

RELAPSE OF MYASTHENIA GRAVIS AFTER AMOXYCILLIN THERAPY

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The ampicillin group of antibiotics has among been considered to be amongst the safest for use in patient with Myasthenia gravis, although a report in 1971 suggested that such might not be the case¹. A recent report suggested that ampicillin may aggravate Myasthenia gravis in patients and experimental animals². We report a case of clinically controlled Myasthenia gravis, in whom the administration of amoxicillin had a deleterious effect.

CASE REPORT

A 29 year old male developed diplopia and ptosis in March 1985, followed by nasal speech and limb weakness one month later. All symptoms were worse in the evenings, and were aggravated on exertion. The diagnosis of Myasthenia gravis was confirmed by a positive edrophonium test. Repetitive stimulation of the right ulnar nerve at 3 Hz produced a decrement of the right abductor digiti minimi action potential by more than 10%. Anti-acetylcholine receptor antibody tests were negative. A CT scan of the thorax was normal. All other tests were within normal limits. Treatment was started with Pyridostigmine 60 mg orally, which was increased upto 9 tablets daily for clinical control. He underwent thymectomy in June 1985. Cephalexin 1 gm was administered I.V. 6 hourly for 5 days during the post-operative period because of a mild chest infection. He made an uneventful recovery. Histology of the thymus gland showed hyperplasia. He was discharged on Pyridostigmine 60 mg 5 times daily. On subsequent follow ups, he was symptom free and had resumed all his activities, including his favourite sport of water skiing. In February 1986, he developed a productive cough, diagnosed as bronchitis, which passed off in a few days with symptomatic treatment. The patient was otherwise completely all right, except that he did not feel 'in top form'. In April 1986, he had a similar attack of bronchitis. This time he was treated with Amoxicillin 250 mg orally three times daily, in addition to the previously prescribed symptomatic treatment. Two days later he noticed easy fatigability. On the third day he developed nasal speech, diplopia, weakness in the lower limbs, and breathlessness on exertion. An edrophonium test was then positive. Amoxicillin was continued for 7 days. When an increase in Pyridostigmine did not relieve his symptoms, he was readmitted to the hospital. On examination, he exhibited the complex clinical picture of Myasthenia gravis. Stimulation of the right ulnar nerve at 3 Hz disclosed a peak decrement of 43% of the right abductor digiti minimi action potential. Antiacetylcholine receptor antibody test was again negative. When he did not respond to a large dose of Pyridostigmine (120 mg 3 hourly), a course of gamma globulin infusions (400 mg/kg body weight per infusion) was started. His clinical status gradually improved and he became completely symptom free within one month. At this stage stimulation of the right ulnar nerve at 3 Hz showed a maximum decrement of 30% of the compound muscle action potential of the right abductor digiti minimi. Since then, he has been able to resume all his normal activities, including water skiing. He continues to have an infusion of gamma globulin once a month, and is being maintained on Pyridostigmine 60 mg 5 tablets daily.

DISCUSSION

Myasthenia gravis is known to be aggravated by certain antibiotics, e.g. the aminoglycoside group, tetracyclines etc.³⁻⁵ So far penicillins have been considered safe in these patients. Ampicillin and amoxicillin are broad spectrum penicillins, with almost identical structures and properties.⁶ Recently, Argov et al² have shown that the administration of ampicillin aggravated Myasthenia gravis in two patients. They also demonstrated that ampicillin increased the preexisting electrical decrement in rabbits with experimental autoimmune myasthenia gravis. Circumstantial evidence points to amoxicillin being responsible in the present instance. It was not considered justifiable on ethical grounds to subject this patient to a challenge of amoxicillin. The evidence incriminating amoxicillin was that he deteriorated within 48 hours of taking it, whereas he did not deteriorate during the first attack of bronchitis untreated by antibiotics. Moreover, cephalexin was administered during and after thymectomy without having any adverse effects. The mechanism by which the ampicillins act on the neuromuscular junction is not known. More clinical and experimental evidence is needed to determine the effects of the ampicillins on the neuromuscular junction. Until then, we recommend that they be used with extreme caution in myasthenia gravis.

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