Laryngospasm secondary to a multinodular goitre

Mohd Sazafi MOHD SAAD [1], Esa KAMARUZAMAN [2], Mazita AMI [1]
[1] Department of Otorhinolaryngology-Head and Neck Surgery, Faculty of Medicine, Universiti Kebangsaan Malaysia, [2] Department of Anaesthesiology & Critical Care, Faculty of Medicine, Universiti Kebangsaan Malaysia, Malaysia.

ABSTRACT

Laryngospasm is a serious event that can result in partial or complete upper airway obstruction. It is a common complication during recovery phase of anaesthesia, resulting from acute irritation of the vocal folds. We report a rare case of laryngospasm secondary to a multinodular goitre that settled after treatment. We postulate that the enlarged thyroid gland can lead to direct irritation of the laryngeal nerve as possible cause of repeated episodes of laryngospasm in this patient. To our knowledge, this is the first reported case of a multinodular goitre as the underlying cause of laryngospasm.

Keywords: Airway obstruction, laryngospasm, multinodular goitre

INTRODUCTION

Laryngospasm is a serious event that can result in partial or complete upper airway obstruction. [1] It is a frequent complication during the recovery phase of anaesthesia as a result of irritation of the vocal folds. [2] Patients frequently describe a sudden onset of difficulty in breathing with stridor. These episodes are brief and typically resolve within a few minutes. However, they can be extremely distressing to the patients. Some patients may also lose consciousness during these episodes. Fortunately, loss of consciousness may lead to the relaxation of the larynx as a result relieving the airway obstruction. The underlying pathological abnormality is believed to be increased sensitivity of the laryngeal mucosa which can be exacerbated by laryngopharyngeal reflux of gastric content. [3] Other explanations for the hypersensitivity of the larynx leading to the development of this mal-adapted reflex arc are focal epileptic activity, neural instability, and aberrant re-innervations following neuropraxia. [3] The diagnosis is based primarily on history and physical examination. However, examination is usually unremarkable between episodes except for possible presence of supra-glottic

Correspondence author: MS MOHD SAAD
Department of Otorhinolaryngology-Head & Neck Surgery, Universiti Kebangsaan Malaysia Medical Centre, Bandar Tun Razak 56000, Cheras, Kuala Lumpur, Malaysia
Tel: +60192644427 Fax: +60391456675
E mail: drsazafi@yahoo.com

inflammation and inter-arythenoid oedema.

CASE REPORT

A 57-year-old lady presented with sudden onset of dyspnoea and chest pain that was followed by lost of consciousness at home. She had a two-day history of low grade fever and sore throat. Her past medical history was relevant for hypertension, type 2 diabetes mellitus and a multi-nodular goitre. The multinodular goitre was diagnosed few years previously but she had declined any surgical treatment. She was a non-smoker and denied any history of drug allergies or asthma. Upon presentation at the emergency department, her Glasgow Coma Scale was 3/15 and she was immediately intubated. Laboratory biochemistry and full blood count results were within normal limits. Arterial blood gas analysis showed mild respiratory acidosis with a pH of 7.153 (normal range 7.34 to 7.44). Chest radiography showed cardiomegaly and left lower lobe haziness consistent with pneumonia. Electrocardiography showed evolving ischaemic changes. Therefore, the diagnoses of acute coronary syndrome and community acquired pneumonia were made.

Following treatment, her condition improved and she was extubated later during the following day. However a couple of hours post-extubation, she became increasingly tachypnoeic and hypoxic and subsequently became unresponsive. She was then re-intubated and transferred to the intensive care unit for further management. A flexible nasopharyngolaryngoscopy was performed and this revealed minimal oedema of the vocal cords and supraglottis which was attributed to repeated intubations. A flexible bronchoscopy through the endotracheal tube showed a normal trachea and main bronchi with no tracheomalacia. A computed tomographic (CT) scan of neck revealed minimal retrosternal extension of the multinodular goitre but no airway compression (Figure 1a).

Extubation was again attempted a few days later after a course of intravenous steroids to reduce the oedema. However, this failed as a result of respiratory distress. Subsequently, a direct laryngoscopy and tracheobronchoscopy was repeated under general anaesthesia and these were again normal (Figure 1b). A diagnosis of laryngospasm was made. She was extubated in the operating

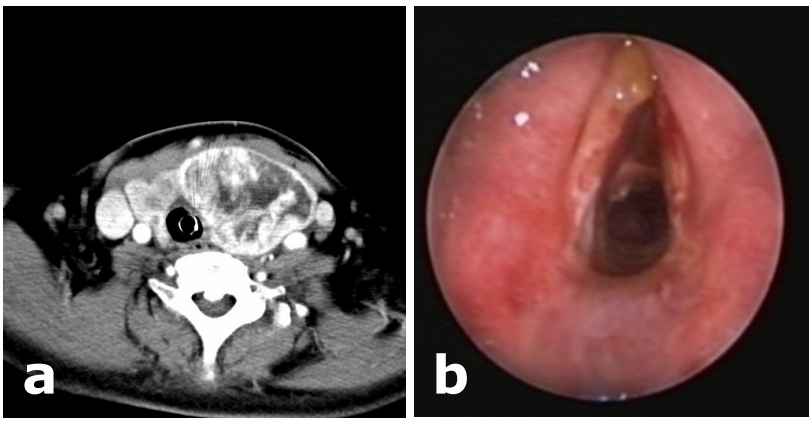

Fig. 1: a) Axial computed tomographic image of the neck showing a multinodular goitre with minimal retrosternal extension and no compression on to the tracheal wall (arrow: endotracheal tube seen within the tracheal lumen), b) Endoscopic image of the larynx showing oedematous vocal cords with minimal slough at the subglottis due to intubation.

room and placed on non-invasive ventilation. Investigations to ascertain the underlying cause for laryngospasm that include thyroid and electrolytes including calcium profiles were within normal limits. A skin prick test showed no possible allergens and a 24 hour double probe pH study was also normal.

The patient was transferred to the general ward two days later after no further episodes of respiratory distress. However in the ward, patient developed another episode of acute respiratory distress and lost of consciousness and needed intubation. Based on previous normal findings and investigations, the patient was counseled for thyroidectomy in view of the multinodular goitre being the possible cause for repeated episodes of laryngospasm. She underwent a total thyroidectomy and tracheostomy. Intraoperative findings revealed an enlarged thyroid gland mainly of the left lobe with no external compression of tracheal wall down until the carina. There was no evidence of tracheomalacia. Histopathological examination showed multinodular goitre with no malignancy. She was weaned off the ventilator the following day and made rapid post-operative recovery. The tracheostomy was successfully decannulated a week later. Since the operation there has been no further episode of laryngospasm after follow-up of seven months.

DISCUSSION

Laryngospasm is an abrupt glottis closure resulting from reflex constriction of the intrinsic laryngeal muscles, resulting in sudden gasping for breath and wheezing. It consists of prolonged glottis closure reflex mediated by the superior laryngeal nerve. [4] If not treated immediately, patient may present with upper airway obstruction and positive pressure ventilation of the lung will be impossible. This will subsequently lead to hypercarbia, hypoxia, cardiac collapse, and death. Laryngospasm is also one of the known complications seen in the peri-operative period especially during induction of anaesthesia or during extubation. Olson and Hallen studied the incidence of laryngospasm in 136,929 patients over an 11 years period and found an overall incidence of laryngospasm of 8.6 per 1,000 in the adult patients with higher incidence of 27.6 per 1,000 in children. [5]

Common initiating factors are hyperactive airway like in case of upper respiratory tract infection. Other common triggering factors are painful stimulation, primary vagal hypertonicity, insufficient depth of anaesthesia on endotracheal intubation or combination of either preceding with or without some irritant such as blood, mucus, laryngoscope blade, suction catheter, surgical debris or other foreign body. [4] It usually manifests with stridor, tachypnoea, tachycardia, increase in pharyngeal secretions, phonetic inability or no airflow despite ventilatory effort. Standard management of laryngospasm in peri-operative period includes giving continuous positive airway pressure with 100% oxygen, removal of trigger factor (i.e. secretion, blood etc), low dose depolarising muscle relaxant (suxamethonium 0.3 mg/kg) to relieve muscle spasm and inevitably intubation if all standard therapy fails. Other than mechanical stimulation, episodic laryngospasm is often associated with gastro-oesophageal reflux disease. [6] However in our patient, the 24-hour pH study was normal.

Some studies hava also suggested

that laryngospasm can be due to laryngeal stimulation from a focal seizure due to irritation to the brain. Under certain conditions, a sensory stimulus originating from the larynx or a more distant structure can trigger prolonged motor activity in the intrinsic laryngeal muscle. Clinically, laryngospasm produce strong glottic closure that is maintained well beyond the length of the stimulus, just as focal cerebral stimulus can generalise into grand mal seizure. [7]

The repeated laryngospasm after a series of extubations, normal endolaryngeal findings and improvement in symptoms since thyroidectomy seem to suggest that the observed episodes of laryngospasm in our patient may be related to nerve irritation by the multinodular goitre. We postulate that compression of the laryngeal nerves by the goitre may have resulted in the irritation of the nerve. A previous study had shown that stimulation of the superior laryngeal nerve can produce low threshold evoked potentials in the adductor fibres of recurrent laryngeal nerve. [7] To our knowledge, this is the first case describing multinodular goitre as the underlying cause of laryngospasm in the literature.

In conclusion, laryngospasm is a serious event that can lead to a life threatening situation. Awareness of the various factors that can precipitate laryngospasm is paramount in the management of patients with recurrent laryngospasms. This case highlighted that an enlarged thyroid gland can induce laryngospasm probably through irritation of the laryngeal nerve.

REFERENCES

1: Donlon JV. Anaesthesia for eye, ear, nose and throat surgery. Churchill Livingstone, Philadelphia; 2000: 2183.

2: Visvanathan T, Kluger MT, Webb RK, Westhorpe RN. Crisis management during anaesthesia: laryngospasm. Qual Saf Health Care 2005; 14:e3.

3: Maceri DR, Zim S. laryngospasm: An atypical manifestation of severe gastroesophageal relux disease (GERD). Laryngoscope 2001; 111:1976-9.

4: Mevorach DL. The management and treatment of recurrent postoperative laryngospasm. Anaesth Analg 1996; 83:1110-1.

5: Olsson GL, Hallen B. Laryngospasm during anaesthesia. A computer –aided incidence study in 136,929 patients. Act Anaesthesiol Scand 1984; 28:567-75.

6: Loughlin CJ, Koufman JA. Paroxysmal laryngospasm secondary to gastroesophageal reflux. Laryngoscope 1999; 109:1503-5.

7: Sasaki M, Sasaki CT. Laryngeal spasm: A neurophysiologic re-definition. Ann Otol Rhinol Laryngol 1977; 86:150-7.