Acute epiglottitis in adults

Franziska Wick, Peter E. Ballmer, Alois Haller
Medizinische Klinik, Kantonsspital Winterthur

Summary

Acute epiglottitis can be a serious life-threatening disease because of its potential for sudden upper airway obstruction. It is a well-recognised entity in children but it is uncommon in adults and therefore is often misdiagnosed.

In this retrospective study we present twelve cases of acute epiglottitis in adults. The diagnosis was made by visualisation of the epiglottis using fibreoptic laryngoscopy. The illness was managed using a standardised management protocol (see Appendix). The most frequent symptoms were odynophagia (100%), inability to swallow secretions (83%), sore throat (67%), dyspnoea (58%) and hoarseness (50%). Body temperature was elevated (>37.2°C) in 75% and 50% of the patients had tachycardia (>100 bpm). The supposedly typical sign of stridor was found in only 42% of the cases.

A routine oropharyngeal examination does not exclude epiglottitis, 44% of our patients had a normal oropharynx and the diagnosis could only be made following fibreoptic laryngoscopy. Nasotracheal intubation was necessary in four patients. A 40-year-old man with sore throat, hoarseness, cough and odynophagia was initially seen by a physician. With the suspected diagnosis of an infection-induced exacerbation of bronchial asthma, he was treated with antibiotics, paracetamol und corticosteroids. On admission six hours later the patient was in coma. The diagnosis was not made until conventional oral endotracheal intubation (without a tracheotomy set placed at the bedside) was attempted. Unfortunately the intubation failed and the patient died.

Medical management of epiglottitis in adults includes antibiotics, NSAIDs and possibly inhalation with adrenaline. The maintenance of an adequate open airway is the main concern in adults as well as in children. Although most adults have no signs of airway obstruction, the clinical threshold for insertion of an airway should remain low, as it is the only way of preventing death.

A high index of suspicion is needed to recognise this rare disease correctly and patients must be admitted to a hospital with intensive care facilities, where the diagnosis can be confirmed and intubation performed if necessary and thus reduce the mortality rate.

Key words: acute epiglottitis; odynophagia; sore throat; visualisation of the epiglottis; fibreoptic laryngoscopy; airway placement

Introduction

Acute epiglottitis in adults is often referred to as supraglottitis as the inflammation is generally not confined to the epiglottis but can also affect supraglottic structures such as the pharynx, uvula, base of the tongue, aryepiglottic folds or the false vocal cords.

Acute epiglottitis is classically described as a haemophilus influenzae type b bacterial infection of the epiglottis in children [1]. In adults only 20% of epiglottitis is caused by haemophilus influenzae [1, 2]. Recent epidemiological studies have recorded a decline in the incidence of epiglottitis in children since the haemophilus b conjugate vaccines were introduced [3, 49, 50]. In Switzerland the annual disease frequency of meningitis and epiglottitis among 0–4 year olds decreased drastically by approximately 80% following the initiation of vaccination in 1990 [49]. In contrast, a steady increase in adult cases was noted (1975: 0.78/100’000; 1992: 2.9/100’000) [3, 52]. Overall mortality for adult epiglottitis is higher, (being estimated at 4–7% [4, 5]), than in children (2–3%) [3, 51], largely due to misdiagnosis and inappropriate treatment [6]. Using a well-standardised management of acute epiglottitis in children, including airway stabilisation, mortality could be reduced from 7.1 to 0.9% [15]. The use of the same management scheme in adults is the subject of controversial discussion.

Abbreviations:
NSAID: non steroidal antiinflammatory drug
ICU: intensive care unit

No financial support to declare.
In our retrospective study we present twelve cases with acute epiglottitis. We evaluate the clinical presentation and course using a standardised management protocol (Appendix).

**Patients and methods**

The charts of all adult patients with acute epiglottitis treated over a 2-year period from 1996–1998 in Kantonsspital Winterthur were studied retrospectively. Each record was reviewed by detailing clinical presentation, results of microbiological evaluation, method of airway management, use of antibiotics and the clinical outcome under a standardised management on the ICU (see Appendix).

There were nine men and three women with a mean age of 47.9 years (range: 24–76).

The diagnosis of epiglottitis was established in all cases by fibreoptic laryngoscopy in local anaesthesia followed by nasotracheal intubation in four cases (see Appendix). The diagnosis of acute epiglottitis was made if there was oedema of the epiglottis, of the aryepiglottic folds or the arytenoid soft tissue (fig. 1 and 2).
In our study five patients initially presented in the emergency room, seven patients had been previously seen by a physician. In only two of them was acute epiglottitis diagnosed, other diagnoses were asthma (2), pneumonia (1), upper airway infection (1) and angina with pharyngitis. Three patients had already been treated with antibiotics (two with amoxicillin/clavulanic acid, one with ceftriaxone). Where asthma was suspected a therapy with beta agonists, histamine antagonists and

Results

In our study five patients initially presented in the emergency room, seven patients had been previously seen by a physician. In only two of them was acute epiglottitis diagnosed, other diagnoses were asthma (2), pneumonia (1), upper airway infection (1) and angina with pharyngitis. Three patients had already been treated with antibiotics (two with amoxicillin/clavulanic acid, one with ceftriaxone). Where asthma was suspected a therapy with beta agonists, histamine antagonists and

Table 1
Symptoms of patients with acute epiglottitis (n = 12). † patient who died, √ correct diagnosis when admitted.

<table>
<thead>
<tr>
<th>Patients</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>Intubation</th>
<th>No intubation</th>
<th>total %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>51</td>
<td>32</td>
<td>51</td>
<td>31</td>
<td>37</td>
<td>76</td>
<td>65</td>
<td>54</td>
<td>74</td>
<td>24</td>
<td>40</td>
<td>40</td>
<td>mean: 42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intubation</td>
<td>No intubation</td>
<td>total %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odynophagia</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>12/12 (100%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inability to swallow secretions</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>10/12 (83%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sore throat</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>8/12 (67%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>7/12 (58%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hoarseness</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>6/12 (50%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emesis</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>4/12 (33%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cough</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>3/12 (25%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muffled voice</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>3/12 (25%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2
Signs of patients with acute epiglottitis (n = 12). † patient who died, √ correct diagnosis when admitted.

<table>
<thead>
<tr>
<th>Patients</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>Intubation</th>
<th>No intubation</th>
<th>total %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever (&gt;37.2°C)</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>9/12 (75%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tachycardia (100 bpm)</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>6/12 (50%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tachypnoea</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>5/12 (42%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pharyngitis</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>8/12 (67%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swelling of the epiglottis</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>7/12 (58%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervical lymph nodes</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>6/12 (50%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swelling of supraglottic tissue</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>5/12 (42%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inspiratory stridor</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>x</td>
<td>5/12 (42%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
steroids had been started. The time between a previous physician visit and admission was variable and ranged from 6 hours to 2 days.

Five patients (42%) were smokers and 3 (25%) had hypertension.

The symptoms and signs are summarised in tables 1 and 2. All of the patients had odynophagia, often with inability to swallow secretions (tables 1 and 2).

Once epiglottitis was suspected the patient was immediately transferred to the ICU accompanied by a doctor (see Appendix). After monitoring, rectal NSAIDs and adrenaline inhalations were administered if there were signs or symptoms of airway obstruction. The diagnosis was made in all cases by visualisation of the epiglottis by fiberoptic nasolaryngoscopy under local anaesthesia, followed by nasotracheal intubation in four conscious patients. (Appendix). A tracheotomy/cricothyrotomy set was always placed at the bedside. Duration of intubation was on average 4, 5 days (range 1, 5–6 days). Extubation was carried out when evidence of severe systemic inflammation had declined and the patient was able to breathe around the tube with the cuff deflated.

In 11 patients (91%) the white cell count was greater than $10 \times 10^9/\text{l}$ (mean $17.2 \pm 5 \times 10^9/\text{l}$, range $10.2–26 \times 10^9/\text{l}$). Pharyngeal cultures were performed in 2 patients (17%). They were both positive for haemophilus influenzae, once with additional streptococcus milleri. Blood cultures were performed in 10 patients (83%) and were positive in 3 (25%) – twice for pneumococci, once for haemophilus influenzae. All were treated with antibiotics (amoxicillin/clavulanic acid) and with rectal NSAIDs. Four patients (33%) received corticosteroids. Five patients (42%) inhaled with adrenaline.

A 40-year-old man (patient 11) with a sore throat, hoarseness, cough and odynophagia was initially seen by a physician. With the suspected diagnosis of an infection-induced exacerbation of bronchial asthma he was treated with antibiotics, paracetamol and corticosteroids. Six hours later on admission the patient was already in coma. The diagnosis was not made before an attempt at conventional oral endotracheal intubation (without tracheotomy set placed at the bedside). Unfortunately the intubation failed and the patient died.

**Discussion**

Acute epiglottitis is an infectious disease of the epiglottis and/or supraglottic structures, which may progress rapidly to complete airway obstruction. The diagnosis of adult epiglottitis is often delayed [7]. In our study seven patients had been seen previously by a physician. In only two of them was acute epiglottitis diagnosed. Increased clinical awareness, careful airway protection and appropriate antibiotic therapy of acute epiglottitis should be effective in minimising mortality in adults.

**Epidemiology**

In contrast to the incidence of acute epiglottitis in children the incidence in adults has shown a steady increase (1975: 6.1/100’000; 1992 0.3/100’000) [3, 52] (fig. 3).

Swiss and Californian data show the same trend. Following the initiation of vaccination for Haemophilus b, the incidence of epiglottitis in children decreased drastically [7, 49]. During the same time, the incidence of acute epiglottitis in adults in California has remained relatively stable, with a mean incidence of 1.8/100 000 [7].

In the literature most authors describe a preponderance of male subjects (59–70%) [3, 8, 9], corresponding to our observations with a male to female ratio of 9:3. Five of our twelve patients were smokers, a possible reason for the male preponderance. Another risk factor was hypertension (25%). In the literature frequent underlying medical conditions are diabetes mellitus, hypertension and alcohol abuse [3, 7]. Seasonal incidence varies from series to series [3, 7, 10].

---

**Figure 3**

Clinical presentation

Most adults who present with acute epiglottitis complain of sore throat and odynophagia [3, 9]. Our patients all suffered from odynophagia. Cervical lymph nodes and diffuse swelling of the throat are not specific for acute epiglottitis but in combination with tachypnoea and inspiratory stridor the diagnosis of acute epiglottitis should be considered.

The differential diagnosis of adult epiglottitis includes infectious processes such as mononucleosis, diphtheria, pertussis, croup, tonsillitis, Ludwig’s angina with retropharyngeal, parapharyngeal and peritonsillar abscesses, tracheobronchitis, subglottic laryngitis, as well as non-infectious diseases such as allergic reactions, angioneurotic oedema, foreign body aspiration, reflex laryngospasm, laryngeal trauma, tumours, hydrocarbon aspiration, systemic lupus erythematosus and inhalation of toxic fumes or superheated steam [11, 14].

Diagnostic procedures

Fibreoptic laryngoscopy is the most accurate way to diagnose acute epiglottitis. A routine oropharyngeal examination does not exclude epiglottitis [7]. About one third of the patients with proven epiglottitis have a normal oropharynx. 44% of our patients had a normal oropharynx and the diagnosis could not be made until fibreoptic laryngoscopy was performed. Laryngoscopic examination can be performed safely in adults in contrast to children [4, 6, 9, 11]. Death due to laryngospasm and airway compromise during laryngoscopic examination has not been described in adults [9, 12]. Soft-tissue x-ray films to diagnose acute epiglottitis are controversial. Sensitivity and specificity vary from author to author between 98% [15] and 38% [16]. In our opinion x-rays should not be done because of a potential delay in diagnosis and the danger of airway obstruction. Computed tomography or MRI is not recommended to establish the initial diagnosis of epiglottitis, but rather to exclude complications such as peritonsillar abscess, abscesses of the deep neck space, lingual tonsillitis, laryngitis or an ingested foreign body [17]. CT findings in acute epiglottitis may be swelling of the supraglottis, obliteration of surrounding fat planes and thickening of the platysma muscle and prevertebral fascia [17]. In our study, computed tomography was carried out in two patients with a prolonged course: in one it showed a thickness of the vallecula and in the other a massive swelling of supraglottic structures.

In 11 of our patients (91%) the white-cell count was greater than 10×10⁹/L (mean 17.2 ± 5 × 10⁹/L, range 10.2–26×10⁹/L), corresponding to the data of Mayo-Smith et al. with an elevation in 80% (>10×10⁹/L) [4]. Review of the literature revealed positive blood-cultures in 31% [18]. Trollfors et al. [1] showed that the addition of serology and PCR to blood cultures doubled the power of verifying the aetiology of acute epiglottitis in adults: detection of haemophilus influenzae in 26%, pneumococci in 22% and group A streptococci in 9% [11]. The aetiology remained unknown in 43% [1]. In our study blood cultures were performed in 10 patients (83%) and were positive in 3 (25%) – twice for pneumococci, once for haemophilus influenzae. All were treated with antibiotics (amoxicillin/clavulanic acid) and with rectal NSAIDs. In our experience microbiological studies were of little importance as they did not influence the management of disease. Throat cultures are even less informative than blood cultures [10, 19]. Whereas paediatric epiglottitis is commonly caused by haemophilus influenzae, in adults a variety of microbes are found (table 3).

Complications

A prolonged clinical course suggests that a complication such as epiglottic abscess, uvulitis or pneumonia has developed [3, 29–31]. Three of our patients had pneumonia, one an epiglottic abscess and one herpangina.

Therapy

Intravenous antibiotics should be started immediately and should cover haemophilus influenzae, s. aureus, streptococcus and pneumococcus (amoxicillin/clavulanic acid or a third generation cephalosporin). Symptomatically NSAIDs can be helpful. All of our patients were treated with (amoxicillin/clavulanic acid) and with rectal NSAIDs. Corticosteroids have often been recommended for epiglottitis. However, there are no

Table 3

Organisms that can cause acute epiglottitis in adults [4, 13, 14, 20–28].

<table>
<thead>
<tr>
<th>Organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteroides melanogenicus</td>
</tr>
<tr>
<td>Branhamella catarrhalis</td>
</tr>
<tr>
<td>Enterobacter cloacae</td>
</tr>
<tr>
<td>Fusobacterium necrophorum</td>
</tr>
<tr>
<td>Haemophilus parainfluenza</td>
</tr>
<tr>
<td>Kingella kingae</td>
</tr>
<tr>
<td>Neisseria meningitidis</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
</tr>
<tr>
<td>Pneumococci</td>
</tr>
<tr>
<td>Streptococcus milleri</td>
</tr>
<tr>
<td>Streptococcus pyogenes</td>
</tr>
<tr>
<td>Vibrio vulnificus</td>
</tr>
<tr>
<td>Group A streptococci</td>
</tr>
<tr>
<td>Citrobacter diversus</td>
</tr>
<tr>
<td>E. coli</td>
</tr>
<tr>
<td>Haemophilus influenzae</td>
</tr>
<tr>
<td>Klebsiella pneumoniae</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
</tr>
<tr>
<td>Pasteurella multocida</td>
</tr>
<tr>
<td>S. aureus</td>
</tr>
<tr>
<td>Pneumococci</td>
</tr>
<tr>
<td>S. viridans</td>
</tr>
<tr>
<td>Viruses</td>
</tr>
<tr>
<td>Candida (in immunocompromised patients)</td>
</tr>
<tr>
<td>Aspergillus (in immunocompromised patients)</td>
</tr>
</tbody>
</table>
controlled data on their usefulness in this setting. The use of corticosteroids did not reduce the need for intubation, the duration of intubation, the duration of ICU stay or the duration of hospitalisation [3, 9]. In our study corticosteroids showed no effect. However, the relatively small number of patients observed does not allow a firm conclusion in regard to the use of corticosteroids. In our series five patients inhaled adrenaline while waiting for laryngoscopy. None of them had to be intubated. However, data demonstrating the efficacy of adrenaline inhalations are still not available.

The role of airway intervention in adults is controversial. There are authors who prefer a conservative management with antibiotics, corticosteroids and humidified oxygen [10, 18–41], others plead for an aggressive airway management with early intubation [6, 40, 42–46]. Mortality among children has dropped from 7.1 to 0.9% since the use of prophylactic airway intervention [15]. A mortality of 1–7% [4, 5, 7] among adults has been described but in patients with acute respiratory obstruction it was 17.6% [47]. Respiratory distress, stridor, sitting erect, inability to swallow secretions and deterioration within 8–12 hours are the major signs and symptoms associated with the need for intubation [3, 7, 48]. When in doubt we think early intubation is the safest approach to preventing death.

References


Correspondence:
Franziska Wick, MD
Kantonsspital Winterthur
Medizinische Klinik
Brauerstrasse 15
CH-8400 Winterthur
e-mail: wick.f@bluewin.ch
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