LETTER TO THE EDITOR

Flaccid Paralysis Due to Chronic Baclofen Overdosage

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Oral baclofen taken at recommended doses (up to 80 mg per day in many national formularies) usually has only a mild clinical effect on spasticity reduction. On account of potential adverse events, baclofen is usually considered as a second-line therapy in stroke patients. It may, nonetheless, be used as a first-line therapy in multiple sclerosis (MS) and spinal cord injury-related spasticity (Yelnik et al., 2009). On the other hand, when intrathecally delivered, baclofen is an effective treatment for spasticity, especially in spinal injury patients and in MS. A single injection at a dose as low as 50 µg leads to complete flaccidity.

We report the case of a 58-year old man with left lower limb spastic paresis related to a parasagittal meningioma surgically treated 12 years ago. It had remained a distal paresis without sensory impairment. No active movement of the foot was possible, with spastic dystonia-induced varus and toe clawing during walking. For walking, an ankle-foot orthosis was required. The patient benefited from local treatment by Botulinum injections about twice a year. In July 2012, in order to reduce his alcohol consumption, a psychiatrist proposed baclofen treatment, slowly increasing from 10 to 200 mg per day. When we were consulted on 10th October, after 1 month of daily absorption of 200 mg, the patient was confused, complained of memory disorders and was virtually paralyzed, unable to stand up alone or to walk. There was a dramatic decrease of left lower limb motor control and also right impairment. Moreover, deep tendon reflexes were absent in the lower limbs and hard to find in the upper limbs. Urinary incontinence was occasionally observed. The patient was admitted to our department.

Brain imagery (magnetic resonance imaging (MRI)) was normal except for the known remaining parasagittal hypodensity. Spinal and caudal imagery (MRI) revealed a lumbar canal narrowed by arthrosis and discal projection. Lower limb electromyography revealed axonal neuropathy, probably alcohol-related. Evoked sensory cortical potentials did not show any abnormalities of the central pathways. Baclofen was reduced by 10 mg every 2 days. Six weeks after admission, all reflexes were present, with the left exaggerated; motor control had been regained as spastic dystonia of the lower limb; walking was possible anew; urinary control was normal and neuropsychological tests revealed no memory disorders except some elements of dysexecutive syndrome.

Since imagery and electrophysiological exams failed to explain the disorders and because complete recovery occurred as the baclofen was reduced, it would appear highly probable that the high previous doses of baclofen had caused the temporary transformation of spastic paresis into flaccid paralysis.

It is unusual to observe flaccidity with oral baclofen, except during acute intoxication with extremely high doses (450–1250 mg) after suicidal or recreational use (Ghose et al., 1980; Haubenstock et al., 1983; Perry et al., 1998). The association in our patient of relatively high doses with a mild alcohol-related neuropathy may have occasioned this particularly pronounced effect with major functional consequences. Since use of high doses of baclofen is being practised by some in the treatment of alcohol addiction (Rigal et al., 2012), it is important that unwanted effects are be brought to the attention of all concerned parties.

REFERENCES