Case Report

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Spasmodic dysphonia presenting in a post trauma tracheostomised patient with inadequate laryngeal muscle relaxation

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ABSTRACT

Spasmodic Dysphonia is a chronic long-term voice disorder, with a very rare incidence of 1 per 100,000 cases in which the movement of vocal cords is both forced and strained resulting in hoarse, quivery and jerky voice. We present a very rare and interesting case of spasmodic Dysphonia that was adequately relaxed after administration of muscle relaxants but had undue contractions of adductor group of muscles at the level of vocal cords. Little is known about the genetic basis of the disease but symptoms improve when the kinetic output of the laryngeal muscles is reduced either by unilateral recurrent laryngeal nerve section, or by botulinum injections into the affected muscles.

Key words: Inadequate Laryngeal Muscle Relaxation, Post Tracheostomy, Spasmodic Dysphonia

INTRODUCTION

Spasmodic Dysphonia is a type of voice disorder which results from involuntary movement of laryngeal group of muscles. Anaesthesia literature provides information and recommendation for providing Anaesthesia to patients with variety of neuromuscular, myotonic and vocal cord disorders. However little is known in the literature regarding the Anaesthesia management of speech disorders like Spasmodic Dysphonia and how these disorders can affect the Anaesthesia management. We present a very rare and interesting case of Spasmodic Dysphonia that was adequately relaxed after administration of muscle relaxants but had undue contractions of adductor group of muscles at the level of vocal cords.

CASE REPORT

A 54 year old male was posted for tracheostomy T tube insertion under general Anaesthesia for long term maintenance. Three weeks before the postulated surgery he was brought to emergency with difficulty in breathing and acute stridor, for which he was tracheostomised. On eliciting the history it was found that the patient had trauma to the anterior neck six months back and was tracheostomised at another hospital and was admitted in Intensive Care Unit. His tracheostomy was de-cannulated after 20 days and patient was discharged from the hospital after one month. The patient remained asymptomatic for few months and was admitted and re-tracheostomised in view of stridor. After shifting the patient to operation theatre, intravenous (IV) access was secured and standard monitors were attached. Patient was pre-oxygenated with 100% oxygen through the Bain's circuit connected to the tracheostomy port and capnometer sampler attached. Patient was administered ranitidine 50 mg, metoclopramide 10 mg, midazolam 1 mg and fentanyl 100 mcg slowly intravenously. Induction of Anaesthesia was done with injpropofol 1.5 mg/kg and inhalation of 3–4% sevoflurane and muscle relaxation was achieved with inj Atracurium 0.5 mg/kg.

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During the performance of direct laryngoscopy, surgeons repeatedly reported a problem with inadequate relaxation of vocal cords. It was revealed that patient had undue contractions of adductor muscle of vocal cord even when he was not breathing as was observed from the movements of the reservoir bag, capnography analysis, and train of four count of 0. Even after increasing the minimum alveolar concentration of sevoflurane to 4% and one more dose of muscle relaxant, surgeons had difficulty inserting tracheal endoscope and complained frequently of inadequate muscle relaxation at the level of vocal cords. Same problem was encountered when tracheostomy T tube was attempted one week prior in the same patient and abandoned because patient was not relaxed with respect to vocal cords. Patient was extubated uneventfully, and 70° endoscopy was done by surgeons to look for etiology. The cords seemed to abduct although not completely, and possibilities of Myasthenia laryngeus/Spasmodic dysphonia were considered as differentials.

On completion of the endoscopy procedure, the patient was reversed (without putting t tube so as to evaluate the patient further) with adequate respiratory efforts. After 1 week of investigations and HD laryngoscopy, diagnosis of Spasmodic Dysphonia was made and he was given botox injections for the same.

DISCUSSION

Spasmodic Dysphonia (SD) is a poorly understood voice disorder in the age group 30-50 years, more common in females than males in which the movement of vocal cords is forced and strained resulting in hoarse, quivery and jerky voice. Spasmodic Dysphonia is rare; some estimates are as low as 1 per 100,000 cases, it is not clear whether the disorder has a genetic basis; most cases are sporadic or secondary to an unknown neurobiological mechanism that produces a chronic abnormality of laryngeal motor neuron regulation during speech[1]. There are vocal interruptions or spasms, periods of no sound (aphonia), and periods when there is near normal voice occurs. Symptoms may be worse when a person is tired or stressed. They may be greatly reduced or even disappear, for example, during singing or laughing.

To the best of our knowledge, we could not trace any evidence of not able to relax fully the adductor group of vocal cord muscles despite the fact that other muscles of the body were paralyzed under the effect of muscle relaxant. The uniqueness of our case encouraged us to share our interesting and rare case of Spasmodic Dysphonia in post trauma tracheostomised patient in which vocal cord group of muscles may be not be relaxed fully with the usual muscle relaxant dosage rather required a higher dosage for the same.

There is only few published case reports in literature related to a case of Spasmodic Dysponia. In the case reports, one patient had recurrent stridor[2] and post extubation laryngospasm in a diagnosed case of spasmodic dysphonia[3]. Treatment approaches in a case of Spasmodic Dysphonia aims around at reducing the kinetic output of laryngeal muscle by either recurrent laryngeal nerve section or by botulinum injections into the laryngeal muscles. Few more approaches recommend avulsing of a long section of the recurrent nerve to prevent re-innervation or bilaterally section and re-innervate the nerve branch going to the thyroarytenoid muscle to the ansacervicalisso as to prevent reinnervation by the recurrent laryngeal nerve. In these treatments, a balance must be achieved between adequately reducing vocal fold hyper adduction while not producing aspiration during swallowing or aphonic speech. These treatment approaches interfere with muscle action rather than blocking abnormal interneuron firing patterns in the laryngeal efferent pathway. There is paucity of literature with respect to Anaesthesia management of cases with Spasmodic Dysphonia, but there are few published case reports suggesting the occurrence of post-operative stridor/laryngospasm in these patients. We present first of its case report, in which we encountered resistance to non-depolarizing blockade at the level of vocal cords.

CONCLUSION

Spasmodic Dysphonia although a very rare entity, can occur in a case of prolonged tracheostomised patient, exact pathophysiology of the disorder is still unknown according to literature. There have been cases of post extubation laryngospasm in such patients, however we encountered resistance to muscle relaxants only at the level of vocal cord muscles and no such resistance in other parts of body was seen. However further studies both at the clinical and genetic level are required to actually assess and know the etiology of the same.

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