CASE REPORT

MYASTHENIA GRAVIS WITH SUPERIMPOSED SPINAL CORD INJURY: A CASE REPORT

Che-Sheng Lin, MD, Juei-Hsiang Wang, MD, Yen-Ho Wang, MD and Shin-Liang Pan, MD, PhD

From the Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital, National Taiwan University, Taipei, Taiwan

Both myasthenia gravis and traumatic spinal cord injury are uncommon disorders and their concurrence is extremely rare. We report here the case of a man with stable myasthenia gravis with spinal cord injury due to a motor vehicle accident. His muscle strength and sensory function in all 4 limbs partially recovered during the initial hospitalization. However, after a later episode of pneumonia and urinary tract infection, muscle strength deteriorated and weakness of the ocular muscles occurred. A relapse of myasthenia gravis was confirmed by the elevated anti-acetylcholine receptor antibody titre. Muscle strength recovered rapidly after control of infection and treatment of myasthenia gravis exacerbation. In this case report, we show that spinal cord injury-related complications, including infection or emotional stress, could trigger a relapse of myasthenia gravis. This highlights the importance of recognizing an exacerbation of myasthenia gravis in spinal cord injury patients with a history of myasthenia gravis, since they are prone to urinary and pulmonary infection. Although exacerbation of myasthenia gravis might prolong hospitalization, the functional outcome of spinal cord injury might not be affected if the exacerbation is diagnosed and treated quickly.

Key words: spinal cord injuries, myasthenia gravis, rehabilitation.


Correspondence address: Shin-Liang Pan, Department of Physical Medicine and Rehabilitation, National Taiwan University Hospital, No. 7 Chung-Shan South Road, Taipei 100, Taiwan. E-mail: panslcb@gmail.com

Submitted December 19, 2007; accepted March 26, 2008

INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder of neuromuscular transmission in which autoantibodies are formed against nicotinic acetylcholine receptors at the neuromuscular junction (NMJ). The clinical symptoms are progressive weakness of the ocular, bulbar, limb, and respiratory muscles following repeated contractions, and recovery of muscle strength after a period of rest (1–2). MG is a relatively uncommon disorder, with a reported annual incidence of 50–125 cases per million people (1, 3). The most common age of onset of MG is the second to third decades in women and the sixth to eighth decades in men. Traumatic spinal cord injury (SCI), which leads to sensorimotor deficits below the level of injury, is also an uncommon event, with an estimated annual incidence of 19 per million people in Taiwan (4) and 30–60 per million people in most industrialized countries (5–6). It is therefore very rare for a patient with MG to have superimposed SCI. We report here the case of a man with MG who suffered from traumatic SCI due to a motor vehicle accident.

CASE REPORT

A 45-year-old man with a 2 year history of MG presented with fluctuating ptosis and diplopia, but no weakness of the facial, neck, or limb muscles. The symptoms were worse after a period of daily activity, especially in the afternoon, and were relieved by rest. The diagnosis of MG was confirmed by a series of studies, including the edrophonium test, repetitive nerve stimulation test, single fibre electromyography, and serum antibodies test. He underwent thoracoscopic thymectomy and received oral pyridostigmine therapy. His symptoms of ocular motor dysfunction then subsided completely and no further neurological symptoms recurred on pyridostigmine treatment.

In May 2006, he suffered weakness of all 4 limbs immediately after a motor vehicle accident. He was sent to the emergency department, where neurological examination revealed markedly reduced muscle strength in the 4 limbs (MMT grade 0–1/5), absence of sensation below the C4 dermatome, absence of tendon reflexes, urine retention, and loose anal tone. These findings suggested a spinal cord lesion. Magnetic resonance imaging (MRI) of the cervical spine showed posterior bulging of the intervertebral disc at C3–4, C4–5 and C5–6, with compression of the dura and cervical cord. T2 weighted images showed a high signal intensity lesion at C3 and C4 in the spinal cord. No spine fracture or brain injury was noted. Based on the diagnosis of traumatic cervical cord injury with tetraplegia, he was immediately treated with high-dose parenteral methylprednisolone. He then underwent laminectomy plus plate fixation for cord decompression and stabilizing the spine. His neurological function improved gradually after the above treatment. Muscle strength in all 4 limbs improved to fair (MMT grade 3/5) and sensory function partially recovered after an 8-week inpatient rehabilitation programme. At discharge after the first hospitalization, he was able to walk with the aid of a walker.
Discussed in the text.