Subglottic stenosis and acute airway obstruction

Sir,
A 22-year-old female patient presented to the Emergency Department (ED) with acute breathlessness, stridor, agitation and altered sensorium. On applying pulse oximeter, her oxygen saturation (SpO₂) was 74% on room air, which only marginally improved with supplemental O₂. On auscultation, there were bilateral rhonchi. Patient’s medical records revealed subglottic laryngeal stenosis. A definitive tracheostomy was planned after securing airway with an emergency endotracheal intubation. A difficult airway cart was readied and an ear, nose & throat (ENT) surgeon was called to be standby for an emergency tracheostomy during endotracheal intubation in ED. Direct laryngoscopy revealed a modified Cormack and Lehane grade 3. A 4 mm un-cuffed endotracheal (ETT) portex tube was successfully negotiated through the vocal cords, beyond the obstruction after repeated unsuccessful attempts to intubate with 5 and 4.5 millimeter (mm) ETT. At that moment, the loose ETT connector suddenly got disconnected from the tube. At the same moment, patient took a deep inspiratory breath, dragging the ETT along. On direct laryngoscopy, a miniscule rim of the proximal part of the disconnected ETT was visible just below the glottic opening. After oropharyngeal suctioning, adjusting head and neck position and proper external laryngeal manipulation, the tube was successfully retrieved using a pediatric magill’s forceps.

Another 4 mm un-cuffed ETT was passed through the vocal cords, tube tightly secured and patient was ventilated with 100% oxygen. Her SpO₂ picked up and became 100%. Subsequently definitive surgical tracheostomy was performed under local anesthesia and mild sedation with 8 mm cuffed tracheostomy tube to bypass the obstruction. Bilateral air entry was checked to be equal and adequate following administration of steroids and bronchodilators. Her sensorium was restored to normal with adequate spontaneous respirations. Her vital parameters were normal throughout (except for initial sinus tachycardia) and subsequently, her SpO₂ became 100% on room air as well. She was later shifted to ward on T-piece after nebulization, tracheostomy suction and medical management.

On detailed evaluation of her past history, she had pregnancy-induced hypertension and generalized edema during the last trimester of her pregnancy (3 months back). She developed postpartum jaundice and sepsis due to retained products of conception after normal vaginal delivery. She underwent management in the intensive care unit of a peripheral hospital for her condition for
15 days, with intubation and ventilatory support, the records of which were not available. Two months later, she developed progressive hoarseness of voice and paroxysmal stridor, for which she consulted a local ENT surgeon. Video laryngoscopy findings performed then revealed: “Less movement of left Vocal cord on phonation. Normal morphology of larynx with narrowing of trachea just below the vocal cords, and was negotiable with a 5 mm bronchoscope only. Mucosa was inflamed and edematous. No growth was present. Rest of trachea, carina, bronchi and bronchioles were normal” [Figures 1 and 2].

The patient was further investigated after been sent to the ward, with cervical X-rays (antero-posterior and lateral), indirect laryngoscopy (IDL) and computed tomography (CT) scan. On IDL, the glottic chink was inadequate and left vocal cord was immobile. On fiber-optic laryngoscopy, bilateral arytenoids were boggy and edematous. On X-ray neck, pre-vertebral soft-tissue shadow appeared increased in thickness, mildly compressing the trachea. On contrast-enhanced CT of the neck, thickening of soft-tissue was noted in glottic and subglottic region, leading to narrowing of lumen. No obvious abnormal post-contrast enhancement was seen [Figures 3 and 4].

Her post-tracheostomy course was uneventful in the ward, where her tracheostomy tube size was sequentially reduced and was finally decannulated. She was later discharged home after speech therapy and advised to follow-up regularly in ENT out-patient department.

Patients with acquired stenosis are diagnosed from a few days to 10 years or more following the initial injury. The majority of cases are diagnosed within a year. Symptoms\(^1\) include dyspnea (may be on exertion or with rest, depending on the severity of stenosis), stridor, hoarseness, brassy cough, recurrent pneumonitis, cyanosis. Management in all these situations needs to be prompt and definitive.\(^2\) Since patient presented with acute airway obstruction possibly precipitated by a recent respiratory tract infection and consequent further narrowing,
Only a 4 mm ETT could be passed into the trachea, which relieved the obstruction momentarily.

In our case, the problem was in the loose connection between the ETT and the standard connector. With patient taking a deep breath at the same time, there was a strong intrathoracic pressure pulling the un-cuffed ETT into the trachea. Postpartum patients who were on prolonged endotracheal intubation are more likely to develop subglottic stenosis.[3] A high index of suspicion is warranted with the onset of respiratory symptoms following a history of intubation, regardless of the duration of intubation. This case highlights the fact that subglottic stenosis can present as acute airway obstruction.

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