An unusual cause of tracheal stenosis

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Purpose: To report a large chronic tracheal foreign body, causing tracheal stenosis in an 11-yr-old girl.

Clinical Features: The history was suggestive of obstructive airways disease with secondary bronchiectasis. Physical findings were crepitations and rhonchi all over the chest. Blood gases were normal. Chest X-ray showed bronchiectasis and a ventilation perfusion scan identified a tracheo-esophageal fistula. During anesthesia to confirm this, intubation and ventilation were difficult because of tracheal stenosis. The hypoventilation resulted in severe hypercarbia and acidosis. A subsequent CT scan showed a stenosis of 2 mm diameter and 1 cm length in the middle third of trachea, bronchiectasis, and an air filled pocket between the trachea and esophagus. PFT showed a severe obstruction. Antitubercular treatment which was started on the presumptive diagnosis of tuberculous stenosis and tracheoesophageal fistula caused a delay with deterioration of patient from intermittent dyspnea to orthopnea with severe hypecarbia and acidosis. The anesthetic management of the tracheal reconstruction was difficult due to her moribund condition even after medical treatment, the short length of the trachea above the obstruction, its severity and lack of resources for alternative techniques. A large foreign body was found lying obliquely in the trachea dividing it into an anterior narrow airway mimicking a stenosed trachea , and a wider posterior blind passage.

Conclusion: The anesthetic consequences were peculiar to the unexpected etiology of the stenosis and poor general condition of the patient. Minor details like the tracheal tube bevel and ventilatory pattern became vitally important.

Objectif : Rapporter le cas d'un important corps étranger trachéal causant une sténose de la trachée chez une fillette de 11 ans.

Éléments cliniques : L'histoire évoquait une maladie obstructive chronique et des bronchectasies associées. L'examen physique a révélé des crépitations et des ronchus diffus au thorax. Les gaz du sang étaient normaux. Les radiographies pulmonaires ont montré des bronchectasies et la scintigraphie de ventilation et de perfusion a identifié une fistule trachéo-œsophagienne. Pendant l'anesthésie, réalisée pour confirmer la présence d'une fistule, l'intubation et la ventilation ont été difficiles à cause de la sténose trachéale. L'hypoventilation a provoqué une hypercapnie et une acidose sévères. La tomographie subséquente a montré une sténose de 2 mm de diamètre sur I cm de long au tiers moyen de la trachée, une bronchectasie et une poche d'air entre la trachée et l'œsophage. Les épreuves fonctionnelles respiratoires (EFR) ont montré une obstruction importante. Le traitement antituberculeux, fondé sur l'hypothèse retenue de sténose tuberculeuse et de fistule trachéo-œsophagienne, a causé un délai pendant lequel l'état de la patiente s'est détérioré, la dyspnée intermittente devenant une orthopnée accompagnée d'hypercapnie et d'acidose sévères. L'anesthésie pratiquée pour la reconstruction de la trachée a été difficile étant donné l'état grave de la patiente, même après le traitement médical, la longueur réduite de la trachée au-dessus de l'obstruction, la sévérité de cette obstruction et le manque de ressources pour l'utilisation de techniques de remplacement. Un important corps étranger a été trouvé. Il reposait obliquement dans la trachée, la divisant en une voie aérienne antérieure étroite, rappelant une trachée sténosée, et en un passage postérieur aveugle, plus large.

Conclusion : Les conséquences anesthésiques ont été particulières, compte tenu de l'étiologie inattendue de la sténose et de l'état général de la patiente. Des détails mineurs comme le biseau du tube trachéal et le schéma ventilatoire ont ainsi pris une importance vitale.

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UCCESS of tracheal reconstruction¹⁻¹⁰ depends on careful planning. We anesthetized a patient twice for severe tracheal stenosis. The relevant findings when the stenosis was unexpectedly diagnosed during the first anesthetic and anesthetic management for delayed tracheal reconstruction follow.

Case Report

An 18 kg 11-yr-old girl was admitted for intermittent breathlessness with normal activities between attacks, for a year. The onset had been sudden. She had been unsuccessfully treated for asthma, aspiration pneumonitis and tuberculosis. Physical findings, airway grading, exercise tolerance (she could climb two floors with ease), blood chemistry and blood gases were normal. Chest X-ray showed bilateral bronchiectasis. Pulmonary function testing was not done. A ventilation - perfusion scan suggested an H - shaped tracheoesophageal fistula. During esophagoscopy to confirm this, tracheal intubation, even with a 4 mm *id* tube was impossible due to an obstruction 1.5-2 cm below the cords. An uncuffed 6 mm tube placed above the obstruction enabled adequate chest excursion, audible breath sounds and oxygen saturation. The P_{FT}CO₂ could not be recorded because of a large leak around the tube. It returned after resumption of spontaneous ventilation at the end of esophagoscopy showing a $P_{FT}CO_2 > 100 \text{ mmHg}$, PaCO₂ -127 mmHg and pH 6.9. However, 10 min of spontaneous respiration restored normalcy and the trachea was extubated.

A CT scan showed stenosis of 2 mm diameter and 1 cm length in the middle third of the trachea, bronchiectasis and an air filled pocket, between the trachea and esophagus. Flow volume loops showed severely flattened inspiratory and expiratory curves. Spirometry was < 40% of predicted values. Surgery was delayed to allow antitubercular therapy. Two months later she was admitted with severe breathlessness, upper respiratory tract infection and 3 kg weight

| TABLE | Blood | gases |
|-------|-------|-------|
|-------|-------|-------|

loss. Chest X-ray was same as before. Blood counts, renal and liver function tests were normal. Arterial blood gases are shown in the Table. After 15 dy of antibiotics, mucolytics, bronchodilators and physiotherapy, she was posted for tracheal reconstruction.

After 5 μ g·kg⁻¹ glycopyrrolate *im* and EMLA, preoxygenation in sitting position, ECG, NIBP, pulse oximeter and capnograph nasal sampling line were attached (Table). Intermittent trilene inhalation facilitated placement of arterial and central venous lines, bilateral superior laryngeal nerve blocks and superficial cervical plexus blocks with bupivacaine 0.5%. The upper airway was sprayed with 1 ml lidocaine 4%. She was awake during these procedures. The systolic blood pressure range was 140 to 110 mmHg and heart rate was 120-140 bpm. Perioperative blood gases are shown in the Table.

Propofol 1 mg·kg⁻¹ bolus followed by 1 mg·kg⁻¹·min⁻¹ infusion and halothane 0.5% in oxygen were given. After a glossopharygeal nerve block with 1 ml lidocaine 1% and succinylcholine 2 mg·kg⁻¹ a cuffed 6 mm *id* armoured nasotracheal tube was passed into the trachea above the stenosis. Part of the inflated cuff was above the vocal cords. Spontaneous ventilation, on its return, was uneven so ventilation was controlled with atracurium 0.5 mg·kg⁻¹ bolus followed by infusion of 0.5 mg·kg⁻¹·hr⁻¹ and isoflurane in oxygen. The stenosis was sectioned and the trachea reanastomosed around a 7.5 mm *id* nasotracheal tube. After 1 mg neostigmine and 0.6 mg atropine spontaneous respiration maintained normal blood gases.

The excised segment of the trachea showed an anterior narrow passage for ventilation and a posterior blind pouch. (Figures 1 a,b). Between them, there was a middle mobile part of two thick septa enclosing a foreign body, a thin circular plastic disk of 1 cm diameter with serrated edges and multiple perforations (Figure 2). Recovery was uneventful after trachea extubation at 72 hr.

| THE HE BIOOR Subes | | | | | | | |
|--|--------|---------|-------|---------|---------|----------|-------|
| Time | PO_2 | PCO_2 | pН | HCO_3 | TcO_2 | ABE | Sa O2 |
| At second admission on O ₂ . | 240 | 70 | 7.1 | 29 | 31 | -8 | 99 |
| Scheduled for surgery off O ₂ . | 90 | 50 | 7.3 | 30 | 31 | -6 | 94 |
| Pre Induction, preoxygenation | 380.4 | 52.7 | 7.363 | 29.5 | 31.1 | 3.0 | 99.8 |
| Post Induction just intubated | 251.3 | 66.5 | 7.244 | 27.9 | 29.9 | -1.1 | 99.5 |
| Post intubation hand ventilation I:E !:1 | 297.5 | 45.0 | 7.227 | 18.1 | 19.5 | -9.1 | 99.6 |
| Post Intubation, spontaneous ventilation | 293.1 | 70.2 | 7.235 | 28.8 | 30.9 | -0.6 | 99.5 |
| Post Intubation controlled ventilation I:E !:1 | 244.4 | 43.5 | 7.393 | 26.1 | 27.5 | 1.1 | 99.6 |
| Stenosis dissection | 390.9 | 112.8 | 6.998 | 26.3 | 66.7 | -6.39 | 99.7 |
| No tracheal handling during laryngeal drop | 246 | 65.4 | 7.195 | 24.4 | 26.4 | -5.1 | 99.5 |
| Ventilation through distal tracheal segment | 173.5 | 41.0 | 7.425 | 26.7 | 27.9 | -2.399.3 | |





FIGURE 1A A line diagram showing the retrospective representation of the foreign body enclosed in grannulomatous tissue the trachea. Note the narrow airway anteriorly, the grannuloma in the middle and the posterior blind passage mistaken for a tracheo esophageal fistula.

The long bevel of a PVC tube is obstructed by the septum.

Discussion

Observation of the foreign body in this patient, suggested that when lying vertically it would not have obstructed the trachea but, when horizontal, it could block most of the trachea causing severe breathlessness and explained the intermittent attacks. Development of septae around the foreign body in response to continued irritation probably led to a gradual division of the trachea into an anterior stenosed airway and a posterior blind passage.

As tuberculosis is endemic in India, the posterior blind passage was mistaken for a tuberculous tracheoesophageal fistula, with recurrent pulmonary aspirations to explain the fever with respiratory distress and bronchiectasis. In retrospect, this delay for antitubercular therapy in the absence of a tissue diag-

FIGURE 1B A representation of a tracheal armoured tube with a short bevel, the bevel staying unobstructed above the blind passage.

nosis coinciding with the critical phase in the progression of the obstruction resulted in gross deterioration of the patient's condition.

When the patient was agitated, her breathlessness was alarming. In retrospect, the mobile upper end of the septum enclosing the foreign body probably moved anteriorly to narrow the airway to the 2 mm shown in the CT, and this explains the swings between normalcy and severe stridor. Also, obstruction of the tracheal tube by the septum (Figure 1a) explains the need for a high inspiratory pressure during the first anesthetic. (Figure 1a) In addition, the type of endotracheal tube bevel was important (Figure 1c) because the 1 cm bevel of a 6 mm tube in a 1.5 cm airway worsened the leak and increased hypoventilation.

During the second anesthetic, controlled ventilation using a cuffed armoured tube with a short bevel, ensured the maximum length of tube in the trachea



FIGURE 1C A representation of the usual type of stenosis where the bevel cannot get obstructed.

whilst avoiding intubation of the blind passage (Figure 1b) and leak. The unavailability of a fibreoptic bronchoscope, HFO HFJ and risks of rigid bronchoscopy precluded their use. The use of local and topical anesthesia and trilene analgesia avoided the agitation which may have precipitated severe stridor.

Conclusion

The anesthetic management of a patient with foreign body tracheal stenosis is described. The intermittent sever obstruction was caused by realignment of the



FIGURE 2 The foreign body, a plastic disc which is a part of `RAKHI' tied on the wrist for religious reasons.

foreign body within the trachea. Attention was paid to minute details of anesthetic management such as the length of the bevel of the tracheal tube, steps to safely minimize anesthesia, the choice of ventilatory pattern and the judicious use of anesthetic agents assumed great importance.

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