


Comprehensive Intensive Care Management of Sepsis Secondary to Submandibular Abscess Complicated by Ventilator-Associated Pneumonia

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ABSTRACT

Introduction: Submandibular infections encompass a spectrum ranging from cellulitis and phlegmon to abscess formation, often originating from odontogenic sources. A submandibular abscess represents a localized collection of pus within the submandibular space, while phlegmon refers to a diffuse, non-suppurative inflammatory process that may precede abscess formation. These conditions pose significant clinical concern due to their proximity to vital structures and potential for rapid spread through deep neck fascial planes. If not promptly treated, such infections can spread through cervical fascial planes, resulting in complications such as mediastinitis, pulmonary infection, sepsis, multiorgan failure, and death. Early recognition and prompt management are therefore critical to prevent severe morbidity and mortality.

Case Description: A 35-year-old man was referred to the emergency department for extensive bilateral submandibular and submental abscesses of odontogenic origin. The patient had previously undergone incision and drainage, tracheostomy, and odontectomy of the gangrenous mandibular molars at previous hospital. Despite these interventions, the patient developed pneumonia with purulent discharge from the tracheostomy site. Imaging revealed the extension of the infection into the parapharyngeal and retropharyngeal spaces, requiring repeat surgical debridement. Following surgery, the patient was admitted to the intensive care unit (ICU) and required mechanical ventilation. The ICU course was complicated by right-sided pleural empyema necessitating thoracotomy with decortication and chest tube placement, as well as ventilator-associated pneumonia caused by multidrug-resistant organisms. Management involved aggressive source control, advanced airway management, broad-spectrum antimicrobial therapy, optimized nutritional support and intensive respiratory physiotherapy.

Conclusion: This case underscores the complexity of managing submandibular abscess complicated by sepsis and ventilator-associated pneumonia. Early recognition, prompt surgical intervention, meticulous airway management, and comprehensive multidisciplinary intensive care are crucial for improving outcomes in patients with extensive deep neck infections and critical respiratory complications.

Submandibular Abscess, Ventilator-Associated Pneumonia, Sepsis, Ludwig's Angina, Deep Neck Infection, Intensive Care Unit

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Keywords

INTRODUCTION

Sepsis, defined as life-threatening organ dysfunction resulting from a dysregulated host response to infection, remains a major global health challenge and is responsible for approximately 20% of all deaths worldwide [1]. It is estimated that more than 31.5 million cases of sepsis occur annually, including nearly 19.4 million severe cases and over 5.3 million deaths, making sepsis one of the leading causes of mortality among hospitalized

patients and those admitted to intensive care units (ICUs), despite substantial advances in early recognition, serial intervention, and antimicrobial therapy [1]. The clinical burden of sepsis is particularly pronounced in critically ill patients, in whom delayed diagnosis or inadequate source control may precipitate rapid progression to multi-organ failure and death.

Sepsis may arise from a wide spectrum of infectious sources, including odontogenic, respiratory, intra-abdominal, soft tissue, urinary tract, intracranial, and hematogenous infections [2]. Among these, submandibular abscesses represent a severe form of deep neck infection, most commonly originating from odontogenic pathologies. Such infections may rapidly progress to diffuse cellulitis or abscess formation involving the submandibular, submental, and sublingual spaces, a condition classically described as Ludwig's angina [3]. Owing to its anatomical proximity to the upper airway, Ludwig's angina carries a high risk of acute airway compromise, frequently necessitating emergency airway intervention, including tracheostomy, and may be further complicated by mediastinitis and thoracic extension [4].

Pulmonary complications are frequently encountered in patients with severe neck infections who require prolonged mechanical ventilation. Ventilator-associated pneumonia (VAP) remains one of the most common ICU-acquired infections and is associated with increased morbidity, prolonged ICU length of stay, antimicrobial resistance, and mortality [5]. In addition, ongoing infection and systemic inflammatory responses may lead to pleural empyema, acute respiratory distress syndrome (ARDS), and multiorgan dysfunction [4,5]. Jankowska et al. reported that up to 59% of deep neck infections were of odontogenic origin, with all cases demonstrating positive bacterial cultures, often compounded by impaired host immunity [3].

Therefore, effective management of sepsis secondary to submandibular abscesses requires early and aggressive source control, timely administration of intravenous broad-spectrum antimicrobial therapy, appropriate hemodynamic resuscitation, and comprehensive supportive ICU care, including lung-protective mechanical ventilation, optimized analgesia, nutritional support, and thromboprophylaxis, to improve clinical outcomes [2]. This case report describes the diagnosis and intensive care management of a patient with sepsis secondary to submandibular abscess complicated by ventilator-associated pneumonia, highlighting the evidence-based strategies for managing this complex and life-threatening condition in the ICU setting.

CASE DESCRIPTION

A 35-year-old man (body weight 80 kg, height 170 cm) was referred to the emergency department from a private hospital after one week of inpatient treatment for extensive bilateral submandibular and submental abscesses of odontogenic origin. The patient had previously undergone incision and drainage, tracheostomy, and odontectomy of the gangrenous mandibular molars (teeth 37–38 and 47–48). Despite these interventions, respiratory distress persisted, and on postoperative day three, purulent and blood-tinged discharge was noted around the tracheostomy site. The patient had a known hepatitis B surface antigen (HBsAg) positivity without ongoing antiviral therapy and no other documented comorbidities or allergies.

Sixteen days prior to admission, the patient experienced progressive right-sided facial and mandibular swelling accompanied by pain, dysphagia, and dyspnea, preceded by an untreated bilateral lower molar toothache. On arrival, he was alert (Glasgow Coma Scale E4M6Vt) but in marked respiratory distress. Vital signs showed a blood pressure of 110/72 mmHg (mean arterial pressure 84 mmHg), heart rate of 152 beats/min, respiratory rate of 32 breaths/min, oxygen saturation of 91% on a non-rebreather mask at 15 L/min, and a body temperature of 36.8°C. Physical examination revealed extensive bilateral submandibular swelling extending to the cervical region, with induration and tenderness.

Initial laboratory investigations demonstrated severe leukocytosis (25,700/mm³), hemoglobin 13.7 g/dL, normal platelet count, hyponatremia (133 mmol/L), elevated urea (90.2 mg/dL), and a serum lactate level of 2.6 mmol/L. Contrast-enhanced cervical and thoracic imaging revealed bilateral submandibular abscesses with right-sided predominance, extension into the parapharyngeal and retropharyngeal spaces, a patent tracheostomy tube, bilateral bronchopneumonia, and bilateral pleural effusions without evidence of pneumothorax or pneumomediastinum (Figure 1). Based on the clinical, laboratory, and radiological findings, sepsis secondary to extensive odontogenic submandibular abscess was diagnosed.

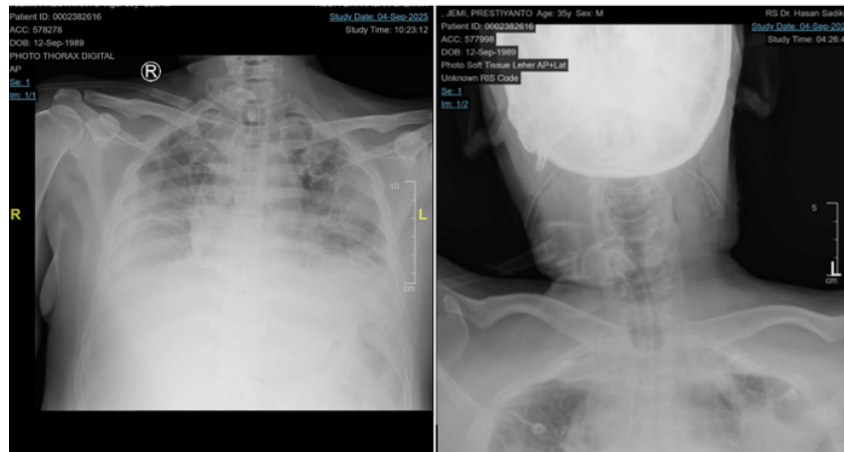


Figure 1. Preoperative cervical imaging and chest radiography revealed bilateral submandibular abscesses with deep neck extension and pulmonary involvement, necessitating a tracheostomy.

Empirical antimicrobial therapy with intravenous ceftriaxone and metronidazole was initiated to provide broad aerobic and anaerobic coverage. The patient subsequently underwent repeat extensive incision and drainage of bilateral submandibular and submental abscesses with parapharyngeal and retropharyngeal extensions. The surgical procedure lasted approximately two hours, with an estimated blood loss of 900 mL and no intraoperative hemodynamic instability. Postoperatively, the patient was transferred to the intensive care unit (ICU) for advanced airway management and sepsis control. Serial laboratory monitoring revealed a peak leukocyte count of 30,980/ μ L on ICU day one, followed by a gradual decline. Renal function initially deteriorated (maximum urea/creatinine 101 mg/dL / 1.22 mg/dL), but improved from ICU day five. Serum lactate levels showed a consistent downward trend (2.4, 2.1, and 1.4 mmol/L on ICU days 1, 3, and 18, respectively). Serial chest radiographs revealed progressive bilateral bronchopneumonia and increasing right pleural effusion (Figure 2).

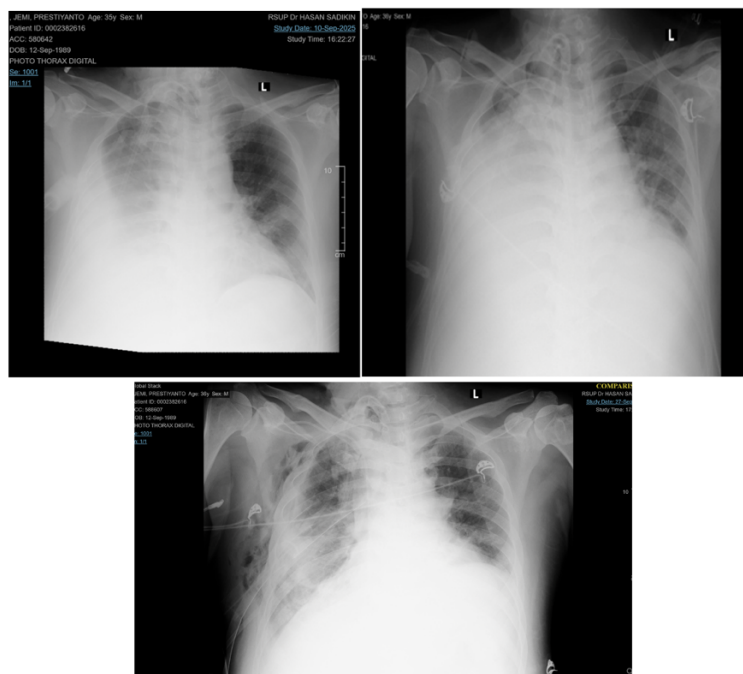


Figure 2. Serial chest radiographs demonstrating the progression of bronchopneumonia and pleural effusion.

The patient required 28 days of ICU care. Initial mechanical ventilation was provided using pressure-synchronized intermittent mandatory ventilation (P-SIMV) with an FiO_2 of 70%, inspiratory pressure of 14 cmH₂O, positive end-expiratory pressure (PEEP) of 6 cmH₂O, and an inspiratory-to-expiratory ratio of 1:2, achieving tidal volumes of 482–510 mL and an oxygen saturation of 100%. Ventilatory support was gradually

reduced to pressure support ventilation; however, subsequent clinical deterioration occurred owing to infectious complications. During the ICU stay, the patient developed ventilator-associated pneumonia caused by multidrug-resistant organisms, including *Acinetobacter baumannii*, carbapenem-resistant *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*. Additional complications included tracheostomy tube leakage requiring resuturing on ICU day 21 and progressive right-sided pleural empyema necessitating thoracotomy with decortication and chest tube placement on ICU day 23. The patient was successfully weaned from mechanical ventilation to T-piece oxygen at 6 L/min on ICU day 25 and transferred to a high-care unit on day 28. Sedation was limited to the early ICU phase using dexmedetomidine at 0.2 µg/kg/h (Richmond Agitation–Sedation Scale –1 to –2) and discontinued by ICU day three. Analgesia was maintained using a low-dose morphine infusion (5 µg/kg/h) to ensure ventilator comfort. Antimicrobial therapy was escalated based on microbiological culture results to meropenem, amikacin, levofloxacin, and high-dose ampicillin–sulbactam, followed by de-escalation to doxycycline and vancomycin according to pleural fluid culture sensitivities. Thoracic computed tomography performed on ICU day 14 confirmed bilateral pleural empyema with right-sided predominance and associated lung atelectasis (Figure 3).

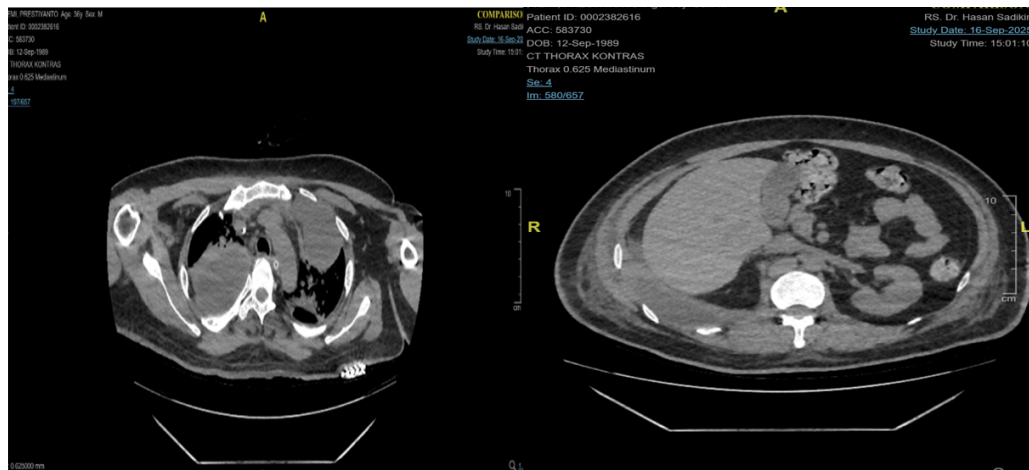


Figure 3. Chest computed tomography revealed bilateral pleural empyema with right-sided predominance and lung atelectasis.

Table 1. Clinical Timeline and Key Interventions

Hospital Day	Clinical Events	Key Interventions	Outcomes
-16 to -7	Progressive mandibular swelling, pain, dysphagia, dyspnea	—	Worsening odontogenic infection
-7 to 0	Treatment at referring hospital	Incision and drainage, tracheostomy, odontectomy	Persistent respiratory distress
0	Sepsis suspected; purulent tracheostomy discharge	Imaging; empirical antibiotics	ICU referral
1	Extensive deep neck infection	Repeat incision and drainage	Postoperative ICU admission
1-3	Acute respiratory failure	Mechanical ventilation (P-SIMV → PSV)	Initial stabilization
6	Persistent pneumonia; sputum retention	Bronchoscopy; tracheostomy upsizing	Improved airway clearance
7-14	Worsening pulmonary infection	Antibiotic escalation	Diagnosis of VAP
21	Tracheostomy air leak	Tracheostomy resuturing	Airway stabilization
23	Right pleural empyema	Thoracotomy and decortication	Lung re-expansion
25	Respiratory improvement	Weaning to T-piece	Ventilator liberation
28	Clinical stabilization	Transfer to high-care unit	Recovery phase

DISCUSSION

This case demonstrates the considerable clinical complexity of sepsis secondary to extensive odontogenic deep neck infection within the spectrum of Ludwig's angina, further complicated by ventilator-associated pneumonia (VAP), pleural empyema, and infection with multidrug-resistant (MDR) organisms. Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection and remains a major global health burden despite advances in critical care, antimicrobial therapy, and surgical management [1]. Current estimates indicate that sepsis continues to account for a substantial proportion of intensive care unit (ICU) admissions and mortality worldwide, underscoring the importance of early recognition, prompt intervention, and continuous reassessment [1,2].

Deep neck infections of odontogenic origin represent a particularly severe source of sepsis because of their propensity for rapid progression along the cervical fascial planes. Odontogenic infections typically arise from pulp necrosis or periodontal disease and may extend into the submandibular, sublingual, and submental spaces, forming phlegmon or abscesses that are characteristic of Ludwig's angina [6]. The anatomical relationship between the mandibular molars and mylohyoid muscle explains the frequent involvement of the submandibular space and the high risk of airway compromise. Extension into the parapharyngeal and retropharyngeal spaces significantly increases the risk of mediastinal spread and thoracic complications, including pleural infection and empyema, which are associated with increased morbidity and mortality [7,8]. In the present case, persistent purulent discharge and clinical deterioration following the initial surgical management strongly suggested inadequate source control, which is a key determinant of poor outcomes in deep neck infections [4].

Airway compromise is the most immediate and life-threatening complication of Ludwig's angina. Therefore, early airway evaluation and proactive airway control are fundamental aspects of management, particularly in patients presenting with progressive floor-of-mouth swelling, dysphagia, drooling, or respiratory distress. Tracheostomy is often required in advanced disease when anatomical distortion and edema make endotracheal intubation hazardous or impractical. Although tracheostomy effectively mitigates the risk of acute airway obstruction, prolonged use of invasive airway devices is associated with impaired secretion clearance, bacterial colonization, and an increased risk of lower respiratory tract infections. These factors likely contributed to the development of VAP in this patient [9,10].

According to the Sepsis-3 framework, sepsis is characterized by acute organ dysfunction attributable to infection, with the Sequential Organ Failure Assessment (SOFA) score supporting clinical risk stratification [1]. In this case, the absence of refractory hypotension and lack of vasopressor requirement indicated sepsis without progression to septic shock. Nevertheless, the principles of sepsis management remain unchanged across severity spectra: early antimicrobial therapy, hemodynamic optimization, and timely source control are essential to prevent deterioration [1,2]. The Surviving Sepsis Campaign emphasizes the rapid initiation of appropriate therapy and repeated clinical reassessment to improve outcomes [2]. In this patient, repeat cervical surgical debridement was critical for controlling the deep neck source, while subsequent thoracic source control via thoracotomy and decortication proved decisive once pleural empyema was confirmed. Marked clinical improvement following thoracic intervention supports the evidence that unresolved infected collections perpetuate systemic inflammation, impair respiratory mechanics, and delay liberation from mechanical ventilation [7,8].

VAP is among the most common ICU-acquired infections, occurring at least 48 h after the initiation of invasive mechanical ventilation, and is associated with prolonged ICU stay, increased antimicrobial resistance, and higher mortality. Diagnosis remains challenging and should rely on an integrated assessment of radiographic abnormalities, systemic inflammatory response, respiratory deterioration, and microbiological evidence, when available [10]. The Clinical Pulmonary Infection Score (CPIS) may assist in bedside assessment, with scores greater than six suggesting a higher likelihood of VAP, although CPIS should not replace clinical judgment or microbiological confirmation. In the present case, early CPIS elevation supported suspicion of VAP, and subsequent cultures identified MDR Gram-negative organisms, including

Acinetobacter baumannii, carbapenem-resistant *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*, substantially complicating antimicrobial management [11,12].

Antimicrobial therapy for odontogenic deep neck infections should initially provide broad-spectrum coverage against oral aerobic and anaerobic pathogens, followed by escalation or de-escalation guided by culture results and clinical response [4-6]. The stepwise antimicrobial approach applied in this patient—empirical therapy, escalation in response to MDR pathogens, and subsequent de-escalation based on pleural fluid cultures—aligns with the established antimicrobial stewardship principles in ICU practice [13,14]. Prolonged hospitalization, repeated surgical interventions, invasive airway devices, and extended mechanical ventilation are well-recognized risk factors for MDR pathogen acquisition, emphasizing the need for strict infection prevention measures and early microbiological surveillance [13].

Several important clinical lessons can be learned from this case. First, odontogenic deep neck infections may progress despite initial surgical intervention; persistent symptoms or purulent tracheostomy discharge should prompt early reassessment for inadequate source control and occult deep space extension [4-6]. Second, prolonged mechanical ventilation and tracheostomy significantly increase the risk of VAP and MDR infections, highlighting the importance of prevention bundles, structured surveillance, and timely culture-guided antimicrobial therapy [10-12]. Third, pleural empyema should be actively considered in patients with worsening pleural opacification or failure to wean from mechanical ventilation, as timely thoracic source control may be pivotal for clinical recovery [7,8].

Serum lactate measurement is widely used to assess systemic perfusion and response to resuscitation in sepsis, although lactate should not be interpreted as a direct surrogate for tissue perfusion [15,16]. In this patient, declining lactate levels were consistent with an improvement in global perfusion. Fluid resuscitation strategies should aim to restore adequate perfusion while minimizing fluid overload, particularly in patients with pulmonary complications [16]. Dynamic indices of fluid responsiveness, such as passive leg raising, offer greater predictive accuracy than static parameters and may guide individualized fluid therapy [17]. In settings where advanced monitoring is limited, simple bedside perfusion markers, including capillary refill time, have been associated with outcomes in sepsis-related hyperlactatemia and may serve as useful adjuncts [18-20]. As this was a single case report, the generalizability of these findings is inherently limited. The absence of institution-specific antibiogram data and additional standardized severity scoring beyond SOFA may further constrain the extrapolation. Nevertheless, this report provides clinically relevant insights into the multidisciplinary ICU management of complex odontogenic sepsis complicated by VAP and pleural empyema, emphasizing the critical roles of repeated source control, vigilant airway and respiratory management, and coordinated, evidence-based critical care.

CONCLUSION

Sepsis secondary to submandibular phlegmon complicated by ventilator-associated pneumonia is a rapidly progressive and life-threatening condition that requires early recognition and decisive multidisciplinary management. Prompt airway stabilization, timely sepsis-directed therapy, and adequate surgical source control are essential to prevent clinical deterioration and improve patient outcomes. Delayed or incomplete source control may sustain systemic inflammation, prolong mechanical ventilation, and increase the risk of secondary complications such as pleural empyema and multidrug-resistant infections. Comprehensive intensive care management, including lung-protective ventilation, individualized hemodynamic support, culture-guided antimicrobial therapy, and coordinated critical care, is crucial for optimizing recovery and survival in patients with severe odontogenic sepsis.

DECLARATIONS

None

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The Authors agree to be published in the Journal of Society Medicine.

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors have made substantial contributions to this case report. was responsible for patient management, data collection, and drafting the initial manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

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