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Revista Española de Anestesiología y Reanimación 69 (2022) 105–108

Revista Española de Anestesiología y Reanimación

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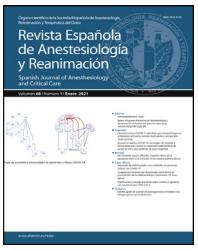

CASE REPORT

Late bilateral vocal cord palsy following endotracheal intubation due to COVID-19 pneumonia☆

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Received 26 October 2020; accepted 19 November 2020
Available online 3 February 2022

KEYWORDS
Vocal cords paralysis;
Intubation intratracheal;
Pneumonia viral;
COVID-19;
Tracheotomy;
Respiratory distress syndrome

Abstract Vocal cord paralysis is a rare but severe complication after orotracheal intubation. The most common cause is traumatic, due to compression of the recurrent laryngeal nerve between the orotracheal tube cuff and the thyroid cartilage. Other possible causes are direct damage to the vocal cords during intubation, dislocation of the arytenoid cartilages, or infections, especially viral infections. It is usually due to a recurrent laryngeal nerve neuropraxia, and the course is benign in most patients. We present the case of a man who developed late bilateral vocal cord paralysis after pneumonia complicated with respiratory distress due to SARS-CoV-2 that required orotracheal intubation for 11 days. He presented symptoms of dyspnea 20 days after discharge from hospital with subsequent development of stridor, requiring a tracheostomy. Due to the temporal evolution, a possible contribution of the SARS-CoV-2 infection to the picture is pointed out.

PALABRAS CLAVE
Parálisis de los pliegues vocales;
Intubación intratraqueal;
Neumonía viral;

Parálisis bilateral tardía de cuerdas vocales trasintubación endotraqueal por neumonía COVID-19

Resumen La parálisis de cuerdas vocales es una complicación poco frecuente, aunque severa, tras una intubación orotraqueal. La causa más frecuente es la traumática, debido a la compresión del nervio laríngeo recurrente entre el maguito del tubo orotraqueal y el cartílago

☆ Please cite this article as: Curros Mata N, Alvarado de la Torre S, Carballo Fernández J, Martínez Morán A, Álvarez Refojo F, Rama-Maceiras P. Parálisis bilateral tardía de cuerdas vocales trasintubación endotraqueal por neumonía COVID-19. Rev Esp Anestesiol Reanim. 2022;69:105–108.

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Infecciones por coronavirus;
Traqueotomía;
Síndrome de dificultad respiratoria del adulto

tiroides. Otras posibles causas son lesión directa de las cuerdas vocales durante la intubación, luxación de los cartílagos aritenoides e infecciones, sobre todo víricas. Suele deberse a una neuroapraxia del nervio laríngeo recurrente, y el curso es benigno en la mayoría de los pacientes. Presentamos el caso de un varón que desarrolló una parálisis de cuerdas vocales bilateral tras una neumonía complicada con distrés respiratorio por SARS-CoV-2 que requirió intubación orotraqueal durante 11 días. Presentó clínica de disnea a los 20 días del alta hospitalaria con desarrollo posterior de estridor, siendo necesaria la realización de una traqueostomía. Debido a la evoución temporal, se apunta una posible contribución de la infección por SARS-CoV-2 al cuadro.

Introduction

Bilateral vocal cord palsy is a rare but serious complication after tracheal intubation (TI)[1]. Vocal cord palsy is usually due to injury[2], although in the context of viral disease it can be caused by viral neurotropism[3].

SARS-CoV-2 coronavirus disease is characterized by severe pneumonia that may require prolonged mechanical ventilation, and the virus can also affect the nervous system[4,5]. We report the case of a patient who developed late bilateral vocal cord palsy that was diagnosed after receiving mechanical ventilation for SARS-CoV-2 pneumonia.

Case report

The patient was a 74-year-old man with a personal history of cholecystectomy and prostatectomy, who had no previous respiratory symptoms. He developed symptoms of fever, cough and myalgia, and was diagnosed with COVID-19 infection by polymerase-transcriptase chain reaction (PCR) on a nasopharyngeal swab, and initially received outpatient treatment. Eight days after diagnosis, he was admitted to the hospital due to progressive dyspnoea. Physical examination on admission revealed tachypnoea with 24 rpm and hypoxaemia (peripheral oxygen saturation [SpO_2] 85%). The chest X-ray revealed bilateral pneumonia, and blood tests revealed lymphopaenia with 0.54×10^9/L lymphocytes, D-dimers 648 ng/mL, interleukin-6 53.7 pg/mL and C-reactive protein (CRP) 7.66 mg/dL. Tests for pneumococcal antigenuria and *Legionella* were negative.

The patient remained on the ward for 7 days, receiving treatment with lopinavir/ritonavir, hydroxychloroquine, ceftriaxone, and azithromycin. Due to a lack of clinical improvement and worsening of inflammatory markers, 3 doses of tocilizumab were added on the third day of admission. The patient's condition progressed to dyspnoea at rest, with bilateral crackles and PaO_2/FiO_2 ratio of 145, so he was admitted to the Resuscitation Unit for pneumonia complicated by respiratory distress (ARDS).

On admission, the patient presented tachypnea 32 rpm, hypoxaemia and significant work of breathing. He was intubated with a number 8 tracheal tube (TT) and was connected to mechanical ventilation, adding 1 mg/kg IV methylprednisolone to his existing regimen. Labs showed elevated acute phase reactants (ferritin 4163 ng/mL, LDH 971 IU/L, D-dimers 8714 ng/mL) and chest X-ray showed worsening of bilateral pulmonary infiltrates.

He required vasoactive support with low-dose norepinephrine (<0.1 mcg/kg/min) for the first 24 h, and received 4 cycles of prone positioning until his supine PaO_2/FiO_2 ratio was greater than 300 mmHg. An additional complication was worsening of renal function in the first 24 h (peak creatinine of 1.75 mg/dL) that improved with fluid intake and furosemide. The TT became obstructed with mucous on the sixth day of admission and was changed. After extubation, 11 days after admission to the unit, he developed acute confusional syndrome that required treatment with quetiapine and resolved within 24 h.

He remained in the unit for 13 days, and 2 negative COVID PCR tests were obtained from bronchial secretions prior to transfer to the ward. He remained on the ward for a further week, with dyspnoea or respiratory distress, and was in good general condition on discharge from hospital: baseline SpO_2 98% and basal hypoventilation with no adventitious sounds on auscultation.

During telephone follow-up to his home he reported good evolution, being able to lead an active life and walk several kilometres each day. Twenty days after hospital discharge, he developed progressive nocturnal cough, dyspnoea, and supine breath sounds that improved when standing. He went to the Emergency Department and was evaluated by a pulmonologist, who ruled out lung involvement. Bronchodilator treatment was started, but due to the persistence of the condition, he was assessed by the ENT specialist on day 35 after discharge, at which point the patient had already developed stridor. A fibreoptic endoscopy study revealed bilateral palsy of the vocal cords in the medial position, and head neck and chest computerized tomography was unremarkable. Given the progression of the symptoms of dyspnoea and stridor, the patient was admitted to hospital and a tracheotomy was scheduled. Before performing the tracheotomy, the patient tested positive again for SARS-CoV-2 on PCR. The serology study showed IgM negative and IgG positive for COVID-19, cytomegalovirus, Epstein Bar virus, herpes simplex virus, and varicella zoster virus, as well as a

negative PCR for HIV. A new PCR for SARS-CoV-2 performed 2 days later was negative. The patient was discharged with a tracheostomy tube 4 days after admission.

Discussion

TI is a very common, usually safe procedure in critically ill patients. However, it is not entirely free from complications, some of which, such as vocal cord palsy, can be serious. This rare complication occurs in 1 in every 1500 intubations (0.07%)[1], and although complications are more frequent after prolonged intubation, vocal cord injuries can also occur after short periods[6]. Risk factors for vocal cord palsy include age over 50 years, diabetes mellitus, and high blood pressure, together with intubation lasting more than 6 h[1,6].

Various mechanisms can be involved in vocal cord palsy secondary to TI. The most widely accepted cause is compression of the inferior or recurrent laryngeal nerve between the cuff of the TT and the posterior thyroid cartilage[2,7]. The recurrent laryngeal nerve divides into 2 branches at the level of the cricoid cartilage. The posterior branch, which innervates the cricoarytenoid and interarytenoid muscles, does not come into contact with cuff of the tube. The anterior branch, however, which innervates most of the vocal cord abductor muscles, can be compressed between the cuff of the TT and the thyroid cartilage lamina, causing medial or paramedian vocal cord paralysis[7,8]. This results in hoarseness and/or respiratory stridor, unlike bilateral palsy of the recurrent nerve, which causes airway obstruction. Vocal cord palsy is usually unilateral, and 70% of injuries affect the left cord, probably because the TT is taped to the right corner of the mouth[1,6].

Other mechanisms described are direct injury to the vocal cords during intubation, particularly traumatic intubation, or dislocation of the arytenoid cartilage[3]. The recurrent laryngeal nerve can also become elongated when the neck is hyperextended during intubation, or can be injured as a result of TT movement through the cords during ventilation[3,8]. Measures that can prevent this complication include choosing an appropriately sized TT, performing atraumatic TI, monitoring TT cuff pressure, and monitoring for possible malposition of airway devices[9]. In our case, the TT was correctly sized for the patient, both the initial intubation and the orotracheal tube change were non-traumatic, cuff pressure was monitored regularly, and prone positioning, carried out in accordance with the Unit's protocol, was uneventful. Despite these precautions, any of these events could potentially have contributed to the patient's condition.

The time of onset of symptoms — 30 days after extubation, ruled out mechanical compression as the injury mechanism. In unilateral vocal cord palsy, symptoms (dysphonia, hoarseness) usually appear within 24 h of extubation, and the diagnosis is usually made 2 weeks later, with a range of between 1–30 days[1]. The symptoms of bilateral palsy, as reported here, usually occur between 30 min and 36 h after extubation[8]. The late onset of symptoms also made it necessary to rule out the presence of a new tumour or abscess secondary to pneumonia, and we therefore performed a head chest and neck CT scan, which was unremarkable.

When vocal cord involvement is secondary to upper airway tract infection, the symptoms of cough, dysphonia, vocal fatigue, odynophagia and/or vocal cord palsy appear late, within a median of 22 weeks[10]. Infection, particularly viral infection, should also be considered as another possible injury mechanism, as case reports of vocal cord palsy in non-intubated patients have also been published[3]. Viral serology was performed to rule out this hypothesis, and showed no recent infection by the most common viruses.

Coronaviruses are also neurotropic, and given the similarly of different coronaviruses, it is likely that SARS-CoV-2 can induce neural lesions[11], and patients such as ours, with severe infection leading to severe pneumonia, are more likely to develop neurological symptoms[4,11]. Two main pathways have been proposed for the entry of neurotropic respiratory viruses into the CNS: the hematogenous route (across the blood–brain barrier) and the neuronal retrograde route from peripheral nerves[5]. The latter is believed to be responsible for the appearance of anosmia and ageusia in more than 5% of SARS-CoV-2 patients[11]. For this reason, we included possible reactivation of the coronavirus in our diagnostic options, since cases of viral reactivation after hospital discharge have been published[12], and the patient presented a positive PCR at the time of vocal cord palsy diagnosis. However, the serology study reported negative IgM and positive IgG for SARS-CoV-2, and 2 further PCR tests (one after tracheostomy and the other a few days later) were negative, so the patient was diagnosed with non-active COVID, and the positive PCR before the tracheostomy was attributed to the presence of viral fragment in the upper airway with no infectious potential.

Post-TI vocal cord plasy is usually benign, and 68.4% of patients make a full recovery[7]. As it is usually due to neuropraxia of the recurrent laryngeal nerve, functional recovery is rapid[8]. In the case reported here, the vocal cord palsy persisted at the 3-month follow-up, although the patient is now able to talk and swallow.

In conculsion, we present a case of bilateral vocal cord palsy after tracheal intubation in a patient who developed pneumonia complicated by ARDS due to SARS-CoV-2. After ruling out the presence of a tumour or compressive infectious complications, viral reactivation or reinfection, the most likely mechanism was nerve compression during intubation, although we were unable to rule out a neurological complication associated with coronavirus infection, a disease that is not yet fully understood. Bilateral vocal cord palsy secondary to TI, though rare, should be taken into consideration if symptoms such as dyspnoea or stridor appear after extubation, even though these can be a late development, as was the case in our patient.

Conflict of interests

The authors have no conflict of interest to declare.

References

1. Kikura M, Suzuki K, Itagaki T, Takada T, Sato S. Age and comorbidity as risk factors for vocal cord paralysis associated with tracheal intubation. Br J Anaesth. 2007;98:524–30.

2. Wason R, Gupta P, Gogia AR. Bilateral adductor vocal cord paresis following endotracheal intubation for general anaesthesia. Anaesth Intensive Care. 2004;32:417–8.
3. Marie JP, Keghian J, Mendel I, Gueit I, Dehesdin D, Andrieu-Guitrancourt J. Post-intubation vocal cord paralysis: the viral hypothesis. A case report. Eur Arch Otorhinolaryngol. 2001;258:285–6.
4. Wu Y, Xu X, Chen Z, Duan J, Hashimoto K, Yang L, et al. Nervous system involvement after infection with COVID-19 and other coronaviruses. Brain Behav Immun. 2020;87:18–22.
5. Paniz-Mondolfi A, Bryce C, Grimes Z, Gordon RE, Reidy J, Lednicky J, et al. Central nervous system involvement by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). J Med Virol. 2020;92:699–702.
6. Hurtado Nazal C, Araneda Vilches A, Vergara Marín C, García Contreras K, Napolitano Valenzuela C, Badía Ventí P. Paralisia de cordas vocais após intubação endotraqueal: uma complicação incomum da anestesia geral. Rev Bras Anestesiol. 2018;68:637–40.
7. Evman MD, Selcuk AA. Vocal cord paralysis as a complication of endotracheal intubation. J Craniofac Surg. 2020;31:119–20.
8. Cherian A, Balachander H, Nagappa M, Rajagopal V. Bilateral vocal cord palsy following endotracheal intubation: a case report. J Clin Anesth. 2010;22:562–4.
9. Endo K, Okabe Y, Maruyama Y, Tsukatani T, Furukawa M. Bilateral vocal cord paralysis caused by laryngeal mask airway. Am J Otolaryngol. 2007;28:126–9.
10. Rees CJ, Henderson AH, Belafsky PC. Postviral vagal neuropathy. Ann Otol Rhinol Laryngol. 2009;118:247–52.
11. Tassorelli C, Mojoli F, Baldanti F, Bruno R, Benazzo M. COVID-19: what if the brain had a role in causing the deaths? Eur J Neurol. 2020;25, http://dx.doi.org/10.1111/14275.
12. Ye G, Pan Z, Pan Y, Deng Q, Chen L, Li J, et al. Clinical characteristics of severe acute respiratory syndrome coronavirus 2 reactivation. J Infect. 2020;80:14–7.