Miller Fisher syndrome in an elderly man

SIR—A 76-year-old man was admitted with sudden onset of ataxia and diplopia. He had had coryzal symptoms 48 h previously. On examination, he had dysconjugate eye movements, dysphonic speech and an ataxic gait. His supinator, biceps and ankle jerks were absent bilaterally. Over the next 3 days, he developed bilateral partial ptosis, complete ophthalmoplegia, proximal right arm weakness and severe ataxia. A clinical diagnosis of Miller Fisher syndrome was made.

Cerebrospinal fluid examination was normal. Magnetic resonance imaging did not show any significant abnormalities. Nerve conduction studies were highly suggestive of a demyelinating peripheral neuropathy affecting all four limbs. Anti-GQ1b antibodies were present in his serum.

He was treated with a course of intravenous immunoglobulin. He gradually improved and was mobile independently on discharge 3 weeks after admission. By 6 weeks post-discharge his eye movements were almost complete and he no longer had ptosis or any significant balance problems.

Miller Fisher syndrome is one of a spectrum of acute demyelinating inflammatory polyneuropathies which include Guillain–Barré syndrome, acute ophthalmoplegia and Bickerstaff’s encephalitis. It is characterized by ataxia, areflexia and ophthalmoplegia [1]. Although the onset of symptoms is usually acute, it can take 5–10 days for the full picture to become apparent. Between 60 and 80% of patients have an antecedent upper respiratory tract infection [2], Miller Fisher syndrome usually follows a benign course [3] and most patients make a good recovery within 10 weeks.

Miller Fisher syndrome can occur at any age, but most commonly presents in the fourth and fifth decades. In a review of reported cases of Miller Fisher syndrome [4], the median age of onset was 48 years. In a recent study [2], which included 110 patients with Miller Fisher syndrome, the median age of onset was 41 years.

Acute-phase IgG antibodies to GQ1b and GT1a gangliosides are strongly associated with Miller Fisher syndrome [5, 6] and may play a role in the pathogenesis of the condition [7]. Cerebral imaging is used primarily to rule out other conditions within the differential diagnosis. The protein concentration in cerebrospinal fluid is often raised. Evidence of demyelination may be found on electromyography.

Treatment is mainly supportive. Both intravenous immunoglobulin and plasmapheresis show benefit in
Guillain–Barré syndrome, and have also been used in Miller Fisher syndrome. Plasmapheresis is unsuitable for older patients, who have impaired cardiac function.

This man developed the characteristic features of Miller Fisher syndrome. His case is unusual in that this syndrome is uncommon in people over 70 years.

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