CASE REPORT

A rare presentation of delayed bilateral vocal cord paralysis post radiation treatment

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SUMMARY

Bilateral vocal cord palsy is a life-threatening condition that can occur due to a plethora of causes. Radiotherapy associated vocal cord immobility is one of the rarest causes associated with this condition and has only been reported a handful of times. We present a case involving a 73-year-old man with a history of glottic carcinoma who had undergone radiotherapy 4 years ago. He presented to the emergency department with a 5-day history of shortness of breath, odynophagia, noisy breathing, low grade fever and sore throat. Further examination via bedside flexible nasopharyngolaryngoscopy (FNPLS) revealed no bulging of the lateral or posterior pharyngeal wall. However, his bilateral vocal cord abductors were immobile in a median position with an almost slit like opening. His epiglottis, arytenoids and bilateral false cords were oedematous with an obvious pooling of secretion at both pyriform fossae. There was no clinical or radiological evidence of tumour recurrence. He was diagnosed with bilateral vocal cord palsy with supraglottitis. An emergency tracheostomy and subsequent direct laryngoscopy were performed to secure his airway. Throughout his hospital stay, he also suffered from other conditions such as community acquired pneumonia, Forrest III ulcer at the pylorus, type 2 myocardial infarction and pulmonary embolism. He was successfully treated for those medical conditions and discharged after a period of 12 days. Radiotherapy induced bilateral vocal cord palsy is not a common finding and can be life threatening if left untreated. Knowledge regarding this condition will aid in diagnosing and treating patients early, producing a better outcome.

INTRODUCTION

Radiation therapy is one of the most effective and suitable therapies available for head and neck cancer. With early laryngeal cancer especially those in stage I and stage II being primarily a local disease, radiotherapy is the principal treatment modality to preserve the larynx. Only more advanced laryngeal cancers are treated via a combination of surgery and radiotherapy or chemotherapy. Radiotherapy itself comes with its fair share of complications and has many documented side effects, including mucositis, skin reactions, decreased salivary flow, soft tissue fibrosis, osteoradionecrosis and perichondritis. Most of the complications occur early but some may occur years after radiation therapy. Radiotherapy-induced peripheral nerve palsies have been reported although quite rarely, usually after a latent period of 1-5 years. There are two mechanisms that explain these injuries, with the first being radiation-induced scarring along the course of the recurrent laryngeal nerve, and the second being vascular insult with ischemia and obliteration of small capillaries leading to degenerative changes.

CASE PRESENTATION

A 73-year-old Chinese man with a previous history of laryngeal cancer treated with radiotherapy presented to the emergency department with the chief complaint of noisy breathing for the past 5 days, associated with sore throat, odynophagia, hoarseness, occasional low-grade fever, and shortness of breath. The emergency department team started the patient on nasal prong oxygen at 3l/minute and administered intravenous dexamethasone 8mg and nebulised adrenaline, before referring the patient to both the otorhinolaryngology, and anaesthetic team respectively.

Further history from the patient revealed that he had underlying diabetes mellitus and hypertension for more than 10 years with no proper follow-up, purchasing oral hypoglycaemic agents and anti-hypertensive medications from a private pharmacy. He was also previously diagnosed with glottic carcinoma (T1NoMo) in 2018 and was treated via radical 3D radiotherapy to the larynx amounting to 55 Grays in 20 fractions. He was under regular follow-up with the hospital until the covid pandemic struck. Prior to the pandemic, his findings during his follow-ups were unremarkable. He otherwise has no known allergies.

On examination, the patient had a full GCS with stridor, tachycardia and tachypnoea. There was a predominant breathiness quality in his voice. He was unable to complete sentences. His vital signs were, blood pressure:156/98 mmHg, pulse: 120 beats per minute, respiratory rate: 40 times per minute, temperature: 36.5 Celsius and oxygen saturation: 98% under nasal prong. Auscultation revealed equal air entry bilaterally with transmitted sounds. A chest radiograph was done, which revealed bilateral lower zone haziness. At the same time his white cell count was 27.2 (normal value: 4.0-10.0) and C-reactive protein was 84.33 (normal value: <5.00), suggestive of community acquired pneumonia (CAP), and he was administered intravenous ceftriaxone 2gm per day.

Bedside flexible nasopharyngolaryngoscopy (FNPLS) was done, and the summarised findings are as follows: epiglottis,

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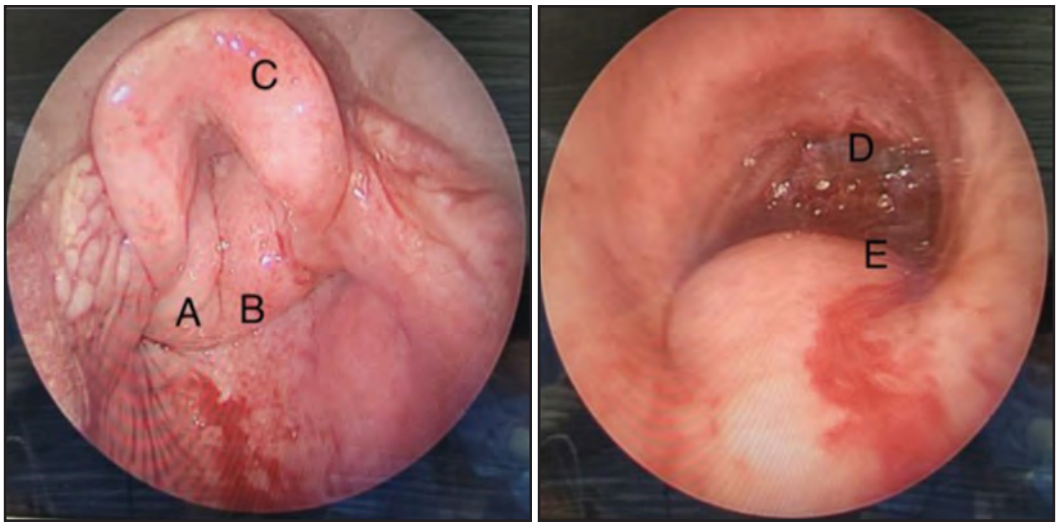

Fig. 1: The direct laryngoscopy findings intraoperatively; A: Left arytenoid, B: Right arytenoid, C: Epiglottis, D: Tracheostomy tube, E: Posterior tracheostomy wall

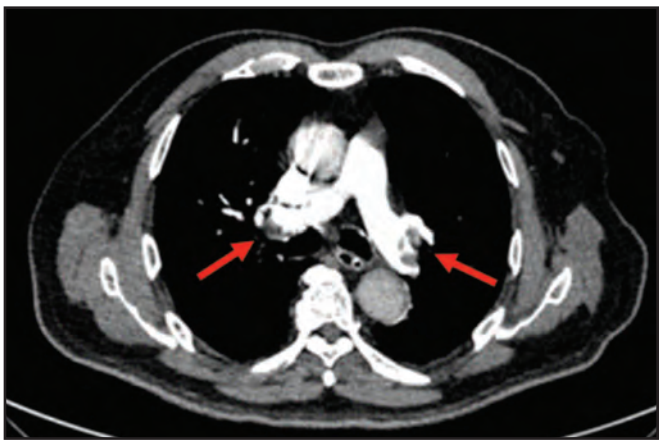

Fig. 2: CTPA showing filling defect in bilateral pulmonary arteries (indicated by red arrows)

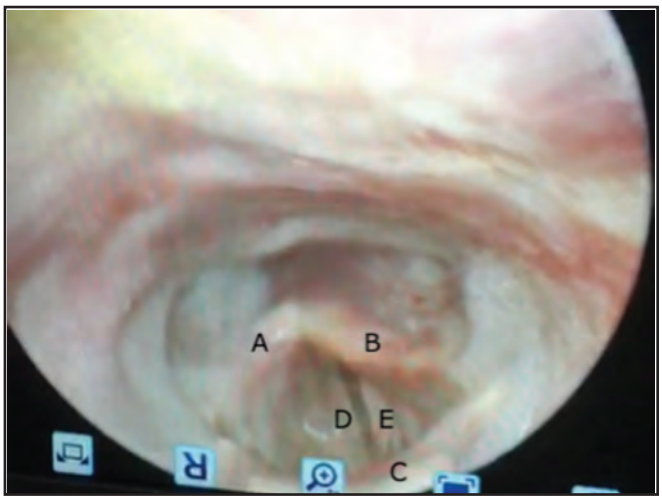

Fig. 3: Bilateral abductor palsy with slit like airway still seen during review of the patient; A: Right arytenoid, B: Left arytenoid, C: Epiglottis, D: Right vocal cord, E: Left vocal cord

arytenoids and bilateral false cords were oedematous; secretion pooling at both pyriform fossae; bilateral vocal cords abductor palsy; and vocal cords in median position with an almost slit like opening. There was no narrowing of the lateral or posterior pharyngeal wall, and no masses were noted. Our provisional diagnosis for the patient was bilateral vocal cord palsy secondary to radiotherapy with supraglottitis. The rest of his physical examination was unremarkable.

The patient was then immediately taken to the operation theatre whereby he was intubated via awake fibreoptic nasal intubation and given a STAT dose of IV cefuroxime 1.5gm. We then proceeded with an emergency tracheostomy to secure his airway, and a direct laryngoscopy examination under anaesthesia to look for any masses or lesions. Direct laryngoscopy findings were unremarkable, there was no clear mass on inspection of the laryngeal subsites, the visualised subglottis was normal until the carina, and cricoarytenoid joints were palpated with no evidence of fixation or subluxation. Post procedure, he was transferred to the intensive care unit (ICU). Despite being a popular treatment choice for the management of bilateral vocal cord palsy, posterior laser cordectomy was not offered for this patient due to his multiple comorbidities and underlying medical issues.

Post emergency tracheostomy, the patient had coffee ground secretion from his nasogastric tube. He was then diagnosed with a small Forrest III ulcer at the pylorus following an esophagogastroduodenoscopy (OGDS). Hence, he was started on a proton pump inhibitor, intravenous pantoprazole 40mg daily.

He was also diagnosed with type 2 myocardial infarction and pulmonary embolism over the next few days of his admission. His computed tomography pulmonary

angiogram (CTPA) showed filling defects involving bilateral main pulmonary arteries extending to their segmental and subsegmental branches. He was treated with a combination of clopidogrel and enoxaparin for his condition.

After 12 days of hospital admission, he was allowed to be discharged home. A bedside FNPLS showed that the supraglottitis had resolved however his vocal cord mobility remained the same, with bilateral true cords in median position with no abduction. He was also scheduled for an outpatient computed tomography (CT) brain to upper thorax to assess for any radiologic evidence of recurrence and to assess the course of both recurrent laryngeal nerves. The findings of the CT scan revealed no evidence of any recurrence at the larynx and the course of both the recurrent laryngeal nerves were not impinged by any lesion or mass. The findings in the brain revealed old multifocal infarcts but no evidence of any lesion. Two weeks post discharge his FNPLS findings remained unchanged.

DISCUSSION

The larynx is an important structure that is involved in phonation, respiration, and deglutition. It is innervated by two branches of the vagus nerve, which are the recurrent laryngeal nerve and the superior laryngeal nerve. The recurrent laryngeal nerve innervates four of the intrinsic laryngeal muscles: thyroarytenoid, posterior cricoarytenoid, lateral cricoarytenoid, and interarytenoid muscles. Injury to any one of these nerves may lead to vocal fold paresis or paralysis. The difference between the two is that vocal fold paresis implies various degrees of vocal fold hypomobility due to neurological injury or from weakness of the nerves, whereas vocal fold paralysis implies complete vocal fold immobility due to the neurological injury.[1] Since the vocal fold is crucial for survival, its impairment can cause complications such as dysphagia or aspiration. When bilateral vocal cords are paralysed, airway obstruction becomes the cause for concern.[2] When this happens, securing the airway via a tracheostomy or intubation is of utmost importance.

There are many different aetiologies of vocal fold paralysis. The most common causative factor is due to surgery, and data from previous studies demonstrate between 25 to 58% of cases are linked to surgery, notably thyroidectomy. Neoplasia is the second most common aetiology, ranging from 7 to 17% of cases.[2] A common cause of vocal fold paralysis which is avoidable is from intubation, which contributes between 2 to 18% of cases. Radiation induced neuropathies in head and neck cancer is a rare complication, contributing to only 1 to 9% of cases.[3] The time lag on the other hand can be up to 35 years, and the most commonly affected cranial nerves are the vagus, trigeminal, spinal accessory, oculomotor, abducens, optic, and hypoglossal.[4] Other causes include neuromuscular disease, viral infections such as coronavirus disease, autoimmune disease and toxicity.

We were able to rule out coronavirus as a potential cause because the patient's voice was normal prior to the event and his covid polymerase chain reaction (PCR) results were negative. As neuromuscular disease can also be one of the causes of bilateral vocal cord palsy, peripheral nerve conduction and an MRI brain would help to rule out neuromuscular causes. However, those tests were not performed as a full neurologic examination was carried out and no abnormalities were detected. At the same time the patient also did not complain of any neurologic symptoms. Another helpful examination would be the utilisation of a laryngeal electromyography (LEMG) unit, which records the electrical activity produced by the laryngeal muscles and gives specific information as to whether the nerve input into a particular muscle is normal or abnormal. This will allow us to differentiate whether the aetiology is due to neural injury or from cricoarytenoid joint ankylosis. We however do not possess an LEMG unit in our centre. Hence, despite the CT scan showing no abnormality over the region of the cricoarytenoid joint, there does remain a distinct possibility that this may be related to a neuromuscular disease.

A literature search associating radiotherapy and delayed vocal fold paralysis produced several results. In 1995, Stern et al reported three cases of vocal fold palsy 21 to-34 years after radiotherapy,[4] whereas Lin et al mentioned that six out of 19 subjects who developed radiation induced cranial nerve neuropathies suffered from recurrent laryngeal nerve palsy, with a latency period that extended up to 20 years.[5] Prepageran in 2005 also reported a case of bilateral vocal cord immobility 15 years after head and neck radiotherapy in a patient with laryngeal carcinoma.[6] In 2012, Jaruchinda et al reported a rate of 7.14% of vocal fold paralysis in a group of 70 people who underwent head and neck radiotherapy, with the time lag varying between 14 to 35 years.[7] A newer study in 2015 by Crawley and Sulica reported 10 cases of vocal fold paralysis causing dysphonia and dysphagia, with an onset of paralysis between 1 and 27 years after irradiation therapy.[8]

Unilateral vocal cord palsy is more commonly seen on the left side due to the longer course of the left recurrent laryngeal nerve. The paralysed vocal cord is unable to adduct and abduct, leading to glottic incompetence. Even though the contralateral unaffected vocal cord can abduct, it is usually insufficient to produce normal phonation oscillation bilaterally. Therefore, patients with unilateral vocal cord palsy often present with dysphonia such as hoarseness, vocal fatigue, weak or breathy voice and sometimes even cough or aspiration. Bilateral vocal cord palsy on the contrary presents with normal or near normal phonation with respiratory distress, spanning from mild stridor upon exertion to life threatening airway obstruction necessitating emergency tracheostomy.[9] Our patient presented with a life-threatening shortness of breath due to bilateral vocal fold immobility with superimposed infection (supraglottitis) 4 years after completion of radiotherapy to the larynx. Since he missed his follow-ups during the covid pandemic, it is difficult to determine if there was one vocal cord that had been affected before the other. At the same time, antibiotics were necessary not only to treat his pneumonia but also his supraglottitis. Unfortunately, during his hospital stint, he developed other acute life-threatening conditions such as community acquired pneumonia, Forrest III ulceration, type 2 myocardial infarction and pulmonary embolism. These conditions are usually associated with physiologic stress,

which was most probably triggered by his acute upper airway obstruction. However, despite suffering from various other medical problems throughout his stay, our focus in this case report is on the rare complication of delayed vocal fold paralysis following radiotherapy.

CONCLUSION

As radiotherapy can potentially cause laryngeal neuropathy years after treatment, patients who have undergone radiotherapy are encouraged to seek immediate consultation at their nearest otorhinolaryngologist if they notice any changes to their voice or experience any form of breathing difficulty. This is because although neuropathies secondary to radiation are relatively rare, they are usually permanent and can significantly affect patients' quality of life with minimal potential for spontaneous recovery. Therefore, as clinicians, it is crucial that these patients are followed up regularly on a lifelong basis so that we can identify, detect, and treat this condition early.

CONSENT

Written informed consent was taken from the patient from the start of managing the case for publication.

CONFLICT OF INTEREST

There was no conflict of interest.

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