Subglottic stenosis: A case report and discussion

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A case report of subglottic stenosis in a 3-year-old, 12 kg, female is presented. The patient's medical history is discussed, and the difficulties in anesthetic management are described. The literature is reviewed regarding incidence, pathogenesis, etiology, and prophylactic measures regarding this complication of endotracheal intubation.

The patient's history in this case included numerous surgical procedures for correction of multiple congenital cardiac anomalies. Repair of a ventricular septal defect (VSD) at age 1 1/2 years was complicated by destruction of ventricular conduction pathways, resulting in third degree heart block. An internal pacemaker was placed. However, after the generator pack was changed at age 3 years, infection forced its removal.

The patient did well for a short time, but was then admitted in a postictal state. She exhibited third degree heart block with occasional runs of premature ventricular contractions (PVCs), as well as moderate pulmonary edema. The patient was in a low perfusion state, and was brought to the OR for a left thoracotomy for placement of another internal pacemaker. Upon induction of general anesthesia with IV ketamine and halothane by mask through a nonrebreathing (NRB) system, the patient suffered severe bradycardia and hypotension. Resuscitation measures, which included intubation and ventilation with 100% O₂ closed chest cardiac massage, and an IV drip infusion of isoproterenol, were successful and the planned surgery was carried out.

Postoperatively, the 4.0mm cuffed oral endotracheal tube with 3cc of air in the cuff, was left in place. The patient was ventilated with a Bennett MA-1 respirator. Her recovery was complicated by congestive heart failure (CHF) and positive end expiratory pressure (PEEP) was necessary because of significant pulmonary edema. Also the patient extubated herself three times which necessitated reintubation, all by the nasotracheal approach. She eventually improved and was extubated five days post-op. She returned to the ward and was subsequently discharged.

Case Report

Approximately six weeks postoperatively the patient was readmitted in moderate to severe respiratory distress characterized by marked respiratory effort and inspiratory stridor at rest, chest retraction, and slight cyanosis. A diagnosis of subglottic stenosis was sup-
ported by xerogram studies which revealed a narrowing of the subglottic tracheal lumen at the cricoid ring.

The patient was brought to the OR for laryngoscopy, bronchoscopy, and tracheostomy. No premedication was requested. Monitoring devices included: Roche Arteriosond® blood pressure cuff, EKG, precordial stethoscope, and rectal temperature probe. Her vital signs were BP 80/40, pulse 72 (pacemaker rate), respirations 45/min, temperature 100°F. General anesthesia was induced with IM ketamine, followed by halothane and 100% O₂ by mask through a NRB system. Early assisted ventilation was necessary and high positive airway pressure was required to adequately ventilate the patient.

Normally a 4.5 to 5.0mm endotracheal tube would be the appropriate choice for a patient this size and age. However, with knowledge of the tracheal narrowing, intubation was first attempted with a 4.0mm uncuffed endotracheal tube. Exposure of the glottis was carried out with ease. The glottis and vocal cords appeared normal, but the endotracheal tube would not pass more than a few millimeters past the vocal cords.

The patient was reoxygenated by mask and intubation was attempted with a 3.5mm uncuffed endotracheal tube, again with the same unsuccessful results. Management of the patient’s airway was becoming difficult, and she seemed to be becoming slightly more cyanotic. At this point the surgeon was asked to pass the Sanders rigid ventilating bronchoscope into the trachea. This was accomplished and adequate ventilation was attained. This maneuver dilated the extremely small airway opening caused by the stenosis. Successful oral intubation was finally accomplished with a 3.0mm uncuffed endotracheal tube, which fit snugly in the subglottic area. Tracheostomy was performed, the patient emerged from anesthesia uneventfully, and was taken to surgical ICU fully awake.

Discussion

In children, the incidence of subglottic tracheal stenosis causing functional respiratory impairment following prolonged endotracheal intubation (greater than 24 hours) has been reported to be in the range of 2-5%. The incidence of subglottic stenosis is less in adults—1-2%. Children are more prone to develop this complication due to the smaller size of their airway, especially at the rigid cricoid ring, and the high intolerance of their subglottic tissue to foreign material. Although the incidence of subglottic stenosis does vary positively with age, there is only a poor correlation between age and the degree of stenosis. It is obvious that this 3-year-old female patient, who was intubated for approximately 5 days, fell into a category where subglottic stenosis may most frequently occur.

In just a short time after intubation, irritation by the endotracheal tube will produce an inflammatory response. This reaction progresses the longer the patient remains intubated. The mucosa becomes hyperemic and can eventually ulcerate to expose the cricoid cartilage and/or the cartilaginous tracheal rings. This irritation, and the usual presence of infection, will soften and destroy the cartilaginous structures. The most severe trauma due to prolonged intubation is usually seen in the cricoid region due to its rigidity, and in the anterior trachea, since it is not as distensible as the post tracheal wall.

Subglottic stenosis is the result of the healing process following such damage. Mucosal tissue undergoes dense proliferative scar formation. Damaged cartilage does not heal by primary regeneration, but by formation of a shrinking fibrous bridge. Because this healing process takes time, signs and symptoms October/1978 523
of upper airway obstruction secondary to stenosis are usually not seen for weeks or months after extubation.\textsuperscript{4,22,23} Subglottic membrane formation has been reported as early as 18 to 24 hours after extubation,\textsuperscript{2} however, early symptoms of respiratory obstruction are usually secondary to subglottic edema.\textsuperscript{10,24,25} In this case report, the patient was readmitted in respiratory distress at approximately 5 weeks post extubation.

Clinical symptoms of respiratory distress attributable to significant subglottic stenosis include dyspnea, chest retraction, and inspiratory stridor, at rest. This lack of effort tolerance indicates a marked degree of stenosis since patients with normal lungs can have a normal exercise tolerance with their tracheal lumen diameter decreased by 50\%. Another sign is a repetitive and irritative cough caused by the stenosis preventing the patient from raising sputum from the trachea into the oral pharynx.\textsuperscript{26} This patient exhibited these signs of severe subglottic stenosis.

Xerograms indicated the cause of this patient's respiratory distress was due to subglottic stenosis. Stenosis in these studies is depicted as a reduction of the subglottic tracheal lumen due to the projection of a thin membrane into the lumen at almost a 90\(^\circ\) angle from the wall.\textsuperscript{15} (See accompanying photographs.)

Although there are arguments for the safe use of low pressure cuffed endotracheal tubes,\textsuperscript{17,19,27,28} cuff pressure, especially when excessive, seems to be one of the primary causes of tracheal damage.\textsuperscript{6,14} Cuff wall pressure has an inverse relationship to perfusion of tracheal mucosa,\textsuperscript{29} with capillary flow being blocked when cuff wall pressure exceeds the normal filling pressure of the arteriolar capillary—about 32 torr.\textsuperscript{30} Even with low pressure cuffed endotracheal tubes, with cuff wall pressure less than this, tracheal injury has been reported.\textsuperscript{31} Considering the potential harmful effects of cuffed endotracheal tubes (even those with low pressure cuffs) and the vulnerability of the pediatric patients' subglottic area to trauma, the use of uncuffed endotracheal tubes is advocated for children.\textsuperscript{2,32}

It has been further advocated that

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\caption{Xerogram—shows lateral view of subglottic stenosis.}
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\caption{Xerogram—shows anterior view of subglottic stenosis.}
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one use an endotracheal tube a size smaller than the largest tube which can be passed through the larynx easily.\textsuperscript{2,10} This allows an air leak around the tube and helps prevent irritation and pressure necrosis, particularly at the cricoid ring.\textsuperscript{10} It is important to remember that there is variation in tracheal size of children of the same age.\textsuperscript{8} One should not totally rely on age charts or formulas for choosing the correct size endotracheal tube. The patient presented in this case report was intubated numerous times. Some intubations were done with cuffed endotracheal tubes which were inflated, particularly while the patient required mechanical ventilation. Repeated intubations, especially if difficult and if the subglottic mucosa is already irritated and inflamed, can cause further trauma and contribute to the development of subglottic stenosis.\textsuperscript{5,6,8,32} The use of a cuffed endotracheal tube is not necessary to provide effective positive pressure ventilation.\textsuperscript{32}

Endotracheal tube and cuff material can be a source of tissue toxins which add to tracheal damage. However, the implantation tested (IT) polyvinyl chloride (PVC) endotracheal tubes, such as were used in this patient, are probably the best material in respect to preventing tissue irritation.\textsuperscript{32,34} These are disposable tubes and are not reused. Endotracheal tubes which have been sterilized for reuse have been incriminated as a cause of laryngotracheal irritation. Such irritation can be caused by the chemicals used for sterilization or an irritant produced from a reaction between the chemical cleaner and the endotracheal tube.\textsuperscript{35}

Endotracheal tube movement within the trachea, in a piston and shearing manner, can contribute to subglottic trauma and lead to stenosis.\textsuperscript{5,12,14,30,32} Such mechanical trauma is not only due to patient laryngeal motor activity,\textsuperscript{2,4,10} but can occur when a patient is being mechanically ventilated. This movement occurs with each cycle of the ventilator and is associated with shifting of the ventilator tubing. It may also be seen intraoperatively due to movement of the anesthesia breathing tubes. Whether one is using an oral or nasal endotracheal tube, it must be securely attached to the patient and the ventilation hoses or breathing tubes should be well supported. The use of a nasal endotracheal tube may allow less movement of the endotracheal tube, particularly that caused by swallowing and other laryngeal motor activity.\textsuperscript{4} Gross motor activity of the intubated patient, who is on a ventilator, can be prevented by using sedatives and/or muscle relaxants.\textsuperscript{2} Neither sedatives nor muscle relaxants were used in this patient.

During some of the time this patient was intubated, it was noted that she tended to keep her neck extended. This postural response to intubation by children is common. Such extension causes cervical lordosis, and the spine forces the cricoid cartilage forward against the endotracheal tube.\textsuperscript{13} It also decreases the distensibility of the posterior tracheal wall by adding the support of the cervical vertebrae.\textsuperscript{32} This may cause the endotracheal tube to exert even more pressure on the anterior wall of the trachea. This positional reaction to intubation by children is another cause of laryngotracheal trauma.

Proper humidification and temperature control of inspired gases being delivered through an endotracheal tube is important. Delivery of dry gases causes the loss of the tracheal mucosa's protective lubricating properties and increases the friability of the tracheal epithelium.\textsuperscript{36} Such dehydration also inhibits ciliary action and may lead to retention of thick dry secretions which are difficult to suction. Such secretions are a media for infection.\textsuperscript{5,37} They can also cause obstruction of endotracheal tubes, especially in small diameter pediatric size tubes.\textsuperscript{38} Ideally, inspired gases should be 30-36°C with an absolute humidity of 30-40mgm H\textsubscript{2}O/L of dry gas.\textsuperscript{37} Adequately warmed and humidified air was delivered to this patient during the period she was intubated.
Vigorous measures should be followed to prevent localized tracheal infection in the intubated patient. Cultures should be taken, antibiotic therapy initiated when indicated, and sterile tracheal suction should be performed to remove secretions. The correlation between localized tracheal infection and tissue damage which may lead to subglottic stenosis is highly suggestive. Also, the presence of infection allows the subglottic tissue to be more easily traumatized by the endotracheal tube. The use of micro-filters may help prevent infection from airborne contamination. The normal physiological filtering mechanism of the upper respiratory tract is of course bypassed when a patient is intubated. Micro-filters can be inserted between the patient and the respirator to help prevent infection from airborne organisms. Most pathogenic organisms vary in size between 0.5 microns and 3 microns in diameter. Normally airborne infection is not spread by individual bacteria, but by clumps of bacteria. It is recommended that such filters be used against particles of 0.5 microns or more in diameter. The patient in this case report received tracheal suction as needed and sterile technique was observed. No tracheal cultures were done during the time this patient was intubated, however, she had received antibiotic therapy as a prophylactic measure against infection at the surgical site. The MAI ventilator has a built-in micro-filtering system 99.97% effective against organisms of 0.3 microns and larger.

Systemic factors play an important role in the occurrence of tracheal damage in the intubated patient. The patient's preoperative low perfusion state, intraoperative hypotensive episode, as well as postoperative cardiovascular instability, could have been contributing factors in the development of this patient's subglottic stenosis. Cardiovascular stability is necessary to assure adequate perfusion of the tracheal mucosa and to preserve its integrity.

The use of some vasopressors can also adversely alter tracheal mucosal perfusion and inhibit its resistance to pressure and irritation. This patient did receive an isoproterenol infusion when she suffered cardiovascular collapse. However, this is a beta adrenergic stimulating drug, so theoretically it does not cause vasoconstriction of the tracheal mucosa's vasculature.

There is a direct relationship between steroid therapy and the occurrence of subglottic stenosis after prolonged endotracheal intubation. However, this occurs only with very high doses of steroids. This patient did not receive steroids during the time she was intubated. However, she was given a normal daily dose of Decadron® for five days, starting three days after she was extubated. The steroid therapy in this case was probably not a significant contributory factor.

Summary

Subglottic stenosis can be a complication of prolonged endotracheal intubation, especially in children. Due to the nature of the healing process, signs and symptoms of respiratory distress usually do not occur until weeks or months following extubation. Management of anesthesia for emergency tracheostomy may be difficult, especially in regard to placement of an endotracheal tube. A full range of endotracheal tube sizes should be available. The availability of a rigid ventilating bronchoscope was of significance in this case.

Subglottic stenosis with involvement of the cricoid region, such as described in this patient, unfortunately commits the patient to chronic tracheostomy with few surgical options. The anesthetist can take steps to lessen the degree of laryngotracheal trauma in the intubated pediatric patient, and help prevent the occurrence of subglottic stenosis. These steps include:

1. Perform careful and delicate intubation, preferably by the nasal route when the patient will be intubated for more than 24 hours.

2. Use sterile uncuffed IT/PVC endo-
tracheal tubes. Lubricants, if used on the endotracheal tube, should also be sterile.
3. Use an endotracheal tube which allows a small air leak, usually one size smaller than the largest endotracheal tube which can be passed through the cricoid ring.
4. Secure the endotracheal tube well, and support the ventilator/anesthesia tubing to prevent movement or accidental removal of the endotracheal tube.
5. Minimize the patient's laryngeal motor activity with sedatives and/or muscle relaxants, but be aware of the possible dangers.
6. Minimize the duration of intubation.
7. Provide adequate temperature and humidity control of inspired gases.
8. Correct the common postural response to intubation by supporting the patient's head in an unextended, neutral position.
9. Take steps to prevent, as well as quickly identify, localized tracheal infection and treat it aggressively when it does occur.
10. Keep the trachea free of excessive secretions by adequate suction, using sterile technique.
11. Use micro-filters to eliminate airborne contaminants in inspired air.
12. Prevent cardiovascular instability while the patient is intubated. Be aware of adverse effects of alpha adrenergic drugs on tracheal mucosal perfusion.
13. Be aware of the adverse effect on healing response associated with large doses of steroids.

REFERENCES


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