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Case report
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Acute epiglottitis from diphtheria: the forgotten but fatal disease

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Abstract

Diphtheria is an infectious disease caused by the gram-positive bacillus *Corynebacterium diphtheriae*. It is transmitted through direct contact with infectious respiratory secretions or skin lesions and was a significant cause of morbidity and mortality worldwide prior to the introduction of vaccination in the 1930s. We report the case of a 12-year-old boy with no history of vaccination who presented with acute upper airway obstruction secondary to respiratory diphtheria. He required endotracheal intubation and was admitted to the intensive care unit. Despite supportive care, the patient developed cardiogenic shock within 24 hours and ultimately succumbed to the infection. This case underscores the importance of maintaining a high index of suspicion for diphtheria in unimmunized patients presenting with acute airway obstruction, tonsillar exudates, and cervical swelling. It also highlights the critical need for effective interdepartmental communication, early diagnostic testing, and timely access to diphtheria antitoxin to improve outcome of this potentially fatal disease. Effort to promote community awareness on national immunization program is imperative on achieving herd immunity to suppress transmission and reduce morbidity associated with vaccine-preventable disease.

Keywords: acute upper airway obstruction, diphtheria, paediatric endotracheal intubation

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Introduction

Diphtheria is a high mortality, virulent infectious disease which has transitioned from a significant public health threat to a rare occurrence due to robust implementation of national immunization programs in Malaysia and worldwide.[1] The increasing observable trend of antivaccination sentiment poses a challenge in achieving herd immunity. Sporadic cases of diphtheria still occur, particularly in the unimmunized population. Given its rarity, it poses a significant challenge for clinicians on prompt recognition, accurate diagnosis, and delivery of expedient management to improve survival outcomes. Diphtheria manifests as a clinical spectrum of disorders precipitated by the absorption and dissemination of diphtheria toxin. Disease progression ranges from localised infection of oropharynx and acute airway obstruction to severe toxin-mediated damage of myocardium, renal tissue, and the peripheral nervous system, all of which may culminate in mortality.[2] We present a case of diphtheria in an unvaccinated child who initially presented as acute epiglottitis, developed cardiogenic shock rapidly, and succumbed to the infection.

Case presentation

A 12-year-old boy with weight of 37 kg, unimmunized since birth, presented with fever and cough for 2 days and shortness of breath for 1 day. There was a history of contact with a sick family member. The patient presented to the emergency department in the middle of the night with tachypnoea at 40 breaths per minute and hypoxia at SpO_2 83% on room air that improved to 100% on high-flow mask oxygen. He also had stridor and hoarseness of voice. The Glasgow Coma Scale was full and examination showed bilateral submandibular lymphadenopathy and bilateral tonsillar enlargement grade 3 covered with minimal slough. Initial evaluation showed oropharyngeal and laryngopharynx inflammation and oedematous epiglottis.

The patient was referred by the ENT to the ICU team for intubation in view of potential airway collapse with provisional diagnosis of acute epiglottitis. A throat swab was obtained for culture and sensitivity, with diphtheria included in the differential diagnosis. However, this information was not conveyed to the ICU and pathology teams.

In view of potential difficult airway management, intubation with video laryngoscopy (VL) in a controlled environment by an anaesthesiologist was planned in the operation theatre. Intravenous (IV) sugammadex 16 mg/kg was on standby prior to induction and a supraglottic device (laryngeal mask airway size 3) was prepared. The ENT team was present and equipment for emergency front-of-neck access was on

standby in case the patient went into cannot intubate/cannot ventilate scenario. His vital signs in the operating theatre were blood pressure 102/59 mmHg, SpO_2 99% on high-flow mask 15 L/min, and respiratory rate of 35 breaths per minute. Anaesthesia induction was performed using IV fentanyl 50 mcg, IV glycopyrrolate 360 mcg, IV ketamine 50 mg, and IV propofol 50 mg. Once controlled bag mask ventilation was achieved, IV rocuronium 20 mg was administered.

VL revealed a swollen, oedematous epiglottis and inflamed oropharyngeal mucosa that bled easily on contact, obscuring the glottic view, as shown in Figure 1, left. Single attempt intubation was achieved using a size 5 endotracheal tube with a C-MAC video laryngoscope (Karl Storz, Tuttlingen, Germany) with D-blade, as shown in Figure 1, right. Arterial blood gas post intubation was 7.36/39/106/22/-3.2 with lactate of 1. The patient was sedated, ventilated, started on IV ceftriaxone and IV azithromycin as empirical treatment for acute epiglottitis and atypical infection. IV dexamethasone 6 mg was started to reduce airway oedema. Despite resuscitation and escalation of vasopressors, including norepinephrine, vasopressin and adrenaline, he remained hypotensive with oliguria. ENT reassessment noted persistent exudates over the tonsils and uvula.

Eight hours in ICU, the patient became tachycardiac with increasing vasopressor and developed metabolic acidosis. Serial blood investigations, shown in Table 1, demonstrated raised inflammatory markers and worsening of renal function, indicating sepsis-induced or toxin-mediated nephropathy. Electrocardiogram on ICU admission showed sinus rhythm (Fig. 2). Bedside echocardiography demonstrated poor myocardial contractility with a small pericardial effusion, consistent with diphtheritic myocarditis. He developed pulseless electrical activity arrest, briefly achieved return of spontaneous circulation, but suffered recurrent cardiac arrests. After 31 minutes of total resuscitation, he was pronounced deceased 24 hours after initial resuscitation in the emergency department. The cause of death was septic shock secondary to acute epiglottitis with myocarditis and multiorgan failure.

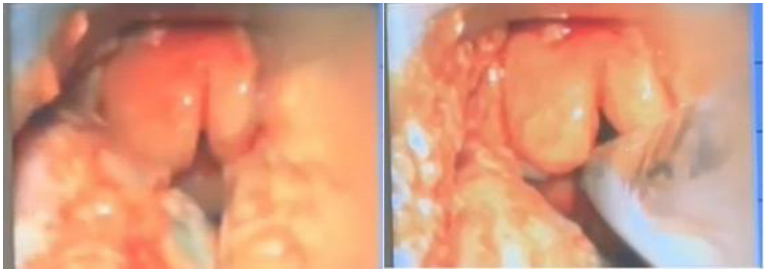

Fig. 1. (Left) Inflamed and oedematous epiglottis obscuring the glottic opening. *(Right)* Glottic view post intubation.

Table 1. Blood investigation results in ICU

Parameter	Time		
	00:00 H	05:00 H	17:00 H
Hb	16.6	14.3	
WBC	11.72	14.4	
PLT	163	162	
Urea	12.9	14.8	21.4
Na	125	128	126
K	5.1	5.1	4.8
Creatinine	111	128	190
Ca	2.28	2.02	
Mg	0.97	0.99	
PO4	1.73	1.65	
ALP	223	210	
ALT	13	15	
CRP	110	96	

Hb: Haemoglobin; WBC: White blood count; PLT: Platelets; Na: Sodium; K: Potassium; Ca: Calcium; Mg: Magnesium; PO4: Phosphate; ALP: Alkaline phosphatase, ALT: Alanine amino-transferase; CRP: C reactive protein

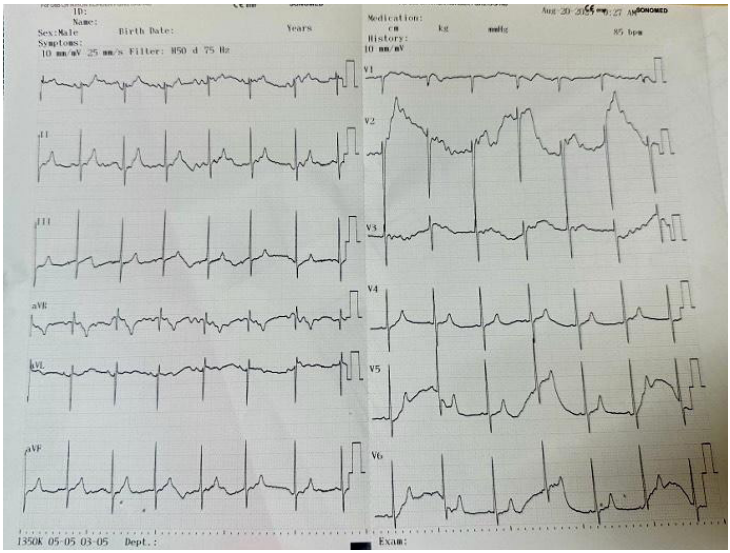

*Fig. 2.*Electrocardiogram on ICU admission showed sinus rhythm.

Throat swab and tracheal aspirate cultures were only available after the patient passed away and confirmed toxigenic *Corynebacterium diphtheriae*, sensitive to erythromycin and vancomycin. Concomitantly, he also had incidental findings of positive COVID-19, although ventilation had not been an issue throughout his ICU stay. Retrospectively, there was a delay in processing the throat swab culture and sensitivity results as the urgency was not highlighted to the pathology team. Thus, this delayed the diagnosis and timely administration of diphtheria antitoxin, which might have changed patient's outcome.

Discussion

This case highlights the challenges of recognising and managing fulminant diphtheria. Several clinical features strongly pointed towards diphtheria in this patient, including the absence of immunisation since birth with origin from a rural district of Sabah, the presence of acute upper airway obstruction with stridor and tonsillar slough, marked cervical lymphadenopathy with submandibular swelling producing a characteristic "bull neck" appearance, and oropharyngeal exudates involving the tonsils and uvula. Although, severe pseudomembrane and "bull neck" of diphtheria preceded cardiomyopathy with positive predictive value of approximately 80%, the textbook presentation is not necessarily recognisable, especially in a rare disease and at tertiary urban centre such as Kuala Lumpur.[2]

Due to the high incidence of acute upper airway obstruction in diphtheria, anaesthesiologists play a critical role in airway assessment and management.

Airway management in patients with upper airway obstruction demands meticulous preoperative planning and multidisciplinary communication. Backup plans are essential and must account for the specific aetiologies of primary plan failure. These strategies involve awake techniques, induction of general anaesthesia (GA) with potential "wake up" contingency, or invasive rescue techniques should emergent reversal of anaesthesia be deemed inappropriate.

Awake techniques, which include awake fibreoptic intubation, awake VL, and awake tracheostomy offer the distinct advantages of preserving spontaneous ventilation and natural airway patency. However, the use of awake techniques is largely restricted to cooperative, non-distressed patients, rendering them less feasible in the paediatric population like in our case. While the use of GA and paralytic agents in context of airway obstruction has historically been a point of contention, evidence showed that neuromuscular blocking agents improve ventilation in stridulous patients with glottic pathology.[3] GA with IV induction is often preferred

for distressed paediatric patient with acute, severe airway obstruction. Conversely, inhalational induction carries risk of precipitating a complete collapse of partially obstructed airway. Furthermore, the delayed clearance of volatile agents in these patients may preclude rapid return of consciousness.[3]

Comprehensive rescue protocols that incorporate supraglottic airway devices, needle cricothyroidotomy, emergency front-of-neck access, or rigid bronchoscopy should be discussed with the ENT team prior to intervention. Any failure to secure airway must prompt immediate transition to subsequent management plan.[4]

The diphtheria toxin is responsible for systemic manifestations, particularly myocarditis, which is the most common cause of death. Myocarditis often presents within 1–2 weeks of respiratory symptoms but may occur earlier in severe disease,[5] as in this case. Skogmar *et al.* reported that systemic manifestations, such as myocarditis, often accompany severe disease, occurring in approximately 10–20% of patients; myocardial damage in such cases is linked to toxin-mediated pathogenesis.[6] Resuscitation, including fluid responsiveness, should be assessed systematically using serial echocardiography, lactate trends, base deficit monitoring, and repeated evaluation of clinical perfusion.[7] In patients who continue to deteriorate despite fluid boluses, early utilization of invasive cardiac output monitoring may be warranted to guide therapy and avoid fluid overload.[7]

The diphtheria antitoxin remains the only intervention capable of neutralising the circulating toxin. Early administration of the antitoxin may prevent worsening of fulminant toxaemia, in addition to antibiotics and supportive care. However, once severe toxin-mediated myocarditis is established, mortality remains high despite intensive management.[5] While most isolates remain penicillin-susceptible, elevated penicillin minimum inhibitory concentrations are rarely observed in both North American and global surveillance studies. Zou *et al.* reported that elevated penicillin minimum inhibitory concentrations are rare in *C. diphtheriae*. Of 45 isolates evaluated, only 1 showed intermediate susceptibility by updated Clinical and Laboratory Standards Institute Criteria.[8] This reinforces that true phenotypic and genotypic penicillin non-susceptibility in diphtheria is uncommon. Even in our neighbouring country, Sariadji *et al.* examined *C. diphtheriae* strains from Indonesia and found that 10.5% were resistant to penicillin and 5.3% resistant to erythromycin—an unexpected pattern given that these drugs remain first-line therapy.[9] Such unexpected antibiotic resistance may have contributed to the poor clinical outcome and underscores the importance of obtaining culture confirmation, not relying solely on empirical therapy.

Several important reminders emerge from this case for all teams managing critically ill patients with suspected infectious airway disease. First, it is essential to

maintain a high index of clinical suspicion in unimmunised paediatric populations. The presence of airway obstruction, tonsillar exudates, and cervical lymphadenopathy in a patient with no prior history of immunisation should immediately prompt consideration of diphtheria diagnosis. Second, flagging the diagnosis across teams is vital. When diphtheria is considered in the emergency department or by ENT specialists, this must be clearly documented and explicitly communicated to the anaesthesia and ICU teams so that it is not overlooked during urgent airway or resuscitation management. Third, early microbiological sampling is paramount. Throat swabs and tracheal aspirates should be collected promptly, with the specific suspicion of diphtheria clearly relayed to the laboratory to expedite confirmatory testing. Fourth, awareness and availability of diphtheria antitoxin should be ensured, as airway security is the immediate priority, but timely antitoxin administration can be lifesaving; all teams should know how and where to access antitoxin within their hospital setting. Fifth, effective communication is fundamental when managing rare and high-mortality diseases such as diphtheria. Integrated coordination between the emergency medicine, ENT, anaesthesia, ICU, infectious diseases, and microbiology teams is necessary to mitigate diagnostic delay. Finally, the importance of vaccination advocacy cannot be overstated. This case underscores that routine childhood immunisation remains the most effective preventive measure against diphtheria and its potentially fatal complications.[1]

Conclusion

Diphtheria, a rare but fatal disease in unimmunised children, can mimic acute epiglottitis and should be considered in unimmunised children presenting with upper airway obstruction and pseudomembranous tonsillitis. Key clinical criteria such as cervical lymphadenopathy, oropharyngeal exudates, and rapid progression to systemic toxicity may point to the diagnosis. Early team communication and timely access to antitoxin can be lifesaving. Vaccines are the safest and most cost-effective public health intervention to reduce morbidity of vaccine-preventable diseases. Effective public health communication and enhanced public literacy regarding the national immunisation program are imperative.

Declarations

Informed consent for publication

Informed consent was obtained from the patient's mother for the publication of the patient's data and clinical images.

Competing interests
None to declare.

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None to declare

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