

Iatrogenic Tension Pneumothorax after Fiberoptic-guided Intubation in a Pediatric Patient: A Case Report

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ABSTRACT

Fiberoptic-guided intubation (FOI) has been an indispensable component of difficult airway management especially in instances where anatomical limitations precluded use of conventional direct laryngoscopy. Its use, however, is not without risks.

This paper presents a 4-year-old female with a limited mouth opening scheduled for an elective oral commissurotomy who developed signs and symptoms of tension pneumothorax immediately following a successful fiberoptic nasotracheal intubation. Passive insufflation of high-flow oxygen through a flexible fiberoptic bronchoscope preloaded with a tight-fitting endotracheal tube led to accumulation of air. This caused lung hyperinflation and subsequently, pneumothorax.

Keywords: anesthesia, airway management, intubation, tension pneumothorax, complications

INTRODUCTION

Fiberoptic intubation (FOI) has become an integral part of difficult airway management guidelines after this technology was introduced.¹ FOI is principally performed when securing the airway is deemed difficult through direct laryngoscopy due to a variety of anatomic limitations such as limited mouth opening.^{2,3}

Although the indications for adult and pediatric FOI are similar, there are profound differences in the considerations for these age groups.³ Pediatric FOI is more challenging because of the smaller airways of pediatric patients, making visualization technically difficult. The lack of cooperation of this age group adds to this difficulty which sometimes requires sedation during FOI. Furthermore, pediatric patients have higher oxygen consumption that may lead to rapid desaturation, necessitating intervention in between attempts at FOI.⁴

Fiberoptic intubation is considered generally safe with invaluable life-saving capacity. However, the technique is not devoid of potentially fatal risks. There are no established statistics yet on its complications due to little available literature coming mostly from case reports. Some of these reported complications are epistaxis, laryngotracheal trauma, laryngospasm and aspiration.² Tension pneumothorax has been described in several fiberoptic-assisted procedures involving the airway.⁵⁻⁹ Its incidence is rare, estimated to be just 1%, caused by tracheal perforation or excessive oxygen insufflation during FOI.^{2,8}

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Although uncommon, tension pneumothorax is a life-threatening condition which can cause hypoxia, hemodynamic instability or death.⁶ Due to its catastrophic and rapidly deteriorative nature, early recognition and management of tension pneumothorax in a patient undergoing FOI are

keys to successful resuscitation of patients who encounter this complication.

CASE PRESENTATION

An otherwise healthy 4-year-old female had a history of accidental caustic ingestion 2 years ago. Chemical burns on the mouth resulted to tissue adhesions after wound healing, causing a limited mouth opening of 2 cm (Figure 1). Due to this, her oral intake was limited to liquefied, pureed or crushed food. This also led to dental health issues as well as speech delay.

The patient weighed 18 kg and had a development at par with her age group. Physical examination revealed no remarkable findings. Cervical mobility was not limited. Thyromental distance was adequate. There were no dyspnea, audible stridor, or any signs of airway obstruction. A pre-operative endoscopic examination of the oral cavity revealed no major anatomic deformities caused by the previous caustic ingestion. After consult and planning with the otorhinolaryngology service, she was scheduled for oral commissurotomy. Since direct laryngoscopy was considered impossible, nasal FOI with minimal sedation was planned.

The patient, accompanied by her mother, was received in the OR awake, alert and slightly agitated. Standard ASA monitors were attached. Topical lidocaine (40 mg/ml) and oxymetazoline (0.5 mg/ml) were sprayed on the nares. Since the oral cavity cannot be directly visualized, the spray nozzle



Figure 1. The patient's airway assessment showing a mouth opening of 2 cm.

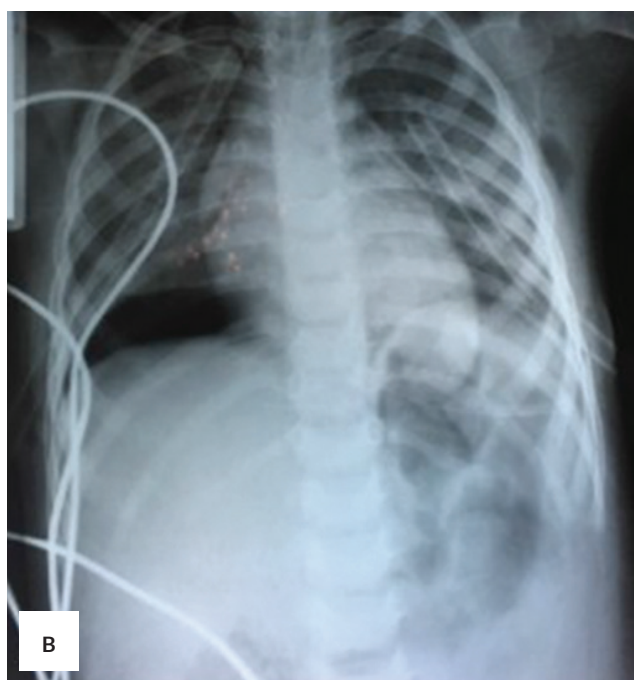
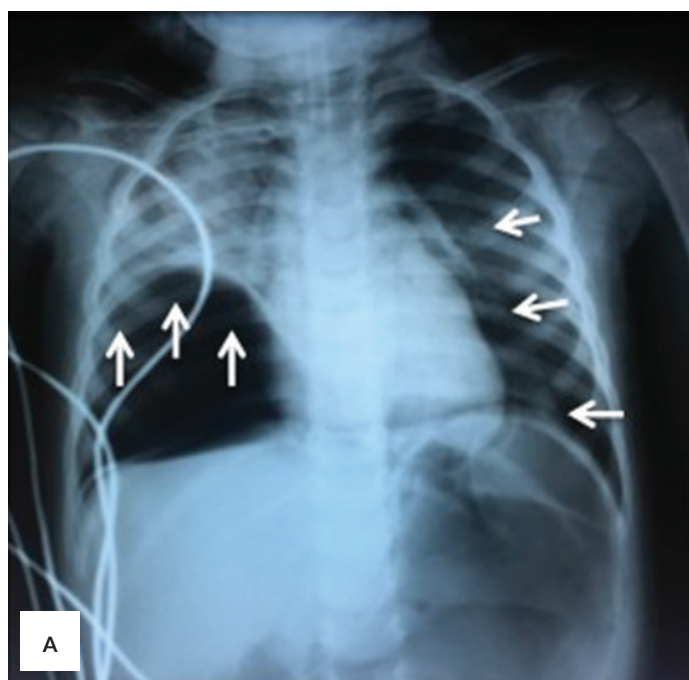


Figure 2. (A) Chest radiography after the initial emergency needle decompression, prior to bilateral chest tube thoracostomy. There are lucent areas devoid of lung markings with visible pleural line (arrows) in both hemithoraces. There is collapse of both lung parenchymas reflective of atelectasis and slight rightward shift of mediastinum. Findings suggest pneumothorax. (B) Chest radiography post-bilateral chest tube thoracostomy. Images show better aeration and re-expansion of the lungs.

was directed to the posterior oral cavity to aim for the uvula and posterior pharyngeal wall. Oxygen supplementation at 6 L/min via Hudson facemask (approximately 44% FiO₂) was given. Atropine 0.3 mg (0.02 mg/kg) was given as an anti-sialogogue. Midazolam 1.8 mg (0.1 mg/kg) and ketamine 18 mg (1 mg/kg) were given in increments to achieve a Ramsay sedation scale score of 6, while maintaining spontaneous respiration.

The head of the bed was elevated at 30 degrees and the Hudson facemask was removed prior to the attempt at FOI. A pediatric flexible fiberoptic bronchoscope (FOB) (Karl Storz Intubation Fiberscope 3.7 x 65) with an outer diameter of 3.7 mm, preloaded with a wire-reinforced endotracheal tube (ETT) with 5.0 mm internal diameter (Portex) was used. The suction port of the scope was connected to a vacuum tubing to facilitate suctioning of secretions while visualizing the structures. The scope was inserted into the right nares with the anesthesiologist facing the head of the patient. Anatomic structures were easily identified as they were not deformed and the glottic opening was visualized. Difficulty was encountered in passing the FOB through the moving glottic opening. Without visualizing the carina, the ETT was advanced through the FOB, and the FOB was subsequently removed. Upon auscultation and observation of capnograph tracing, it was noted to be an esophageal intubation. The ETT was immediately removed and the patient was ventilated with 100% FiO₂ using an anesthesia mask. A brief period of desaturation with an SpO₂ of 85% occurred which was immediately corrected to an SpO₂ of 100% upon ventilation.

Prior to the second attempt, secretions were suctioned and the suction port of the scope was connected to the auxiliary oxygen port at 5 L/min to provide oxygen supplementation and facilitate clearance of secretions on the camera. A more experienced anesthesiologist performed the second FOI. The ETT was successfully placed in 5 minutes, as evidenced by direct visualization of the carina, bilateral breath sounds, ET-CO₂ of 35 mmHg and SpO₂ of 99%. Propofol, 30 mg (1.6 mg/kg) and atracurium 5 mg (0.27 mg/kg) was given to deepen anesthetic depth and control ventilation. Sevoflurane inhalation was then initiated at 3 vol% to maintain anesthetic depth.

A few minutes after intubation, SpO₂ decreased to 80% and upon auscultation, there were absent breath sounds bilaterally. The FOB was reinserted to recheck placement of ETT, which confirmed proper placement. Suspecting bronchospasm, 8 puffs of salbutamol (100 mcg/dose) was administered via ETT, increase in sevoflurane concentration to 5 vol% and ketamine 36 mg (2 mg/kg) administration were simultaneously done. Oxygen saturation continued to drop and breath sounds were still inaudible bilaterally after the management. BP was noted to decrease to 60/40 mmHg. At this point, SpO₂ further decreased to 60%. Highly considering pneumothorax, needle decompression was immediately performed using a gauge 24 IV cannula inserted

perpendicularly at the 2nd intercostal space, midclavicular line of both right and left hemithoraces. After needling, the SpO₂ improved to 70-80%. Chest x-ray was done which confirmed the diagnosis of pneumothorax (Figure 2) and the patient was immediately referred for bilateral chest tube thoracostomy.

While waiting for the thoracic surgeon, SpO₂ decreased again to 60% and HR decreased to 60 bpm. Epinephrine 18 mcg (1 mcg/kg) was administered intravenously and chest compressions were started. After the first cycle, the patient's rhythm was pulseless ventricular tachycardia. Shock was delivered at 2 J/kg and chest compressions were resumed. After another cycle of compression, return of spontaneous circulation was established with BP of 70/40 mmHg, HR of 110 bpm and SpO₂ of 70%.

During the chest compressions, needle decompressions were again performed bilaterally with gauge 20 IV cannulas. This time, the needles were connected to an adult IV tubing (macroset), with the other end submerged under a sterile normal saline to achieve a water seal. The needles were secured until the thoracic surgeon arrived to perform chest tube thoracostomy. During this time, SpO₂ were noted to improve to 90%.

After the bilateral chest tube insertion, vital signs remained stable with BP of 80-90/50-70 mmHg, HR of 90-120 bpm and SpO₂ of >90%. The procedure was deferred and the patient was transferred to the pediatric critical care unit. She was extubated after 8 days and the chest tubes were removed after 13 days, just prior to the patient's discharge. No further surgery was planned as of this writing. Patient was referred to rehabilitation medicine for outpatient management.

DISCUSSION

Surgery under local anesthesia was difficult for this patient, as gaining cooperation is difficult in her age group.^{4,10} While sedation could facilitate cooperation, it could also blunt the protective reflexes of the airway from foreign body.^{11,12} An airway secured with ETT was preferred to protect it from secretions and blood that may accumulate during the surgery. General endotracheal anesthesia was therefore deemed appropriate for the procedure.

Since the mouth was also the operative site, a nasotracheal approach was planned to avoid disruption of the operative field with an ETT.¹³ In addition, the ETT could be anchored more securely at the nose and decrease the risk of accidental extubation during surgical manipulation. The limited mouth opening of 2 cm precluded the use of direct laryngoscopy for this patient.^{2,3} A flexible pediatric fiberoptic intubating scope was used because it was the available airway equipment that could access the trachea through the nose.

Deeper levels of sedation are associated with backward displacement of oropharyngeal structures which will make FOI technically difficult.¹³ While an awake approach is ideal,

minimal sedation was given to facilitate cooperation during FOI. In addition, the smaller anatomic structures would also entail a need for an immobile patient due to significant changes in the camera view brought about by subtle movements.⁴ Sedation was titrated to maintain spontaneous respirations as respiratory depression during FOI may result in more rapid desaturation due to higher oxygen consumption in pediatric patients.⁴ This is further compounded by the increased collapsibility of airways, causing obstruction.¹⁴

This rapid desaturation was observed during the first attempt at FOI. It should be noted that during the initial pass of the fiberoptic scope, the patient did not receive any oxygen supplementation. Considering the previously mentioned factors, oxygen supplementation was then provided through the suction port of the fiberoptic scope during the second attempt to secure the airway. Aside from serving as a source of oxygen supplementation, this technique of passively insufflating oxygen also cleared the secretions from the camera lens and expanded the airway structures which may have collapsed, thus further facilitated the procedure.^{3,15-17} Hence, as seen in the case presented, the second attempt at FOI was easily performed.

However, after the successful intubation, the patient desaturated, which was mistakenly diagnosed and managed as bronchospasm. Recognition of pneumothorax was difficult at this point, as it was more challenging among asleep and anesthetized patients, owing to absence of subjective complaints.^{8,18} In addition, intraoperative signs and symptoms associated with pneumothorax are non-specific and could also be attributed to bronchospasm, ETT dislodgement and airway obstruction.^{8,19} The diagnosis can be confirmed through chest radiography. Another rapid diagnostic tool to confirm the diagnosis would be through point-of-care ultrasonography.²⁰ It was found to be more sensitive (87%) and specific (99%) compared to radiography in detecting pneumothorax.²¹ The absence of a characteristic gliding sign confirms the presence of pneumothorax. Although this technique offered more convenience and was relatively faster to perform, accurate diagnosis still depended on the skill of the operator.²⁰ During the incident, chest radiography was more readily available; hence, was used to confirm the diagnosis.

It is important to take note that the absence of a confirmatory diagnostic tool should not be the reason to delay the management. It is advisable to proceed with the live-saving interventions based on a high-index of suspicion, as further delay is lethal and catastrophic.^{18,19,22,23}

Pneumothorax occurs when there is a breach in the integrity of the visceral, parietal or mediastinal pleura.²⁴ In the case presented, the probable cause of parenchymal injury is lung hyperinflation. As described, the suction port was used to insufflate oxygen at 5 L/min. The internal diameter of the ETT relative to the external diameter of the fiberoptic bronchoscope is an important determining factor in the dynamics of air flowing through the suction port to the rest of airway.^{3,6,16} An ETT that is tightly fitted to a flexible

fiberoptic bronchoscope would limit escape of insufflated air. In this case, a pediatric flexible fiberoptic bronchoscope (Karl Storz Intubation Fiberscope 3.7 x 65) with a diameter of 3.7, pre-loaded with a wire-reinforced ETT with 5.0 mm internal diameter (Portex) was used. Current reports have not established yet a safe margin between the diameter of the FOB and inner diameter of ETT, but recommend a markedly smaller FOB size relative to the ETT.^{6,25} In this case, a smaller available option was a neonatal FOB (Karl Storz Intubation Fiberscope 2.8 x 5) with an outer diameter of 2.8 mm.

A high oxygen flow of 5 L/min through an FOB with a tightly fitting ETT allowed air entry that exceeded air escape. This led to lung hyperinflation and subsequently, pneumothorax.^{3,6,16} Regarding oxygen supplementation, the recommended replacement should only provide for the oxygen consumption of the lungs which is estimated to be at 3-5 mL/kg/min for a pediatric patient (54-90 mL/min for this patient).²⁴ There are no studies supporting a safe range of oxygen flow through the suction port for supplementation purposes yet, but current recommendations suggest a flow not more than 5-6 L/min in adults and even less in pediatric patients.^{6,16,25} Oxygen supplementation could have been delivered through other means such as continuous positive airway pressure, T-piece and nasal prongs applied on the other nares.^{6,26} An endoscopic mask could also deliver oxygen supplementation and allow passage of FOB through a port.²⁶

Air accumulated precipitously within the thoracic cavity due to controlled ventilation. Asleep patients receiving controlled ventilation do not generate negative pressure that counteracts the increasing effects of positive intrapleural pressure caused by pneumothorax. These patients receive inspiratory pressures of more than +20 cm H₂O.²⁴ With each positive pressure ventilation, more air passes through the pleural defect, contributing to the rapid accumulation of air.^{20,23,27} For this reason, asleep and ventilated patients exhibit deterioration earlier compared to awake, spontaneously breathing patients.

The rapid increase in intrapulmonary pressure creates a mechanical compression causing decreased venous return, decreased pulmonary artery pressure and subsequently, decreased cardiac output, exhibited in the case as abrupt desaturation and hemodynamic collapse.²⁴

In the setting of pneumothorax during mechanical ventilation, a pathway for air exit should be established immediately, as continuous air leak will rapidly progress to tension pneumothorax.^{20,23} Needle decompression is a therapeutic initial intervention, releasing the mechanical tension causing hypoventilation and hemodynamic collapse.²⁸⁻³⁰ This was immediately performed on the patient once pneumothorax was suspected. With the first bilateral needle decompression, the symptoms were initially relieved with a return of breath sounds. However, the therapeutic benefit of the intervention was only brief. This could be explained by the ongoing air leak through the pleural defect, which

exceeded the escape through a small bore needle.³⁰ There was also a suspicion of catheter kinking which prompted removal of the catheters for replacement. This, however, removed the only points of air exit, initiating re-accumulation of air and contributed to the patient's deterioration.²⁴ Using a large bore needle, such as gauge 14-18 could have prevented this.^{16,24,30}

It was, therefore, deemed necessary to maintain a patent pathway of exit for the accumulated air. Hence, the needle catheterization was maintained while waiting for the chest tube thoracostomy. The catheters were connected to a water seal in order to prevent air-reentry and aid in restoring the appropriate pressure within the pleural cavity.³¹ As seen in the case, this intervention was able to stabilize the patient while waiting for the definitive treatment.

CONCLUSION

Providing high flow oxygen supplementation via the pediatric fiberoptic suction port and use of a tight-fitting pediatric fiberoptic in an endotracheal tube during fiberoptic intubation can cause lung hyperinflation leading to iatrogenic tension pneumothorax. As demonstrated in this report, a high index of suspicion and immediate management deterred an otherwise fatal outcome.

Statement of Authorship

Both authors contributed to the conceptualization of work, acquisition and analysis of data, drafting and revising, and approval of the final version submitted.

Author Disclosure

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