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Case Report

Severe *Staphylococcus aureus* Pneumonia With Bronchial Mucosal Necrosis in an Electronic Cigarette User: A Case Report

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ABSTRACT

Severe *Staphylococcus aureus* pneumonia and electronic cigarette or vaping product use-associated lung injury (EVALI) may present with overlapping clinical and radiological findings, complicating etiological assessment. We report the case of a 29-year-old woman with a history of smoking, e-cigarette use and cannabis use who developed extensive S. aureus pneumonia complicated by acute respiratory failure. Chest computed tomography demonstrated diffuse bronchial wall thickening, small-airway filling, tree-in-bud centrilobular opacities and extensive multifocal consolidations. The patient required invasive mechanical ventilation and underwent extubation on the second hospital day, followed by reintubation on the third hospital day because of respiratory deterioration. Her course was complicated by ventilator asynchronies and spontaneous pneumothorax requiring chest drainage. Bronchoscopy on the fifth hospital day revealed bronchial mucosal edema, inflammation and necrosis, with necrotic material. Tracheal secretion and bronchial lavage cultures identified S. aureus, with growth exceeding 10,000 colony-forming units per milliliter in the bronchial lavage. Treatment included meropenem and vancomycin, subsequent antimicrobial adjustment according to susceptibility testing, individualized ventilatory support and management of the pleural complication. The reported outcome was favorable, with sufficient respiratory recovery to discontinue invasive mechanical ventilation and continue rehabilitation. Vaping-related lung injury could have coexisted with bacterial pneumonia. This case highlights the importance of microbiological investigation, serial reassessment and an integrated approach to severe pulmonary disease in patients with a history of vaping.

Keywords: Acute respiratory failure; Bronchial mucosal necrosis; Community-acquired pneumonia; Electronic cigarettes; Mechanical ventilation; *Staphylococcus aureus*

Introduction

Community-acquired pneumonia (CAP) remains an important cause of morbidity and mortality and may progress to acute respiratory failure requiring invasive ventilatory support. Among the etiological agents associated with severe presentations, *Staphylococcus aureus* is notable for its ability to cause rapidly progressive pulmonary infection, with multilobar involvement and, in certain cases, parenchymal necrosis, cavitation and pleural complications[1-3]. Necrotizing forms represent particularly severe presentations, requiring early recognition, microbiological identification and appropriate antimicrobial treatment[3].

Concurrently, the increasing use of electronic cigarettes (e-cigarettes) has raised concerns regarding their acute and chronic respiratory effects. E-cigarette or vaping product use-associated lung injury (EVALI) was recognized during the outbreak that occurred in the United States in 2019. This syndrome may manifest as fever, cough, dyspnea, chest pain, hypoxemia and extensive radiological abnormalities, with presentations ranging from pulmonary infiltrates to severe respiratory failure[4-9]. Although the association between tetrahydrocannabinol (THC)-containing products and the EVALI outbreak has been extensively documented, particularly regarding the presence of vitamin E acetate in certain formulations, other components and mechanisms of lung injury remain under investigation[6,8,10,11].

The evaluation of patients with suspected vaping-related lung injury is particularly challenging because of the absence of a specific diagnostic test and the overlap of clinical and radiological manifestations with infectious diseases. EVALI is considered a diagnosis of exclusion, requiring investigation of alternative or concomitant causes of lung injury, including bacterial and viral infections[4-7,12,13]. In this context, identification of an infectious agent does not automatically exclude the possibility of concomitant vaping-related lung injury, provided that clinical evidence supports this hypothesis. However, e-cigarette exposure alone does not establish a diagnosis of EVALI or allow causality to be attributed to the observed lung injury[4-6].

The association between severe bacterial pneumonia, exposure to vaping products and pulmonary complications therefore constitutes a clinical scenario requiring an integrated diagnostic and therapeutic approach. Progression to acute respiratory failure may require invasive mechanical ventilation, continuous monitoring of respiratory mechanics and interventions directed at complications, while microbiological identification guides adjustment of antibiotic therapy[3,14,15]. Additionally, spontaneous pneumothorax (barotrauma) in patients with severe pulmonary infection may represent a relevant complication, particularly in cases associated with destruction of lung tissue[1,3].

In this context, the present report describes the case of a young adult woman with a history of smoking, e-cigarette use and cannabis use who was admitted with extensive *Staphylococcus aureus* pneumonia, acute respiratory failure, extubation failure, necrotizing changes in the bronchial mucosa and pneumothorax. The objective is to present the clinical course, radiological and microbiological findings, complications during ventilatory support and outcome after treatment, discussing the relevance of investigating vaping exposure in patients with severe pulmonary involvement.

Case Presentation

A 29-year-old woman with a history of bipolar disorder was receiving pharmacological treatment with topiramate 50 mg at night, lamotrigine 100 mg once daily and divalproex sodium 500 mg, one tablet in the morning and two at night. She had an 11-year history of smoking, together with e-cigarette and cannabis use. She was referred from an urgent care unit after several days of marked fatigue, myalgia and unmeasured fever, followed by rhinorrhea, dry cough, progressive dyspnea and severe right-sided chest pain that was respiration-dependent and exacerbated by certain movements. At the referring unit, she exhibited lip cyanosis, tachypnea and a peripheral oxygen saturation of 83% on room air. Initial therapeutic measures included salbutamol, ipratropium bromide, oseltamivir, ceftriaxone, azithromycin and hydrocortisone. Rapid tests for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and influenza were negative. Despite these measures, respiratory failure progressed, requiring orotracheal intubation and invasive mechanical ventilation.

On the first hospital day, chest computed tomography (CT) **(Figure 1)** demonstrated diffuse bronchial wall thickening, small-airway filling and centrilobular opacities with a tree-in-bud pattern. Extensive areas of consolidation were identified, involving almost the entire left lower lobe, as well as the medial segment of the middle lobe, the posterior segment of the right upper lobe and the basal segments of the right lower lobe. The pleural spaces were described as potential spaces. The main pulmonary artery was normal in caliber and no filling defects were observed in the evaluated pulmonary arteries, including their lobar and segmental branches. There was no lymphadenopathy and cardiac dimensions were normal. Collectively, the findings were consistent with extensive infectious pulmonary involvement, with multifocal and small-airway involvement. The radiological patterns described in the literature on e-cigarette-associated lung injury are heterogeneous and may overlap with other pulmonary diseases, including infections[6,7,13].

On the second hospital day, in view of the initial clinical course orotracheal extubation was performed. However, the patient developed respiratory deterioration over the subsequent 24 hours, progressing on the third hospital day to respiratory fatigue and worsening blood gas parameters. The heart rate, acidosis, consciousness, oxygenation and respiratory rate (HACOR) score was 6 points .[14] Following discussion with the patient and her family regarding the clinical deterioration and the need to reinstitute invasive ventilatory support orotracheal reintubation was performed.

Mechanical ventilation was delivered in volume-controlled mode, with a tidal volume of 360 mL, positive end-expiratory pressure (PEEP) of 8 cmH_2O, respiratory rate of 28 breaths per minute and fraction of inspired oxygen (FiO_2) of 35%. Peak pressure was 29 cmH_2O, plateau pressure was 24 cmH_2O and driving pressure was 16 cmH_2O. The recorded minute ventilation was 10.1 L/min, with an inspiratory-to-expiratory ratio of 1:1.7. Ventilator asynchronies were observed, including premature cycling and double triggering, along with an airway resistance of 13.8cmH_2O/L/s and respiratory compliance of 22mL/cmH_2O, according to the clinical records. The ratio of arterial oxygen partial pressure (PaO_2) to the fraction of inspired oxygen (PaO_2/FiO_2) was 171, indicating substantial impairment

of oxygenation. Considering the severity of pulmonary involvement, suspected preexisting structural abnormalities and the unsatisfactory response to ventilatory measures, an individualized ventilatory strategy was adopted, with a low tidal volume and a higher respiratory rate, together with continued sedation and neuromuscular blockade for approximately 24 hours. Protective ventilation strategies are recommended in the management of patients with acute respiratory distress syndrome (ARDS), with parameters individualized according to the clinical condition and respiratory mechanics[15].

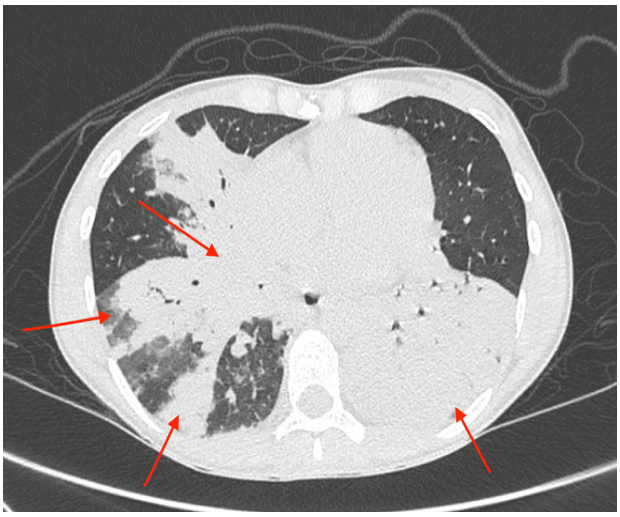


Figure 1: Chest computed tomography obtained on the first hospital day. Extensive multifocal consolidations involving almost the entire left lower lobe, the medial segment of the right middle lobe, the posterior segment of the right upper lobe and the basal segments of the right lower lobe, accompanied by diffuse bronchial wall thickening, small-airway filling and centrilobular tree-in-bud opacities. These findings are consistent with extensive pulmonary infection and associated small-airway involvement. Red arrows indicate areas of pulmonary opacity and consolidation visible in this image.

Also on the third hospital day, a tracheal secretion culture identified *Staphylococcus aureus*. Given the severity of the condition and the microbiological result, antibiotic therapy with meropenem and vancomycin was initiated. The patient developed worsening ventilatory status, requiring further adjustments to ventilator settings and continued neuromuscular blockade. During the investigation of respiratory deterioration, bedside lung ultrasonography (point-of-care ultrasound, POCUS), followed by chest radiography, demonstrated findings suggestive of spontaneous pneumothorax due to barotrauma. Chest drainage was performed to treat the pleural complication and support respiratory stabilization. Pneumothorax and other pleuropulmonary complications have been described in reports of severe staphylococcal pneumonia, including necrotizing presentations[1,3].

On the fifth hospital day, bronchoscopy **(Figure 2)** revealed edema, inflammation and necrosis of the bronchial mucosa. A substantial amount of purulent secretions and necrotic material was observed in the airways, particularly in the upper lobes and extensive suctioning and bronchoalveolar lavage were performed. The bronchial lavage culture yielded *Staphylococcus aureus* at more than 10,000 colony-forming units per milliliter (CFU/mL), corroborating the identification of the organism previously isolated from the tracheal secretions. The literature describes severe presentations of *S. aureus* pneumonia, including necrotizing forms, although characterization of these presentations depends on integrating clinical, microbiological, radiological and, when available, histopathological findings[1-3]. Following the microbiological and antimicrobial susceptibility testing results, antibiotic therapy was adjusted according to the susceptibility profile of the isolate.

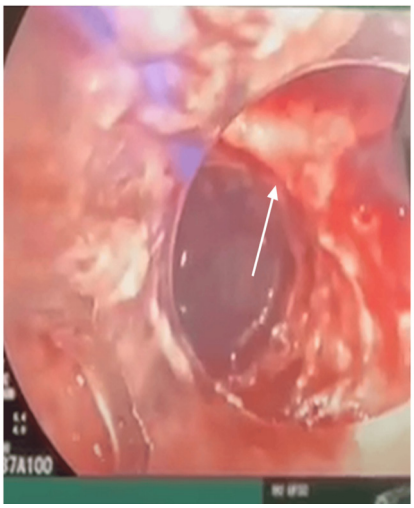


Figure 2: Bronchoscopic findings on the fifth hospital day. Edematous and erythematous bronchial mucosa with adherent whitish material, consistent with the purulent secretions and mucosal necrosis described in the bronchoscopy report. The white arrow indicates an area of bronchial mucosa.

The reported final outcome was favorable, with sufficient respiratory recovery to discontinue invasive mechanical ventilation and continue the rehabilitation process.

Discussion

Severe *Staphylococcus aureus* pneumonia and e-cigarette-associated lung injury are diagnoses that may share clinical and radiological manifestations, making etiological assessment particularly challenging. EVALI (e-cigarette or vaping product use-associated lung injury) was recognized as a syndrome of acute lung injury associated with the use of electronic vaping devices, with presentations that include cough, dyspnea, chest pain, fever, hypoxemia and pulmonary infiltrates. The absence of a specific diagnostic marker and the overlap with respiratory infections require systematic investigation of alternative and concomitant causes[4-7].

The association between EVALI and tetrahydrocannabinol (THC)-containing products was particularly relevant during the 2019 outbreak in the United States. Identification of vitamin E acetate in the bronchoalveolar lavage fluid of affected patients provided important evidence regarding the involvement of this compound in many of the investigated cases, although it does not allow the same exposure to be attributed to all patients with vaping-associated lung injury[6,8,10]. Furthermore, the histopathological patterns described in EVALI include acute lung injury, diffuse alveolar damage and organizing pneumonia, frequently with predominantly small-airway changes. However, these findings are nonspecific and must be interpreted in the clinical and microbiological context[6,12].

In the present case, the history of e-cigarette and cannabis use, together with acute respiratory failure, extensive pulmonary consolidations and endobronchial abnormalities, makes it

pertinent to consider a possible contribution of vaping exposure to the severity of pulmonary involvement. However, the tracheal secretion and bronchial lavage cultures, with *S. aureus* growth exceeding 10,000 CFU/mL, support the relevance of a bacterial infection. Thus, the condition cannot retrospectively be classified as confirmed EVALI based solely on the available data. The hypothesis of coexisting vaping-associated lung injury and bacterial pneumonia remains possible but unproven.

Another important aspect is the possibility of staphylococcal pneumonia with necrotizing features. Some strains of *S. aureus*, including those producing Panton-Valentine leukocidin, are associated with severe presentations involving multilobar consolidations, tissue destruction and respiratory complications. However, the presence of bronchial mucosal necrosis on bronchoscopy does not, by itself, establish pulmonary parenchymal necrosis and neither virulence factor testing results nor a description of cavitation on computed tomography were available[1-3].

The clinical course was also marked by extubation failure, the need for reintubation, ventilator asynchronies and pneumothorax. The HACOR score of 6 points contributed to the assessment of respiratory deterioration, while identification of pneumothorax by bedside ultrasonography, followed by radiographic confirmation and chest drainage, illustrates the importance of serial reassessment in critically ill patients. Ventilation with reduced tidal volumes and control of inspiratory pressures is consistent with the principles of protective ventilation in patients with acute respiratory distress syndrome, although the indication and parameters should be individualized according to pulmonary mechanics and the clinical condition[14,15].

Finally, clinical improvement following targeted antimicrobial treatment, management of the pleural complication and ventilatory support reinforces the importance of an integrated approach. The literature on EVALI nevertheless highlights persistent gaps concerning the mechanisms of injury, long-term pulmonary consequences and interactions between vaping exposures and respiratory infections. In this context, the case contributes to the discussion regarding the need to systematically investigate e-cigarette use in young adults admitted with respiratory failure and extensive pulmonary infiltrates[6,7,13].

Conclusions

This case illustrates severe *Staphylococcus aureus* pneumonia complicated by acute respiratory failure, extubation failure, bronchial mucosal necrosis and pneumothorax in a young woman with a history of smoking, e-cigarette use and cannabis use. Microbiological identification and antimicrobial susceptibility testing guided antibiotic therapy, while individualized ventilatory support and chest drainage addressed the respiratory and pleural complications. The favorable respiratory outcome underscores the importance of an integrated approach and serial reassessment in patients with severe pulmonary infection.

A history of vaping should be systematically investigated in young adults presenting with acute respiratory failure and extensive pulmonary infiltrates. However, exposure alone does not establish EVALI or demonstrate a causal relationship with the severity of pulmonary disease. In this patient, microbiological findings supported *S. aureus* pneumonia, whereas concomitant vaping-associated lung injury remained possible but unproven. Similarly, bronchial mucosal necrosis should not be equated with pulmonary parenchymal necrosis without corroborating evidence.

Declarations

Ethics approval and consent to participate

According to the authors, ethics committee approval was not required for this case report. The patient provided written informed consent.

Consent for publication

The patient provided informed consent for the publication of this case report. No personally identifiable information is included in the manuscript and patient confidentiality has been fully preserved.

Availability of data and materials

The data and materials related to this case are held at the Polydoro Ernani de São Thiago University Hospital, Federal University of Santa Catarina, Florianópolis, Brazil.

Conflicts of interest

All authors declared no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work and no other relationships or activities that could appear to have influenced the submitted work.

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Authors' contributions

The source manuscript attributes concept and design, acquisition, analysis or interpretation of data, drafting and critical review to Laura Vicenzi Souza, Bruno Zanardo Faggion, Lucas Prudente Vieira Motta, Ana Laura Spillere Budny, Lucca Antonini Savas, Bruno Coelho Siniscalchi, Luiz Henrique Barros Santos Pontelli and Leticia Buque Santos; supervision to Laura Vicenzi Souza. All authors should review and approve the final manuscript.

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