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Diagnosis and treatment of tension pneumothorax under anesthesia: A case report

An 80-year old woman with a history of tracheal stenosis, tracheostomy, and 3 months of increasing respiratory distress underwent tracheal dilatation under general anesthesia with jet ventilation. Tracheal dilatation was successfully performed via suspension laryngoscopy and jet ventilation. During emergence the patient developed decreased oxygen saturation, hypotension, and respiratory distress, requiring intubation and ventilatory support. During tracheostomy, anterior chest subcutaneous emphysema led to a diagnosis of tension pneumothorax. Chest tube placement facilitated tracheostomy and improved ventilatory and circulatory parameters. This article discusses the diagnosis and treatment of a tension pneumothorax under general anesthesia.

Jet ventilation, spontaneous rupture of blebs or bullae, surgical trauma, or barotrauma are the 4 most likely explanations for a tension pneumothorax in this patient. Jet ventilation can cause pneumothorax, pneumomediastinum, or subcutaneous emphysema. Chronic obstructive pulmonary disease may cause blebs or bullae, which might rupture when exposed to positive pressure ventilation. Tissue trauma during dilatation or tracheostomy may cause a pneumothorax when positive pressure ventilation is employed. Barotrauma from high peak inspiratory pressure, rigid bronchoscopy, dilatation procedure, or jet ventilation may cause a pneumothorax. Prompt diagnosis and treatment will markedly decrease associated morbidity and mortality.

Key words: General anesthesia, jet ventilation, otolaryngology surgery, subcutaneous emphysema, tension pneumothorax.

The occurrence of a tension pneumothorax, especially while the patient is under general anesthesia, can be a life-threatening event. Onset can be subtle and slow or obvious and rapid, but the severity of an undiagnosed tension pneumothorax could ultimately lead to death if unrecognized and not immediately treated. The addition of positive pressure during mechanical ventilation can cause a pneumothorax to progress into a tension pneumothorax.

Vigilance during general anesthesia warrants the recognition of patients at risk for tension pneumothorax. Early recognition of clinical symptoms and prompt treatment will reduce morbidity and mortality. This article will discuss the occurrence, diagnosis, and treatment of a tension pneumothorax during general anesthesia.

Case summary

An 80-year-old woman, ASA physical status III (165 cm, 89 kg), was scheduled for tracheal dilatation because of subglottic stenosis and persistent stridor. Preoperative vital signs were heart rate, 106; blood pressure, 122/55 mm Hg; respirations, 20. Medical history included non–insulin-dependent diabetes, hypothyroidism, hypertension, angina, coronary artery disease, hiatal hernia, sleep apnea, and a remote significant smoking history. An echocardiogram indicated normal left ventricular function with moderate aortic insufficiency. The patient denied having drug allergies and was taking levothyroxine, furosemide, and lansoprazole. Previous surgeries included hysterectomy, cholecystectomy, right thyroidectomy, and 3-vessel coronary artery bypass graft in 1998. The recovery following the coronary artery bypass graft was complicated by respiratory insufficiency requiring tracheotomy and prolonged ventilator support. Just 6 months before the procedure described in this article, the patient's trachea was decannulated and the stoma surgically closed. However, in the 3 months before the procedure, the patient had become increasingly short of breath with any activity, which resulted in her transfer for further evaluation and treatment.

In the operating room the patient's electrocardiogram leads II and V5, blood pressure, and Sa_{O_2} were monitored. Following preoxygenation for 4 minutes, a Sellick maneuver was applied, and anesthesia was induced with 180 mg of sodium pentothal, 100 µg of fentanyl, and 80 mg of succinylcholine. Suspension laryngoscopy was performed by the otolaryngologist, and 0.8% end-expired concentration of isoflurane, supplemented by an additional 50 µg of fentanyl, 2 mg of midazolam, and 30 mg of rocuronium, was used to maintain anesthesia using jet ventilation. The jet ventilation was instituted with adequate chest excursion and oxygenation achieved between 20 to 25 cm water pressure. An F_{IO_2} of between 0.6 and 0.8 maintained oxygen saturation greater than 94% throughout the dilatation procedure.

At the conclusion of the procedure, neuromuscular blockade was fully reversed (verified by train-of-4 assessment and 5-second sustained tetany) with 3.0 mg neostigmine and 0.6 mg glycopyrrolate, and the patient was breathing spontaneously. The patient developed increased difficulty breathing during emergence. A mask and 100%

oxygen were used to assist the patient with ventilation and to improve oxygen saturation. Direct laryngoscopy revealed obstruction during exhalation above the level of the dilatation. The otolaryngologist decided that an endotracheal tube was needed and requested insertion of a small tube to avoid further tissue trauma and edema formation that could extend to the supraglottic region. A size 5.5 uncuffed endotracheal tube was atraumatically placed, resulting in equal breath sounds by auscultation. No air leak could be detected, and oxygen saturation rapidly improved.

Tidal volume was adequate at 8 mL/kg, and peak inspiratory pressures registered 23 to 26 cm of water pressure during ventilation. The electrocardiogram displayed sinus tachycardia at a rate of 115 to 130 beats per minute, which persisted after anesthesia was deepened with 1% end-expired concentration of isoflurane and 100 µg of fentanyl. Because of the patient's coronary artery disease history, a total of 5 mg metoprolol was titrated intravenously to achieve a heart rate in the 80 to 90 beat-per-minute range. Emergence was again attempted, but the patient was unable to maintain satisfactory oxygenation without assistance because of the small endotracheal tube. The otolaryngologist elected to perform a tracheostomy to eliminate potential airway obstruction.

Approximately 20 to 25 minutes after the beginning of the tracheostomy procedure, the patient had an episode of hypotension with a sustained systolic blood pressure reading of 68 mm Hg. Since she had been receiving nothing by mouth for several hours, and the short duration of the procedure precluded significant fluid replacement, it was decided to administer a 500-mL bolus of lactated Ringer's solution and 50 µg of phenylephrine. The systolic blood pressure improved to the 110 to 120 mm Hg range for approximately 15 minutes. The placement of the tracheostomy was difficult due to scar tissue and anatomical alterations from the previous tracheostomy. The tracheostomy tube was repeatedly placed into a false pocket, and oxygen saturation began to deteriorate. Aggressive manual ventilation with 100% oxygen was required to maintain oxygen saturation at 88%. Depression of the ST segment was noted on the electrocardiogram. Anesthesia and surgery personnel worked together to achieve optimal surgical access for placement of the tracheostomy tube. Despite multiple attempts, the tracheostomy tube was not successfully placed, and the patient began to demonstrate further hemodynamic deterioration. Additional intravenous access was obtained, and a second 500-mL bolus of lactated Ringer's solution was administered. Additional phenylephrine was titrated in 50-µg increments to maintain blood pressure, but little improvement was noted.

The difficulty in maintaining oxygenation was explained when palpation of the right chest wall revealed diffuse subcutaneous emphysema resulting in a differential diagnosis of pneumothorax. Absence of breath sounds on the right side and a chest radiograph confirmed the diagnosis. Chest tube placement and evacuation of intrapleural air resulted in significant improvement in cardiopulmonary status. The tracheostomy tube was then inserted successfully into the tracheal passage, evidenced by symmetrical chest expansion and positive end-tidal carbon dioxide.

The patient was transported to the intensive care unit in stable condition. There was no evidence of perioperative myocardial infarction as evidenced by stable 12-lead electrocardiogram and negative cardiac enzymes. On postoperative day 1, the patient was weaned off the ventilator and was breathing without difficulty on 0.40 F_{IO_2}. Breath sounds were equal bilaterally, and the chest radiograph showed that the right lung appeared well expanded. On postoperative day 2, the patient was transferred from the intensive care unit to the surgical floor. The patient's recovery course was uncomplicated, and she was discharged (with her tracheostomy) 8 days later.

Discussion

A pneumothorax is an accumulation of air within the pleural space that can occur in a number of situations. Pneumothorax is a potentially life-threatening event that may precipitate respiratory and/or cardiovascular collapse. A simple pneumothorax may develop into a tension pneumothorax when positive pressure ventilation is incorporated. A tension pneumothorax occurs when a 1-way leak into the pleural space causes an increase in intrapleural pressure. Cardiovascular involvement, ranging from hypotension to complete cardiovascular collapse, occurs as a result of compression of major vessels and is a late sign indicative of the presence of a tension pneumothorax. Diagnosis of tension pneumothorax can be difficult, and significant patient compromise may occur before the diagnosis is made. Signs and symptoms of a tension pneumothorax include decreased or absent breath sounds on the affected side, asymmetric chest movement, jugular venous distention, tracheal deviation and mediastinal shift, a reduced pulmonary compliance, and subcutaneous emphysema. Detection in an anesthetized patient presents a more difficult diagnostic challenge, with tachycardia and hypotension frequently being the first evident clinical signs.[1] During

general anesthesia, any sudden or unexplained deterioration in blood pressure, heart rate, oxygen saturation, or pulmonary compliance suggests of tension pneumothorax.[2]

Four possible etiologies could have caused this particular pneumothorax: jet ventilation, spontaneous rupture of blebs or bullae, surgical trauma during tracheostomy, or barotrauma from high ventilatory pressures. The questions of exactly when and how the pneumothorax occurred in this case remain unanswered.

Jet ventilation is an alternative method of ventilation that can be used as a short-term replacement for tracheal intubation. It is an ideal technique during laryngeal procedures, such as tracheal dilatation, tracheal resection, and bronchoscopy. Jet ventilation uses the Venturi effect and the Bernoulli principle to entrain room air with the anesthetic gas mixtures and deliver it by pressure through an injector at 2 to 12 L/min.[3] Simply stated, the Bernoulli principle incorporates flow and pressure. The greater the velocity (flow), the less lateral pressure is exerted on the wall of the tube (trachea). The jet ventilator is a portable, hand-operated device that can deliver oxygen and inhaled anesthetic agents via supraglottic or subglottic routes. In this case, supraglottic jet ventilation was instituted via a rigid bronchoscope. It is powered by regulated pipeline or cylinder oxygen pressure and can be adjusted by a controllable pressure-reducing valve and toggle switch. When the toggle switch is depressed, a jet of oxygen entering the bronchoscope entrains air, and the air-oxygen mixture resulting at the distal tip of the bronchoscope emerges at a pressure to provide adequate ventilation and oxygenation.[4] Inspiratory:expiratory ratio should allow for adequate passive exhalation, and pulse oximetry should be monitored continuously.[5]

Advantages of this type of system include uninterrupted ventilation, optimal surgical visualization, a motionless field, suitability for use in all age groups, and efficiency that allows for a longer bronchoscopic examination. Jet ventilation also has disadvantages. Disadvantages of jet ventilation include barotrauma (pneumothorax, pneumomediastinum, or subcutaneous emphysema), gastric dilation with possible regurgitation, drying of mucosal surfaces, and complete respiratory obstruction.[5] Another disadvantage is the variability of F_{IO_2} at the distal end of the bronchoscope secondary to entrainment of air during ventilation. Jet ventilation is most suited for patients with unobstructed airways and normal lung and chest wall compliance.[5] It may be difficult or impossible in extremely obese patients and patients with poor pulmonary compliance, making adequacy of ventilation difficult to assess.[6]

With this patient's history of smoking and respiratory difficulties prior to this procedure, the coexistence of chronic obstructive pulmonary disease and possible rupture of blebs or bullae must be considered. Chronic obstructive pulmonary disease is one of the most common pulmonary disorders encountered in anesthesia practice.[7] Its prevalence increases with age and is strongly associated with a history of cigarette smoking.[7] Patients can be asymptomatic or mildly symptomatic and exhibit expiratory airflow obstruction to varying degrees. Maldistribution of ventilation pulmonary blood flow results in shunt or dead space. The continued destruction of pulmonary capillaries and alveolar damage may cause development of blebs or bullae. Rupture of these alveolar blebs or bullae can result in pneumothorax.[4] Chronic obstructive pulmonary disease and its associated pathologies placed this patient at an increased risk of pneumothorax.

Inadvertent penetration or dissection of the trachea during tracheal dilatation procedures are possible causes of pneumothorax. During the dilatation described in this article, several dilating instruments of increasing size were introduced into the stenotic area. The otolaryngologist found tracheal dilatation to be technically difficult, and a small dissection of tracheal tissue cannot be definitively ruled out as a potential cause of the pneumothorax.

During tracheostomy, an incision is made transecting the trachea, at which time the tip of the tracheal tube is positioned cephalad to the incision. Ventilation during this period is difficult because of the large leak through the trachea.[8] Aggressive hand ventilation and high peak inspiratory pressures during surgical manipulation of the trachea could have caused dissection resulting in a 1-way leak. Increased airway resistance secondary to the stenotic tracheal lesion and narrow diameter of the endotracheal tube contributed greatly to our difficulty in maintaining adequate oxygenation.

Barotrauma is an all-inclusive term that ranges from subcutaneous emphysema to life-threatening complications such as pneumothorax and tension pneumothorax with the primary causative factor being high peak inspiratory pressure.[9] There is a high incidence of barotrauma associated with peak inflation pressures exceeding 40 cm water pressure. Insertion of a rigid bronchoscope can cause a significant positive end expiratory pressure effect and may result in barotrauma.[4] Provided there is not a tight fit between the bronchoscope and the airway, the risk of barotrauma is low.[4]

Persistent hypotension is a result of tension pneumothorax.[10] Episodes of hypotension during surgery can be attributed to several etiologies. During normal spontaneous ventilation, intrathoracic pressure is negative. Once positive pressure ventilation is established, a decrease in cardiac output may occur secondary to decreased preload; mean arterial pressure decreases as a result. Patients with myocardial dysfunction or valvular disease may experience a more profound effect during positive pressure ventilation.[11] In addition, patients with compromised intravascular volume may be more susceptible to these effects.[11] Prior to mechanical ventilation, the patient's blood pressure ranged from 100 to 135 mm Hg systolic, and hypotension did not occur until she was placed on the ventilator. Improvement in blood pressure after the fluid bolus led us to believe she was dehydrated. Also, isoflurane, fentanyl, and metoprolol, alone or in combination, could decrease sympathetic outflow leading to vasodilation and result in low blood pressure.

It should be emphasized that any patient who deteriorates under anesthesia and has wounds to the abdomen, lower neck, or ribs should be assumed to have a tension pneumothorax until proved otherwise.[12] Continuous assessment and monitoring of chest expansion and breath sounds at all times are necessary to diagnose airway complications such as tension pneumothorax in the early stages. Not only is it necessary to monitor for complications, it is a standard of care. An esophageal precordial stethoscope is useful in identifying changes in ventilation. An anesthetic combination of propofol or sodium thiopental with a short-acting opioid and a short-acting muscle relaxant, such as mivacurium or succinylcholine infusion, is appropriate for patients undergoing tracheal dilatation and bronchoscopy. Consider endotracheal intubation with a small microlaryngeal tube or uncuffed endotracheal tube following bronchoscopy. This will facilitate suctioning and allow the patient to recover gradually from anesthesia.

Conclusion

The occurrence of a tension pneumothorax, especially while under general anesthesia, can be a life-threatening event. The most likely explanations for a tension pneumothorax during jet ventilation are spontaneous rupture of blebs or bullae, surgical trauma, or barotrauma related to inspiratory pressure. Prompt diagnosis and treatment will markedly improve patient prognosis and significantly decrease morbidity and mortality, which is almost guaranteed if a tension pneumothorax is left untreated. This paper discussed the diagnosis and treatment of a tension pneumothorax under general anesthesia.

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