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Life-Threatening Suspected Intraoperative Bronchospasm During Tracheostomy in a Morbidly Obese Patient: A Case Report

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Abstract

Suspected severe intraoperative bronchospasm presenting with a silent chest and ventilation failure is a life-threatening emergency. We present a case of a morbidly obese patient (body mass index (BMI) 49.3 kg/m^2) who developed suspected severe bronchospasm accompanied by profound hypotension during tracheostomy following prolonged endotracheal intubation and was successfully managed with immediate intervention. This case highlights the importance of rapidly differentiating mechanical airway obstruction, perioperative anaphylaxis, and bronchospasm in severe ventilatory failure. Early recognition and prompt administration of intravenous (IV) epinephrine, along with proactive airway management, are critical in high-risk patients.

Categories: Anesthesiology
Keywords: bronchospasm, epinephrine injection, morbidly obese patient, prolonged intubation, tracheostomy

Introduction

Bronchospasm during general anesthesia can present across a wide clinical spectrum, ranging from mild expiratory wheezing and elevated airway pressures to severe, life-threatening respiratory failure [1]. It is relatively common, with a reported incidence of approximately 0.2% in the general surgical population, increasing to 2%-7% in patients with asthma or reactive airway disease [2]. Severe, life-threatening bronchospasm is much less common but may occur even in patients without a prior history of airway disease [1]. Differentiating bronchospasm from mechanical airway obstruction can be particularly challenging in an already intubated, obese patient during a tracheostomy, as demonstrated in this case [3]. Most notably, the presentation of a *silent chest* - characterized by a complete cessation of airflow without audible wheezing- together with a sudden drop in end-tidal carbon dioxide ($EtCO_2$) to near zero, resulting in a flat capnogram, constitutes a time-critical medical emergency. Importantly, both a silent chest and a flat capnogram may result from several life-threatening conditions, including acute mechanical airway obstruction, severe bronchospasm, and perioperative anaphylaxis. Without prompt recognition and immediate intervention, this catastrophic event can lead to fatal outcomes [4].

Reports of sudden intraoperative complete ventilation failure with a silent chest during tracheostomy in an already intubated, morbidly obese patient are scarce. This case highlights the diagnostic challenge of rapidly distinguishing mechanical airway obstruction from other life-threatening causes, including severe bronchospasm and perioperative anaphylaxis. We describe its recognition, differential diagnosis, and management.

Case Presentation

This case report was approved by the Institutional Review Board of Kosin University Gospel Hospital (IRB No. KUGH 2026-06-018), which waived the requirement for informed consent in accordance with the IRB policy for retrospective case reports. A 66-year-old morbidly obese female patient (height, 156 cm; weight, 120 kg; body mass index (BMI), 49.3 kg/m^2) was transferred to the operating room for an elective tracheostomy to facilitate prolonged ventilatory support. She was classified as American Society of Anesthesiologists physical status III. Her past medical history included hypertension, diabetes mellitus, and residual right hemiparesis secondary to an intracranial hemorrhage that had occurred 15 years previously. She had no history of asthma, chronic obstructive pulmonary disease, previous bronchospasm, atopy, drug allergy, or underlying cardiac disease and was a lifelong non-smoker. Preoperative transthoracic echocardiography demonstrated preserved left ventricular systolic function (ejection fraction, 72.6%) without significant structural or valvular abnormalities. Fifteen days before the tracheostomy, she had been admitted to the intensive care unit (ICU) with acute respiratory failure secondary to sepsis caused by left acute pyelonephritis, complicated by acute kidney injury. She required endotracheal intubation and mechanical ventilation. After clinical improvement, she was extubated four days before surgery. Despite high-flow nasal oxygen therapy initiated immediately after extubation, she developed tachypnea (respiratory rate, 30-40 breaths/minute) and agitation. Post-extubation laryngeal edema was suspected, and intravenous hydrocortisone (50 mg) was administered; however, her condition did not improve. She

subsequently developed sudden apnea with oxygen desaturation and hypotension, requiring emergent re-intubation. Because prolonged weaning from mechanical ventilation was anticipated, tracheostomy was planned. A preoperative chest radiograph revealed bilateral pleural effusion, pulmonary edema/pneumonitis in both lungs, cardiomegaly, and an endotracheal tube with its tip above the carina (Figure *1*).

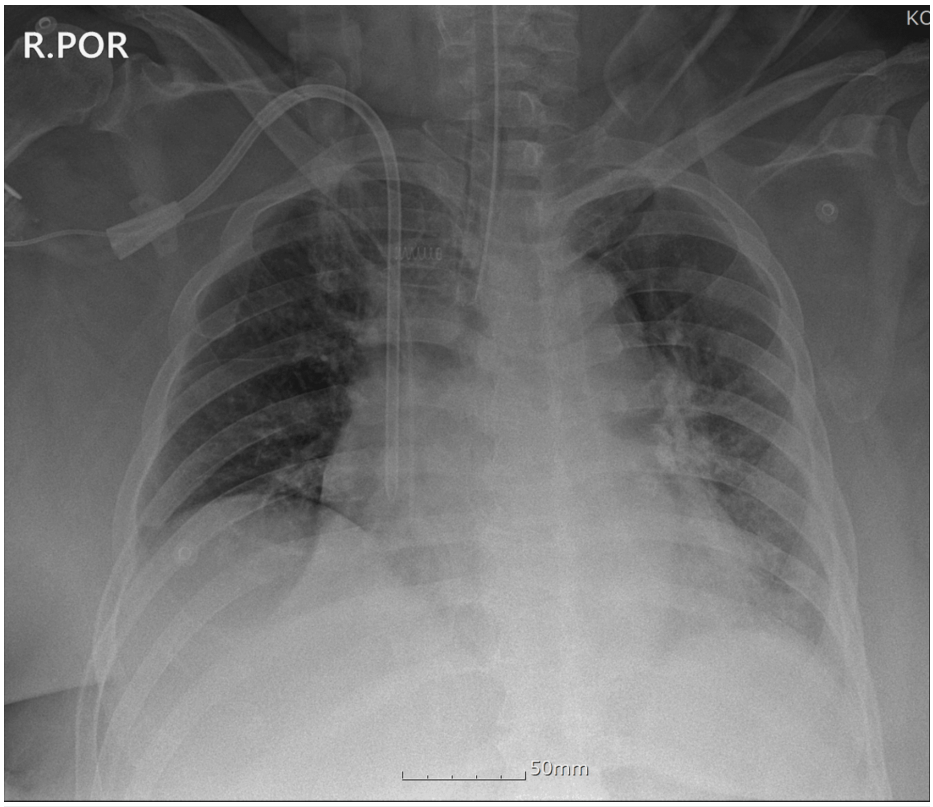


FIGURE 1: Preoperative chest radiograph.

The anteroposterior portable chest radiograph shows an endotracheal tube and a central venous catheter in place. Notable findings include cardiomegaly, diffuse bilateral opacities consistent with pulmonary edema or pneumonitis, and bilateral pleural effusions. The endotracheal tube tip is positioned above the carina.

On the day of surgery, the patient arrived at the operating room already intubated via the existing oral endotracheal tube, awake, and receiving manual ventilation with a bag-valve device. No premedication was administered. Pre-induction vital signs were stable: blood pressure (BP), 110/70 mmHg; heart rate (HR), 80 beats/minute; and peripheral oxygen saturation (SpO_2), 96%. General anesthesia was induced with propofol (120 mg), rocuronium (100 mg), and a remifentanil bolus (1 µg/kg), and was maintained with 2.0% sevoflurane and a continuous infusion of remifentanil (0.1 µg/kg/minute).

The depth of anesthesia was monitored using a Bispectral Index (BIS) monitor, with BIS values maintained between 30 and 60 throughout the surgery. Although pre-induction hemodynamics were within an acceptable range, a low-dose norepinephrine infusion (0.03-0.06 µg/kg/minute) was started prophylactically to mitigate vasodilatory hypotension associated with propofol and remifentanil administration and to maintain a mean arterial pressure above 70 mmHg in the setting of sepsis and recent acute kidney injury. Mechanical ventilation was set to a fraction of inspired oxygen (FiO_2) of 0.4, a respiratory rate of 15 breaths/minute, a tidal volume of 450 mL, and a positive end-expiratory pressure of 6 cmH2O. From the beginning of the surgery until the occurrence of the event, the $EtCO_2$ was maintained at 39-41 mmHg, peak inspiratory pressure (PIP) at 25 cmH_2O, and plateau pressure at 24 cmH_2O.

Thirty minutes after the start of surgery, the PIP increased to 30 cmH $_2$O, and the $EtCO_2$ waveform began to distort. At this time, vital signs were BP 121/69 mmHg, HR 98 beats/minute, and SpO_2 95%, but the $EtCO_2$ increased to 44 mmHg. Five minutes later, the $EtCO_2$ sharply dropped to 0 mmHg, presenting a flat capnogram. The surgical procedure was immediately halted. Suspecting a circuit disconnection or a mechanical obstruction, we inspected the endotracheal tube (ETT) and breathing circuit. The ETT was intact without any kinking and was securely connected to the breathing circuit, which in turn was appropriately connected to the mechanical ventilator.

We increased the FiO_2 to 1.0 with a fresh gas flow of 10 L/minute and switched the ventilation mode from volume-controlled to manual mode. However, manual positive pressure ventilation was completely impossible due to severe resistance, conveying a sensation of complete airway occlusion. The surgical field was assessed, and dissection had reached only the muscle layer due to the patient's thick cervical subcutaneous fat, with no exposure of the trachea. Lung auscultation revealed absent or severely diminished breath sounds bilaterally. The position and depth of the ETT were re-verified under video laryngoscopic visualization, confirming correct intratracheal placement. An endotracheal suction catheter passed easily through the full length of the tube and yielded a small amount of secretion, with no improvement in ventilation. To exclude residual neuromuscular blockade or chest wall rigidity, an additional 20 mg of rocuronium was administered without effect.

Differential diagnoses, including pneumothorax, mucus plugging, pulmonary embolism, and anaphylaxis, were considered and rapidly assessed at the bedside. Endobronchial tube migration was excluded by direct visualization, confirming appropriate ETT position. Mucus plugging was considered less likely given the minimal secretions and lack of response to suctioning. Pneumothorax was deemed unlikely based on the absence of unilateral clinical signs. Although pulmonary embolism and anaphylaxis were considered, the overall clinical context, including the intraoperative sudden respiratory deterioration and absence of characteristic supporting features, made these conditions less likely.

Four minutes after the onset of the event, the SpO_2 progressively declined to 69%, and the BP decreased to 90/25 mmHg with an HR of 100 beats/minute. Based on the overall clinical findings, severe bronchospasm was considered the most probable diagnosis in the clinical context at that time. Given the rapid respiratory and hemodynamic deterioration consistent with a peri-arrest state, 1 mg of IV epinephrine was administered as a resuscitative intervention (Figure *2*).

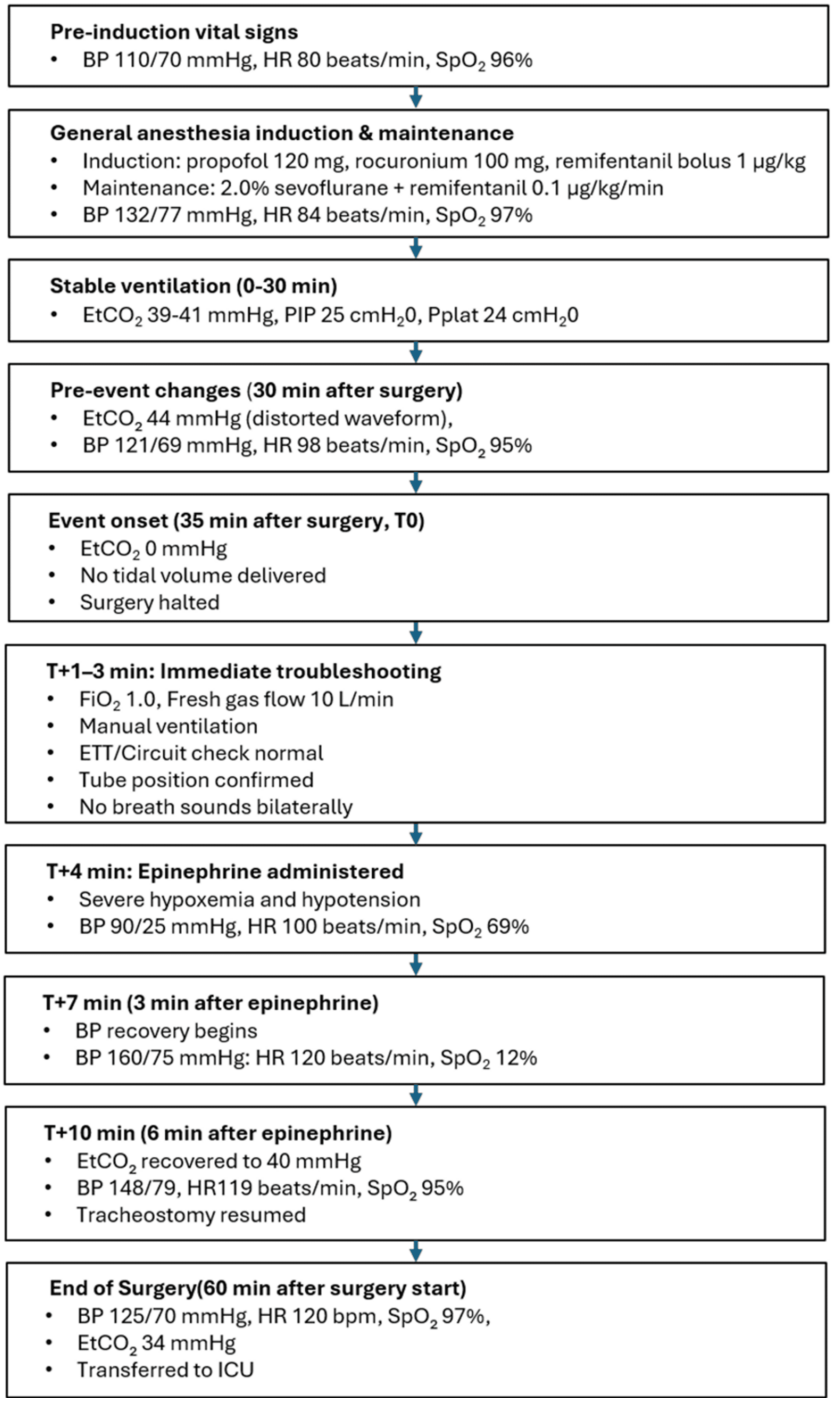


FIGURE 2: Clinical timeline of suspected intraoperative bronchospasm and management during tracheostomy.

The event onset (T0) occurred 35 minutes after surgery initiation. Changes in $EtCO_2$, oxygenation, hemodynamics, and key interventions are presented chronologically from anesthesia induction to ICU transfer.

Image created by the authors using Microsoft PowerPoint (Microsoft Corp., Redmond, WA).

BP, blood pressure; HR, heart rate; SpO_2, peripheral oxygen saturation; $EtCO_2$, end-tidal carbon dioxide; FiO_2, fraction of inspired oxygen; FGF, fresh gas flow; ETT, endotracheal tube; PIP, peak inspiratory pressure; Pplat, plateau pressure; ICU, intensive care unit

Two minutes after epinephrine administration, BP and SpO_2 remained critically low at 85/25 mmHg and 27%, respectively, with an HR of 105 beats/minute. Three minutes after epinephrine administration, BP

rebounded to 160/75 mmHg, and HR increased to 120 beats/minute, although SpO_2 remained extremely low at 12%. Six minutes after epinephrine administration, the extreme airway resistance gradually subsided during continuous manual ventilation attempts, and $EtCO_2$ reappeared on the capnogram. The capnographic waveform progressively normalized, with the $EtCO_2$ recovering to 40 mmHg. SpO_2 rebounded to over 95% (Figure 3).

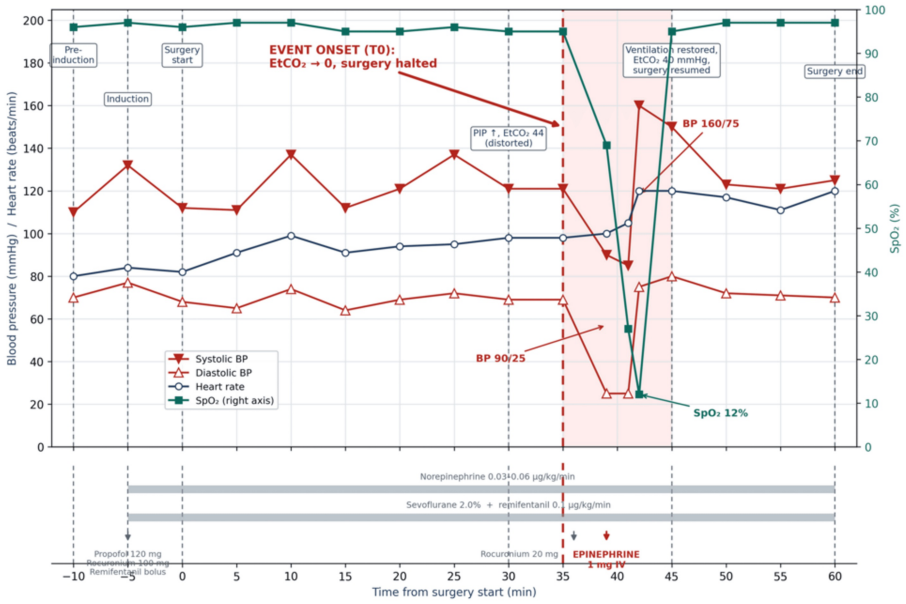


FIGURE 3: Intraoperative hemodynamic trends and SpO_2 during the event.

Intraoperative trends in blood pressure (BP; systolic and diastolic), heart rate, and SpO_2 (right axis) during the event, plotted against time from the start of surgery. The shaded band marks the critical period from event onset (T0, 35 minutes; $EtCO_2 \rightarrow 0$, surgery halted) to restoration of ventilation and resumption of surgery at 45 minutes. BP (nadir 90/25 mmHg) and SpO_2 (nadir 12%) recovered rapidly after 1 mg of intravenous epinephrine. Continuous and bolus medications are shown in the lower panel.

$EtCO_2$, end-tidal carbon dioxide; PIP, peak inspiratory pressure; BP, blood pressure; SpO_2, peripheral oxygen saturation

The patient rapidly improved following immediate airway management and intravenous epinephrine administration. Therefore, formal activation of the extracorporeal membrane oxygenation protocol or consultation with additional surgical teams was not deemed necessary in this clinical context.

An arterial blood gas analysis obtained approximately 10 minutes after the event, after ventilation had been restored, showed pH 7.279, $PaCO_2$ 48.2 mmHg, PaO_2 73.1 mmHg, $HCO3^-$ 22.1 mmol/L, base excess -4.8 mmol/L, and SaO_2 92.8%, suggestive of a mixed respiratory and mild metabolic acidosis.

Although the surgery had been paused to minimize surgical stimulation, the surgeon resumed the tracheostomy after confirming the recovery of the patient's hemodynamic status and oxygenation. Because the life-threatening airway event had occurred immediately beforehand, once the trachea was opened, the existing orotracheal tube was promptly exchanged for a cuffed endotracheal tube inserted through the tracheostomy stoma, prioritizing rapid re-establishment of a secure airway over placement of a formal tracheostomy tube at that stage. The removed tube showed no evidence of obstruction along its length. After stabilization, anesthesia was re-established with sevoflurane and remifentanil, and an adequate anesthetic depth was maintained under BIS monitoring throughout the remainder of the procedure, which was completed uneventfully approximately 10 minutes later.

Before transfer from the operating room, the final vital signs were BP of 125/70 mmHg, HR of 120 beats/minute, SpO_2 of 97%, and $EtCO_2$ of 34 mmHg. Postoperatively, to minimize further airway stimulation, the patient was transferred to the ICU in a sedated state (BIS 44) on a dexmedetomidine infusion, with the endotracheal tube maintained through the tracheostomy stoma.

In the ICU, she recovered steadily with gradual sedation tapering. She was liberated from mechanical

ventilation on postoperative day 7, maintaining SpO_2 96% on room air, after which the endotracheal tube was exchanged for a Portex tracheostomy tube. The tracheostomy tube was removed on postoperative day 20, and the tracheostomy stoma was surgically closed before hospital discharge. The patient recovered without significant neurological complications and was discharged on postoperative day 30.

Discussion

Sudden intraoperative loss of breath sounds (silent chest) and ventilation failure represent a diagnostic challenge, as they may be associated with several life-threatening conditions, including severe bronchospasm, perioperative anaphylaxis, and mechanical airway obstruction such as endotracheal tube kinking or secretion-related blockage [1]. Furthermore, failure to initiate appropriate early treatment can lead to profound hypotension and irreversible brain damage [4].

As in this case, when $EtCO_2$ is lost, peak pressure increases, and ventilation becomes difficult during surgery, the possibility of bronchospasm should be considered; FiO_2 should immediately be increased to 1.0, ventilation switched to manual bagging, and the surgery, along with any other stimulating activities, must be halted [1]. Next, mechanical obstruction must be ruled out. A kinked, blocked (by secretion, mucous plug, or cuff herniation), or misplaced tracheal tube, or an occlusion in the breathing circuit can mimic the symptoms of bronchospasm and must always be checked first [1,5]. In our case, we checked the breathing circuit, inspected the endotracheal tube for kinking, and verified the tube position using a video laryngoscope. Importantly, an endotracheal suction catheter passed freely through the entire length of the tube while aspirating secretions. We also inspected the removed tube for blockade. Because rapidly confirming or ruling out mechanical obstruction is crucial in such emergencies, our medical team performed these assessments simultaneously to systematically exclude potential mechanical causes. Taken together, these assessments enabled the rapid exclusion of mechanical ob■ struction and facilitated consideration of alternative causes of ventilation failure. To make this rapid troubleshooting process clearer, we summarize our systematic assessment protocol in Figure *4*.

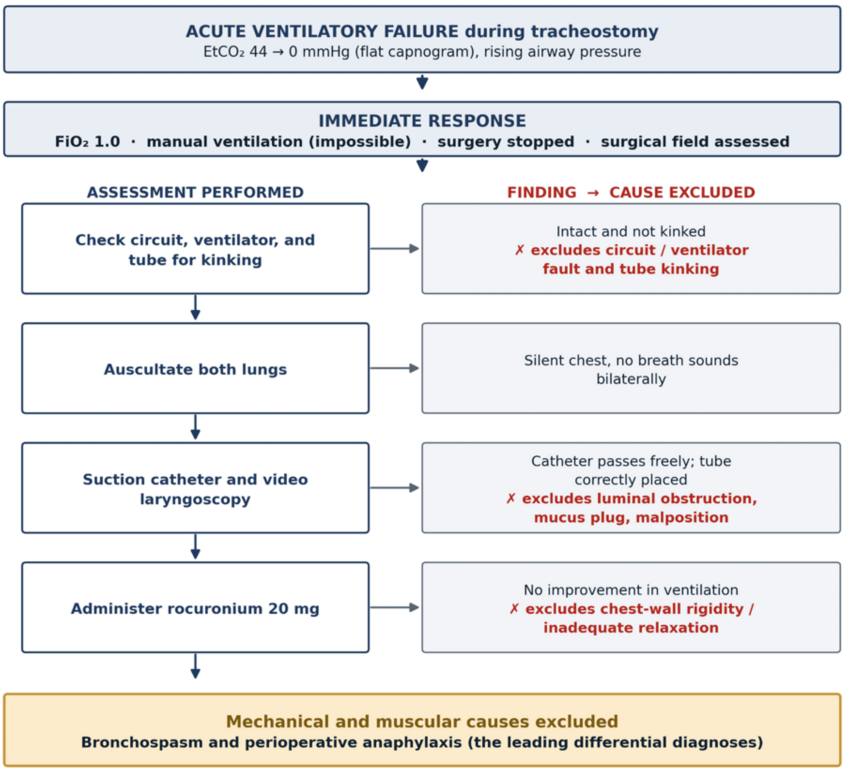


FIGURE 4: Systematic, stepwise exclusion of causes of the acute intraoperative ventilatory failure in the present case.

After the immediate response (FiO_2 1.0, manual ventilation, cessation of surgery, assessment of the surgical field), the breathing circuit, ventilator, and endotracheal tube were checked (intact, not kinked); the lungs were auscultated (silent chest bilaterally); a suction catheter and video laryngoscopy confirmed a patent, correctly positioned tube; and a further dose of rocuronium gave no improvement, excluding a muscular cause. With mechanical and muscular causes excluded, bronchospasm and perioperative anaphylaxis remained the leading differential diagnoses.

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$EtCO_2$, end-tidal carbon dioxide; FiO_2, fraction of inspired oxygen

Once mechanical obstruction has been ruled out, bronchospasm should be suspected, and immediate management should include deepening anesthesia using intravenous or inhalational agents such as propofol, ketamine, or sevoflurane, in addition to adjuncts such as intravenous lidocaine and dexmedetomidine that may reduce airway reactivity and improve bronchial tone, while simultaneously calling for help and initiating pharmacological treatment [1]. Under most clinical circumstances, inhaled salbutamol is recommended as a first-line treatment for intraoperative bronchospasm, and increasing the concentration of a volatile anesthetic such as sevoflurane, a potent bronchodilator, remains an appropriate early intervention when even limited ventilation may still allow delivery of the agent [1]. Although $EtCO_2$ is typically elevated during airflow obstruction, such as bronchospasm, if airflow obstruction becomes sufficiently severe to produce near-complete airway obstruction, effective alveolar ventilation may be markedly reduced, resulting in a transient loss of the capnogram until ventilation is restored. In our patient, however, the silent chest was consistent with near-complete cessation of airflow, which likely rendered inhaled or volatile therapies unreliable; therefore, intravenous therapy was prioritized as escalation treatment to ensure rapid and dependable drug delivery [6]. With the patient in a peri-arrest state (SpO_2 12%-27%, BP 85/25 mmHg) but maintaining a perfusing rhythm under general anesthesia with immediately available IV and full monitoring, we administered a 1-mg IV bolus of epinephrine due to imminent cardiovascular collapse. Although intramuscular epinephrine is generally recommended as the first-line route in most cases of anaphylaxis, IV administration may be considered in life-threatening peri-arrest conditions such as severe bronchospasm with hemodynamic collapse in the operating room, where rapid onset and titratable effects are required. In this case, it was administered as a rescue intervention rather than a routine first-line therapy.

Severe bronchospasm and grade 3-4 perioperative anaphylaxis can present almost identically, including

bronchoconstriction, abrupt loss of $EtCO_2$, and profound hypotension, often without cutaneous manifestations; therefore, anaphylaxis was considered an important differential diagnosis [7]. Indeed, the absence of skin findings does not exclude anaphylaxis, as cutaneous signs may be absent in severe reactions or obscured intraoperatively [7]. In our patient, no urticaria, erythema, or angioedema was observed; however, such manifestations may be absent even in severe anaphylaxis, and their absence does not definitively rule out this diagnosis [7].

Although the 35-minute delay in symptom onset and the subsequent clinical recovery, despite administration of an additional 20 mg of rocuronium during the event to exclude chest-wall rigidity, makes rocuronium-induced anaphylaxis less likely, it cannot be completely excluded in the absence of confirmatory diagnostic testing, including serum tryptase measurement and formal allergy evaluation [8].

In considering possible causes, no new perioperative allergen was identified at the time of deterioration. The prophylactic antibiotic had been administered earlier after a negative skin test, and no colloids or chlorhexidine were used. The patient also had a strong predisposition to airway hyperreactivity, including prolonged intubation, recent re-intubation with suspected laryngeal edema, pneumonitis, and morbid obesity.

Taken together, the clinical findings may be more consistent with severe bronchospasm; however, perioperative anaphylaxis remains an important differential diagnosis and cannot be definitively excluded in the absence of biochemical confirmation, including serum tryptase measurement.

The β2-agonist effect of epinephrine dilates the obstructed airways, while its α1 and β1 effects potently increase the depressed myocardial contractility and blood pressure, thereby restoring circulation to the brain and systemic organs [4,9]. In our case, the extreme airway resistance resolved and the $EtCO_2$ waveform recovered following IV epinephrine administration; however, the clinical response was delayed by approximately 6 minutes. This delay is most plausibly explained by impaired drug delivery during profound circulatory failure (BP 90/25 mmHg, essentially a low-flow shock state): blood pressure recovered first, followed by resolution of airway resistance and return of the capnogram, a sequence consistent with restored circulation enabling the drug to reach airway β2-receptors [10,11]. Particularly in the setting of profound hypotension, a delay in clinical effect may occur because of impaired delivery of the administered drug to its target receptors. Accordingly, early administration of epinephrine should be strongly considered when bronchospasm is suspected, and blood pressure is progressively decreasing [10,11]. Prompt administration of epinephrine may be critical in preventing further deterioration and may have important implications for patient survival [12].

The patient had been intubated for 15 days before surgery and underwent extubation followed by re-intubation four days preoperatively. Preoperative chest radiography demonstrated pneumonitis. This pre-existing pneumonitis may have contributed to airway inflammation and increased airway hyperresponsiveness, potentially making the patient more susceptible to bronchospasm during airway manipulation. Furthermore, a prolonged indwelling endotracheal tube can act as a persistent foreign body within the airway [13], and the resulting mechanical compression induces chronic inflammation and edema of the airway mucosa [14]. This chronic inflammatory environment leads to an abnormal increase in mucus secretion, rendering the airway even more hyperreactive [15]. In this case, surgical stimulation during the subcutaneous dissection phase, as well as other airway-related manipulations, may have served as a possible trigger for reflex bronchoconstriction in an already markedly hyperreactive airway [12]. Therefore, in high-risk patients, the possibility of bronchospasm should always be anticipated, and appropriate preparatory measures should be implemented.

Morbid obesity itself acts as a risk factor for bronchospasm due to increased airway hyperresponsiveness resulting from chronic systemic inflammation [16]. In addition, the excessive adipose tissue accumulated in the chest wall and abdomen significantly reduces chest wall compliance [17]. When combined with the extreme increase in airway resistance during severe bronchospasm, this may result in critically difficult manual ventilation [17]. Furthermore, with a BMI of 49.3 kg/m^2, the patient in this case exhibited a markedly reduced functional residual capacity and increased basal oxygen consumption relative to metabolic demand, predisposing to rapid oxygen desaturation during apnea [17]. The abrupt decrease in oxygen saturation to 12% during the silent chest event, characterized by complete loss of breath sounds due to bronchospasm, likely reflects these physiological characteristics of morbid obesity [4,18]. Moreover, air trapping-induced elevation of intrathoracic pressure may have impaired venous return, contributing to hypotension. Together with severe hypoxemia, this can precipitate life-threatening circulatory collapse [1]. Overall, this case highlights that morbid obesity may amplify both the development of bronchospasm and the rapidity of subsequent respiratory and hemodynamic deterioration once severe airway obstruction occurs.

Conclusions

This case describes a suspected intraoperative bronchospasm in a patient with multiple perioperative risk factors, including morbid obesity and a complex clinical course requiring prolonged airway management. However, perioperative anaphylaxis could not be definitively excluded and remains an important differential

diagnosis given the overlapping clinical features. This case highlights the importance of maintaining a broad differential diagnosis when sudden, severe airway and hemodynamic compromise occur during anesthesia. Rapid recognition, continuous monitoring, and prompt management are essential in such high-risk situations to ensure timely and appropriate treatment and to prevent progression to life-threatening respiratory and circulatory collapse.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Donghee Kang, Ju Deok Kim, Dongyul Lee, Ji Wook Kim

Critical review of the manuscript for important intellectual content: Donghee Kang

Supervision: Donghee Kang, Ju Deok Kim

Drafting of the manuscript: Ju Deok Kim, Dongyul Lee, Ji Wook Kim

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. The Institutional Review Board (IRB) of Kosin University Gospel Hospital issued approval KUGH 2026-06-018. This case report was approved by the Institutional Review Board of Kosin University Gospel Hospital (IRB No. KUGH 2026-06-018), which waived the requirement for informed consent in accordance with the IRB policy for retrospective case reports. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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