ACUTE EPIGLOTTITIS IN CHILDREN
Review of 27 patients

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SUMMARY

Of 27 children treated for acute epiglottitis, 26 survived. Eighteen patients treated by nasotracheal intubation were devoid of sequelae, whereas of six patients in whom tracheotomy was performed, one developed an ugly scar and two had slight tracheal stenosis at the tracheostomy site. In 22 of the 27 children the diagnosis of the referring physician was wrong, causing serious delay in securing the airway. We conclude that short-term nasotracheal intubation and antibiotic prophylaxis is the optimal treatment for acute epiglottitis in children. However, such patients are often in a critical condition, and it is essential that a well-planned procedure for examination and treatment is established in each hospital.

Acute epiglottitis is still one of the most dangerous infectious diseases in children because of a rapid progress to airway obstruction, exhaustion and sudden death from hypoxia. The condition is rare and, in our experience, is mistaken too often for pseudo-croup or other less dangerous conditions. This may cause serious delay in establishing an artificial airway. While there is agreement that the airway must be secured in children with acute epiglottitis, the debate continues on whether to perform tracheotomy or endotracheal intubation (Tarkkanen and Kohonen, 1972; Heldtander and Lee, 1973; Sweeny, Allan and Steven, 1973; Tos, 1973; Milko, Marshak and Striker, 1974; Adair and Ring, 1975; Battaglia and Lockhart, 1975; Schuller and Birck, 1975).

We have reviewed our experience of 27 children suffering from acute epiglottitis: 18 were treated by nasotracheal intubation and six by tracheotomy. In a follow-up study we have sought evidence of permanent vocal cord or tracheal injury or other sequelae.

PATIENTS AND METHODS

The hospital records of 27 children treated for acute epiglottitis between January 1, 1971 and June 30, 1977 were reviewed. During the first 2 years of this period, tracheotomy was performed routinely, but from 1973 all patients with acute epiglottitis and needing support of the airway were managed with endotracheal tubes passed through the nose or the mouth.

The patients and their parents were interviewed at between 2 months and 5½ yr after the illness. A physical examination and x-ray examination of the trachea were performed with particular reference to evidence of damage to the vocal cords or trachea and psychological sequelae of the treatment.

RESULTS

Details of the children studied are shown in table I.

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>2-</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>3-</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4-</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>5-</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>6-</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7-</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>8-</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>9-</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>15-</td>
<td>0</td>
<td>1*</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>10</td>
<td>27</td>
</tr>
</tbody>
</table>

* Dead on arrival at the hospital.

The diagnosis of the referring physicians was correct in only five of the 27 patients, the majority being misdiagnosed as pseudo-croup or laryngitis (table II).

All patients had respiratory difficulty of varying degree and, although many of the early records were incomplete, the diagnosis was confirmed in all instances by direct laryngoscopy. However, as we intensified our teaching about the management of epiglottitis to the house staff, more of the records show a typical history of epiglottitis:

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Table II. Diagnosis of referring physicians in 27 patients with acute epiglottitis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudocroup/laryngitis</td>
<td>14</td>
</tr>
<tr>
<td>Epiglottitis</td>
<td>5</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>2</td>
</tr>
<tr>
<td>Asthma</td>
<td>1</td>
</tr>
<tr>
<td>Coryza</td>
<td>1</td>
</tr>
<tr>
<td>Foreign body</td>
<td>1</td>
</tr>
<tr>
<td>Dyspnoea/stridor</td>
<td>2</td>
</tr>
<tr>
<td>Cardiopulmonary arrest</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
</tr>
</tbody>
</table>

Sudden onset of high fever (mean of 20 rectal temperatures recorded on admission was 39.4 °C, range 38.5-40.6 °C)
dysphagia in older children
refusal to take food in infants and small children
drooling and a thick muffled voice in a quiet, pale child who preferred to sit, often leaning forward.
The blood leucocyte count was obtained in only seven patients before antibiotic therapy was started. The mean value was $18 \times 10^9$ cells litre$^{-1}$ (range 13-25 $\times 10^9$ cells litre$^{-1}$).

In 25 of the 27 patients bacteriological specimens from the pharynx, epiglottis, nose, trachea or blood were studied. *Haemophilus influenzae* was cultured from one or more of these sites in 12 patients. Blood cultures were positive for *H. influenzae* in five of nine patients; three of the four negative cultures were taken after antibiotic treatment had started. One blood culture revealed a massive growth of *Staphylococcus aureus*; this blood culture was from a patient who had *H. influenzae* in the tracheal secretions. Two patients had beta-haemolytic streptococci in cultures from the pharynx.

Treatment

Ampicillin was used in doses varying from 200 to 400 mg kg$^{-1}$ per 24 h. One patient with *H. influenzae* in the tracheal culture was still febrile after 3 days of treatment with ampicillin in an adequate dosage, and he was then given chloramphenicol, which produced a rapid decrease in temperature to normal. Unfortunately, the antibiotic sensitivity of the cultured *H. influenzae* was not determined.

Two patients had received antibiotics before admission to hospital and were not fevered, with only minor respiratory distress and moderate oedema of the epiglottis. They did not require an artificial airway (table III).

Twenty-five patients required an artificial airway. In 1971 and 1972 tracheotomy was performed on six patients, under halothane anaesthesia with orotracheal intubation.

In 1973, a 15-year-old girl arrived in the emergency department 45 min after suffering respiratory arrest at home, about 36 h after the onset of fever and a sore throat. On the way to hospital her father attempted mouth-to-mouth ventilation, but he was not able to ventilate the lungs. On direct laryngoscopy the epiglottis was grossly enlarged (“like a mandarin”), but it was possible to perform orotracheal intubation, and adequate spontaneous circulation was established 20 min later. The patient showed the features of brain death during 2 days of subsequent artificial ventilation and then suffered a further cardiac arrest.

Nasotracheal intubation was performed in 18 patients during the past 3.5 yr. Two of these patients had shallow breathing and were cyanosed and unconscious on arrival. They were taken immediately to the operating room and an oral endotracheal tube was inserted without anaesthesia. A third patient arrived with only moderate respiratory difficulties and diazepam was given rectally to treat anxiety. The resulting sedation caused the patient to lie down, and he soon became apnoeic, cyanosed and unconscious. The anaesthetist inserted an oral endotracheal tube rapidly without anaesthesia. The orotracheal tubes in these three patients were replaced by nasotracheal tubes under halothane in oxygen anaesthesia. The remaining 15 patients received an inhalation induction

Table III. Duration of treatment of epiglottitis. Mean value and range (in parentheses)

<table>
<thead>
<tr>
<th>Intervention</th>
<th>No. of patients</th>
<th>Duration of intubation (h)</th>
<th>Duration of hospital stay (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tracheotomy (1971-72)</td>
<td>6</td>
<td>80 (72-96)</td>
<td>6 (6-10)</td>
</tr>
<tr>
<td>Nasotracheal intubation* (1974-77)</td>
<td>17</td>
<td>33 (13-72)</td>
<td>6 (4-9)</td>
</tr>
<tr>
<td>Nasotracheal intubation (1976-77)</td>
<td>9</td>
<td>27 (17-37)</td>
<td>6 (4-9)</td>
</tr>
<tr>
<td>Antibiotic agent only† (1972 and 1974)</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

* Patients treated with nasotracheal intubation, except one who had *H. influenzae* resistant to ampicillin. The tube was removed on day 5 after 2 days of treatment with chloramphenicol. Duration of hospital stay 8 days.
† These two patients had received antibiotics before admission and had only moderate oedema of the epiglottis, and only slight respiratory distress which did not require intubation of the trachea.
of anaesthesia with nitrous oxide, oxygen and halothane, or halothane in oxygen, and nasotracheal intubation was performed whilst anaesthesia was maintained with halothane in oxygen. Ventilation was assisted usually with a bag and mask with the patient in the sitting position during induction of anaesthesia. The gastric contents were not aspirated before induction of anaesthesia, since we assumed the stomach was empty as a result of the dysphagia. In no instance did vomiting occur. Neuromuscular blocking drugs were not given. In many instances we were guided to the laryngeal inlet by mucous bubbles appearing in the cleft between the oedematous aryepiglottic folds and the swollen epiglottis. Five of the six patients treated by tracheotomy and two of the patients treated with nasal endotracheal tubes received steroids parenterally.

**Outcome**

The mean duration of intubation in the tracheotomy group was 80 h (range 72–96 h) and mean hospital stay was 8 days (range 6–10 days). The mean duration of intubation of 17 of the 18 patients in the nasotracheal tube group was 33 h (range 13–72 h). In one patient, however, the nasotracheal tube was retained for 5 days. This patient had *H. influenzae* in the tracheal secretions, but did not become afebrile following treatment with ampicillin. The tube was removed on the 3rd day in spite of the rectal temperature being 38–39 °C and the epiglottis being red and swollen, and it had to be reinserted. Chloramphenicol therapy was commenced and the patient became afebrile but the epiglottis remained swollen. Two other patients managed to remove the tube themselves 7 and 13 h after nasotracheal intubation because of an inadequately secured tube and insufficient sedation. In all four patients the nasotracheal tubes were reinserted easily.

**Complications**

There were no serious complications during treatment with an artificial airway (table IV). In two patients with nasal endotracheal tubes extubation of the trachea occurred accidentally soon after the initial intubation, but this was caused probably by inadequate insertion of the tube. Two other patients managed to remove the tube themselves 7 and 13 h after nasotracheal intubation because of an inadequately secured tube and insufficient sedation. In all four patients the nasotracheal tubes were reinserted easily.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Tracheotomy</th>
<th>Nasotracheal intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accidental extubation</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Transient hoarseness after extubation</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Ugly tracheostomy scar</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Tracheal stenosis by x-ray (slight)</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Permanent vocal cord damage</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Psychological sequelae</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

On x-ray examination, two of 18 patients with a nasotracheal tube and one of six patients with a tracheostomy had evidence of lung collapse (table IV). Pneumonic infiltrations on the initial chest x-ray, probably associated with the disease and not complications of the treatment, were present in one of the patients with a tracheostomy and nine of the 18 patients with a nasal tube.

Two patients had slight stridor after extubation. This resolved after a short time following treatment with nebulized racemic adrenaline. Five patients had transient hoarseness after removal of the nasal endotracheal tube (table IV).

**Follow-up study**

One of six patients developed an ugly scar at the tracheostomy site. X-ray examination of the trachea of 24 patients 2 months to 5 yr after treatment with an artificial airway showed slight tracheal stenosis at the site of the stoma in 2 patients who had a tracheostomy. Neither of the patients with radiologically detectable tracheal stenosis had dyspnoea, either on exertion or during periods of upper airway infection.

In the follow-up study there was no evidence of permanent vocal cord damage in any of the patients treated with artificial airways.

The parents of four of the patients managed with nasotracheal tubes thought their children had a slight change of voice after treatment, and although their voices appeared normal to us, the children were referred to an otolaryngologist. Following indirect laryngoscopy in three patients and direct laryngoscopy in one patient he reported normal findings.

None of the patients have shown any obvious adverse psychological effects of their experiences. The patient with the ugly scar is the only one with a possible psychological problem following treatment; she is teased by her friends about the scar.
DISCUSSION

It is well known that boys suffer from acute epiglottitis more commonly than do girls, and this is confirmed in our study. However, our patients were younger than is usual in this condition. More than half of our 27 patients were less than 3 years old, and more than 80% were less than 5 years of age. These findings are similar to those of Blanc and others (1977).

The classical history and symptoms of acute epiglottitis were found in many of our patients: a sudden onset of high fever with dysphagia or refusal to take food in a quiet, obviously sick child; respiratory difficulties; a preference for a sitting position, frequently leaning forwards; drooling; a thick, muffled voice and often no conspicuous cough. Once seen, this state is recognized easily on subsequent occasions and may be differentiated from the restless, noisy, afebrile child with a hoarse, barking dry cough of laryngitis or pseudo-croup (Battaglia and Lockhart, 1975; Blanc et al., 1977).

H. influenzae was cultured in specimens from only 12 of 27 patients, but may have been the causative agent in all patients, since a positive culture depends upon several technical factors (Margolis, Colletti and Grundy, 1975). H. influenzae resistant to ampicillin has not been reported in Norway, and unfortunately the antibiotic sensitivity of the H. influenzae cultured from the only patient in our series not responding to ampicillin was not determined. Ampicillin should be the drug of first choice still for epiglottitis in Norway, although chloramphenicol is now the initial antibiotic given by Oh and Motoyama (1977). Steroids were not used in most of our patients and appear to be of no value in this condition (Tos, 1973).

Apart from the 15-year-old girl, who had a respiratory arrest at home, treatment was successful in all patients and there were no serious sequelae. However, delay in securing the airway as a result of a lack of appreciation of the problems (table II) might have resulted in disaster in some of the patients. The most important outcome of our study is an increased awareness of epiglottitis among hospital personnel and primary physicians in our district, and the development of a safe procedure for evaluation and treatment of patients suspected of having the disease. An outline of this regime is given in the Appendix and a more detailed account may be obtained from the authors.

Provided this regime or a similar one (Heldtander and Lee, 1973; Oh and Motoyama, 1977) is followed, we believe that short-term nasotracheal intubation (rather than routine tracheotomy) and antibiotic therapy is the optimum treatment of children with acute epiglottitis. As we gained experience it became apparent that the period of intubation could be reduced to less than 24 h in most patients. This is in agreement with the experience of Adair and Ring (1975) and Shann and others (1975), who stated that the intubation time for most children could probably be reduced to 8–12 h. The importance of this finding is evident: the risk of damage to the vocal cords and the tracheal mucosa should be small. Hilding (1971) found only minimal mucosal changes in a few patients when the period of intubation lasted less than 48 h.

The primary physician

If a child is suspected of having acute epiglottitis, the physician is urged to avoid inspecting the epiglottis with a tongue depressor. Instead, he should accompany the patient to hospital, keeping the patient in a sitting position and leaning forward. Sudden death from epiglottitis is probably caused more often by hypoxia than by complete airway obstruction (Johnson, Sullivan and Bishop, 1974; Adair, 1975; Adair and Ring, 1975). The patient should be given oxygen, preferably by directing an oxygen flow over the patient’s face, thus avoiding a mask which may distress the patient and increase oxygen consumption. The patient should not be given sedatives which produce drowsiness and cause him to lie down, allowing the epiglottis to fall posteriorly over the laryngeal inlet and producing an increased risk of airway obstruction. The patient should not be given injections because anxiety and crying, in response to the injection, increase oxygen consumption, aggravate the hypoxaemia and increase the danger of sudden death (Adair, 1975). If the period of transport to hospital is long, and the patient is still able to swallow liquids, ampicillin should be administered by mouth.

If respiratory arrest occurs, the patient should be ventilated artificially with oxygen. Airway obstruction is usually not complete, and it has been possible to resuscitate patients with mouth-to-mouth ventilation (Johnson, Sullivan and Bishop, 1974; Adair and Ring, 1975).

Examination and treatment of patients with acute epiglottitis

An outline of our programme for the examination and treatment of patients with acute epiglottitis is shown in the Appendix.

The patient suspected of having acute epiglottitis should be held in the sitting position and disturbed as little as possible until all equipment and personnel are ready for immediate tracheal intubation or
ACUTE EPIGLOTTITIS IN CHILDREN

tracheotomy. The diagnosis is confirmed by direct laryngoscopy by a senior anaesthetist.

Unless the patient is in severe respiratory distress, inhalation anaesthesia is induced with halothane in nitrous oxide in oxygen and deepened by assisted ventilation using halothane in oxygen only.

No neuromuscular blocking drugs are given; for as long as the patient breathes spontaneously mucous bubbles are usually seen at the laryngeal inlet, and this facilitates orientation during intubation. A nasal endotracheal tube is placed in the trachea and taped securely using tincture of benzoin or mastic solution on the skin and tube. The patient is sedated with morphine and diazepam or a mixture of pethidine, chlorpromazine and promethazine given i.v. as needed.

When the patient has become afebrile for a few hours, following treatment with ampicillin i.v., the tube may be removed even if the epiglottis remains swollen.

This programme is similar to those used by Oh and Motoyama (1977) in 12 patients, and by Heldtander and Lee (1973), who have treated more than 100 children with acute epiglottitis by nasotracheal intubation without complications (P. Lee, personal communication).

APPENDIX

OUTLINE OF TREATMENT PROCEDURE FOR ACUTE EPIGLOTTITIS

All children with respiratory distress are examined in an emergency room fully equipped for establishing an artificial airway.

When a child is in severe respiratory distress, the resuscitation team is called immediately to the emergency department.

Suspect acute epiglottitis when the child:

has respiratory distress, a sore throat and fever

is sitting, leaning forward

is drooling, with a thick muffled voice.

Procedure

Child sits on the lap of one of the parents.

Anaesthetist inspects the epiglottis using a laryngoscope. If epiglottitis is verified, anaesthesia is induced with halothane in nitrous oxide in oxygen, and continued with assisted ventilation with halothane in oxygen.

Give atropine i.v. or i.m.

Intubate the trachea via the nose.

Tape the tube securely, using tincture of benzoin or mastic solution.

Extubate if the epiglottis is pale, even if it remains swollen.

When the child has been afebrile for 6–12 h, inspect the epiglottis under anaesthesia delivered through the tube.

Keep the patient well sedated with morphine and diazepam i.v., or a mixture of pethidine, chlorpromazine and promethazine i.v.

Inject saline into the tube, bag-ventilate and suction every hour.

Measure rectal temperature every 2 h.

Extubation

When the child has been afebrile for 6–12 h, inspect the epiglottis under anaesthesia delivered through the tube. Extubate if the epiglottis is pale, even if it remains swollen.

ACKNOWLEDGEMENTS

We thank Professor O. Garborg and Drs K. Evensen and K. Dalby for valuable help in the follow-up study.

REFERENCES


Sur les 27 enfants qui ont été traités pour épiglottite aiguë, 26 ont survécu. Dix-huit malades qui avaient été traités par intubation nasotrachéale n’ont eu aucune sequelle, alors que sur les six malades sur lesquels on avait pratiqué une trachéotomie, l’un d’eux en a conservé une cicatrice inesthétique et deux autres une sténose trachéale à l’emplacement même ou l’on a effectué la trachéotomie. Dans le cas de 22 enfants sur 27, le diagnostic du médecin traitant était erroné, ce qui a entraîné un fâcheux retard dans le dégagement du passage d’air. Nous en concluons que l’intubation nasotrachéale à court terme et l’administration d’antibiotiques constituent le traitement principal de l’épiglottite aiguë chez les enfants. Les malades de ce genre sont néanmoins souvent dans un état critique et il est essentiel de déterminer dans chaque hôpital une procédure bien planifiée, pour l’examen et le traitement.