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Anesthetic Considerations for a Unique Presentation of Acute Tophaceous Pulmonary Gout: A Case Report

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Abstract

Gout is a common inflammatory condition characterized by deposition of crystals in tissues throughout the body. Diagnosis is based on clinical features and may be confirmed by visualization of negatively birefringent monosodium urate crystals under polarized light microscopy. In cases of gout, polarized light microscopy can reveal negatively birefringent monosodium urate crystals. Though gout is common in joints few cases have been described elsewhere in the body. This paper describes a male patient at his early 80s who developed acute tophaceous pulmonary gout. This case report will highlight an uncommon manifestation of gout and discuss the anesthetic management considerations for these patients.

Glossary of Terms

CT: Computerized Tomography; VATS: Video-Assisted Thoracoscopy; DLT: Double Lumen Tube; NSAIDs: Non-Steroid Anti-Inflammatory; ASA: American Society of Anesthesiologists

Introduction

Acute gout flares, which are characterized by uric acid plasms concentrations > 6.8 mg/dL, typically present with severe monoarticular pain, erythema, and swelling. At least 80% of initial flares involve a single joint, most often at the base of the first metatarsophalangeal joint or the knee [1]. On rare occasions, however, uric acid may build up in other parts of the body.

Hyperuricemia is a predisposing factor for gout development and it ultimately stems from one of two pathways: Under excretion of urate or overproduction of urate [2]. Hyperuricemia is also associated with an increased risk of hypertension, metabolic syndrome, type 2 diabetes, coronary artery disease, left ventricular hypertrophy, atrial fibrillation, myocardial infarction, stroke, heart failure, and chronic kidney disease [2,3]. Risk factors for hyperuricemia include male gender, advanced age, obesity, purine-rich diet, alcohol use, hypertension, diuretic use, congestive heart failure, and chronic kidney disease [4].

Gout-associated lung disease is a rare type of gout that typically presents with pleural effusions and tophaceous gout buildup in the lungs. Symptoms can present similarly to those of interstitial lung disease, with shortness of breath, labored breathing, chest pain, dry cough, and wheezing. Gout, particularly in the lungs, is important to diagnose before surgery as it can influence a patient's welfare throughout the perioperative period. Therefore, anesthesia providers must be mindful of a diagnosis of gout when determining an anesthetic plan, which may include the preoperative selection of gout prophylaxis, the intraoperative choice of anesthetics, and the postoperative management of painful flares.

Case Presentation

A male patient at his early 80s was admitted to the emergency department for worsening shortness of breath and orthopnea. His medical history included atrial fibrillation, cardiomyopathy, type 2 diabetes mellitus, stage 3 chronic kidney disease, and hypertension. A computed tomography (CT) scan showed a large pericardial effusion and left pleural effusion, which prompted placement of chest tube and performance of a subxiphoid pericardial window the following day. Nearly 1600 cc of blood-tinged fluid was removed; subsequent cytology did not provide any further diagnoses. The patient began exhibiting signs of clinical improvement, and so the chest tube was removed one day after its initial placement and he was discharged home.

The patient initially did well at home, but he slowly began to redevelop worsening shortness of breath. Twenty-seven days after surgery, he re-presented to the emergency department with increased shortness of breath. On physical examination temperature was 37 °C, heart rate (HR) was 72 beats/min, respiratory rate

(RR) was 19 per minute, noninvasive blood pressure (NBP) was 134/68 mmHg, SaO_2 was 94% on room air. He had an elevated white blood cell count of 12 × 10^9/L, elevated lactate level of 3.1 mmol/L, creatinine level of 1.6 mg/dL, uric acid level of 10.2 mg/dL, and BNP level of 3900.

The chest X-ray performed in the emergency department revealed near complete opacification of the left hemithorax, which had worsened since the previous visit. A CT scan revealed a large left pleural effusion with compression atelectasis of the left lung. On auscultation, breath sounds were decreased on the left side. He was placed on a 3 L/minute nasal cannula and saturated at 97% to 100% oxygen. The patient was admitted overnight for a video-assisted thoracoscopy (VATS) and re-insertion of the chest tube. He had preoperative transthoracic echocardiography, which showed normal systolic function, LVEF 45%, left atrium and right atrium dilation, small circumferential pericardial effusion, no evidence of hemodynamic compromise.

Anesthesia was induced with intravenous propofol (2 mg/kg), rocuronium, and fentanyl. A 35F left-sided double lumen tube (DLT) was inserted without difficulty. The placement of the DLT was confirmed using a bronchoscopy. Anesthesia was maintained with inhaled sevoflurane and oxygen.

During the VATS, 2000 cc of blood-tinged fluid was drained from the left pleural cavity. The left lung was found to be friable, with deposits of thick white material covering the pleura. Dense adhesions were present between the lower lobe of the left lung and the parietal pleura. Polyserositis with involvement of pericardium and lungs was seen. The lung was biopsied, and samples of the white material were collected for cytologic review. The patient's vital signs were stable during the surgery. Three chest tubes were placed: 1 anteriorly, 1 posterior to the apex, and 1 in the middle of the diaphragm area. The patient was extubated at the end of the surgery and transferred to the post-anesthetic care unit.

Surgical pathologic review of the white material found non-necrotizing granulomatous changes with abundant negatively birefringent crystalline material. The crystalline material was coarse, with red compensated polarization and characteristics of calcium-pyrophosphate-dihydrate. The fluid from the pleura had a normal white blood cell count, with no inflammatory cells. Surgical pathology findings and physical findings confirmed the diagnosis of pulmonary tophaceous gout. Endocrinology, rheumatology, and infectious disease services assisted with managing this acute flare for the remainder of the patient's hospitalization. He began receiving 40 mg of methylprednisolone every 8 hours for 2 days, then his dose transitioned to 8 mg twice daily. Additionally, he began receiving 0.6 mg of colchicine and 200 mg of allopurinol for maintenance. The patient was discharged in postoperative day 10. There is no violation of HIPPA for this case report.

Discussion

Gout has a reported worldwide prevalence ranging from 0.1% to 10% [4]. The disease is characterized by monosodium urate crystal deposition with a potential for buildup, which causes tophi. The patient discussed in this report had a unique case characterized by an atypical initial presentation of a recurring pulmonary effusion coupled with a lack of prior personal or family history of gout. The patient had multiple risk factors for hyperuricemia (male, advanced age, hypertension, DM, cardiomyopathy, and chronic kidney disease). Pericardial effusion and plural effusion were diagnosed in the emergency department. We suspected that the patient's pericardial effusion was caused by uremic pericarditis. This assumption was based on the data showing high incidence of uremic pericarditis among patients with untreated chronic kidney disease [5]. Many patients with uremic pericarditis are asymptomatic, and a pericardial fluid cytologic exam is not diagnostic [5]. This patient's initial hospital admission for pericardial effusion was not pulmonary gout presentation.

Most of hyperuricemic patients (85%-90%) could have asymptomatic monosodium urate (MSU) crystal deposits [6]. Alcohol intake, purine-rich meals, and heavy meals can all induce acute MSU crystallization [7]. These studies may explain our patient's clinical presentations. He had asymptomatic MSU deposits in his pleura by hyperuricemia. The alcohol intake or heavy meals triggered the acute gout flare and second emergency room visit. Ultimately, however, modern understanding of the pathogenesis of gout is still incomplete [8].

To our knowledge, pleural effusion as the initial presentation of gout has not been previously reported. Pleural effusions are diagnosed with a chest radiograph, thoracentesis, and chest tube placement. If thoracentesis does not provide a definitive diagnosis, further investigation is recommended using VATS with pleural and lung biopsies [9]. This patient's pulmonary gout diagnosis was unknown before surgery, it was challenging for the anesthesia providers to develop specific anesthetic plans for pulmonary gout preoperatively. During the surgery, the surgeon identified the crystals and suggested the diagnosis, anesthesia providers quickly modified the anesthetic plan. The anesthesia providers may consider pulmonary gout as a differential diagnosis if the patient has multiple risk factors for gout. Depositions of monosodium urate crystals can induce an acute inflammatory response; therefore, it can pose a significant risk for intraoperative and postoperative complications.

During the preoperative period, anesthesia providers must obtain a thorough patient history, especially of the cardiac, pulmonary and renal diseases, to ensure that the patient's comorbidities have been optimized for an elective procedure. Preoperative laboratory evaluations should include a complete blood count, metabolic panel, hemoglobin A1C, electrocardiogram, workup for coronary artery disease, and chest radiography. If a patient has uncontrolled hypertension, diabetes, renal failure, or suspected ischemic heart diseases, consult with a specialist accordingly to evaluate and optimize management of the medical condition before surgery.

Colchicine should be discontinued the morning of the surgery because of the risk of kidney injury and immune system suppression via inhibition of microtubules, but allopurinol may be continued [10]. Though fasting is important for reducing aspiration, prolonged fasting and starvation for patients with gout may alter uricacid levels. Patients should be encouraged to eat a light meal at least 6 hours before anesthesia is administered; in addition, patients should be encouraged to consume clear fluids, including oral carbohydrate drinks, up until two 2 hours before anesthesia initiation.

Intraoperatively, Standard American Society of Anesthesiologists (ASA) monitoring procedures should be followed. Invasive blood pressure monitoring should be considered for patients with severe cardiovascular and pulmonary diseases. A central venous line can be considered for high-risk surgical patients and critically ill patients.

The use of intraoperative anesthetic drugs must also be carefully considered. One study found that, at 1, 2, and 3 hours after induction and on postoperative day 1, the concentration of urinary uric acid was significantly increased with use of propofol compared to use of sevoflurane [11]. Gout's effects on the pharmacokinetics and pharmacodynamics of other anesthetic agents, such as etomidate, ketamine, muscle relaxants, or local anesthetics, has not yet been studied. The surgery itself may also precipitate a gout flare.

Intraoperatively, blood loss, fluids, and temperature regulation are all important are all important aspects to consider avoiding gout flare-up. Excessive blood loss can cause metabolic acidosis and change systemic pH, which increases uric acid formation. Maintaining normothermia and avoiding hypothermia are also important because decreased temperature can theoretically precipitate crystal formation at an increased rate compared to normothermia and can increase vessel vasoconstriction. Regarding fluid control, intravascular volume expansion from fluid infusion can dilute uric acid and decrease urate reabsorption, but the absence of fluid infusions will do the opposite and increase risk of gout flare [12]. If a patient has renal impairment, kidney function can be preserved by maintaining normovolemia and normal kidney perfusion. Sodium bicarbonate can be used to alkalinize the urine and reduce uric acid levels [13]. Increased length of surgical time is also associated with gout flares [12].

Postoperatively, one study found that approximately 44.3% of patients with known history of gout developed gout flares during the postsurgical period at a mean interval of 3.7 days after surgery [10]. However, the authors did not investigate the relationship between anesthesia and postsurgical gout flares [10]. Furthermore, 2 risk factors were identified for postsurgical gout flare: presurgical uric acid level ≥ 9 mg/dL and the magnitude of uric acid level changes before and after surgery [12].

An anesthesia provider's treatment of gout flares focuses on eliminating excess uric acid via non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine. Pain management medications for gout include NSAIDs, glucocorticoids, and colchicine. If pain is not controlled by non-opioid medications, then narcotics are recommended [14]. Postoperative nausea and vomiting can be prevented by multimodal prophylaxis- i.e, administering simultaneously administering multiple antiemetics, such as ondansetron, scopolamine patches, and dexamethasone. Presurgical control of serum uric acid levels and prophylactic administration of colchicine will help prevent gout attacks postoperatively [12].

Conclusion

This patient had a unique presentation of tophaceous gout in the lung pleura. When managing lung disease of unknown etiology, practitioners should consider pulmonary gout as a differential diagnosis. This patient's hyperuricemia, though previously asymptomatic, was likely tied to underlying chronic kidney disease that prohibited proper excretion of uric acid. Despite the patient's negative personal and family history for gout, gout was diagnosed via analysis of pleural fluid. For anesthesiologists caring for a patient with a history of gout, key modifications in anesthetic care plans are essential throughout the entire perioperative period to avoid or minimize acute gout flares.

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Conflicts of Interest

None.

Author Contributions

Lauren Pomerantz helped with performing the literature review, writing the report, and editing the report; Benjamin Eslahpazir helped with collecting the case data, editing the report, and obtaining consent; Evan Huang helped writing the report, and editing the report; Jeffrey Huang was the attending anesthesiologist on the case and helped with performing the literature review, writing the report, editing the report, and obtaining consent.

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