Adult epiglottitis: an under-recognized, life-threatening condition

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Epiglottitis in the adult can be fatal and should be treated with the same degree of concern and suspicion in respect of airway patency as in children. We present three cases of adult epiglottitis in which the airway was lost prior to or during the intervention of an anaesthetist. We suggest that an emphasis on conservative management is distracting and belies the serious nature of this disease.

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Epiglottitis is an inflammation of the supraglottic structures that can occur at any age. There is a reported increase in the incidence of epiglottitis in the adult population.\(^1\) In contrast to the aggressive management of epiglottitis in children, a more conservative approach has been advocated in adults.\(^2\) We present three cases of adult epiglottitis where the airway was lost either before or during intervention of an anaesthetist.

Case reports

Case 1

A 41-yr-old male presented with a sudden onset of sore throat, stridor and dyspnoea. He was previously fit but regularly smoked heroin. On arrival to the accident and emergency department, he rapidly developed complete airway obstruction resulting in a respiratory arrest. Manual ventilation by facemask proved ineffective and no i.v. access could be secured. Laryngoscopy was impossible because of trismus. Intralngeal succinylcholine 100 mg was injected and permitted laryngoscopy. A large mass arising from the epiglottis was observed, completely obscuring the larynx. Following oxygenation via an emergency cricothyroidotomy, the trachea was intubated nasally using a fibroptic bronchoscope. Intravenous cefotaxime was commenced. A surgical tracheostomy was performed under general anaesthesia and the patient admitted to a high dependency area on the otolaryngology ward. A lateral soft tissue neck radiograph (Fig. 1), MRI scan (Figs 2 and 3) and fibroptic nasendoscopy (Fig. 4) were performed once the airway had been secured. The patient made a full recovery. Candida albicans was eventually cultured from the mass.

Case 2

A 73-yr-old male was admitted with less than a 24-h history of sore throat, dysphagia and dyspnoea associated with mild inspiratory stridor. Fibroptic nasendoscopy in the accident and emergency department revealed a swollen, hyperaemic epiglottis. Blood cultures were taken and he was commenced on i.v. cefotaxime and dexamethasone 4 mg. Four hours later, on the otolaryngology ward, his respiratory status deteriorated rapidly. A surgical tracheostomy was attempted under local anaesthesia but the procedure abandoned when the patient became hypoxic and began convulsing. The anaesthetist was able to intubate the trachea orally in the unconscious patient, prior to transfer to the operating room for conventional tracheostomy. The patient was admitted to the intensive care unit and made an uneventful recovery. The trachea was decannulated 4 days later. Two blood cultures taken on the day of admission both grew Neisseria meningitidis, later typed as Group Y. There was no other positive microbiology and virology screen was normal.

Case 3

A 56-yr-old man was admitted to the accident and emergency department with acute onset of a sore throat and dysphagia. He was sitting up, dyspnoeic, and drooling saliva. A flexible nasendoscopy showed an oedematous, enlarged epiglottis. An i.v. line was inserted, blood cultures taken and Augmentin 1.2 g and hydrocortisone 200 mg were given intravenously. The patient was transferred to the operating theatre. During an inhalational induction of anaesthesia with sevoflurane, he developed complete airway

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Fig 1 Lateral soft tissue radiograph demonstrating the ‘thumb print’ sign (E). The ‘vallecula’ sign (V) can be determined by identifying the base of tongue (B) and tracing it inferiorly to the hyoid bone (B). If there is no pocket of air extending almost to the hyoid bone and roughly parallel to the pharyngotraheal air column, then the diagnosis of epiglottitis is confirmed. Laryngeal inlet is noted (L).

Fig 2 Sagittal MRI of case 1. The prominent swollen epiglottis (E) is clearly visible above the laryngeal inlet (L). The tracheostomy (T) is also noted.

obstruction. Intravenous succinylcholine 100 mg was administered and orotracheal intubation was attempted but failed. An emergency cricothyroidotomy was then performed using a 13-gauge transtracheal catheter. Jet ventilation was commenced and continued whilst a surgical tracheostomy was performed. The trachea was decannulated 3 days later. Blood cultures were negative.

Fig 3 Coronal MRI of case 1. Again the enlarged epiglottis (E) can be seen above the laryngeal inlet (L).

Fig 4 Fibreoptic nasendoscopy view in case 1. Unlike the classic cherry red epiglottis in children, adult epiglottitis is characterized by diffuse inflammation of aryepiglottic structures. Both the epiglottis (E) and the arytenoids (A) are clearly inflammed. The laryngeal inlet (L) is noted.

Discussion

Adult epiglottitis has an incidence of between 1–4 per 100 000 per annum\textsuperscript{3,4} and is described as having a low morbidity and mortality.\textsuperscript{5} Nonetheless, mortality in adults is around 7%, and some reports suggest it is as high as 20%.\textsuperscript{3} Hingorani and colleagues\textsuperscript{6} observed that a similarly high mortality in children prompted a change to an interventional practice. Even though adult epiglottitis is unpredictable and occasionally catastrophic in outcome, conservative management is still preferred by many authors.\textsuperscript{2,5}
**Haemophilus influenzae** type B is found in as few as 17% of adult patients with suspected epiglottitis. There is also a high rate of negative blood cultures possibly suggestive of a viral cause (although only herpes simplex has been reported). Meningococcal epiglottitis is extremely rare and we present only the seventh reported case; the first was reported in 1995. Alternatively, epiglottitis may occur following mechanical injury such as the ingestion of caustic material or the inhalation of hot objects, smoke or vapours. Epiglottitis following illicit drug use has been described as a result of the accidental inhalation of a heated object. Although there has been an association with cigarette smoking, there are no reports linking epiglottitis and the smoking of heroin. The causal organism or factor is, therefore, often less identifiable in adults than in children.

An adult with epiglottitis usually presents with symptoms of sore throat and painful dysphagia. Drooling and stridor are infrequent presenting signs. In fact the presence of stridor, dyspnoea and a short duration of symptoms prior to presentation, are all described as predictors of airway loss in the adult with epiglottitis. This is, however, controversial. For example, Wolf presented 30 patients in whom stridor was present and who were subsequently successfully treated conservatively. On the other hand, Mayo-Smith described a patient who had no history of stridor, yet suddenly developed airway obstruction and died. Indeed the three cases we present support the fact that the disease presentation and progression is variable and that there are no reliable markers that predict the need for invasive airway support.

The diagnosis of epiglottitis is essentially clinical but can be supported by indirect laryngoscopy. Typically, there is diffuse swelling of the aryepiglottic structures unlike the classic cherry red epiglottis in children. Otherwise, once the airway is deemed safe, a lateral, soft tissue radiograph may show a thickening of the epiglottis (‘thumb print sign’; see Fig. 1). Ducic and colleagues have proposed the ‘vallecula sign’ to improve the diagnostic accuracy of soft tissue radiographs. This stepwise approach attempts to identify the vallecula as it nears the level of the hyoid bone. In the absence of a ‘deep and well-defined vallecula’, the radiological findings support the diagnosis of epiglottitis.

If the clinical diagnosis of epiglottitis is made and the airway judged to be at risk, intervention should not be delayed by attempts to obtain cultures or radiographs. Treatment should begin promptly with intravenous antibiotics. Steroids have no accepted place and the benefit of epinephrine, either nebulized or intramuscular, has yet to be confirmed.

Either orotracheal intubation or tracheostomy may be performed under local anaesthesia but both are potentially stimulating procedures which may precipitate sudden loss of the airway. General anaesthesia may be performed with an inhalational induction but can be complicated by a relatively prolonged excitation phase in adults (as occurred in case 3). Friedman therefore recommended a rapid sequence induction with the facility to perform a criothyroid puncture if intubation proves difficult. Bag and mask ventilation can simply worsen or complete the airway obstruction and should be avoided.

Neuromuscular blocking drugs are traditionally avoided in epiglottitis (although succinylcholine was listed on a recent protocol for the management of paediatric epiglottitis). We used succinylcholine in two of the above case reports, including one intralinguinal injection. Although this route is described in children, we could find no reference to its use in adults.

In summary, a belief that epiglottitis is rare in adults has contributed to misdiagnosis and high mortality rates. We present three cases that are typical of adult epiglottitis, in that there is no one identifiable causal agent or factor that would allow rationalisation of a particular therapy. To advocate conservative management belies the aggressive nature of this disease. We believe there should be a greater emphasis on early interventional support of the airway.

### References