Acute airway distress secondary to iatrogenic injury during Tracheostomy

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Abstract Laceration and perforation of the membranous trachea are uncommon. We present a case of bilateral Pneumothorax, which developed subsequent to iatrogenic injury during tracheostomy.

Keywords Pneumothorax • Tracheostomy

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Introduction

Complications of tracheostomy are numerous [1,2]. One of the less common complications is injury to the pleural dome or perforation of the posterior tracheal wall. This complication however is not uncommon in children or individuals with short and fatty neck because of the peculiar anatomic differences of airway. We present here a case of bilateral pneumothorax, which developed following iatrogenic injury to the posterior tracheal wall in a nine-year-old boy.

Case report

A nine-year-old male patient presented to us with right side posttraumatic temporomandibular joint ankylosis. Routine investigations were done. He was subsequently taken up for ankylosis release and interpositional arthroplasty with costochondral graft. Following intravenous (IV) access, the patient was premedicated with glycopyrrolate and 8mg Dexamethasone. Infusion of 5% DNS was started and antibiotic prophylaxis administered. Monitoring included electrocardiography, pulse oximetry, end tidal CO₂, noninvasive blood pressure and temperature. Informed consent was obtained for awake intubation. Tracheostomy standby and jet ventilation were kept ready. 2 drops of xylometazoline were instilled into the more patent right nostril. The upper

airway was anesthetized using right nasal packing with freshly prepared adrenalized 4% lignocaine, 2% viscous gargles and bilateral superior laryngeal nerve block with 2 ml of 2% adrenalized lignocaine on each side. A blind awake nasal intubation was attempted after applying cricoid pressure. This was discontinued following two unsuccessful atraumatic attempts. Tracheostomy was planned. A slit in the trachea was made under local anesthesia with number 11 blade after dissection. The tracheostomy tube was stabilized with the strap and no additional sutures were required for the surgical wound. Tracheostomy performed with moderate difficulty according to the operator experience. Bilateral breath sounds were confirmed. Anesthesia was maintained with halothane, 50% N₂O and 50% O₂. Ankylosis was released and a costochondral graft was taken from the right side rib. During the entire operative period the saturation was maintained and nothing significant noted. First postoperative day patient developed severe respiratory distress. On examination, patient was dyspnoeic, pulse–130/Min, was tachypnoeic with short shallow breath, chest expansion was inadequate bilaterally and only minimal breath sounds could be heard in all the lung fields on auscultation. SaO₂ dropped below 80. A pneumothorax was suspected and chest X ray AP view was taken. Chest X-ray showed bilateral pneumothorax Fig. 1. Immediately drainage was done with 16NO Angiocath, lungs expanded

progressively Fig. 2 and 3. He recovered completely the same day. Patient was additionally put on Ryles tube feeding for 10 days, and Tracheostomy decannulated in 10 days.

Discussion

Pneumothorax, the loss of air from the lung into the pleural space, is described as being spontaneous or traumatic in origin. The spontaneous Pneumothorax can be further subdivided into primary (i.e., no underlying pulmonary disease) and secondary (i.e., related to underlying pulmonary pathology). The general pathologic mechanism of the development of spontaneous Pneumothorax occurs from either a visceral pleural tear from rupture of a sub pleural bleb during normal breathing or from hyperinflation of the lung during bronchiolar obstruction resulting in excessive distal airway pressure with subsequent alveolar rupture [3]. Multiple thoracoscopic studies have shown the existence of blebs and bullae in 48–100% of patients with spontaneous Pneumothorax [4,5]. Potential causes for traumatic/iatrogenic pneumothorax within the practice of anesthesia are often related to subclavian catheterization, positive pressure ventilation, supraclavicular nerve blocks, traumatic intubation, abrupt awakening with an endotracheal tube in place, and needle thoracentesis from a misdiagnosed pneumothorax.

Fig. 1 Collapsed lung secondary to pneumothorax

Fig. 2 Progressive expansion of lung after chest drain insertion

Fig. 3 Complete expansion of lung

Pneumothorax can be caused by 1 of the following 4 mechanisms:

(1) Extra thoracic trauma (i.e., closed-chest compressions), (2) Spontaneous rupture of alveoli, (3) Disruption of fascial planes in the neck (i.e., traumatic intubation), or (4) Abnormally high intrathoracic pressure (i.e., valsalva from protracted coughing). Tension pneumothorax occurs when there is continuous loss of air from the lung into the pleural space with no escape route. This results in a shift of the mediastinal contents to the opposite side.

In this case, there was no need for closed chest compressions, essentially eliminating the first cause of pneumothorax. Spontaneous rupture of alveoli is uncommon but not rare. Frequently in tall, thin males,

Fig. 4 Anatomic difference of airway in an adult and child

sometimes having a history of smoking or inhalation drug abuse [6]. Blind nasal tracheal intubation was unsuccessful and discontinued after 2 atraumatic attempt utilizing moderate pressure applied to the cricoid cartilage (Sellick Maneuver). The tube did not appear to engage any resistance from either the piriform sinus or vallecula, which could lacerate the hypopharynx. The other reason to rule out the intubation as the cause for the pneumothorax was the absence of pain in the throat and lack of any emphysema in the neck. There was no excess pressure to the distal airways, if pneumothorax occurred during this period, symptoms probably would have developed earlier.

Ruling out the anesthetic causes left us with two other most likely reasons, injury

during tracheostomy or pleural injury during costochondral graft harvest. Laceration and perforation of the membranous trachea are uncommon but they have been described after tracheostomy [7,8]. Children make up the majority of patients sustaining such tracheal injuries because of the difference in the anatomy of airway. Cricoid cartilage is the narrowest portion in the infants' airway (Fig. 4). It's narrower at this level in children than in adults, where the glottis is the narrowest part. Therefore the chances of injury to the posterior tracheal wall are much greater in children during tracheostomy. Likelihood of pleural dome injury is also high in children because of its high cervical placement. In this case the blind nasal intubation was already attempted atraumatically and then the tracheostomy was carried out. An Iatrogenic injury to the posterior wall of trachea was the most likely reason for the bilateral pneumothorax. After the injury, the inflation of tracheostomy tube cuff would have further separated membranous trachea leading to pneumothorax secondary to Pneumomediastinum. However the other likely cause for perforation of the pleura, the costochondral graft harvest was ruled out, because there was; (1) no obvious air leak during harvest of the graft (2) Post graft harvest we checked for any puncture with positive pressure ventilation and (3) Pneumothorax was bilateral which is unlikely with unilateral graft harvest, unless there is a great trauma leading to Pneumomediastium and then Pneumothorax. Combined with relative inexperience of the operator and the use of the pointed blade, the injury to the posterior tracheal wall was the most likely reason. Since pneumothorax developed secondary to the pneumomediastinum with relatively small injury, the pneumothorax was delayed. Looking for the exact site of the tracheal wall injury, using fibre-optic bronchoscopes via the tracheostomy tube before decannulation is difficult with tube in place.

The injury was managed conservatively with underwater drain and by deflating the cuff. Patient was additionally put on Ryle's tube feeding for 10 days. During performing airway access technique one should be familiar with prevention, identification and management of acute airway perforation. In this case there was more than one reason to suspect risk of airway injury, however there was failure to recognize it immediately. Although the patient recovered without sequelae, a higher

index of suspicion and careful surveillance may have prevented the further complication. Tracheostomy in children therefore needs utmost attention to the peculiar anatomy and care taken to use smaller size instruments than the regular adult ones.

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