

A SWOLLEN THROAT TO METABOLIC CRISIS: A CASE OF DIABETIC KETOACIDOSIS TRIGGERED BY EPIGLOTTITIS

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CASE DESCRIPTION

A 56-year-old Malay woman with no known medical history presented with a four-day history of severe dysphagia, hoarseness, and vomiting. She denied respiratory distress or foreign body ingestion. Examination revealed dehydration, an injected pharyngeal wall and right-sided cervical tenderness. Flexible nasoendoscopy demonstrated epiglottic swelling with bilateral vallecula obliteration, consistent with epiglottitis. Blood gas analysis showed severe metabolic acidosis and hyperglycemia with elevated serum ketones. Urinalysis confirmed glycosuria and ketonuria, and renal profile revealed mild acute kidney injury. She was diagnosed with DKA precipitated by acute epiglottitis.

The patient was started on aggressive intravenous fluid resuscitation, insulin infusion, and broad-spectrum antibiotics (intravenous ceftriaxone). Airway management was prioritized with continuous monitoring for respiratory compromise in the ICU. She remained stable and did not require intubation. Over the next 48 hours, her metabolic acidosis resolved, and her symptoms of epiglottitis improved.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes mellitus characterized by hyperglycemia, ketonemia, and metabolic acidosis. It is often precipitated by infections such as urinary tract infections or pneumonia. However, upper airway infections like epiglottitis are rarely reported as triggers. Epiglottitis, a rapidly progressive bacterial inflammation of the epiglottis and supraglottic structures, poses a dual threat of airway obstruction and systemic inflammatory stress, which may precipitate metabolic crises, particularly in undiagnosed diabetics.

DISCUSSION

The physiologic stress of infection, compounded by dehydration and insulin deficiency, can precipitate DKA. While infections are common precipitants, epiglottitis is rarely implicated. The combination of inflammation, hormonal dysregulation, and impaired oral intake synergistically contributed to the development of DKA in this case. Timely recognition and a multidisciplinary approach focusing on airway vigilance and metabolic stabilization were key to a favorable outcome.

CONCLUSION

This highlights the rare but serious coexistence of epiglottitis and DKA. Clinicians should consider atypical infectious triggers in cases of unexplained metabolic decompensation and concurrent management of airway and metabolic complications are essential to prevent morbidity and mortality.

KEYWORDS

Diabetic ketoacidosis; Epiglottitis; Upper airway infection; Airway management



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