ORIGINAL ARTICLE

Laryngeal Dysfunction in Acute Organophosphorus and Carbamate Poisoning

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Abstract

Background: Organophosphorus (OP) and carbamate pesticides are widely used for crop protection. We describe the spectrum of laryngeal abnormalities in patients admitted to the intensive care unit (ICU) with acute OP and carbamate poisoning as there is limited information on it.

Materials and methods: Consecutive patients admitted to the ICU with acute OP and carbamate poisoning over 20 months (December 2014–July 2016) were recruited. Patients were followed up post-discharge if they had undergone tracheostomy or developed hoarseness of voice or stridor following extubation. Asymptomatic individuals who consented underwent laryngoscopy after ICU discharge. The primary outcome was the development of laryngeal dysfunction. Other outcomes included length of stay, need for ventilation, mortality, tracheostomy, and time to decannulation of tracheostomy.

Results: Of the 136 patients recruited, 71 (52%) underwent laryngoscopy. The overall mortality rate was 9.6%. Of the 71 patients who underwent laryngoscopy, 18 had abnormal findings, which included unilateral or bilateral vocal cord paresis or palsy ($n = 14$) and/or aspiration ($n = 9$), subglottic stenosis ($n = 1$), tracheal stenosis ($n = 1$), or arytenoid granuloma ($n = 1$). Laryngeal dysfunction was associated with the ingestion of a dimethyl OP compound ($p = 0.04$) and quantum consumed ($p < 0.001$). Patients with laryngeal dysfunction had significantly ($p = 0.004$) longer hospital stay (19.1 ± 10.7 vs 11.8 ± 8.3 days).

Conclusion: Laryngeal dysfunction is not uncommon in OP and carbamate poisoning and is associated with the ingestion of larger quantity of a dimethyl OP compound and longer hospital stay. Otorhinolaryngologists could be involved early to help identify these abnormalities and initiate an appropriate treatment to ensure a functional voice and good airway.

Keywords: Aspiration, Organophosphorus poisoning, Poisoning, Subglottic stenosis, Vocal cord paralysis.

Indian Journal of Critical Care Medicine (2022): 10.5005/jp-journals-10071-24096

Introduction

Deliberate ingestion of pesticide is a leading cause of suicide in India. Among the commonly used pesticides, poisoning with organophosphorus (OP) compounds and carbamates is frequent because of easy availability. The incidence is high in India and more so in Tamil Nadu.[1]

Three distinct patterns of neuromuscular weakness are described: acute weakness (type I paralysis), which occurs within a few minutes to few hours after exposure; intermediate syndrome (type II paralysis), which generally occurs after 24 hours and lasts up to 1–2 weeks; and organophosphate-induced delayed polyneuropathy (OPIDP) (type III paralysis), which occurs after several weeks following exposure.[2,3] Type I weakness is due to cholinergic overstimulation, type II weakness is due to neuromuscular dysfunction, and type III paralysis is a result of covalent inhibition of the neuropathy target esterase (NTE).[3–5]

There are several case reports[6–9] and one small case series[10] that describes laryngeal dysfunction in the setting of OP poisoning. This has been observed not only in type II paralysis[6] or failed extubation[7] but also as a late manifestation with delayed-onset laryngeal dysfunction.[8,10]

In one report, laryngeal electromyography (EMG) ruled out cricoarytenoid joint injury or arytenoids dislocation and confirmed bilateral laryngeal paralysis.[6] Given the paucity of the literature on the topic, this study was undertaken to describe the laryngeal abnormalities in OP poisoning and the factors associated with its occurrence.

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How to cite this article: Mani GS, Mathews SS, Victor P, Peter JV, Yadav B, Albert RRA. Laryngeal Dysfunction in Acute Organophosphorus and Carbamate Poisoning. Indian J Crit Care Med 2022;26(2):167–173.

Source of support: Nil

Conflict of interest: None

Materials and Methods

This cohort study was conducted in the medical intensive care unit (ICU) of a tertiary care university-affiliated teaching hospital in South India over 20 months (December 2014–July 2016). Patients who presented following deliberate ingestion of an OP compound or carbamate were included. The diagnosis of OP or carbamate poisoning was made based on the container brought by the patient in the setting of clinical signs of OP/carbamate

poisoning in combination with reduced pseudocholinesterase activity (reference range 3000–8000 IU/L). Patients with unknown poisoning were also included if they manifested cholinergic signs and had evidence of cholinesterase suppression. Nonconsenting patients, those with preexisting laryngeal pathology, pregnant women, mentally challenged individuals, and those with a previous admission requiring ventilatory support were excluded.

Patients were managed using standard protocols for the management of OP poisoning that included atropine and supportive therapy for organ dysfunction.[11,12] Oximes were not used.[13] Patients with hypotension (systolic blood pressure <90 mm Hg) were fluid resuscitated. If they continued to be hypotensive despite adequate fluid resuscitation, they were started on an appropriate vasoactive agent, based on the clinical assessment of the etiology of shock. Patients with respiratory failure were managed with noninvasive or invasive ventilation as appropriate. Patients who were invasively ventilated received analgo-sedation as a combination of morphine or fentanyl along with midazolam. Endotracheal or tracheostomy tubes with high-volume, low-pressure cuffs were used with protocolized periodic check of the cuff pressure to ensure that cuff pressures did not exceed 30 cm water. Sedation was ceased prior to extubation.

All patients were assessed for readiness for extubation using a combination of factors that included the assessment of (a) airway (using cuff leak and cough test), (b) adequacy of breathing (ensuring a reasonable respiratory rate on minimal ventilatory supports and a forced vital capacity (FVC) of at least 15 mL/kg), (c) stable hemodynamics (on minimal or no inotropes), (d) normal electrolytes (potassium and phosphorus), and (e) a Glasgow Coma Scale of at least 7T/10 and no evidence of significant proximal or neck muscle weakness. In the setting of OP poisoning, given our experience that neuromuscular weakness could fluctuate in a span of few hours, at least two assessments of muscle function (proximal and neck muscle) and FVC were done (minimum 6 hours apart) prior to extubation. Where there was still an element of doubt on ventilatory reserve, a T-piece trial was given for a period of not exceeding 1–2 hours prior to extubation.[14]

Patients who failed extubation were trialed on noninvasive ventilation or re-intubated depending on the clinical situation. The cause for failed extubation was assessed as to whether it was due to airway-related issues or ventilatory failure/fatigue. Post re-intubation, if the cause was ascertained to be due to an airway problem (e.g., laryngeal dysfunction), a surgical tracheostomy was performed by the otolaryngologist either in the ICU at the bedside or in the operating theater if there was a perceived higher risk of complications. Percutaneous tracheostomy was not considered in this setting. If the reason for failed extubation was ventilatory fatigue, the patient was reassessed after a further period of ventilatory support for readiness for extubation. In the absence of airway compromise, if ventilatory failure persisted, then tracheostomy was done during the second week of ICU admission. Patients who underwent tracheostomy for airway compromise were quickly weaned to a T-piece, when respiratory muscle weakness improved. Once weaned, the patient was transferred from the ICU to the ward for further care. Decannulation of the tracheostomy was done either during hospital stay or after discharge in the outpatient clinic after ensuring that vocal cord function had improved and airway was adequate.

Patients underwent flexible laryngoscopy during hospitalization if they developed hoarseness of voice or stridor following extubation or if they required tracheostomy. Findings of vocal cord paresis or paralysis, aspiration, laryngotracheal stenosis, and granuloma formation were recorded. These patients were advised review in the outpatient clinic for the reassessment of vocal cord function as well as decannulation of tracheostomy if this was not done prior to hospital discharge. Hospital records were reviewed to obtain longer follow-up data, where available. Other patients with severe OP poisoning who did not manifest symptoms of airway compromise were also approached for their willingness to undergo flexible laryngoscopy post ICU discharge.

Cases were defined as patients who developed evidence of laryngeal dysfunction during the course of ICU admission or immediately post ICU discharge. Laryngeal dysfunction was diagnosed if there was direct evidence of vocal cord dysfunction (e.g., vocal cord paresis or paralysis) or indirect evidence of vocal cord dysfunction without overt evidence of cord paresis (e.g., aspiration). Aspiration was defined as entry of secretions past the vocal folds, as evidenced on laryngoscopy.[15] Follow-up laryngoscopy was done to assess the cord mobility as well as sequelae such as granuloma formation or stenosis.

Demographic, treatment, and outcome data of patients who underwent laryngoscopy during the study period were recorded prospectively. Data of all other patients who were admitted with OP/carbamate poisoning during the study period but did not undergo laryngoscopy were obtained *post hoc* from hospital records. The primary outcome of interest was the development of laryngeal dysfunction, which included vocal cord paresis or paralysis, aspiration, stenosis, and granuloma formation. Secondary outcomes included mortality, length of stay, need for ventilation, tracheostomy, and time to decannulation of tracheostomy.

Statistical Analysis

Baseline characteristics were summarized as frequencies (percentages) for categorical variables as mean (standard deviation, SD) or median (interquartile range, IQR) for continuous variables as appropriate. Mann–Whitney *U*-test or Fisher's exact test was used to compare the characteristics of patients who underwent laryngoscopy with those who did not undergo laryngoscopy as well as those who had laryngeal dysfunction and those who did not develop laryngeal dysfunction. All statistical analyses were done using STATA v15.0 (StataCorp, College Station, Texas). The study was approved by the Institutional Review Board and the Ethics Committee of the hospital (IRB No: 9142/2014).

Results

The study cohort of 136 patients (90 males and 46 females) comprised a relatively young population, with a mean ± SD age of 31.3 ± 12.8 years and moderate severity of illness. Diethyl (41.2%) and dimethyl (29.4%) OP were the main compounds ingested. Nearly 90% of patients required ventilation and 24.3% required tracheostomy. The overall in-hospital mortality rate was 9.6%.

When the characteristics of patients who underwent laryngoscopy ($n = 71$) were compared with those who did not ($n = 65$), patients who did not have laryngoscopy were sicker ($p = 0.005$) and had higher mortality (Table 1). On the other hand, duration of ventilation, need for tracheostomy, and length of stay were significantly higher in the group that underwent laryngoscopy (Table 1).

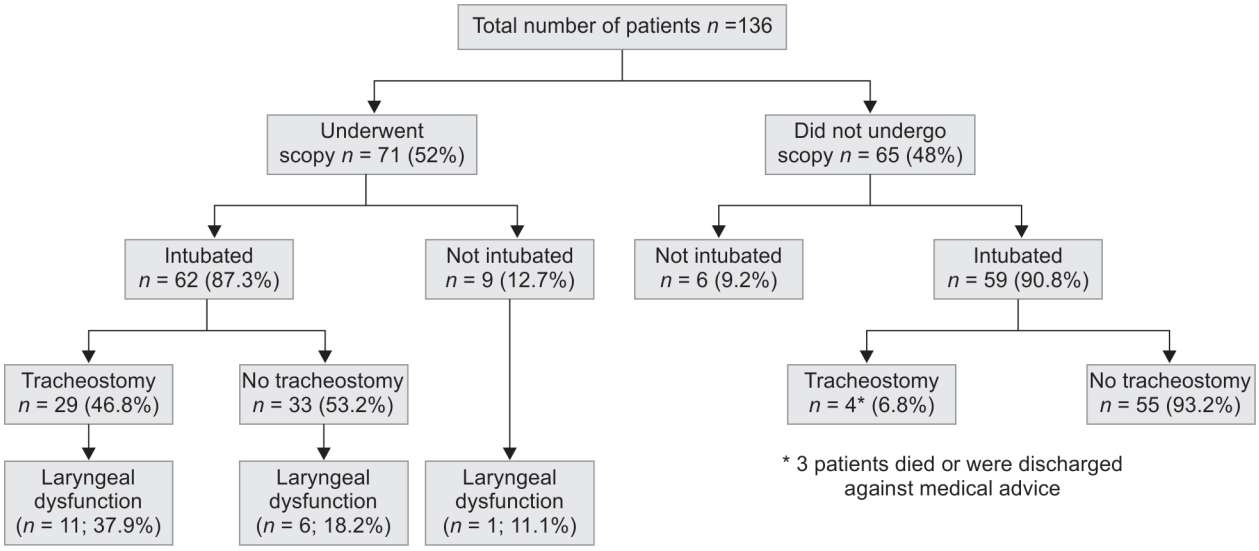
Of the 71 patients who underwent flexible laryngoscopy, 62 (87.3%) were intubated and 9 (12.7%) were not intubated

Table 1: Baseline characteristics and outcomes of the entire study cohort

	All patients (n = 136)	*Laryngoscopy done (n = 71)*	*No laryngoscopy (n = 65)*	*p value*
Age, years	31.3 (12.8)	29.6 (10.6)	33.1 (14.6)	0.12
Gender, Male:Female	90:46	49:22	41:24	0.48
APACHE II score	13.0 (6.2)	11.3 (5.2)	14.6 (7.0)	0.005
Type of compound*				
Dimethyl	40 (29.4)	21 (29.6)	19 (29.7)	0.89
Diethyl	56 (41.2)	31 (43.7)	24 (37.5)	
S-alkyl	7 (5.2)	3 (4.2)	4 (6.3)	
Carbamate	3 (2.2)	1 (1.4)	2 (3.1)	
Unknown	30 (22.0)	15 (21.1)	15 (23.4)	
Amount ingested, mL	86.7 (72.9)	77.4 (47.3)	105.2 (106)	0.09
Cholinesterase, U/L†	122 (89–308)	130 (90–334)	115 (85–297)	0.59
Number ventilated	121 (89)	62 (87.3)	59 (90.8)	0.59
Ventilation duration, days	7.5 (5.9)	9.3 (7.0)	5.5 (3.6)	0.0003
VFD, days	18.4 (8.4)	18.8 (6.6)	17.9 (10.0)	0.54
Number tracheostomy	33/121 (27.3)	29/62 (46.8)	4/59 (6.8)‡	<0.001
Day of tracheostomy	7.8 (3.6)	7.7 (3.4)	8.5 (3.4)	0.67
Day of decannulation; Median (IQR) days~	31 (20.8–47.5)	32 (19.5–48)	30~	—
Length of hospital stay, days	11.2 (8.3)	13.7 (9.5)	8.6 (5.7)	0.003
Mortality*	13 (9.6)	0 (0)	13 (20)	<0.001

All values are mean (standard deviation) unless specified; APACHE, acute physiology and chronic health evaluation; †Median (interquartile range, IQR) pseudocholinesterase; *For these variables, values are the number of patients and the percentages are in parentheses; VFD, ventilator-free days, calculated as (28 – duration of ventilation)—ventilator free days is zero for those who died; ‡Three of these patients either died or were discharged against medical advice: ~Of the four patients who underwent tracheostomy in those who did not undergo scopy, three patients died, hence, the day of decannulation is available in the only survivor

Flowchart 1: Flow diagram of patients recruited to the study



(Flowchart 1). Twenty-nine patients (46.8%) required tracheostomy, of whom 11 (37.9%) had laryngeal dysfunction. In the subset of patients who were intubated and did not undergo tracheostomy, six (18.2%) had evidence of laryngeal dysfunction (Flowchart 1). Among those who were not intubated and consented for laryngoscopy ($n = 9$), one (11.1%) had laryngeal dysfunction.

Table 2 compares the baseline characteristics and outcomes of patients who had evidence of laryngeal dysfunction ($n = 18$) with those of patients who had a normal study ($n = 53$). The quantum of poison consumed and the ingestion of a dimethyl compound were significantly associated with laryngeal dysfunction (Table 2). Patients who developed laryngeal dysfunction had more attempts at intubation ($p = 0.02$), failed extubation ($p = 0.05$), requirement for tracheostomy ($p = 0.04$), and stayed longer in hospital (19.1 ± 10.7 vs 11.8 ± 8.3 days; $p = 0.004$) when compared with those who had no evidence of laryngeal dysfunction.

Table 3 provides clinical details of the 18 patients who developed laryngeal dysfunction. Of note, during the first

Table 2: Baseline characteristics and outcomes of those who underwent flexible laryngoscopy

	All patients (n = 71)	*Abnormal laryngoscopy (n = 18)*	*Normal laryngoscopy (n = 53)*	*p value*
Age	29.6 (10.6)	28.7 (11.6)	29.9 (10.3)	0.68
Gender, Male:Female	49:22	11:7	38:15	0.40
APACHE II score	11.3 (5.2)	11.1 (3.1)	11.4 (5.9)	0.87
Type of compound*				
Dimethyl	21 (29.6)	9 (50.0)	12 (22.6)	0.04*
Diethyl	31 (43.7)	6 (33.3)	25 (47.2)	
S-alkyl	3 (4.2)	1 (5.6)	2 (3.8)	
Carbamate	1 (1.4)	1 (5.6)	0 (0)	
Unknown	15 (21.1)	1 (5.6)	14 (26.4)	
Amount ingested, mL	77.4 (47.3)	127.9 (68.9)	64.8 (29.6)	<0.001*
Cholinesterase, U/L†	130 (90–334)	130 (90–171)	127 (92–368)	0.73
Intubation >1 attempt‿	6/54 (11.1)‡	3/9 (33)	3/45 (6.7)	0.02*
Number ventilated	62 (87.3)	17 (94.4)	45 (84.9)	0.43
Ventilation duration, days	9.3 (7.0)	11.4 (5.5)	8.5 (7.4)	0.16
VFD, days	18.8 (6.6)	16.7 (5.5)	19.7 (6.8)	0.11
Failed extubation‿	12/62‡	6/12 (50)	11/50 (22)	0.05*
Number tracheostomy	29 (40.9)	11 (61.1)	18 (34.0)	0.04*
Day of tracheostomy	7.7 (3.4)	8.9 (2.5)	7.0 (3.7)	0.15
Day of decannulation	37 (22.8)	28.6 (23.4)	41.7 (21.6)	0.14
Length of hospital stay, days	13.7 (9.5)	19.1 (10.7)	11.8 (8.3)	0.004*
Mortality‿	0 (0)	0 (0)	0 (0)	—

All values are mean (standard deviation) unless specified; APACHE, acute physiology and chronic health evaluation; †Median (interquartile range. IQR) pseudocholinesterase; ‿For these variables, values are the number of patients and the percentages are in parentheses; VFD, ventilator-free days, calculated as (28 – duration of ventilation)—ventilator-free days is zero for those who died; *Significant value; ‡Data available only in 54 patients for intubation attempts and for 62 patients for failed extubation

laryngoscopic examination which was done at a median time of 17.5 days (IQR 14.5–24), direct visual evidence of vocal cord dysfunction (unilateral or bilateral vocal cord paresis or palsy) was observed in 14 patients, while 8 patients had evidence of aspiration, with or without vocal cord dysfunction. Four patients had only evidence of aspiration without any overt vocal cord paresis. During the subsequent follow-up laryngoscopy which was done at a median time of 45.5 days (IQR 31–53), one patient each had subglottic narrowing, arytenoid granuloma, and tracheal stenosis and an additional patient had evidence of aspiration. All patients had evidence of vocal cord dysfunction within 4 weeks of poisoning, and 11 patients (61.1%) required tracheostomy. One-third of the patients (6/18) who had evidence of laryngeal dysfunction were lost to follow up. It was interesting to note that one patient who had subglottic narrowing (grade I) had not been intubated; since the airway was adequate, no surgical intervention was required. Another patient who was initially noted to have left vocal cord palsy presented 6 weeks later with stridor. While the vocal cord palsy had recovered, the patient had developed tracheal stenosis. This patient underwent tracheal resection and anastomosis and on follow-up continues to have a good airway.

Discussion

In this cohort study of 136 patients admitted to the ICU with OP or carbamate poisoning, 71 patients (52%) underwent flexible laryngoscopy. In the initial evaluation, 18 patients had evidence of laryngeal dysfunction that included unilateral or bilateral vocal cord immobility and/or aspiration. Delayed complications of intubation that were observed in this series included laryngotracheal stenosis and vocal granuloma. Laryngeal dysfunction was significantly associated with the ingestion of a dimethyl OP compound, quantum ingested, multiple intubation attempts, failed extubation, need for tracheostomy, and longer hospital stay.

Although there are case reports of laryngeal dysfunction in OP poisoning, this study is a descriptive study of the laryngeal dysfunction in a cohort of patients admitted to the ICU with OP or carbamate poisoning. The estimated incidence of laryngeal dysfunction in this cohort of 25.4% is likely to be overestimated or underestimated and hence not reported due to the following reasons. The cohort comprised only patients admitted to the ICU and did not include all patients admitted to the wards with less severe manifestations; only about half underwent laryngoscopy. These introduce a selection bias. Further higher illness severity and mortality in those who did not undergo laryngoscopy could have also altered the incidence of laryngeal dysfunction (Table 1).

It is likely that several factors contribute to laryngeal dysfunction in this setting. The association between the quantum of poison ingested and the type of poison (dimethyl OP) and the development of laryngeal dysfunction is not surprising. Further, analysis was precluded by the fact that 11 different compounds were associated with laryngeal dysfunction (Table 3). Monocrotophos ($n = 3$), dimethoate ($n = 3$), chlorpyrifos ($n = 2$), and phorate ($n = 2$) were the main compounds implicated in our study. In the published reports, several compounds were implicated and no compound was implicated twice in laryngeal dysfunction.[6–10]

The observation of an association between failed extubation and need for tracheostomy and laryngeal dysfunction is also not surprising since extubation failure occurred in these patients despite careful assessment of readiness for extubation through a protocolized checklist. The need for tracheostomy and the delay in decannulation (Table 3) due to vocal cord dysfunction

Table 3: Clinical details and follow-up data of patients with laryngeal abnormalities

No	*Age/sex*	*Compound*	*Type of OP*	*Amt. ingested*	*Vent*	*Vent durn*	*Trach*	*Trach day*	*Day decan*	*Initial laryngoscopy (day)*	*Final laryngoscopy (day)*
1	18/M	Dichlorvos	Dimethyl	100 mL	Yes	3	No	—	—	Left VC palsy (5)	Left VC palsy (NA)
2	24/F	Carbofuran	Carbamate	40 mL	No	—	—	—	—	Left VC palsy (NA)	Subglottic stenosis grade I (NA)
3	16/F	Phorate	Diethyl	80 mL	Yes	8	No	—	—	Left VC palsy (16)	Left VC palsy (NA)
4	50/M	Dimethoate	Dimethyl	250 mL	Yes	10	No	—	—	Left VC palsy (30) Aspiration	Normal (47)
5	45/M	Malathion	Dimethyl	200 mL	Yes	8	Yes	5	17	Aspiration (16)	Normal (21)
6	55/M	Dimethoate	Dimethyl	80 mL	Yes	11	Yes	6	92	Left VC palsy (15)	Left VC palsy (58)
7	19/M	Unknown	—	100 mL	Yes	11	Yes	9	30	Left VC palsy (18)	Left VC palsy Aspiration (NA)
8	23/F	Chlorpyrifos	Diethyl	150 mL	Yes	17	Yes	7	36	Bilateral VC palsy (20)	Bilateral vocal paresis (42)
9	33/M	Phorate	Diethyl	200 mL	Yes	12	Yes	11	13	Aspiration (NA)	Aspiration (NA)
10	20/F	Monocrotophos	Dimethyl	NA	Yes	4	Yes	8	32	Aspiration (17)	Aspiration (31)
11	35/M	Quinalphos	Diethyl	75 mL	Yes	18	Yes	9	32	Bilateral VC paresis (25)	Left VC paresis (53) Right arytenoid granuloma
12	27/M	Triazophos	Diethyl	NA	Yes	22	Yes	14	30	Aspiration (24)	Normal (27)
13	18/F	Prophenofos	S-alkyl	250 mL	Yes	14	Yes	10	8	Left VC palsy (24)	Lost to follow up
14	36/F	Chlorpyrifos	Diethyl	NA	Yes	19	Yes	10	8	Bilateral VC palsy (25) Aspiration	Left VC paresis (44)
15	28/M	Monocrotophos	Dimethyl	NA	Yes	14	Yes	9	17	Bilateral VC paresis Aspiration (22)	Normal (57)
16	28/M	Dimethoate	Dimethyl	NA	Yes	11	No	—	—	Left VC paresis Aspiration (14)	Lost to follow up
17	25/M	Monocrotophos	Dimethyl	200 mL	Yes	5	No	—	—	Left VC palsy (11)	Normal mobile cords Tracheal stenosis (47)
18	17/F	Phosphenetoin	Dimethyl	NA	Yes	6	No	—	—	Left VC palsy (12)	Lost to follow up

OP, organophosphorus; Amt, amount; Vent, ventilation; durn, duration; Trach, tracheostomy; decan, decannulation; VC, vocal cord; NA, not available; day in parentheses describe the day following hospital admission when laryngoscopy was done; NA, data not available

support the hypothesis that vocal cord dysfunction in this setting takes time to resolve. These observations are consistent with the published literature. In two reports,[6,7] failed extubation was due to bilateral vocal cord paralysis. In another report,[8] the patient developed hoarseness post-extubation that progressed to life-threatening stridor. The time to resolution of vocal cord function was 2 weeks to 2 months in these reports, consistent with our observations. The case series[10] of three patients who developed delayed laryngeal symptoms contrasts our cohort; in this series,[10] laryngeal dysfunction occurred 14–26 days after extubation and lasted 4–15 weeks.

One confounding factor needs to be addressed. It is likely that multiple attempts at intubation, particularly by inexperienced trainees, could also result in laryngeal trauma, edema, and failed extubation.[16] Although multiple intubation attempts were associated with laryngeal dysfunction in our study ($p = 0.02$), it was not clear if they were causal for laryngeal dysfunction. Laryngeal edema, trauma, or dislocation of arytenoid cartilage[17] was not observed on flexible laryngoscopy in our patients. Given that laryngeal dysfunction occurred even in un-intubated patients (1/9; 11.1%), it is likely that OP compounds can directly cause laryngeal weakness.

The mechanism of laryngeal weakness is unclear. It is likely that laryngeal weakness is due to the same pathophysiological mechanisms that result in proximal and neck muscles in intermediate syndrome. In one study where laryngeal EMG[6] was performed, widespread fibrillation and positive sharp waves were observed in the muscles innervated by the recurrent laryngeal nerve. There was also a significant reduction in voluntary motor unit action potential during attempts at muscle contraction in the cricothyroid muscles innervated by the external branch of the superior laryngeal nerve. On the other hand, the mechanism of delayed-onset laryngeal dysfunction in the previously published case series[10] is probably different and related to the pathophysiology of OPIDP, wherein the inhibition of the NTE is the likely mechanism for weakness, since this occurred several days after extubation.[10,18,19] The aspiration seen in about 50% of the subjects could be due to the superior laryngeal nerve dysfunction, the subtle findings which can easily be missed or can be attributed to recurrent laryngeal nerve palsy.[20,21] The tracheal stenosis seen in one patient and the arytenoid granuloma in another could be related to post-intubation-induced mucosal injury.[22] The inflammation and scarring in the airway due to mucosal ischemia (decreased capillary perfusion secondary to cuff pressure) could have also been aggravated by hypotension, acid reflux, and

Table 4: Organophosphorus (OP) compounds implicated, categorized as those who developed laryngeal dysfunction and those who did not develop

Compound	*Individual compounds*	*Patients without laryngeal dysfunction*	*Patients with laryngeal dysfunction*
	Di-ethyl compounds		
	Triazophos	17	1
	Chlorpyrifos	15	2
	Phorate	14	2
	Quinalphos	3	1
	Ethion	1	0
	Di-methyl compounds		
Organophosphorus compound	Monocrotophos	20	3
	Dimethoate	4	3
	Dichlorvos	3	1
	Parathion	3	0
	Acephate	1	0
	Malathion	0	1
	Phosphamidon	0	1
	S-alkyl compound		
	Profenofos	6	1
Carbamate	Carbofuran	2	1
Unknown OP	Unknown	29	1

vocal abuse.[22–24] To be noted is the fact that one patient with subglottic narrowing was not intubated.

The following limitations merit mention. This was a single-center study focused only on critically ill OP-poisoned patients. It would have been ideal to answer the association between OP poisoning and laryngeal dysfunction using a case–control study; however, this was planned as a cohort study. Although the plan was to recruit consecutive patients, several patients (around 50%) could not be recruited due to the nonavailability of the principle investigator during weekends or leave as well as the treating clinician's decision to not subject patient to laryngoscopy. Although this problem was circumvented to some extent by the inclusion of the entire cohort of patients admitted to the ICU during the study period through post hoc review of the hospital records, this introduced a selection bias in determining the incidence of laryngeal dysfunction. Hence, we opted to not provide an estimate of the incidence of laryngeal dysfunction as this would have either overestimated or underestimated the incidence. The study was thus restricted to describe the nature of laryngeal dysfunction as a basis for further work using the appropriate study design.

Besides association between the quantum of poison ingested and the type of poison (dimethyl OP), further subgroup analysis was precluded by the fact that 11 different compounds were associated with laryngeal dysfunction (Table 4). Only about 50% of patients were assessed for laryngeal dysfunction. Laryngeal EMG was not done in our study. This would have helped delineate those with cord dysfunction due to the pesticide from arytenoid dislocation due to traumatic endotracheal intubation. Further unmeasured/unknown factors (e.g., solvents) could have also contributed to laryngeal dysfunction. A subset of patients with laryngeal dysfunction were lost to follow up, and hence, recovery of vocal cord function could not be documented in all. Despite these limitations, this study provides a detailed description of the nature of laryngeal dysfunction and the course in these patients.

Conclusion

A functional voice and good airway is vital, particularly in patients who recover from critical illness and return to their homes to make a living. The laryngeal dysfunction noted in those with nonfatal suicidal attempts may be either due to OP neurotoxicity or mechanical trauma due to laryngotracheal intubation. This warrants further study focusing on delineating the mechanisms through detailed EMG studies and extended follow-up periods. The otorhinolaryngologist could facilitate the detection of laryngeal abnormalities and initiate suitable care to ensure a functional voice and an adequate airway.

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