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Multifocal motor neuropathy with abrupt onset and spontaneous recovery

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Sirs: Multifocal motor neuropathy (MMN) is characterized by asymmetric weakness without sensory disturbances, with muscle wasting and fasciculations. It has a slowly, stepwise progressive course. Electrophysiological studies show partial or persistent motor conduction blocks [1].

Although unilateral wrist drop or grip weakness can be a presenting symptom in MMN, an acute onset and almost full recovery within a few days has not yet been described.

A 34 year old man presented with abrupt, painless onset of weakness of his right hand. On examination he only had distal weakness of the right arm: strength in finger and wrist extensors 3/5, abductor pollicis brevis and opponens pollicis 4/5, and intrinsic muscle function 4/5

(MRC). Sensory examination was normal. The deep tendon reflexes were brisk and symmetric, the plantar reflex normal on both sides. Ancillary investigations including MRI imaging of the brain were unremarkable. Nerve conduction studies of the right arm were normal. There was an almost full recovery within 5 days, except for a slight impairment of right-sided rapid finger movements. During three years of follow-up his minor signs remained stable. Then he noticed a gradually progressive weakness of the right arm and left leg. He complained of cramps in the abdominal muscles and all four limbs. Examination revealed fasciculations in the both abductor pollicis brevis and distal weakness of his right arm comparable with the examination three years previously. Additionally there was weakness of the left foot flexors and extensors(4/5). Sensory testing was normal. Deep tendon reflexes had become hypoactive at the right arm and absent at both ankles. Extensive nerve conduction studies showed a bilateral partial motor conduction block of the median nerve in the lower arm, and a complete motor conduction block in the left peroneal nerve. Motor and sensory nerve conduction velocities, distal latencies, and F-waves were within normal values. IgM anti-GM1 antibodies were present (1 : 100). CSF analysis showed 3 white cells and total protein was 0.49 g/L. The diagnosis of MMN was made and the patient was treated with 2 g/kg intravenous immunoglobulin (IVIg) in two days. He showed a clear improvement after IVIg starting within four days after onset of the infusion. Within two weeks there was an almost complete recovery with only slight weakness of finger extension at the right and normal strength of left

foot. There was a relapse after three weeks, which again responded to IVIg. He presently still needs 80 g of IVIg every 4 weeks to avoid full relapses between treatments.

MMN is a chronic demyelinating neuropathy. It is characterized by slowly progressive asymmetrical weakness of the limbs without sensory involvement [2]. The diagnosis of MMN is based on clinical, laboratory and electrophysiological characteristics [2]. The distinguishing electrophysiological characteristics are the presence of multifocal motor conduction blocks outside the usual sites of nerve compression [3–4]. These results of electrophysiological testing and the normal CSF protein are not compatible with the diagnosis chronic inflammatory demyelinating polyneuropathy (CIPD).

This patient's history illustrates that MMN can present with abrupt onset of weakness of one limb and rapid, spontaneous and almost complete recovery. Although the asymmetry is often remarkable in MMN, both limbs are usually involved. The patient had multifocal motor weakness, however, without a clear distribution of a multiple mononeuropathy, the more frequent distribution pattern in MMN. Slow spontaneous remissions in MMN have been reported [5], but a spontaneous recovery within a few days has not yet been described.

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References

1. Pestronk A, Chaudhry V, Feldman EL, et al. (1990) Lower motor neuron syndromes patterns of weakness, nerve conduction abnormalities, and high titers of antiglycolipid-antibodies. *Ann Neurol* 27:316–326
2. Nobile-Orazio E (2001) Multifocal motor neuropathy. *J Neuroimmunol* 115:4–18

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3. Van Asseldonk JTH, Van den Berg LH, Van den Berg-Vos RM, et al. (2003) Demyelination and axonal loss in multifocal motor neuropathy: distribution and relation to weakness. *Brain* 126:186–198
 4. Van Asseldonk JTH, Franssen H, Van den Berg-Vos RM, et al. (2005) Multifocal motor neuropathy. *Lancet Neurol* 4:309–319
 5. Chad DA, Hammer K, Sargent J (1986) Slow resolution of multifocal weakness and fasciculation: a reversible motor neuron syndrome. *Neurology* 36:1260–1263