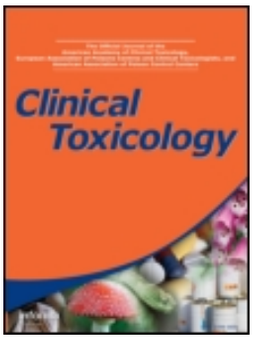

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CASE REPORT

Isolated bilateral vocal cord paralysis with intermediate syndrome after organophosphate poisoning

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Introduction. Muscular weakness affecting predominantly the proximal limb muscles and neck flexors is the cardinal feature of intermediate syndrome with cranial nerve palsies occasionally accompanied. Following acute cholinergic phase of organophosphate poisoning (OPP), only a few isolated cases of vocal cord paralysis have been reported in the past. We describe a case of bilateral vocal cord paralysis which occurred in the wake of a clinical recovery from acute cholinergic crisis in OPP. *Case report.* A 32-year-old woman presented with severe cholinergic crisis after ingestion of an unknown amount of dichlorvos in a suicide attempt. The patient was improved from cholinergic crisis by administration of antidotes. On day 4, she complained of progressive dyspnea and dysphonia after removal of the endotracheal tube. Needle electromyography for neuromuscular confirmation was normal. However, laryngeal electromyography (LEMG) findings were consistent with bilateral laryngeal paralysis suggesting the vagus nerve involvement. Her vocal cord movements were restored to near normal with time and she was discharged on the 20th day after admission. *Conclusions*. Physicians should account for the neurotoxic effects of organophosphate poisoning during the first line management of exposed patients. Isolated bilateral vocal cord paralysis (BVCP) should be excluded as a cause, if dysphonia or respiratory distress occurs after extubation in patients with intermediate syndrome. LEMG in such cases can be an important diagnostic adjunct.

Keywords Intermediate syndrome; Organophosphate; Vocal cord paralysis

Introduction

Neurotoxic effects of organophosphate compounds include three well-defined neurological syndromes known as acute cholinergic crisis, intermediate syndrome, and delayed organophosphate induced polyneuropathy. Of the three neurological syndromes, intermediate syndrome has a feature of muscular weakness affecting predominantly the proximal limb muscles and neck flexors, while cranial nerve palsies are not uncommon. Following acute cholinergic phase of organophosphate poisoning (OPP), only a few isolated cases of vocal cord paralysis have been reported in the past (1,2). We describe a case of bilateral vocal cord paralysis (BVCP) occurred in the wake of a clinical recovery from acute cholinergic crisis. The cause of the paralysis was postulated to be due to the neurotoxicity, a sequela to the persistent cholinesterase inhibition manifested in the intermediate syndrome. With this case, we would like to discuss the importance of ruling in or out a case of OPP in dealing with patients suffering from intermediate syndrome with dysphonia, dyspnea, or respiratory distress.

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Case report

A dichlorvos-poisoned woman was admitted with severe cholinergic crisis and comatose mental status. After gastric decontamination under the endotracheal intubation, she was treated with high doses of atropine (8 mg/ h IV until adequate atropinization was established) and pralidoxime (initially 2 g IV, subsequently doses were repeated every 12 hr for 2 days). The breathing was controlled by assisted mechanical ventilation system. The initial plasma cholinesterase level was 129 U/L (Normal range from 4,100 to 9,900 U/L). Approximately 72 hours after the administration of antidotes, most of cholinergic signs disappeared but the patient was left on mechanical ventilation support due to the signs of mild aspiration pneumonia with weak self-respiratory efforts. On day 4, she only showed mild weakness in spontaneous respiratory efforts, but she could sustain strong handgrip on command, shoulder abduction, and head lift for 5 seconds or more in assessment for muscle strength prior to extubation trial. The deep tendon reflexes were normal. The endotracheal tube was carefully removed, and the atropine infusion was

discontinued. The patient complained of progressive dyspnea and dysphonia but without requiring re-intubation. The plasma cholinesterase level was still suppressed to 1,550 U/L. On day 5, needle electromyography was performed, and sensory and motor conduction velocities of median, ulnar, peroneal, and tibial nerves were normal in conduction study. LEMG in bilateral thyroarytenoid muscles innervated by recurrent laryngeal nerves demonstrated widespread fibrillation and positive sharp waves at rest. Also, cricothyroid muscles innervated by superior laryngeal nerves revealed significant reduction in voluntary motor unit action potential during attempts at muscle contraction. This finding was consistent with bilateral laryngeal paralysis by the motor involvement of the vagus nerve. An otolaryngologist was consulted and a videolaryngoscopic examination showed that both vocal cords remained stationary in the paramedian to intermediate position (glottic gap; about 3 mm), signifying BVCP due to bilateral laryngeal nerve palsy than isolated lesions of the recurrent laryngeal nerve. There was no evidence of foreign body, inflammation, or infection. The paralysis lasted for 15 days since admission and then gradually resolved with time. On day 17, the patient`s vocal cord movements were restored to near normal state on videolaryngoscopy and she was discharged on the 20th day after admission. There was no further development of neurological sequelae was observed during the one month follow-up period.

Discussion

OPP induced intermediate syndrome usually has an acute onset within 24 to 96 hours after the cholinergic effect has worn off with the antidote administrations, and 5 to 18 days are usually required for resolution of symptoms. It usually occurs without fasciculations or other cholinergic manifestations, and the muscles innervated by motor cranial nerves, neck flexors, proximal limb muscles, or respiratory muscles are predominantly affected (3,4). Prolonged suppression of the enzyme acetylcholinesterase is observed in this syndrome.

When the course was reviewed in reference to the features stated above, this case could not be regarded as a continual state of cholinergic crisis, since acute cholinergic symptoms and signs disappeared mostly before it lapsed to the vocal cord paralysis with an exception of weak self-respiratory effort. Moreover, this case could not be categorized under a delayed type polyneuropathy because the onset and recovery of neurologic findings in delayed organophosphate induced polyneuropathy takes 2 to 3 weeks and 6 to 12 months, respectively. The neurologic involvement was confined only to the superior and inferior laryngeal nerve (LN) and originated from the vagus nerve. Vocal fold immobility is caused most frequently by the palsy of LN that controls movement of the laryngeal muscles. It may be caused by a variety of diseases or disorders that mechanically interfere with the movement of the vocal fold. The etiologies of bilateral vocal fold immobility, according to Benninger`s findings in a series of 117 cases, are listed as follows in decreasing order of frequency: surgical trauma (44%), malignancies (17%), endotracheal intubation (15%), neurologic disease (12%), and idiopathic causes (12%) (5). Rarely, organophosphate serves as a toxin resulting in vocal cord immobility to human or cats (1,2,4,6,7). Senanayake et al. reported that vagal nerve palsy was identified in four of ten patients with the intermediate syndrome after organophosphate intoxication. In one patient only the vagus nerve was involved (4).

Although true vocal cord paralysis which follow short-term endotracheal intubation is relatively rare, mechanical vocal fold immobility in conjunction with complications of endotracheal intubation can be caused by arytenoid dislocation or subluxation, or compression injury to the recurrent LN (RLN) induced by an inflated endotracheal tube cuff and the overlying thyroid cartilage (8). In this case, tracheal intubation was neither difficult nor misdirected technically, and the tube was not misplaced or removed accidentally during the initial placement or subsequent positioning period. In regard to the RLN injury, our patient did not have any potential risk factors to cause compression injury to the RLN during or after endotracheal intubation. The endotracheal tube cuff was deflated periodically to attenuate pressure, preventing pressure injury to RLN.

Laryngeal joint injury or arytenoid dislocation is best evaluated by LEMG combined with laryngoscopic examinations. Normal recruitment pattern of LEMG is most commonly found in arytenoid dislocation (9) and also may appear in a patient with prolonged intubation (10). LEMG findings in our case concurrently revealed neuropathic (denervation or partial denervation) patterns in bilateral thyroarytenoid and cricothyroid muscles. This suggests that combined involvement of both the SLN and RLN lead to BVCP, and also that the cause of BVCP is not arytenoid dislocation or subluxation and a compression injury by endotracheal intubation.

Neuropraxy due to trauma is to resolve completely within 8 to 12 weeks after onset of vocal cord paralysis (11). However, our patient was clinically resolved over 2 weeks, which is nearly consistent with required periods for clinical recovery of the intermediate syndrome, after onset of BVCP.

Although plasma cholinesterase (PChE) activity cannot be assumed to mirror the effect of organophosphate compounds at target synapses, we only performed PChE measurements because erythrocyte acetylcholinesterase (AChE) was unavailable at our institution. However, dichlorvos mostly inhibits PChE in response of PChE and AChE to different organophosphates (12). That is, PChE activity, immediately after dichlorvos exposure, is substantially inhibited more than erythrocyte AChE activity (13). The fact that there was a prolonged suppression of PChE supports our view that the finding of isolated bilateral vocal chord paralysis is reflective of an intermediate syndrome.

The presence of acute, seemingly reversible, bilateral vocal cord paralysis as a part of intermediate syndrome has not been previously reported. However, our findings on videolaryngoscopy and LEMG that signified BVCP are more consistent with findings of neurotoxicity induced by prolonged suppression of the enzyme acetylcholinesterase in the intermediate syndrome than that of mechanical laryngeal injury. Although an idiopathic cause of a transient BVCP cannot be completely excluded in this case, the time course and several other presumptive data strongly suggest that the BVCP might have been a unique component of the intermediate syndrome.

Conclusion

We have described an isolated bilateral vocal cord paralysis as a unique component of intermediate syndrome in OPP. Although the exact timing of the onset of the paralysis was unclear in this case, owing to a concomitant endotracheal intubation, the condition of vocal cord paralysis did not last long and it was successfully managed by general supportive measures administered in sitting position. However, due to the potential dangers involved in such cases, physicians should be aware of the occurrence of neurotoxic effects while caring for the patients exposed to organophosphates. Isolated BVCP should be excluded as a cause, if dysphonia or respiratory distress after extubation in patients with intermediate syndrome. LEMG in such cases is to be an important diagnostic adjunct.

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