LETTER TO THE EDITOR

NEUROPATHY ASSOCIATED WITH LANSOPRAZOLE TREATMENT

Proton-pump inhibitors are extensively used worldwide. Rare cases linking omeprazole to the occurrence of neuropathy or myopathy have been reported. Lansoprazole is not known to cause neuropathies. The present case should therefore be of interest.

A 42-year-old woman was referred to our unit by her general practitioner with a 12-month history of burning paresthesias and dysesthesias rising from the soles of the feet to the knees. The symptoms had appeared progressively about 3 months after she had started lansoprazole for heartburn and dyspepsia from hiatus hernia and gastro-esophageal reflux. She described a transient improvement in her sensory symptoms on withdrawal from the drug for a few weeks, but restarted the medication because of her gastric problems. She was a nonsmoker, drank alcohol moderately, used no other drugs, and had otherwise an unremarkable medical history. There was no relevant family history. On examination, tone and power were normal in all limbs. There was no muscle wasting, tenderness, or fasciculations. Light-touch and pin-prick appreciation was impaired distally in a stocking-and-glove distribution. Vibration sense was also diminished distally but only in the lower limbs. Joint position sense was preserved and Romberg's sign was absent. Deep tendon reflexes were all present but diminished. Plantar responses were flexor, and the remainder of the neurological examination was entirely normal. There was no indication of autonomic dysfunction. All routine blood investigations (blood count; plasma electrolytes; renal and liver function tests; thyroid function tests; serum vitamin B_{12}, folate, glucose, creatine kinase, and serum protein electrophoresis) were normal. Auto-antibody screen and antineuronal (Hu) antibodies were negative. Cerebrospinal fluid (CSF) was acellular with a protein concentration of 0.47 g/L (normal range 0.20–0.40) and normal glucose level. Nerve conduction studies showed an axonal, sensory polyneuropathy. The right sural sensory nerve action potential was absent, the left reduced (4 μV; normal 5–20). Compound muscle action potentials were all within the normal range. Motor and sensory conduction velocities, distal latencies, and F-wave latencies were all within normal limits. Needle electromyography (EMG) showed no abnormal spontaneous activity. A nerve biopsy was not performed. At this stage her sensory axonal neuropathy was suspected to be related to the use of lansoprazole.

Lansoprazole was stopped. Significant but only partial improvement of the patient's sensory symptoms occurred within days. In view of the recurrence of her gastric symptoms, she started taking another proton-pump inhibitor, rabeprazole. This did not exacerbate her neuropathic sensory disturbance, but in view of its poor effect on her gastric symptoms, was replaced within weeks by ranitidine. Gabapentin was prescribed in an attempt to ease her paresthesias and dysesthesias, and helped in a dose of 300 mg taken three times daily.

Two years later, the patient had persistent distal sensory loss without weakness but deep tendon reflexes had normalized. Electrophysiological studies showed marked improvement of the sural sensory nerve action potentials, which were now both present with amplitudes of 18 μV on the right and 9.5 μV on the left. Motor studies and needle EMG findings were unchanged.

To our knowledge, only two previous reports have linked a proton-pump inhibitor, namely omeprazole, with a neuropathy. The case reported by Faucheux et al.[1] was the only one with an electrophysiologically documented sensorimotor axonopathy, no electrophysiological data having been provided by Sellapah, who described only the clinical features of a sensory neuropathy that reversed on treatment withdrawal.[3] Myopathic involvement with omeprazole has also been described.[1,2]

In our patient, occurrence of the neuropathic symptoms shortly after initiation of lansoprazole, and clinical and electrophysiological improvement after withdrawal, are both suggestive of causation. The absence of deterioration on rabeprazole does not favor a drug-class side effect, although the short duration of treatment on this other proton-pump inhibitor makes a definite conclusion impossible. Although probably of rare occurrence, awareness that lansoprazole may cause neuropathy is important, particularly in patients requiring treatment and having a preexisting neuropathic disorder. Also, in view of the increasing use of proton-pump inhibitors for gastric protection in neurological patients on long-term steroid therapy, caution is justified with lansopra-

zole, especially in those whose primary complaint is an inflammatory neuropathy (or myopathy).

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