Adult epiglottitis: prompt diagnosis saves lives

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Mortality from adult epiglottitis may be reduced by prompt action, which requires increased awareness of the disease's existence

Acute epiglottitis is a well recognised life threatening infectious disease of children. It is so uncommon in adults that many doctors are unaware of its existence, and few have experience of a case. Failure to diagnose epiglottitis early in adults undoubtedly contributes to its continuing mortality, which may be higher than in children.¹ We report two cases of adult epiglottitis seen over three years in this emergency department. In one case diagnosis was prompt and the patient survived, and in the other epiglottitis was not diagnosed and the patient died.

Case reports

CASE 1

In July 1990 a 35 year old white man referred himself to the emergency department on a weekday afternoon, having suffered for five days with a sore throat that had started two days after returning from holiday in Portugal. In the morning the pain had increased and he had difficulty in swallowing and had experienced rigors. The triage nurse observed that he had a painful throat and was shaking. He had a high fever (39.4°C) and was noted to be only "mildly distressed." His pharynx was inflamed, with minimal tonsillar enlargement; he had bilateral upper cervical lymphadenopathy. A senior house officer diagnosed a bacterial upper respiratory tract infection. Despite the history of rigors no blood cultures or throat swabs were taken, and the patient was discharged with phenoxymethylpenicillin 500 mg to be taken four times daily, having been advised to see his general practitioner if his condition did not improve within 48 hours.

At home six hours later he suddenly complained to his wife that he was unable to breathe and then collapsed. He was in cardiorespiratory arrest when an ambulance arrived and could not be intubated by the paramedical crew. Subsequent resuscitation attempts at hospital were unsuccessful, although he was intubated blindly past an oedematous, inflamed epiglottis.

A swab taken from the epiglottis at necropsy grew *Haemophilus influenzae* type b and commensal flora. The histological appearance of the larynx was of a severe epiglottitis, with inflammatory changes affecting both vocal cords—the laryngeal oedema was sufficient to cause complete obstruction of the airway.

CASE 2

In March 1993 a 37 year old white man referred himself to the emergency department at 3 am with an 18 hour history of painful and difficult swallowing. He had awoken suddenly with a choking sensation, which had improved on sitting upright. He was tachypnoeic, with a respiratory rate of 40 per minute, and was using his accessory muscles of respiration; no stridor or drooling was apparent. His temperature was 37.8°C. Epiglottitis was suspected on clinical grounds and a lateral neck radiograph showed a swollen epiglottis (figure).

Senior otolaryngological and anaesthetic staff advised deferral of elective intubation. He was given 200 mg hydrocortisone intravenously, together with intravenous chloramphenicol (1.2 g) and cefotaxime (1 g). He was transferred to a critical care facility for

airway monitoring. Six hours later, after satisfactory progress, a fiberoptic examination showed a swollen epiglottis. Steady improvement occurred over the next 24 hours; on endoscopic re-examination after 48 hours the epiglottis was only mildly swollen. Treatment was then changed to oral antibiotics and he was allowed home. Blood cultures gave negative results but a peripheral blood film showed a polymorph leucocytosis (total white cell count $20.6 \times 10^9/l$ (83% neutrophils) (range $4.5-11.0 \times 10^9/l$)).

Discussion

Acute infective inflammatory diseases of the upper airway are anatomically distinct: laryngotracheitis, or "viral croup," affects structures below the glottis, while laryngitis of the glottis (which is common) and supraglottitis (which is rare) affect the structures above. The term epiglottitis is often used instead of supraglottitis, but the aryepiglottic folds and arytenoid soft tissues may be affected as well as the epiglottis.²

EPIDEMIOLOGY

The annual incidence of adult epiglottitis has been estimated as 9.7 per million adults (Rhode Island)¹ and 8.8 per million adults (Denmark),³ with a hospital bed occupancy of 0.03 per 1000 adult admissions (Cleveland, Ohio)⁴; this compares with a bed occupancy in the United States of 0.5-0.9 per 1000 paediatric admissions.² Mortality from adult epiglottitis between 1980 and 1991 was 1.1%, rising to 17.6% if respiratory distress was a presenting symptom.⁵

Epiglottitis occurs primarily in temperate climates, but its incidence varies between populations in a given area and between areas in a given population (a significant difference in incidence has been seen in American soldiers attending military hospitals in Panama and Alaska).⁶ The incidence in adults has been increasing since the 1960s,⁷ although the impact of childhood vaccination against *Haemophilus influenzae* type b on invasive infection in adults is as yet unknown. Some studies show a higher rate during the summer and others a higher rate during the winter. A meta-analysis of 32 studies has confirmed a slight male predominance (3:2, male: female)². Any age group can be affected, most children being between 1 and 5 years old and most adults between 20 and 50.⁸

Lateral radiograph showing enlarged soft tissue shadow of epiglottis

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AETIOLOGY

In children *Haemophilus influenzae* type b is nearly always the cause of the infection. It is still the commonest cause in adults; others include *Streptococcus pneumoniae* and *Haemophilus parainfluenzae*, with isolated reports of *Klebsiella pneumoniae* and *Pasteurella multocida* in blood cultures. Bacteraemia is less common in adults than in children.⁹

DIAGNOSIS

General practitioners will often be the first to see patients with epiglottitis, but in one study only one out of 42 cases was correctly diagnosed before admission to hospital.¹⁰ Other patients may present to emergency departments where it is usually a junior doctor who is relied on to make the diagnosis.

A sore throat with pain on swallowing is common but not a good discriminating symptom. Suspicion should be aroused if there is little pharyngeal inflammation. There may be swelling of the uvula,⁹ and respiratory distress, although less common than in children, is a serious sign. Indirect mirror laryngoscopy, or better fiberoptic laryngoscopy, is a more reliable method than lateral neck radiography of showing a swollen epiglottis (radiography is diagnostic in only 79% of cases¹) and does not precipitate airway obstruction—the chief fear in children.⁵ Fever and leucocytosis occur only occasionally.

MANAGEMENT

Once epiglottitis is suspected any further assessment and treatment should be performed in a resuscitation room of the emergency department by senior accident and emergency, otolaryngological, and anaesthetic staff. The patient should be urgently transferred to an intensive care facility. In the absence of respiratory distress the patient may be observed closely and given humidified oxygen; should there be symptoms or signs

of respiratory distress then emergency intubation or tracheostomy is preferred because of the high risk (18.3%) of developing complete airway obstruction. If this is imminent a cricothyrotomy may be life saving until a definitive airway can be established. A helium and oxygen mixture delivered by face mask can also be used as a temporising measure while help to secure the airway is being summoned.

After blood cultures have been taken, antibiotics against *Haemophilus influenzae* should be given intravenously. Ampicillin resistance is quite common and some centres prescribe chloramphenicol as well. Chloramphenicol resistance is not unknown, however; a third generation cephalosporin may be the drug of first choice until sensitivities are known—the choice of antibiotic for the patient in case 2 was not entirely rational. Steroids have been widely used, despite little evidence that they reduce mortality or obviate the need for intubation.⁸ Adrenaline given by nebuliser or parenterally is also of no definite benefit.

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MEMOIR

Lack of moral fibre

Watching a television programme recently on post-traumatic stress at RAF Lyneham I was reminded of my experiences as a medical officer to 59 Squadron on Thorney Island in 1941. The squadron was an operational unit of Lockheed Hudsons in Coastal Command. One of the most disturbing problems was the psychological trauma of returning aircrew from bombing raids. Some refused to face the ordeal again.

We had to classify them as showing lack of moral fibre—a diagnosis that resulted in officers and others being demoted to the lowest ranks. Such draconian measures were considered necessary at that time as we were desperately short of aircrew. The medical officer's task was further aggravated by the aircrew taking the view that those who had never experienced the hazards were not competent to make dispassionate judgments.

Realising that the problem was causing concern at the Air Ministry I offered, through my commanding officer, to go on several bombing raids in an effort to counsel those who were distressed with more authority. The ministry agreed rather reluctantly with several conditions. These included removing my medical insignia, emptying my pockets before flights, and not carrying my identity card but wearing my identification discs—all necessary precautions in case of a forced landing or parachute drop.

I took part in five operational flights in the autumn of 1941, which bombed naval targets in France and the Netherlands. In the last attack we bombed St Nazaire docks and encountered the heaviest antiaircraft barrage I experienced. Viewed from the underbelly turret of the

plane the tracers in different colours seemed to leave the ground very slowly then suddenly pass the aircraft at frightening speed. Evasive action was impossible. On the return journey we faced an added hazard from enemy night fighters. We lost some aircraft but we were fortunate to arrive home unharmed.

This completed my permitted quota involving some 20 hours of operational flying, a small stint in comparison with regular aircrew. I am uncertain whether I achieved my objective, but I did become aware of problems which had not occurred to me before. The long wait between the evening meal and take off was a period of tension. I also noticed that the oxygen masks used over 3000 m were sometimes loose fitting making physical effort difficult and occasionally painful. Aircrew with catarrh—there were no pressurised cabins—suffered symptoms from blocked Eustachian tubes.

So far as I am aware I was the only medical officer in the RAF to participate in a series of operational bombing sorties but other readers may correct me.

Today we refer to post-traumatic stress. The analysis is different, the approach more realistic, the counselling I hope more productive, and the Air Ministry seems to be more appreciative of the problems involved.—J L TRAINER was a medical officer in the RAF Voluntary Reserve, 1939-45

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