Bilateral Vocal Cord Paralysis Following Treatment With Vincristine

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V incristine is a vinca alkaloid with an established role in the treatment of acute lymphoblastic leukaemia (ALL)(1). Vincristine-associated peripheral neuropathy is a well-described entity. In a previous POG 9201 trial, 3.6% patients had significant toxicity(2). It manifests as loss of deep tendon reflexes, neuritic pain, paresthesias, and wrist and foot drop. Less frequently, transient cortical blindness, oculomotor nerve dysfunction, jaw pain, facial palsy, sensorineural hearing loss, and laryngeal nerve paresis have been attributed to vincristine(3). We report a child who developed bilateral vocal cord palsy during induction treatment of ALL.

CASE REPORT

A 14 year-old male with B-cell acute lymphoblastic leukemia (ALL) was being treated with BFM-87 protocol. Ten days after receiving the 4th dose of vincristine (1.5mg/m²), he developed stridor and hoarseness. There were no previous clinical symptoms of neuropathy and no positive history for inherited neuropathies. He was aspirating fluids, which led to pneumonia. A flexible fiber optic endoscope showed both vocal cords to be in abducted position with loss of movement of both vocal cords. A contrast enhanced CT of soft tissue neck was normal. There was loss of deep tendon reflexes. The nerve conduction velocity studies showed motor predominant axonal neuropathy involving the upper and lower extremity. Electromyography of larynx was not done. His subsequent doses of vincristine were stopped. Stridor improved after ten days and hoarseness of voice resolved 35 days after the onset of palsy. Subsequent laryngoscopy and flexible fiber optic endoscope showed normal movement of both vocal cords. The child is currently in reinduction phase, is off vincristine and is doing fine.

DISCUSSION

Vincristine related vocal cord paralysis has been reported infrequently in the literature(4-8). Vincristine neurotoxicity is more severe when more than the recommended dose is given, if the patient is hypersensitive to this drug, if there is pre-existing liver dysfunction or a hereditary neuropathy, and if other drugs such as allopurinol, erythromycin,
isoniazid, mitomycin C, phenytoin, and itraconazole are concomitantly used(3,9-10). Our patient was receiving fluconazole at the time of development of vocal cord palsy. Azoles are known to increase the neurotoxicity of vincristine(9). The pathogenesis of neuropathy is explained by structural changes in the microtubules of peripheral nerves and interference with axoplasmic transport(3). Visualization of the airway confirms the diagnosis and rules out treatable causes of stridor in the febrile, immunocompromised patient(5).

Involvement of vocal cords has been unilateral or bilateral. Vocal cord of left side is predominantly involved. Paralysis appeared in most cases during induction phase only implying that even small doses of vincristine are capable of causing the nerve damage. Few children with generalized neurotoxicity from vincristine including hypotonia, decreased gastrointestinal motility, and painful paresthesias have been described while laryngeal nerve paralysis was the only neurotoxic manifestation in the other patients(4-7). No age is immune. It has been described in infants also(6). All cases resolved spontaneously upon withdrawal of the vincristine. Reintroduction of vincristine led to reappearance of hoarseness in one patient(7). Vinca-alkaloid-induced vocal cord paralysis is a potentially dangerous but reversible lesion. Most of the authors documented that complete recovery of vocal cord paralysis required 6-9 months (4,6,9).

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REFERENCES