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■ Case Report ■
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Airway obstruction during general anesthesia in a premature infant suspecting bronchospasm and/or airway malacia -A case report-

Department of Anesthesiology and Pain Medicine, Yeungnam University College of Medicine, Daegu, Korea

Hyojung Seo, Sangjin Park, and **Haemi Lee**

Airway management is challenging during general anesthesia particularly in small infants. Airway obstruction is prone to occur in premature infants during general anesthesia due to several reasons. We report a case of airway obstruction occurred during the induction of general anesthesia in a 2-month-old infant. Several attempts at endotracheal intubation with positive pressure ventilation resulted in repeated patterns of no end-tidal carbon dioxide output after each trial of endotracheal intubation, but it was reappeared after extubation. However, anesthetic induction with self-respiration and gentle assistance with manual bagging led to a successful intubation. This case was explained by hydromechanics in a collapsible premature airway. **(Anesth Pain Med 2017; 12: 147-150)**

Key Words: Airway obstruction, General anesthesia, Prematurity.

Airway obstruction is not an uncommon phenomenon in premature infants during general anesthesia because the airway is immature and prone to collapse. Airway obstruction may occur in many situations such as bronchospasm, airway malacia, bronchial intubation, tracheal stenosis, external compression to the airway (e.g., vascular ring or mediastinal mass), internal obstruction by mucus, anaphylaxis, etc. Among them, bronchospasm occurs most commonly during the anesthesia especially in the induction stages [1]. Bronchospasm during the induction stage is most commonly caused by airway irritation, often related to intubation [2]. It is characterized by prolonged expiration, wheezing, and increased peak airway pressures.

Airway malacia is a disease of increased collapsibility of the larynx, trachea and/or bronchus due to structural anomalies of the cartilage. This disorder may be found in healthy infants, but it is more common in premature infants. It is generally a benign condition and usually self-limited, because dynamic airway collapse improves as the airway structures mature. The airway that is maintained during spontaneous breathing could be altered by positive airway pressure changes in patients with airway malacia [3]. When the trachea is compressed or functional narrowing occurs, intermittent respiratory obstruction and arterial desaturation may occur [4-6]. Here, we report a case of airway obstruction in a 2-month-old infant suspecting bronchospasm and airway malacia.

CASE REPORT

A 2-month-old boy, weighing 3.7 kg was scheduled to undergo panretinal photocoagulation on both eyes. He was born by cesarean section at gestational age (GA) 31 + 2 weeks at a weight of 1,750 g due to preterm labor, but intubation was not performed, because he had relatively good Apgar scores (5 at 1 min, 8 at 5 min) and good self-respiration. In the pediatric intensive care unit, the infant was cared for in an incubator and nasal oxygen was administered until the 5th day of life. However, he showed frequent oxygen desaturation down to 80%, mostly during feeding, which recovered with simple stimulation. Therefore, aminophylline and theophylline were administered for about 1 month. As no more desaturation patterns were observed and a brain sonogram and an echocardiogram revealed normal structures and functions, the infant was discharged when he was 38-days-old (GA 36 + 4 weeks),

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Corresponding author: Haemi Lee, M.D., Ph.D., Department of Anesthesiology and Pain Medicine, Yeungnam University College of Medicine, 170, Hyeonchung-ro, Nam-gu, Daegu 42415, Korea. Tel: 82-53-620-3361, Fax: 82-53-626-5275, E-mail: hmlee@yu.ac.kr

weighing 2,920 g. But five days later, the infant was readmitted because of an episode of perioral cyanosis while feeding that lasted for 10 seconds and discharged after 2 days because there were no signs of aspiration and his general condition was normal. Ten days later, the infant was readmitted for the treatment of retinopathy of prematurity. During the preoperative evaluation, his mother reported that he still experienced frequent apnea and was restored with light stimulation.

On the day of the operation, the infant was brought to an operation room in his mother's hands. Non-invasive blood pressure, electrocardiography, pulse oximetry, and end-tidal CO_2 ($ETCO_2$) were monitored. The patient was administered 8 vol% of sevoflurane then maintained at 3 vol% for anesthetic induction with 4 L/min oxygen. As there were no difficulties in assisted manual ventilation, 2 mg of rocuronium bromide was injected IV. There was no resistance during the mask ventilation and intubation was attempted with a 3.5 endotracheal tube after full muscle relaxation. However, nothing could be heard during inspiration and expiration and there was no $ETCO_2$ tracing following intubation. Endotracheal tube was removed and mask ventilation was attempted again, but with great difficulty. Therefore, an oral airway device was inserted and mask ventilation was performed using a two-handed maneuver. After spray of salbutamol 200 μg intratracheally, intubation was again attempted with the same tube, but again, $ETCO_2$ could not be traced. As oxygen saturation rapidly decreased into the seventies, an endotracheal tube was extubated and manual ventilation was further attempted, but peripheral oxygen saturation (SpO_2) was barely maintained over 90% under fraction of inspired oxygen 1.0 with high peak inspiratory pressure. $ETCO_2$ could be traced and inspiratory stridor was heard on lung auscultation during mask ventilation, but no $ETCO_2$ could be traced and no inspiratory or expiratory sounds were heard after each trial of intubation. To relieve gastric distension, a 5 Fr. orogastric tube was inserted and air was aspirated intermittently. Although intubation was attempted two more times by a different anesthesiologist, the same patterns repeated; $ETCO_2$ tracings were lost following the intubations. At this time, the depth of the tube was corrected from 11 cm to 8 cm, but no $ETCO_2$ tracing was noted. We then decided to wake the infant, so a reversal agent for muscle relaxation and 0.9 mg of dexamethasone were injected to avoid laryngeal edema. When the self-respiration recovered, the SpO_2 was maintained at 100% on room air and no stridor was heard on lung auscultation. Assuming that ventilation was not problematic with self-respiration, endotracheal intubation was planned under sedation during self-respiration. To maintain spontaneous ventilation, sevoflurane was increased slowly from 3 vol% with minimal manual assistance under a mask. This time, intubation was successful with the same tube and $ETCO_2$ showed a normal tracing. During auscultation after intubation, self-respiration disappeared and manual bagging was gently applied, but ventilation was well maintained. Mechanical ventilation carefully began with positive end-expiratory pressure (PEEP). The surgery was uneventful and the infant was extubated following the procedure without any complications. After 5 hours, the infant's mother reported that he had little difficulty in breathing while feeding, so he received nebulized budesonide and salbutamol. A chest X-ray was taken and it was non-specific (Fig. 1). The infant was discharged without any complications 2 days later.

DISCUSSION

This present case demonstrates a unique pattern of airway obstruction; no capnogram tracing after intubation but reappears under mask ventilation. At first, airway patency was well maintained under gentle mask ventilation, but it was altered by intubation and vigorous positive pressure ventilation to restore oxygen saturation. Oxygen saturation was barely maintained with stridor during manual ventilation under a mask, but there were no $ETCO_2$ outputs or breathing sounds after each trial of endotracheal intubation. The infant was extubated immediately, because misplacement of the endotracheal tube was suspected at first. However, esophageal intubation was ruled out through

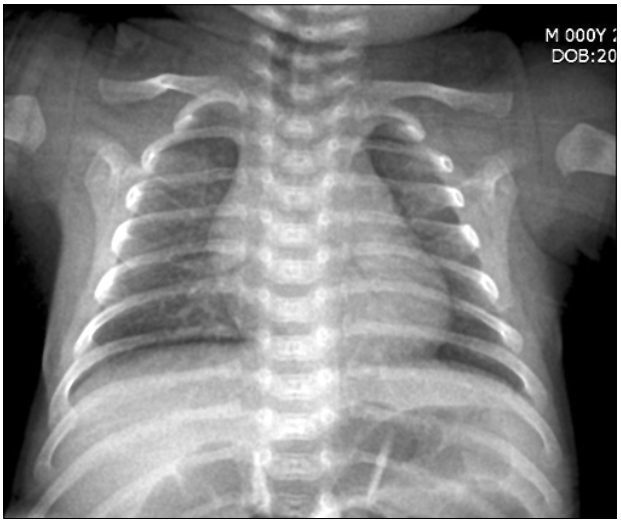

Fig. 1. Chest radiograph taken after the surgery shows non-specific finding.

reattempted intubation after confirmation of the vocal cords and insertion through an orifice without an orogastric tube. Endobroncheal intubation was also ruled out by pulling the endotracheal tube back from a depth of 11 cm to 8 cm, but no difference was found in the results. Endotracheal tube was rechecked whether there is deformity or mucus plug in it, but nothing could be found [7].

Bronchospasm was suspected considering lung auscultation and high peak inspiratory pressure. Infants with upper respiratory tract infection or history of long time ventilatory support may have increased risk of bronchospasm. In this case patient, history of recurrent aspiration during feeding may have had a role in the inflammation of the airway. Breathing sounds could not be heard after intubation, but this is possible without any gas flow in severe bronchospasm [8]. However, other causes might have additively contributed to this phenomenon because the pattern of airway obstruction was unique depending on each trial of intubation as well as preoperative respiratory problems.

Other than bronchospasm, this case could be explained as an example of the occurrence of airway malacia in both extrathoracic and intrathoracic segments. The presence and extent of central airway collapse depends on how rigid the airway is, the magnitude of pressure applied across it, or the transmural pressure across the tracheal wall [9]. Airway obstruction can occur during any phase of respiration but is most pronounced with expiration, because cartilaginous rings of airway fail to provide the necessary rigidity to resist the external force and prevent airway collapse [10]. Therefore, with self-respiration, expiratory stridor is the most common presentation of intrathoracic airway malacia but when cartilaginous immaturity found in the cervical airway or the obstruction is severe enough, it may cause inspiratory airway collapse and inspiratory stridor. In the case of the patient discussed here, the airway was completely obstructed following intubation and partially relieved with vigorous positive pressure ventilation under muscle relaxation. It is assumed that after intubation, the airway obstruction was caused by pressure decline distal to the tube. According to the Bernoulli effects, pressure decreases as the flow increases when passing through a narrow space [11]. The airway passing through an endotracheal tube is much narrower than the oral cavity; therefore, flow will be increased and pressure will further decrease in the endotracheal tube. The faster flow passes to the end of the tube and finally obstruction occurs at the site of malacia outside the tube due to the pressure drop. However, during mask ventilation, inspiratory stridor was heard and ventilation was barely maintained with an irregular $ETCO_2$ tracing. At this time, vigorous manual bagging applied with high air-flow to restore oxygen saturation may cause incomplete laryngomalacia.

There have been previous reports of airway obstruction with airway malacia during general anesthesia. Kwon et al. [12] reported a case of airway obstruction in a premature neonate with suspected tracheomalacia. Airway obstruction occurred after each trial of intubation with small diameter endotracheal tubes or when the tubes were cut. The size and length of the endotracheal tubes were changed to relieve the obstruction. Asai and Shingu [13] reported a case of airway obstruction with hypoxia during emergence from anesthesia due to unexpected tracheomalacia in a child. The obstruction worsened as the child became agitated with labored breathing, and was relieved with soft emergence even though the child did not have any signs of airway obstruction prior to the surgery or after the surgery. These two cases demonstrate that the characteristics of endotracheal tubes and the flow rate may influence ventilation in patients with airway malacia.

Our patient had a high suspicion of airway malacia although he was not diagnosed preoperatively. He was born as a premature and had symptoms of feeding difficulty with apnea and cyanosis which can be seen in airway collapse. During feeding, as the esophagus becomes engorged it may further contribute to tracheal collapse and obstruction. Patients with airway malacia are often asymptomatic and diagnoses are made incidentally during general anesthesia by a bronchoscopic examination. Airway malacia was not confirmed by bronchoscopy in this case, because it was difficult to maintain oxygen saturation during the attack. Rather, in instances of high suspicion of airway malacia, waking the patient and then applying smooth induction and emergence could lead to a successful anesthesia for the surgery.

In conclusion, it is important to conduct a careful preoperative assessment to premature infants. Situations likely to collapse the airway should be minimized, such as proceeding surgery during upper respiratory tract infection, intubating under low plane of anesthesia, using a small diameter endotracheal tube or using high gas flow. Bronchospasm should be diagnosed and treated promptly. When airway malacia is suspected, vigorous manual bagging should be avoided, and the smooth conversion of manual ventilation to mechanical ventilation is recommended. A pneumatic stent using continuous positive airway pressure or PEEP might be helpful in maintaining airway patency.

REFERENCES

1. Westhorpe RN, Ludbrook GL, Helps SC. Crisis management during anaesthesia: bronchospasm. Qual Saf Health Care 2005; 14: e7.
2. Dewachter P, Mouton-Faivre C, Emala CW, Beloucif S. Case scenario: bronchospasm during anesthetic induction. Anesthesiology 2011; 114: 1200-10.
3. Masters IB, Chang AB, Patterson L, Wainwright C, Buntain H, Dean BW, et al. Series of laryngomalacia, tracheomalacia, and bronchomalacia disorders and their associations with other conditions in children. Pediatr Pulmonol 2002; 34: 189-95.
4. Schwartz MZ, Filler RM. Tracheal compression as a cause of apnea following repair of tracheoesophageal fistula: treatment by aortopexy. J Pediatr Surg 1980; 15: 842-8.
5. Blair GK, Cohen R, Filler RM. Treatment of tracheomalacia: eight years' experience. J Pediatr Surg 1986; 21: 781-5.
6. Ahel V, Banac S, Rozmanić V, Vukas D, Drescik I, Ahel VA Jr. Aortopexy and bronchopexy for the management of severe tracheomalacia and bronchomalacia. Pediatr Int 2003; 45: 104-6.
7. Han J, Kim C, Lee SH, Burm J. Complete endotracheal tube obstruc tion with mucus during anesthesia in a child with upper respiratory tract infection : a case report. Anesth Pain Med 2007; 2: 82-4.
8. Choi YJ, Choi SU, Cho EJ, Oh JY, Lim HJ. Severe bronchospasm in a premature infant during induction of anesthesia caused ventilation failure. Korean J Anesthesiol 2013; 65(6 Suppl): S84-6.
9. Hysinger EB, Panitch HB. Paediatric tracheomalacia. Paediatr Respir Rev 2016; 17: 9-15.
10. Tanphaichitr A, Tanphaichitr P, Apiwattanasawee P, Brockbank J, Rutter MJ, Simakajornboon N. Prevalence and risk factors for central sleep apnea in infants with laryngomalacia. Otolaryngol Head Neck Surg 2014; 150: 677-83.
11. Doyle DJ, O'Grady KF. Physics and modeling of the airway. In: Benumof and Hagberg's Airway Management. 3rd ed. Edited by Hagberg CA: Philadelphia, Mosby Elsevier. 2012, pp 92-117.
12. Kwon YS, Lim YH, Park HL, Yoo BH, Woo SH, Yon JH. Reversible airway obstruction caused by changing the size and length of an endotracheal tube in a premature neonate with suspected tracheomalacia -A case report-. Korean J Anesthesiol 2010; 59 Suppl: S30-2.
13. Asai T, Shingu K. Airway obstruction in a child with asymptomatic tracheobronchomalacia. Can J Anaesth 2001; 48: 684-7.