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Nitrofurantoin-induced peripheral neuropathy: A lesson to be re-learnt

Sir,

The widespread use of nitrofurantoin since its availability in 1952 in the treatment of urinary tract infection was soon followed by reports of neurotoxicity attributable to it. Neuropathy has been well documented with short-term use but rarely developed with chronic drug consumption.^[1,2] Clinical association between nitrofurantoin-induced neuropathy and tethered cord syndrome has not been reported. Progressive neuro and urological dysfunction localized to the conus medullaris may also suggest tethered cord. We present a case of peripheral neuropathy induced by nitrofurantoin; confirmed clinically and electrophysiologically with incidental coexistence of tethered cord.

A 16-year-old girl was admitted to our hospital because of weakness and paresthesia. She was well

since two years ago when nitrofurantoin (200 mg/dl) was prescribed for recurrent urinary tract infection elsewhere. Eight months before admission she felt paresthesia in both lower limbs and then it extended to the upper extremities. Five months later she couldn't stand and walk easily without help. Family history was negative.

On neurological examination severe muscular atrophy along with limb deformity and contracture were noted [Figure 1]. Deep tendon reflexes were decreased and trophic changes were present. The EMG/NCV evaluation of both upper and lower limbs revealed severe decrease in amplitude, increased latency and slow conduction velocities compatible with severe sensorimotor, mainly axonal peripheral neuropathy. Sensory nerve action potentials were not detected. The EMG study revealed acute denervation findings in the distal part of the lower limbs and chronic regenerative changes in the proximal portions. Paraspinalis muscles were normal. The MR imaging showed abnormally low settled spinal cord [Figure 2] compatible with tethered cord. Neurogenic bladder due to tethered cord caused urinary retention, infection and obstructive uropathy. Routine workup for diabetes, vasculitis, celiac disease, metabolic and nutritional disorders was negative. Concerning the low serum creatinine, nitrofurantoin was discontinued, three months later paresthesia decreased and motor function recovered partially.

Nitrofurantoin neurotoxicity is associated with a symmetric sensorimotor predominantly axonal polyneuropathy.^[3] The neuropathologic findings include Wallarian degeneration, segmental demyelination resulting in axonal loss, usually distal portions, which is responsible for protein synthesis, secondary to nutritional deficiency or various toxins.^[4] Those proteins are vital to the growth and development of the axon and myelin sheath. The antibacterial activity is probably due to interfering with bacterial carbohydrate metabolism inducing acetyl-coenzyme-A inhibition which is the same mechanism responsible for nitrofurantoin neurotoxicity. Another hypothesis states underlying comorbid diseases which may contribute to subclinical nerve injury aggravated by nitrofurantoin. It may also cause neuropathy by acting as a folic acid antagonist.^[1] About 14% of adverse drug reactions (ADRs) are neurological, the vast majority of them are dose-related. So they can be prevented and/or treated by adjusting the administered dose.^[5] Grushka et al.^[6] reported a woman with "strong pains" who used nitrofurantoin. Within three months of drug withdrawal, pain decreased and her muscle strength recovered. It seems that clinical judgment is the mainstay of ADR diagnosis. It was also shown that the ADR probability scale has consensual



Figure 1: Severe muscular atrophy of the upper (A) and lower limbs (B)



Figure 2: Lumbosacral MRI showed lipoma (short arrows; A) and low conus medullaris (long arrow; B)

and concurrent validity.^[7] Up to now, no drug is available to prevent or cure drug-induced neuropathies.

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