Case Report

Acute airway obstruction, an unusual presentation of vallecular cyst

Address for correspondence:
Dr. Sameer M Jahagirdar,
A 14, Green Avenue Apartment, Point Care Street, Mudaliyarpet,
Puducherry - 605 004, India.
E-mail: dr.sameerjahagirdar786@gmail.com

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Sameer M Jahagirdar, P Karthikeyan[1], Ravishankar M

Department of Anaesthesiology & Critical Care Medicine, and [1]ENT, Mahatma Gandhi Medical College and Research Institute, Puducherry, India

ABSTRACT

A 18-year-old female presented to us with acute respiratory obstruction, unconsciousness, severe respiratory acidosis, and impending cardiac arrest. The emergency measures to secure the airway included intubation with a 3.5-mm endotracheal tube and railroading of a 6.5-mm endotracheal tube over a suction catheter. Video laryngoscopy done after successful resuscitation showed an inflamed swollen epiglottis with a swelling in the left vallecular region, which proved to be a vallecular cyst. Marsupialisation surgery was performed on the 8th post admission day and the patient discharged on 10th day without any neurological deficit.

Key words: Acute supraglottitis, airway management, vallecular cyst

INTRODUCTION

Acute adult supraglottitis is an inflammatory disease of the epiglottis and adjacent structures. It can be rapidly fatal because of the potential for sudden airway obstruction. Early recognition and prompt airway management is of utmost importance to reduce morbidity and mortality.

CASE REPORT

An 18-year-old female, residing 25 km from the hospital, was brought to the emergency department in an unconscious state. History, as given by her mother, included low-grade fever, a small swelling in the neck, and throat pain for 3 days; difficulty in swallowing since 1 day; and difficulty in breathing since the previous evening. She had been on treatment at the local hospital and was referred to us when she developed difficulty in breathing; she had lost consciousness while traveling to our hospital.

On examination in the emergency department, the patient had severe stridor, was unresponsive to calls, and was sweating profusely. Her temperature was 99°F and heart rate 160/minute. The carotid, radial, and other peripheral pulses were not palpable. She had a diffuse swelling over the anterior aspect of her neck. Pulse oximetry showed oxygen saturation <50%. Oxygen by mask was started and an intravenous (IV) line was secured and 500 ml of Ringer's lactate solution was given. An arterial blood sample was taken for arterial blood gas (ABG) analysis. The anaesthetist was called to manage the airway problem. Direct laryngoscopy revealed a swollen epiglottis obstructing the glottic aperture and preventing visualization of the glottis. While probing the area under the swollen epiglottis with a 3.5-mm ID endotracheal tube, it smoothly passed inside at one point. The tube was confirmed to be in the trachea as there was partial ventilation of the lungs and marginal improvement of oxygenation. The length and diameter of the endotracheal tube was not sufficient to permit an Ambu bag to be connected so that the patient could be ventilated. Therefore, an 8 FG suction catheter (with the connector cut off) was first introduced through the 3.5-mm ID endotracheal tube [Figure 1], the endotracheal tube was removed over the catheter and, finally, a 6.5-mm ID cuffed portex endotracheal tube was railroaded over the suction catheter under direct visualization with the

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laryngoscope. The suction catheter was removed and patient was ventilated with oxygen-enriched air using an Ambu bag. Visible chest expansion on ventilation and bilateral breath sounds on auscultation confirmed that the endotracheal tube was correctly positioned in the trachea.

ABG analysis of the sample taken before intubation revealed pH 6.79, pO_2 94 mm Hg, pCO_2 188 mm Hg, and HCO_3 27 mmol/L; the values indicating severe acute respiratory acidosis.

After 15–20 minutes of ventilation, the patient regained consciousness. She was transferred to the intensive care unit (ICU) and connected to a ventilator in pressure-control mode, with inspiratory pressure 20 cm H_2O, PEEP 4, respiratory rate 15/minutes, and inspired O_2 concentration of 50%. Two hours later, on an FiO_2 of 50%, ABG analysis showed pH 7.39, pO_2 72 mm Hg, pCO_2 37 mm Hg, and HCO_3 22 mEq/L. Our provisional diagnosis was acute epiglottitis with negative-pressure pulmonary oedema. Ventilation was continued. We also gave IV (intravenous) ceftriaxone 2 gm/day, IV metronidazole 1 gm/day, and IV dexamethasone 4 mg 6th hourly.

The patient was stabilized over the next 6 hours. The examination under general anaesthesia using 70° nasendoscope revealed oedema of the epiglottis on the left side, of the left aryepiglottic fold, and of the left arytenoids; slough was seen in the vallecular region [Figure 2a and b]. The endotracheal tube was maintained in place and mechanical ventilation was continued for one more day. Repeat video laryngoscopy the next day showed reduction in the oedema of the arytenoids and aryepiglottic folds and the epiglottis on the left; a swelling was noticed on the left side in the vallecular fossa. Glottic opening was normal. The patient was extubated on the operation table after oropharyngeal suctioning. Antibiotics were continued and dexamethasone was tapered and stopped on day 4. As the swelling in the anterior aspect of the neck persisted, computed tomography (CT) of the neck was performed. This revealed a vallecular cyst [Figure 3].

On the 8th day of hospital admission, marsupialisation of the vallecular cyst was done under general anaesthesia. The intraoperative course was unremarkable. The cyst wall and fluid was sent for pathological examination. Culture revealed no growth. The patient was discharged on the 10th day. At discharge she did not have any neurological deficit.

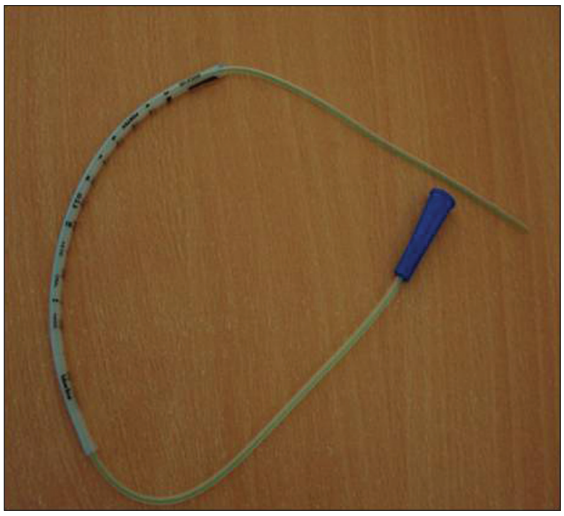

Figure 1: An 8 FG suction catheter with 3.5-mm ID endotracheal tube

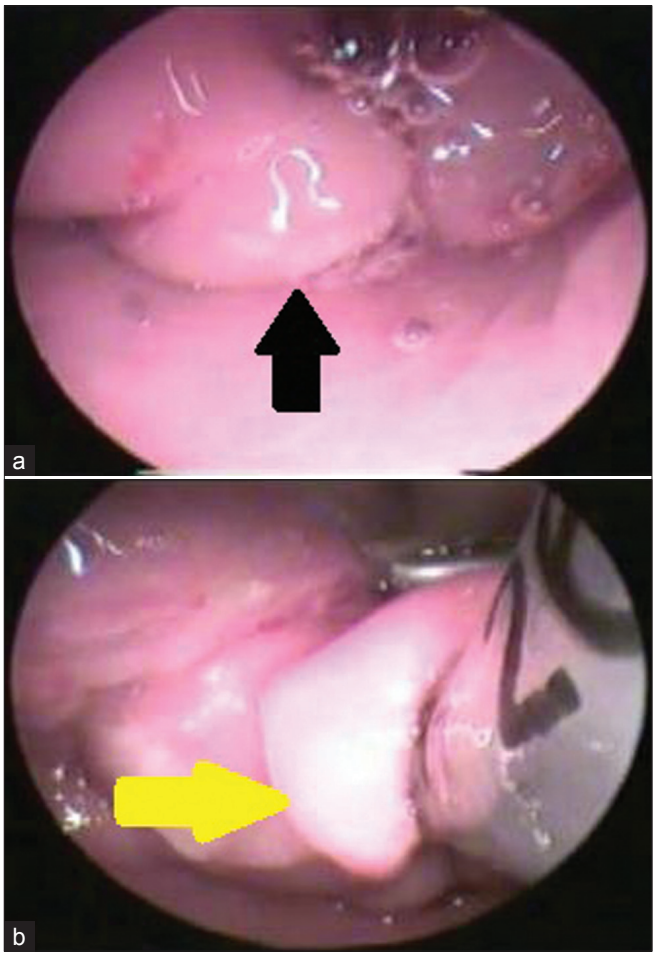

Figure 2: Oedema of (a) left arytenoids and left aryepiglottic fold (arrow) (b) Oedema of lateral part of epiglottis (yellow arrow)

DISCUSSION

Vallecular cysts are the ductal variety of laryngeal cysts. They are caused by obstruction of the submucous duct. As the vallecular space is full of lymphoid and

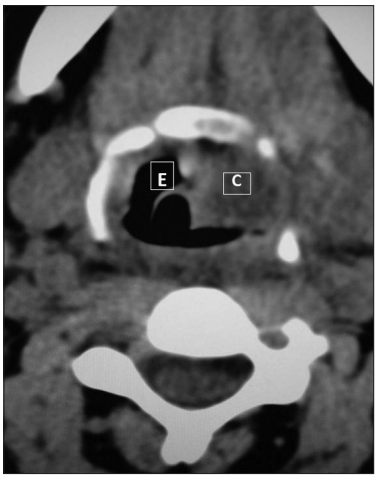

Figure 3: CT image showing displaced epiglottis (E), with the vallecular cyst (C)

glandular tissue which is easily obstructed, vallecular cysts in adults are not rare.[1] Although they are generally asymptomatic, they may occasionally cause stridor, cough, dysphonia, foreign body sensation, and dysphagia. Infection of the cyst may spread to the surrounding structures and cause oedema and inflammation.[2] Early management with antibiotics usually prevents complications.

Acute supraglottitis is an acute inflammation of the supraglottic region of the oropharynx, epiglottis, vallecula, arytenoids, and aryepiglottic folds.[3] Infection with *Haemophilus influenzae* or group A streptococcus is the most common cause in adults. Other etiological agents are bacteria such as pneumococcus species and bacteroides; herpes simplex virus; Candida species; and physical agents such as direct trauma, thermal, and caustic injury.[3,4]

Although acute supraglottitis is primarily a disease of the paediatric age-group, it is being increasingly seen in adults now, probably because of widespread immunization of children against *H. influenzae*. The most consistent presenting symptoms of acute supraglottitis are sore throat and odynophagia. Drooling of saliva and stridor can be the presenting signs of epiglottitis. Other symptoms and signs include hoarse or muffled voice, tenderness over the hyoid bone, cervical adenopathy, fever, cough, and ear pain.[5]

The diagnosis of supraglottitis is essentially clinical and can be supported by indirect, direct, or flexible laryngoscopy. White blood cell count elevation has been reported in 65%–90% of cases, with an average value of 15×10^9/L.[6] Typically, in adults, there is diffuse swelling of the aryepiglottic folds, unlike the classical cherry red epiglottis seen in children. Once the airway has been secured, a lateral soft tissue radiograph can be taken and will show thickening of the epiglottis ('thumbprint sign') and decrease in the vallecular air space as the epiglottis swells and extends anteriorly ('vallecula sign').[7] In the absence of a deep and well-defined vallecula on a soft tissue radiograph supports the, diagnosis of epiglottitis.

Our patient had symptoms of sore throat for 3 days and dysphagia for 1 day. She was treated in a local hospital with oral antibiotics and referred to us when she developed difficulty in breathing. She lost consciousness while being transported to our hospital and arrived in a state of impending cardiac arrest. ABG analysis was indicative of severe respiratory acidosis and loss of consciousness due to CO_2 narcosis. Any further delay in airway management would have been disastrous.

Patients with severe respiratory distress or near total obstruction at the time of presentation should have an airway established immediately. Such patients will be agitated and unlikely to be cooperative during laryngoscopy and oral intubation. They will not be able to lie down supine for a tracheostomy either. Needle cricothyrotomy or emergency cricothyrotomy with a trocar is the immediate management choice. Once oxygenation and air exchange take place the patient will quieten down. These patients and patients with moderate stridor should ideally be managed within the operation theatre complex. The options available to the anaesthesiologist for airway management in adults with acute supraglottitis are awake oral or nasal intubation under local anaesthesia, inhalational induction followed by oral intubation, fiberoptic bronchoscopy with nasotracheal intubation, and tracheostomy under local anaesthesia.[8] An awake intubation avoids the complications related to complete airway obstruction in already compromised airway under general anaesthesia, but demands patient cooperation. However, an attempt of awake intubation can precipitate severe laryngospasm and complete airway obstruction.[9]

Inhalational induction with orotracheal intubation is described as the method of choice in paediatric patients. However, inhalational induction in adults is likely to be slow and there may be a prolonged excitement phase.[10] In the emergency situation, there is a risk of aspiration of gastric contents in patients with a full

stomach. Sevoflurane is to be preferred over halothane as the inhalation induction agent because it causes significantly less airway irritability, coughing, and breath holding and, besides, provides good tracheal relaxation.[11] Fiberoptic bronchoscopy in an awake patient can be carried out under local anaesthesia in adults but requires special skills, especially in a patient with distorted anatomy due to an edematous airway. The passage of a fiberoptic bronchoscope can be difficult through a narrow laryngeal aperture and temporarily causes complete airway obstruction.[12] A guidewire passed through the working channel of the fiberoptic bronchoscope into the trachea can be used for railroading an endotracheal tube over it.[13] A malleable stillete extending beyond the endotracheal tube, an airway exchange catheter, or a bougie can be used as a guide to pass the endotracheal tube through a narrowed supraglottic region. Either orotracheal intubation or tracheostomy may be performed under local anaesthesia, but both are potentially stimulating procedures and may precipitate sudden loss of the airway.[14] An ENT surgeon or a general surgeon should be present during induction of anaesthesia to perform emergency cricothyrotomy and/or tracheostomy if intubation is unsucessful. Such patients should be admitted to the ICU and have serial laryngoscopic examinations done. The airway can be removed when supraglottic oedema subsides.

Our patient had impending cardiac arrest. She was successfully managed by railroading an endotracheal tube over a suction catheter as better airway management devices were not immediately available.

Review of the literature revealed that secondary infection of a laryngeal cyst with spread of infection to the adjacent tissues is not an uncommon occurrence and should be kept in mind when confronted with patients such as this.[1,15]

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