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Atypical Acute Epiglottitis Identified During Emergency Intubation: A Case Report

Denise Ann V. Caballes [1], Scarlett Mia S. Tabuñar [2]

1. Department of Emergency Medicine, University of the Philippines, Philippine General Hospital, Manila, PHL 2. Department of Emergency Medicine, University of the Philippines, College of Medicine/Philippine General Hospital, Manila, PHL

Corresponding author: Denise Ann V. Caballes, deniseann.caballes@gmail.com

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Abstract

Acute epiglottitis in adults can be difficult to recognize when classic upper-airway symptoms are absent and a competing cardiopulmonary process is present. A woman in her late 70s with hypertension and diabetes presented with progressive dyspnea, hoarseness, non-productive cough, orthopnea, bipedal edema, and hypoxemia. She was afebrile and denied odynophagia, dysphagia, or drooling. Stridor was absent on examination. Pneumonia and possible acute decompensated heart failure were initially considered. Despite oxygen therapy, her work of breathing worsened. During rapid sequence intubation, video laryngoscopy unexpectedly demonstrated marked epiglottic and supraglottic edema. An initial 7.5 mm endotracheal tube could not be passed. The otorhinolaryngology service was called and stood by for a surgical airway, after which a 7.0 mm tube was successfully placed without documented peri-intubation complications. Endotracheal aspirate subsequently yielded light growth of *Klebsiella pneumoniae* in the setting of concurrent pneumonia, although the lower respiratory tract source precluded definitive attribution of the supraglottic infection to this organism. The patient improved with antimicrobial therapy, corticosteroids, ventilatory support, and glycemic management and was discharged without recurrent airway symptoms. This case highlights the need to reconsider upper-airway pathology when progressive respiratory distress is accompanied by hoarseness or unexplained voice change, even when cardiopulmonary findings appear to provide an alternative diagnosis.

Categories: Otolaryngology, Emergency Medicine, Infectious Disease
Keywords: adult epiglottitis, diabetes mellitus, difficult airway, emergency airway, klebsiella pneumoniae, supraglottic edema, video laryngoscopy (vl)

Introduction

Epiglottitis, once primarily a paediatric disease, now occurs more commonly in adults following widespread *Haemophilus influenzae* type B (Hib) vaccination. Adult presentations may be variable, and delayed recognition can be clinically significant because of the potential for rapid progression to airway obstruction [1,2]. Diabetes mellitus is an important comorbidity and has been associated with an increased likelihood of airway intervention in patients with acute epiglottitis [3,4]. *Klebsiella pneumoniae* is an uncommon but documented pathogen in adult epiglottitis [5].

In this report, the term atypical presentation is used descriptively to refer to the absence of several commonly recognized upper-airway manifestations of epiglottitis together with the predominance of cardiopulmonary findings that initially suggested an alternative diagnosis. The clinical challenge in this case was not simply the occurrence of adult epiglottitis, but its presentation in a patient with a plausible concurrent lower respiratory and cardiovascular process. Progressive dyspnea, orthopnea, peripheral edema, bilateral crackles, hypoxemia, elevated cardiac biomarkers, and radiographic pulmonary findings initially supported pneumonia with possible cardiac decompensation. At the same time, fever, odynophagia, dysphagia, drooling, and stridor were absent. Marked epiglottic and supraglottic edema became apparent only during video laryngoscopy performed for emergency intubation. We present this case to illustrate practical diagnostic triggers and airway-safety considerations when severe supraglottic disease is obscured by competing cardiopulmonary findings.

Case Presentation

Clinical presentation

A woman in her late 70s with hypertension and diabetes mellitus presented with progressive dyspnea, hoarseness, non-productive cough, easy fatigability, orthopnea, and bipedal edema. She denied fever, odynophagia, dysphagia, or drooling. She had previously been treated at a local hospital for presumed pneumonia and was discharged on unrecalled oral antibiotics, which she had taken for approximately one day prior to admission.

On arrival at the emergency department, blood pressure was 130/80 mmHg, heart rate was 110 beats per

minute, respiratory rate was 30 breaths per minute, temperature was 36.7°C, and oxygen saturation was 86% on room air. Capillary blood glucose was 308 mg/dL. She was in respiratory distress but maintained a patent airway and could speak in short phrases. Pulmonary examination revealed bilateral crackles. Oxygen support was initiated at 15 L/min through a non-rebreather mask. Despite supplemental oxygen, her work of breathing continued to worsen. After discussion of risks and benefits, consent for endotracheal intubation was obtained. The patient's clinical course from the emergency department presentation through discharge and follow-up is summarized in Table *1*.

Clinical phase	Clinical findings	Interventions/ outcome
ED arrival	Blood pressure 130/80 mmHg, heart rate 110/min, respiratory rate 30/min, temperature 36.7°C, SpO2 86% on room air; respiratory distress, bilateral crackles, capillary glucose 308 mg/dL with initial arterial blood gas on FiO_2 0.80: pH 7.25, $PaCO_2$ 53 mmHg, PaO_2 146 mmHg	Oxygen supplementation provided via non-rebreather mask with initial laboratory, ECG, and chest radiograph evaluation
Early ED course	Persistent worsening work of breathing despite oxygen support	Consent obtained for intubation and transitioned to bag-valve-mask ventilation
Airway intervention	Marked epiglottic and supraglottic edema on video laryngoscopy; 7.5 mm endotracheal tube could not be passed	Otorhinolaryngology service called and surgical-airway standby; 7.0 mm endotracheal tube successfully placed
Post-intubation	Airway secured and no documented peri-intubation complication such as desaturation, hypotension, or clinically apparent airway trauma	Piperacillin-tazobactam 4.5 g IV loading dose, azithromycin 500 mg IV, hydrocortisone 250 mg IV with ventilatory support and disposition for ICU admission
Within 24 hours	Serial arterial blood gases showed improving ventilation and decreasing FiO_2 requirement	Continued ventilatory support, insulin therapy, and hemodynamic monitoring
Hospital day 5	Airway swelling improved with successful spontaneous breathing trial	Extubated after otorhinolaryngology service reassessment
Hospital day 10	Stable	Discharged home
2-week follow-up	No dyspnea, hoarseness, or dysphagia; returned to usual activities of daily living	Continued outpatient follow-up

TABLE 1: Clinical timeline from emergency department presentation to follow-up

Airway management

After consent for intubation, respiratory support was transitioned to bag-valve-mask ventilation. Rapid sequence intubation was performed by a senior-level emergency medicine resident using propofol 40 mg IV and rocuronium 40 mg IV. Video laryngoscopy unexpectedly revealed marked edema of the epiglottis and surrounding supraglottic structures. An initial attempt to pass a 7.5 mm endotracheal tube was unsuccessful due to the narrowed supraglottic airway. The otorhinolaryngology service was immediately called. Upon arrival, the team remained on standby for a possible surgical airway while a subsequent attempt with a smaller 7.0 mm endotracheal tube was successfully performed. No peri-intubation oxygen desaturation, hypotension, or clinically apparent airway trauma was documented.

Figure *1* shows the video laryngoscopic view obtained during endotracheal intubation, demonstrating marked edema of the epiglottis and surrounding supraglottic structures.



FIGURE 1: Video laryngoscopic visualization of the epiglottis during emergency intubation

Still frame extracted from a mobile phone recording of the C-MAC video laryngoscope monitor showing a prominent and edematous epiglottis during emergency airway management. Image quality is limited by the secondary recording method.

Investigations

Initial laboratory evaluation showed leukocytosis with neutrophilic predominance, severe hyperglycemia, and markedly elevated inflammatory and cardiac biomarkers. Electrocardiography demonstrated sinus tachycardia with nonspecific ST-T wave changes. Relevant laboratory findings are summarized in Table *2*.

Laboratory parameter	Patient result	Reference value
White blood cell count	28.30 × 10^9/L	4.50–11.0 × 10^9/L
Neutrophils	0.90 (90%)	0.50–0.70 (50%–70%)
Random blood glucose	432.14 mg/dL	74–106 mg/dL
Glycated hemoglobin (HbA1c)	15.4%	3.5%–6.0%
High-sensitivity C-reactive protein	>300 mg/L	1.0–3.0 mg/L
Procalcitonin	1.28 ng/mL	<0.25 ng/mL
High-sensitivity troponin I	364 ng/L	<9 ng/L
N-terminal pro-B-type natriuretic peptide	3,728.81 pg/mL	0–125 pg/mL

TABLE 2: Relevant laboratory findings

Initial arterial blood gas analysis obtained during the emergency department evaluation showed acidemia with hypercapnia while the patient was receiving supplemental oxygen. Serial arterial blood gas measurements showed a progressive reduction in inspired oxygen requirement. These findings are summarized in Table *3*.

Clinical phase	FiO_2	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	HCO_3^-
ED arrival	0.80	7.25	53	146	23.2
1 hour post-intubation	0.70	7.33	36	125	19.0
12 hours post-intubation	0.40	7.40	33	100	20.4

TABLE 3: Serial arterial blood gas results during the early hospital course

Chest radiography demonstrated bilateral reticular and airspace opacities with minimal pleural effusions, supporting the presence of a concurrent pulmonary process. Serial chest radiographs subsequently demonstrated interval regression of the pulmonary opacities.

Differential diagnosis

The initial working diagnosis was community-acquired pneumonia with possible acute decompensated heart failure. Progressive dyspnea, orthopnea, bipedal edema, bilateral crackles, elevated N-terminal pro-B-type natriuretic peptide, leukocytosis, elevated inflammatory markers, and pulmonary radiographic findings supported these diagnoses. Type 2 myocardial infarction was also considered because of the troponin elevation; however, the absence of chest pain and ischemic electrocardiographic changes in the setting of hypoxemia, systemic illness, and increased work of breathing favored supply-demand-related myocardial injury rather than acute coronary thrombosis.

An upper-airway cause was not initially suspected because fever, throat pain, dysphagia, drooling, and stridor were absent, and the patient's hoarseness was initially attributed to respiratory effort. Epiglottitis became apparent only during intubation when video laryngoscopy demonstrated marked epiglottic and supraglottic edema. The endotracheal aspirate subsequently yielded light growth of *K. pneumoniae* in the setting of concurrent pneumonia. Because the organism was isolated from a lower respiratory tract specimen rather than directly from the supraglottic infection, its specific etiologic role in the epiglottitis cannot be definitively established.

Treatment

Initial empiric antimicrobial therapy included piperacillin-tazobactam 4.5 g intravenously as a loading dose and azithromycin 500 mg intravenously. Hydrocortisone 250 mg intravenously was administered for marked supraglottic edema. The patient was transferred to the intensive care unit for close airway monitoring and ventilatory support. Glycemic control was managed with insulin therapy, and supportive measures and hemodynamic monitoring were continued for the systemic illness and associated myocardial injury.

Her airway swelling gradually improved. After successfully completing a spontaneous breathing trial and undergoing reassessment by the otorhinolaryngology service, she was extubated on hospital day five.

Outcome and follow-up

She was discharged home on hospital day 10 in stable condition with instructions for glycemic optimization, cardiopulmonary follow-up, and monitoring for recurrent upper airway symptoms. At her two-week outpatient review, she reported full recovery without dyspnea, hoarseness, or dysphagia and had returned to her usual activities of daily living. Her blood glucose control had improved under the care of her primary physician. No further airway intervention or hospitalization was required, and she remained alive and well without recurrence of symptoms at the latest follow-up.

Discussion

Adult epiglottitis is increasingly recognized in the post-*Haemophilus influenzae* type B (Hib) vaccination era. Although epiglottitis was historically considered a predominantly pediatric disease, adults now constitute an important affected population. Adult presentations may be variable, and acute airway compromise remains a potentially life-threatening complication [1,2]. In the series by Frantz et al., 19 of 129 adults with laryngoscopically confirmed acute epiglottitis required airway intervention [1]. More recently, Felton et al. reported that 12 of 70 adults with acute epiglottitis required acute airway intervention, including three who underwent emergent cricothyroidotomy and one who required tracheostomy [6].

The differential diagnosis was created by the coexistence of credible cardiopulmonary explanations for respiratory distress. Bilateral crackles, elevated N-terminal pro-B-type natriuretic peptide, marked inflammatory abnormalities, and radiographic pneumonia supported pneumonia with possible cardiac decompensation. At the same time, fever, throat pain, dysphagia, drooling, and stridor were absent. Adult epiglottitis may not consistently present with classic symptoms; in the cohort by Felton et al., fever was documented in only 12.9% of patients, while voice changes, odynophagia, and dysphagia were present in 38.6%, 45.7%, and 47.1%, respectively [6]. In this case, hoarseness was present but was initially interpreted in the context of respiratory effort, and supraglottic disease became apparent only during airway evaluation.

From an emergency medicine perspective, this case suggests several practical triggers for reconsidering upper-airway pathology in an adult initially thought to have a cardiopulmonary cause of respiratory distress. These include progressive respiratory compromise despite initial oxygen support, hoarseness or unexplained voice change, deterioration that appears disproportionate to the apparent pulmonary findings, and impending need for airway intervention despite the absence of fever, drooling, dysphagia, odynophagia, or stridor. None of these features alone establishes epiglottitis; rather, their combination should broaden the differential diagnosis and increase preparedness for an unexpectedly difficult airway.

Diabetes mellitus is an important comorbidity in adult epiglottitis. In a retrospective study of 96 patients with acute epiglottitis, Katori and Tsukuda identified diabetes mellitus, stridor, muffled voice, and a rapid clinical course as factors associated with airway intervention [3]. A systematic review and meta-analysis further identified diabetes mellitus as a factor associated with an increased likelihood of airway intervention in adults with epiglottitis [4]. Similarly, Chang et al. reported that adult patients requiring invasive airway support had more systemic comorbidities and greater hyperglycemia than those who did not require airway support [7]. The presence of hyperglycemia and glycated hemoglobin of 15.4% in this patient may therefore have been clinically relevant to disease severity, although causality cannot be inferred from a single case.

Klebsiella pneumoniae (K. pneumoniae) is an uncommon but documented pathogen in adult epiglottitis. Berthiaume and Pien previously reported acute *K. pneumoniae* epiglottitis in an adult requiring antimicrobial therapy and surgical drainage [5]. In this patient, an endotracheal aspirate yielded light growth of *K. pneumoniae* in the setting of directly visualized marked epiglottic and supraglottic inflammation and concurrent pneumonia. This microbiologic finding supports *K. pneumoniae* as a plausible pathogen in the overall respiratory infection.

Airway management is the central concern in severe adult epiglottitis. A recent systematic review and meta-analysis of 56 studies involving 10,630 patients found an overall airway intervention rate of 15.6%, with the rate decreasing over time to approximately 10% in more recent decades. Tracheal intubation was required in 10.2% of patients, and failed intubation occurred in 4.2%, highlighting the high-risk nature of airway intervention in this condition [8]. Clinical and laryngoscopic findings may help identify patients at greater risk of requiring airway intervention. In a retrospective study of 83 patients with acute epiglottitis, Shimizu et al. found that dyspnea, hoarseness, muffled voice, drooling, and severe laryngoscopic narrowing were among the findings associated with airway intervention [9]. In this case, the failed initial passage of a 7.5 mm tube, prompt communication with the otorhinolaryngology service, surgical-airway standby, and successful use of a smaller 7.0 tube illustrate the value of early escalation, alternative airway planning, and interprofessional coordination when unexpected supraglottic narrowing is encountered.

The patient's elevated troponin levels were interpreted in the context of severe systemic illness, hypoxemia, and increased myocardial oxygen demand. The Fourth Universal Definition of Myocardial Infarction

distinguishes myocardial injury from myocardial infarction and defines type 2 myocardial infarction as myocardial ischemia resulting from an imbalance between myocardial oxygen supply and demand unrelated to acute coronary thrombosis [10]. This distinction emphasizes the importance of integrating cardiac biomarkers with symptoms, electrocardiographic findings, and the overall clinical context rather than attributing troponin elevation alone to acute coronary occlusion.

This report has several strengths. The supraglottic abnormality was directly visualized and documented through video laryngoscopy during the episode of respiratory deterioration; the airway sequence, serial arterial blood gases, microbiologic testing, radiographic course, extubation, and short-term outcome were available for review. The case also demonstrates a real concurrent pulmonary process rather than a purely misleading initial diagnosis, which helps explain why upper-airway disease was not initially apparent.

Several limitations should be acknowledged. First, *K. pneumoniae* was isolated from an endotracheal aspirate rather than directly from the epiglottis or supraglottic tissue; its etiologic role in the epiglottis therefore remains uncertain. Second, because the diagnosis was unexpected and made during emergency airway management, no planned pre-intubation fiberoptic examination or targeted supraglottic microbiologic specimen was obtained. Finally, as a single case report, the observations cannot establish causality or determine the diagnostic performance of individual symptoms.

Conclusions

Adult epiglottitis may be difficult to recognize when the clinical picture is dominated by concurrent cardiopulmonary abnormalities and classic upper-airway symptoms are absent. In this patient, progressive respiratory distress, hypoxemia, pulmonary abnormalities, and elevated cardiac biomarkers initially supported pneumonia and possible cardiac dysfunction, while marked epiglottic and supraglottic edema became apparent only during video laryngoscopy for emergency intubation.

Progressive respiratory compromise accompanied by hoarseness or unexplained voice change should prompt consideration of upper-airway pathology even in the absence of fever, odynophagia, dysphagia, drooling, or stridor. When airway intervention is required, clinicians should anticipate the possibility of severe supraglottic narrowing, prepare alternative endotracheal tube sizes and rescue airway strategies, and obtain early surgical-airway support when available. The isolation of *K. pneumoniae* from an endotracheal aspirate provides microbiologic context but does not establish the organism as the definitive cause of the supraglottic infection.

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: Denise Ann V. Caballes, Scarlett Mia S. Tabuñar

Acquisition, analysis, or interpretation of data: Denise Ann V. Caballes

Drafting of the manuscript: Denise Ann V. Caballes

Critical review of the manuscript for important intellectual content: Denise Ann V. Caballes, Scarlett Mia S. Tabuñar

Supervision: Scarlett Mia S. Tabuñar

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