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CASE REPORT

Upper airway obstruction in a Nigerian girl with Diphtheria: Could blind nasotracheal intubation be the minimally invasive alternative treatment in a resource-constrained setting?

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Abstract

Managing upper airway obstruction in patients with diphtheria can be challenging. In a resource-constrained setting with no Intensive Care Units, blind nasotracheal intubation could be lifesaving.
We report an eight-year-old girl who presented with a sore throat, fever, neck swelling and severe pallor. Following the commencement of blood transfusion, she developed respiratory distress associated with restlessness, stridor and gasping for air. Emergency nasotracheal intubation resulted in resolution of the respiratory distress and other symptoms. While evaluating a child with diphtheria, the sudden emergence of restlessness and respiratory distress may signal upper airway obstruction. Urgent blind nasotracheal intubation may be lifesaving.

Background

Diphtheria is a vaccine-preventable infection caused by *Corynebacterium diphtheriae*.[1] It may affect the respiratory system, eyes, skin and other parts of the body.[1] Respiratory diphtheria is the commonest type and is associated with complications such as upper airway obstruction, disseminated intravascular coagulopathy, myocarditis, acute kidney injury and neuritis.[2] Upper airway obstruction is reported to be a leading cause of death.[3] We report a case of an 8-year-old Nigerian girl who was admitted via the children's emergency unit to the Isolation Centre of Yobe State Specialist Hospital (YSSH) Potiskum, Nigeria. She presented on account of sore throat, fever and severe pallor. Shortly after commencement of blood transfusion, she developed respiratory distress necessitating urgent blind nasotracheal intubation with a cuffed tube.

Case Presentation

An 8-year-old girl presented on account of a sore throat, fever, neck swelling of 5 days and severe pallor noted at presentation. There was a history of recent recurrent epistaxis. There was an outbreak of diphtheria in the community, and the continuous community sensitisation programmes by the government made her parents present her to the hospital. No jaundice, passage of smoke-coloured urine or other urinary symptoms. However, there was a positive history of ingesting water-based oral traditional herbs and topical neck ointment with minimal improvement. She was not immunised for diphtheria.

On examination, she was markedly pale, anicteric, not cyanosed and febrile with a temperature of 38.9^0C. She weighed 22kg (86.3% of expected weight) and her height was 119cm (95.2% of expected height). She was tachypnoeic with respiratory rate of 42 cycles/minute but not dyspnoeic, vesicular breath sounds with no crepitations. Oxygen saturation (SpO_2) was 93% in room air. Pulses were bounding, regular and synchronous with rate of 136/min, the normal first and second heart sounds were heard as well as third with gallop rhythm but no murmur. Abdominal examinations revealed a full abdomen with right hypochondrial tenderness, the liver was 5cm palpably enlarged, its surface was firm and smooth. Other organs were not enlarged. Bowel sounds were normoactive. She was conscious, and alert, and with normal tone across the limbs. Throat examination revealed a pseudomembrane along the soft palate and around the Waldeyer ring. A nasal examination revealed a thick greyish discharge in the nasal cavity. Packed cell volume was 18%.

The initial working diagnosis was nasopharyngeal diphtheria with anaemic heart failure. The case was notified. In preparation for a blood transfusion, she was nursed leg up to allow adequate venous return to the heart, commenced on oxygen via nasal prongs at 2 L/minute, and was given IV frusemide 20 mg stat. She was nursed at 45º head up (cardiac position) and commenced a transfusion of fresh whole blood within one hour of arrival. An hour into the transfusion, she suddenly became restless with features of respiratory distress. She had stridor and was gasping for air whilst the SpO_2 dropped to 30%. She was immediately commenced on oxygen and was nebulized with adrenaline with minimal improvement. She was subsequently blind intubated via the nasotracheal route with a size 6.0 mm cuffed endotracheal tube, through which she immediately expelled the psuedomembrane, Figure 1A and her SpO_2 increased to 92%, with the resolution of respiratory distress as well as restlessness. She was on intermittent suctioning and frequent nasotracheal tube cleaning with dry gauze, Figure 1B. She was transfused with 2 units of blood and commenced on IV Erythromycin 250mg 8hrly. She also received IV amoxicillin-clavulanate 600mg 12 hourly for 4 days, and IV diphtheria antitoxin (DAT) 40,000IU in 250mls of normal saline over 4 hours after a test dose. She was on IV fluids 5% dextrose saline, 500 mL 8 hourly for 24 hours and nil *per os*. She was extubated after 24 hours of intubation, Figure 2A, and commenced on oral feeds the next day.

Figure 1. **A**. The Pseudomembrane expelled through the Naso-tracheal tube; B. the patient with naso-tracheal tube inside being clean by the nurse on duty

After the transfusion, her haemoglobin was 18 g/dL, she had leucocytosis with white cell count of 15200 cells/mm^3 (differential count: neutrophil 69.6 %, lymphocytes 20.9 % and monocytes 7.6%) and her platelet count was 200,000 cells/mm^3. The throat swab culture grew *Corynebacterium diphtheriae*. Electrolytes, urea, creatinine, urinalysis and post-extubation ragiograph were normal, Figure 2B

Figure 2. **A.** showing the patient post nasotracheal extubation after 24hrs. **B.** Post-extubation chest radiograph of the patient showing apparently normal radiograph

The patient was discharged home after 4 days of IV Erythromycin and Amoxicillin-clavulanate to complete 14 days of oral Erythromycin and Amoxicillin-clavulanate. The patient was counselled on drug compliance, hand hygiene, and the need to come back for a follow-up. Prophylaxis was provided for her contacts, and her care givers were advised to be advocates for hospital treatment of diphtheria rather than resort to traditional herbs. She was seen in the clinic 4 days after discharge and remained symptom-free and clinically stable.

Discussion

Diphtheria is a vaccine-preventable disease caused by *Corynebacterium spp*; *C. diphtheriae*, *C. ulcerans*, and *C. pseudotuberculosis*.[4] These gram-positive rods can produce diphtheria toxin, an exotoxin that causes potentially fatal cardiac and neurological complications if absorbed systemically. Diphtheria typically affects children under the age of 15.[4] The diagnosis is heralded usually by the presence of adherent pseudo-membrane, a thick greyish/whitish patch. The pseudomembrane is the hallmark of the disease and is characterized by the formation of a dense, grey debris layer of mixture of fibrin, epithelial cells, white blood cells, red blood cells, dead tissues and *Corynebacterium spp*.[5] The treatment usually involves the administration of DAT, antibiotics, nutritional support, and treatment of complications if present.

Upper airway obstruction is the most common cause of death in diphtheria patients.[3] Upper airway obstruction may present as respiratory distress, choking, sudden violent coughing, noisy breathing, snoring, stridor, cyanosis, and worsening oxygen saturation.[5,6] Upper airway obstruction might occur from poor neck positioning, pseudomembrane, secretions, blood clots, nasopharyngeal airway swelling, dislodged food material or foreign body aspiration. The sudden development of respiratory distress after blood transfusion commencement might have resulted from the rush of blood to the neck where the organism was concentrated. This was due to increased affinity by the organism to the red blood cells from the novel HbpA (iron-regulated protein) that bind to haemoglobin and haemoglobin-haptoglobin complex for which the organism uses the complex as iron stores.[7] Thus, obstructing the airway. Securing a patent airway in patients with diphtheria could be lifesaving. Tracheostomy has been repeatedly tried in our isolation centre with variable success rate. Hence, the need to consider another means for relieving the airway.

Pierre-Fidèle Bretonneau is usually credited with having documented in 1825 the first successful use of tracheostomy to relieve laryngeal obstruction caused by diphtheria after two unsuccessful operations in 1818 and 1820.[8] An alternative was sought due to high mortality and excessive bleeding in addition to the need for frequent suctioning. In the 1880s, Dr Joseph O'Dwyer, developed a method of intubating patients orally to survive the life-threatening phase of diphtheria.[9] During the early 19th century, tracheostomy remained the last resort due to the lack of anaesthesia, high risk of infection, and low success rate of the procedure. Since then, various methods such as cricothyrotomy, tracheotomy, orotracheal intubation with mechanical ventilation have been tried in patients with upper airway obstruction from diphtheria with varying successes.

Nasotracheal intubation technique was first described in 1902 by Kuhn.[10] Nair and Joseph in 1975 were the first to describe the use of nasotracheal intubation in 57 children. Fifty recovered without nerve injury.[11] Complications of nasotracheal intubation include epistaxis, bacteraemia, and partial or complete tube obstruction.[12] Contraindications to nasotracheal intubation are base of skull fracture, midface instability and impending respiratory arrest.[12] There is paucity of information on blind nasotracheal intubation for patients with upper airway obstruction from diphtheria in Nigeria and possibly Africa. We suggest that more consideration should be given for using nasotracheal intubation during this life-threatening phase of diphtheria. It is a minimally invasive procedure requiring minimal nursing care, with minimal complications and can be done on the ward. Meanwhile, more studies on the use of nasotracheal intubation and outcomes in patient with diphtheria are needed.

Conclusion

Managing patients with diphtheria and upper airway obstruction can be very challenging, especially in resource constraint settings like ours, where intensive care units and mechanical ventilation are limited. The prompt and diligent use of blind nasotracheal intubation may be lifesaving and may be done on the ward and not necessarily the intensive care unit. The mainstay of treatment of upper airway obstruction is to secure a patent airway. We suggest that blind nasotracheal intubation should be considered in resource-constrained settings in patients with upper airway obstruction, after non-invasive methods have failed. The procedure is easy, fast and requires minimal nursing care. It also has minimal complications compared to more invasive techniques such as tracheostomy. We advise that healthcare workers involved in the care of diphtheria patients are equipped with the skill of nasotracheal intubation as this could be lifesaving. However, more data on its use is required.

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