acids were not changed. From these results, it is clear that DHE affected the serum GH level in our patient.

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Bucillamine may induce Myasthenia Gravis

To the editor: D-penicillamine (D-PC), one of the sulfhydryl compounds, is known to induce myasthenia gravis (MG) in patients with rheumatoid arthritis (RA) (1). Other sulfhydryl compounds, such as pyritinol (2), captopril (3) and tiopronin (4), have also been reported as possible candidates which induce MG in patients with RA. We report here a patient with RA who developed MG during treatment with a new sulfhydryl compound, bucillamine (Rimatil® ). This drug has been used as a disease modifying anti-rheumatic drug in Japan since 1987. This is the first report concerning the possible association of this drug and MG.

The patient was a 36-year-old female with an 8-yr history of RA. From January 21, 1988, she had received D-PC (100 mg/day), and her rheumatic symptoms improved. However, because of the side effect of decreased serum immunoglobulin A, D-PC was discontinued on April 4, 1988. From May 12, 1988, she was given 100 mg/day of bucillamine, and her rheumatic symptoms improved; she received this medication every 2 days from September 1, 1988. In July 1989, she started to feel weakness of extremities. After about 2 wk, she had diplopia, blepharoptosis and weakness of upper limbs which worsened in the evening. She decided to stop taking bucillamine on August 2, 1989 and was admitted to our hospital three days later. She was diagnosed to have MG, based on the clinical symptoms, a 15% decrement in response to repetitive nerve stimulation on the hypothenar muscle, positive tensile tests, and high levels of serum anti-acetylcholine receptor (Ach R) antibodies (89 nmol/l; N < 0.5 nmol/l). Hyperplasia of the thymus was detected on magnetic resonance imaging. Her myasthenic symptoms were improved slightly after discontinuation of bucillamine but she required pyridostigmine bromide (120 mg/day) treatment. Since October 5, 1989, she has undergone plasmapheresis using an IM-T350 immunoabsorbent column 6 times. She showed complete disappearance of myasthenic symptoms after plasmapheresis, and did not need anticholinergic drugs. However she showed myasthenic symptoms again after discharge. So she was treated again by plasmapheresis, steroid pulse therapy and an immunosuppressive agent (Azathioprine). Her clinical symptoms improved but her symptoms and high level of anti-AchR antibody were still present.

It can be suggested that the MG in this patient was associated with the bucillamine treatment, because of the clinical course; the onset of MG occurred during the treatment with bucillamine, and slight, transient improvement of symptoms was seen after discontinuation of the drug.

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