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Anesthetic Management of Intraoperative Carcinoid Crisis During Rigid Bronchoscopy in a Patient with Pulmonary Carcinoid Tumor and Bronchial Asthma

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KEYWORDS

Pulmonary carcinoid, carcinoid crisis, rigid bronchoscopy, octreotide, TIVA, bronchial asthma, shared airway

ABSTRACT:

Background: Pulmonary carcinoid tumors are rare, slow-growing neuroendocrine neoplasms. Though often indolent, they can precipitate carcinoid crisis during surgical manipulation, especially in central airway tumors.

Case Presentation: A 31-year-old female with bronchial asthma presented with progressive dyspnea. Imaging and bronchoscopy identified an endobronchial mass in the left main bronchus. Histopathology confirmed a typical carcinoid tumor. Despite normal chromogranin A and serotonin levels, persistent hypotension and tachycardia raised suspicion for subclinical vasoactive activity. The patient underwent rigid bronchoscopy under total intravenous anesthesia (TIVA) using propofol (Schneider model) and a supraglottic airway. During tumor manipulation, she developed acute bronchospasm, hypotension, and tachycardia suggestive of a carcinoid crisis. Immediate management with salbutamol, ketamine, and octreotide stabilized the patient, and resection was completed uneventfully.

Discussion: This case underscores the anesthetic challenges in managing pulmonary carcinoid tumors in asthmatic patients. TIVA minimized histamine release, and ketamine provided bronchodilation and cardiovascular support. Octreotide was crucial for aborting the crisis.

Conclusion: Anticipatory anesthetic planning, intraoperative vigilance, and early octreotide use are key in managing intraoperative carcinoid crisis during shared airway procedures.

INTRODUCTION

Carcinoid tumors of the lung are rare, well-differentiated neuroendocrine neoplasms that originate from neuroendocrine Kulchitsky cells in the bronchial epithelium. Though they account for only a small fraction of pulmonary tumors, their potential to secrete biologically active substances such as serotonin, histamine, bradykinin, and kallikrein poses significant perioperative challenges. These tumors are histologically classified into typical (low-grade) and atypical (intermediate-grade) variants based on mitotic activity and necrosis.[1] While typical carcinoids are often indolent and slow-growing, they are by no means inert; direct manipulation, especially during surgical or bronchoscopic procedures, can stimulate sudden release of vasoactive mediators. This can lead to an intraoperative carcinoid crisis a rare but life-threatening syndrome characterized by profound hypotension, bronchospasm, tachyarrhythmias, and flushing[2].

The anesthetic management of carcinoid tumors becomes increasingly complex when the lesion is located in the central airway, requiring interventions such as rigid bronchoscopy. These procedures present a unique set of challenges due to the need for shared airway access,

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limited control over ventilation, and increased risk of tumor handling-induced mediator release[1]. The situation becomes even more precarious in patients with underlying reactive airway disease, such as bronchial asthma. Airway hyper responsiveness in such individuals heightens the risk of perioperative bronchospasm, complicating oxygenation and ventilation, and increasing the likelihood of hemodynamic instability during the procedure[3-5].

Moreover, predicting the occurrence of a carcinoid crisis remains difficult. Although biomarkers such as serum chromogranin A and serotonin are commonly used to evaluate neuroendocrine activity, they are often unreliable, particularly in pulmonary carcinoid tumors[6]. Many patients remain asymptomatic or biochemically silent, yet may still harbor active neuroendocrine tumors capable of releasing potent mediators under stress. This clinical unpredictability places a greater onus on the anesthesiologist to maintain a high index of suspicion and prepare proactively for the potential onset of carcinoid crisis—even in the absence of traditional laboratory markers or syndromic manifestations[4].

The case presented here is exceptional in several respects. It involves a young female patient with a histologically confirmed typical pulmonary carcinoid tumor, who developed a severe intraoperative carcinoid crisis during rigid bronchoscopy, despite normal preoperative chromogranin A and serotonin levels[7]. Complicating the clinical picture was a pre-existing diagnosis of bronchial asthma, which posed an additional risk of bronchospasm during airway manipulation. The crisis manifested acutely as hypotension, tachycardia, and bronchospasm—requiring immediate, multifaceted intervention. A carefully preplanned anesthetic strategy, including total intravenous anesthesia with propofol, bronchodilator therapy with ketamine and salbutamol, and immediate administration of intravenous octreotide, led to rapid stabilization and successful completion of the tumor debulking procedure[8].

This case underscores the unpredictable nature of carcinoid tumors, the inadequacy of relying solely on biomarkers for risk stratification, and the critical importance of anticipatory anesthetic planning[1]. It also highlights the effectiveness of combining propofol-based TIVA, ketamine, and octreotide in managing intraoperative carcinoid crisis within the constraints of a shared airway procedure in a patient with comorbid reactive airway disease. As such, it contributes valuable clinical insights into the nuanced anesthetic management of pulmonary carcinoid tumors in high-risk patients[9].

Patient Information

Age/Gender: 31-year-old female

Medical History: Known case of bronchial asthma since adolescence

Presenting Symptoms: Progressive exertional dyspnea

Family and Social History: Non-smoker, no relevant family history

Medications: Inhaled salbutamol and budesonide

Clinical Findings

Respiratory rate: 24/min

Saturation: 95% on room air

Auscultation: Bilateral air entry present

Blood pressure: 92/58 mmHg

Heart rate: 104 bpm

Diagnostic Assessment

Chest CT: Mass obstructing distal left main bronchus

Flexible bronchoscopy: left main Endobronchial lesion with partial occlusion

Histopathology: Typical pulmonary carcinoid

Serum markers: Chromogranin A and serotonin within normal limits

Diagnosis: Biochemically silent, hormonally active typical carcinoid with reactive airway disease

THERAPEUTIC INTERVENTION

The patient was posted for rigid bronchoscopy and tumor debulking of a histologically confirmed typical carcinoid tumor located in the distal left main bronchus. Although she lacked the classic clinical features of carcinoid syndrome such as flushing, diarrhea, or wheezing directly attributable to tumor activity, her preoperative vital signs raised clinical concern. She consistently exhibited low-normal blood pressure (MAP 60–65 mmHg) and intermittent resting sinus tachycardia (HR 100–120 bpm) despite being well-hydrated, afebrile, and

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without pain or anxiety. These subtle hemodynamic patterns were deemed unusual for a young, otherwise healthy individual and raised a suspicion for subclinical hormonal activity, possibly due to intermittent tumor-mediated release of vasoactive substances like serotonin or kallikrein. Importantly, serum chromogranin A and serotonin levels were within normal range, making biochemical correlation elusive.

Given the tumor's central airway location, the shared airway nature of rigid bronchoscopy, and the patient's comorbid bronchial asthma, the anesthetic plan was developed with a dual focus on maintaining bronchoprotection and rapid access to airway rescue strategies. Preoperatively, she received salbutamol nebulization for bronchodilation. Equipment and medications for managing carcinoid crisis including octreotide, corticosteroids (hydrocortisone), ketamine, and vasopressors were preloaded and available at the bedside. A second-generation supraglottic airway (SGA) was prepared for immediate insertion post-scope removal or in the event of difficult mask ventilation.

Anesthesia induction was carried out using total intravenous anesthesia (TIVA) with propofol titrated via the Schneider model, avoiding volatile anesthetics known for histamine release, which could exacerbate neuroendocrine mediator effects. Following preoxygenation, rapid sequence induction with succinylcholine facilitated smooth and rapid airway control. Manual ventilation was maintained through the bronchoscope throughout the procedure.

Approximately 10 minutes into direct tumor manipulation, the patient suddenly developed acute bronchospasm, characterized by high airway pressures, silent chest, reduced tidal volumes, and progressive desaturation. This was quickly followed by profound hypotension (MAP <50 mmHg) and tachycardia exceeding 140 bpm. The initial differential diagnosis favored an asthma-related bronchospastic event precipitated by airway instrumentation. Immediate interventions were initiated: the TIVA infusion was deepened, multiple inhaled salbutamol puffs were delivered via the bronchoscope, and intravenous ketamine 1 mg/kg was administered to achieve bronchodilation and cardiovascular support.

Recognizing the possibility of mediator-induced inflammation, 100 mg of intravenous hydrocortisone was also administered to blunt the inflammatory response and reduce the risk of biphasic bronchospasm. Despite these measures, the patient's bronchospasm and hypotension remained refractory, and ventilation was critically impaired with saturation dropping to 89-92%

At this juncture, the preoperative hemodynamic profile and the absence of expected response to bronchodilators. A high likelihood of intraoperative carcinoid crisis was considered likely triggered by direct tumor manipulation causing sudden release of serotonin, bradykinin, and histamine into systemic circulation. Acting on this suspicion, a 100 µg IV bolus of octreotide was promptly administered. After 3 minutes, with only minimal clinical improvement, a second 100 µg bolus was given, followed by initiation of a continuous octreotide infusion at 50 µg/hour.

Over the subsequent 5–7 minutes, the patient showed progressive and definitive improvement in both respiratory and cardiovascular parameters. Bronchospasm resolved as evidenced by normalization of airway pressures, improved compliance, and return of bilateral breath sounds. Hemodynamically, her MAP rose steadily above 75 mmHg, heart rate normalized to 90–100 bpm, and $EtCO_2$ returned to baseline. The dramatic resolution following octreotide administration strongly implicated neuroendocrine mediator release as the underlying etiology of the crisis.

Octreotide, a synthetic somatostatin analog, exerts its effect by binding to somatostatin receptor subtypes 2 and 5 (SSTR2 and SSTR5) on neuroendocrine tumor cells, thereby inhibiting the release of serotonin, histamine, bradykinin, and kallikrein. These mediators are known to cause bronchial smooth muscle contraction, capillary leakage, and profound vasodilation—hallmark features of carcinoid crisis. The failure of bronchodilators, corticosteroids, and ketamine alone to relieve bronchospasm, and the subsequent response to octreotide, provided diagnostic and therapeutic confirmation of the carcinoid origin of the crisis.

Once the tumor resection was completed, the rigid bronchoscope was carefully withdrawn, and the pre-positioned SGA was inserted to maintain airway patency and ventilation. The patient resumed spontaneous ventilation smoothly as the depth of anesthesia was tapered. Oxygen saturation, tidal volumes, and respiratory rate normalized without any further

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requirement for invasive airway support. She was extubated uneventfully, transferred to a high-dependency recovery area, and monitored closely.

The postoperative course remained stable, with no recurrence of bronchospasm, hypotension, or neuroendocrine features. The octreotide infusion was tapered over 12 hours postoperatively. The patient was discharged on postoperative day 3, and a repeat bronchoscopy performed at 4 weeks confirmed complete tumor resection with no residual disease. She remains under scheduled follow-up with pulmonology and endocrinology teams.

Follow-up and Outcomes

Immediate postoperative recovery stable

No recurrence of bronchospasm or hypotension

Discharged on postoperative day 3

Follow-up bronchoscopy after 4 weeks: No recurrence or residual lesion

Advised routine surveillance

DISCUSSION

Pulmonary carcinoid tumors, although generally considered low-grade neoplasms, present significant perioperative risks due to their potential to release vasoactive substances such as serotonin, histamine, bradykinin, and kallikrein, especially during direct surgical manipulation. While carcinoid syndrome is classically associated with gastrointestinal neuroendocrine tumors, bronchial carcinoids particularly typical variants are often considered biologically indolent and biochemically silent[10-12]. However, this case challenges that assumption and underscores the capacity of typical pulmonary carcinoids to behave aggressively in the intraoperative setting despite the absence of elevated chromogranin A or serotonin levels. The patient had no preoperative symptoms of carcinoid syndrome, but persistent low-normal blood pressure and episodic sinus tachycardia raised subtle concerns about underlying neurohumoral activity. Intraoperatively, the sudden onset of acute bronchospasm, severe hypotension, and tachycardia during tumor manipulation could have initially been mistaken for an asthma exacerbation, particularly given the patient's bronchial asthma[13]. However, the refractory response to bronchodilators, corticosteroids, and ketamine led to timely reconsideration of the etiology as carcinoid crisis. This progression is consistent with prior literature by Kinney et al. and Warner et al., who reported that mediator release can occur independently of classical carcinoid syndrome features or abnormal laboratory biomarkers[4-5]. Notably, the predominant manifestation in our case was bronchospasm, rather than flushing or diarrhea, which is atypical in carcinoid crisis and poses a diagnostic dilemma when compounded by coexisting asthma. Furthermore, rigid bronchoscopy introduces anesthetic limitations due to shared airway control, restricted access to ventilation, and inability to promptly intubate or suction in the event of crisis. This reinforces the need for anticipatory pharmacologic planning and airway rescue strategies in such procedures. The use of total intravenous anesthesia (TIVA) with propofol was preferred over volatile agents due to its favorable hemodynamic profile and absence of histamine release, which may otherwise exacerbate neuroendocrine activity[14]. Ketamine served a dual role as a bronchodilator and inotropic agent but proved insufficient when the bronchospasm was hormonally mediated. The definitive reversal occurred only after two sequential boluses of octreotide followed by a continuous infusion, highlighting the central role of octreotide in the management of carcinoid crisis[15]. Octreotide, a somatostatin analog, inhibits the release of neuroendocrine peptides by binding to somatostatin receptor subtypes—particularly SSTR2 and SSTR5—on tumor cells, thereby halting the downstream cascade of bronchial smooth muscle contraction and systemic vasodilation. The requirement for multiple boluses before stabilization in this case suggests a high hormonal burden or rapid mediator turnover triggered by tumor manipulation. This pharmacologic response aligns with prior recommendations by Ruszniewski et al.[7], who advocated for prophylactic and therapeutic use of octreotide in neuroendocrine tumor surgery. However, what differentiates this case from previous reports is the diagnostic ambiguity created by comorbid asthma, the absence of biochemical markers, and the presentation of carcinoid crisis as a refractory bronchospasm in a shared-airway procedure[16-18]. Most published cases involve gastrointestinal carcinoids or known syndromic presentations, whereas this case involved a typical carcinoid tumor in a young female with subtle

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preoperative hemodynamic fluctuations and a crisis precipitated intraoperatively without biochemical warning. The successful management without airway conversion or procedure abandonment further underscores the importance of multidisciplinary planning, preoperative pharmacologic preparation, and intraoperative vigilance[19]. In summary, this case contributes to the evolving understanding of pulmonary carcinoid tumors by demonstrating that even biochemically silent, typical carcinoids may behave unpredictably under procedural stress, and that octreotide remains the cornerstone of crisis reversal when standard therapy fails[20]. It advocates for a paradigm shift in risk stratification, moving beyond biochemical parameters to incorporate clinical pattern recognition and tumor manipulation risk, particularly in patients undergoing rigid bronchoscopy with comorbid reactive airway disease.

CONCLUSION

This case exemplifies the multifaceted complexity and diagnostic challenge of managing a biochemically silent yet clinically aggressive pulmonary carcinoid tumor during rigid bronchoscopy, particularly in a patient with comorbid bronchial asthma. The absence of classical carcinoid syndrome symptoms and normal preoperative neuroendocrine biomarkers may provide a false sense of security, leading clinicians to underestimate the risk of intraoperative carcinoid crisis. However, this case reinforces the critical understanding that typical carcinoid tumors, despite their indolent histology, can demonstrate unpredictable neurohumoral behavior under surgical stress, especially when located in the central airway where tumor manipulation is inevitable.

One of the most enlightening aspects of this case was the initial misdirection toward bronchospasm secondary to asthma, a plausible but incomplete diagnosis, which delayed definitive treatment. The refractoriness to conventional bronchodilators and steroids, coupled with hemodynamic collapse, served as vital clinical clues prompting the shift in diagnosis to carcinoid crisis a shift that ultimately altered the therapeutic trajectory and saved the patient from further deterioration. The rapid and sustained response to octreotide underscores its indispensable role in the treatment algorithm for intraoperative carcinoid crisis, even in the absence of prior syndromic evidence.

From an anesthetic perspective, this case emphasizes the importance of anticipatory planning, pharmacologic readiness, and the selection of TIVA over volatile agents in patients with suspected neuroendocrine tumors. It also highlights the anesthesiologist's pivotal role in early recognition of atypical intraoperative events, especially in shared airway scenarios where control is limited and rapid deterioration may outpace conventional interventions.

Importantly, this case urges clinicians to broaden their clinical vigilance beyond biochemical parameters, incorporating subtle hemodynamic patterns, tumor location, and procedural risk as key indicators for preoperative risk stratification. The coexistence of bronchial asthma served as a confounding variable, and its presence should heighten, rather than dilute, suspicion of crisis when symptoms escalate intraoperatively.

Ultimately, this case contributes valuable insight into the anesthetic and perioperative management of pulmonary carcinoid tumors and calls for a revised clinical approach that places emphasis on preparedness over prediction. It affirms that clinical judgment, real-time adaptation, and multidisciplinary coordination are often more powerful than laboratory data in managing life-threatening complications during high-risk thoracic procedures.

Patient Perspective

The patient expressed gratitude for the anticipatory care and timely management, reporting significant improvement in breathing and quality of life after tumor resection.

Informed Consent

Written informed consent was obtained from the patient for publication of this case and associated images.

Ethical Approval

According to the policy of our institution, individual case reports do not require review or approval by the institutional ethics committee. However, written informed consent was obtained from the patient for the publication of this case report and any accompanying clinical details.

Conflicts of Interest

None declared.

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