Clinical Problems

Haemophilus Epiglottitis: Nine Recent Cases in Oxford

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Summary

Recent experience of epiglottitis in Oxford confirms the need for doctors in this country—especially family doctors and casualty officers—to be aware of this disease and to be able to recognize it and to take appropriate action immediately. Some important diagnostic and therapeutic implications both of the anatomy of the supraglottic lesion and of the septicaemic component of the illness are discussed.

Introduction

Acute epiglottitis is a severe bacterial infection, potentially fatal within a few hours of onset, which occurs mainly in children aged 2-7 years. In most cases it is possible to show that the causative agent is a type b strain of *Haemophilus influenzae*. Reports from North America, where this condition has been recognized for over 30 years,^{1 2} have established the following points: (a) it is not uncommon there, accounting for about 8% of admissions for "croup" in some series^{3 4}; (b) it is clinically distinguishable from other forms of croup^{5 6}; (c) its very high mortality rate in the absence of appropriate treatment can be reduced to low levels provided that the patients reach hospital alive and are then correctly diagnosed and correctly treated without delay—for example, Berenberg and Kevy⁷ reported two deaths among 39 patients, and Baxter⁴ one death in 103 patients. Phelan and Williams⁸ reported similar findings in Australia.

Recorded British experience has been strikingly different. Until recently reports have been few and the diagnosis has usually been made at necropsy.^{9 10} In the past few years, however, successful treatment of 25 patients has been reported in the British literature.¹¹⁻¹³ Nineteen of the 25 were seen by Jones¹² over a 20-year period, mainly in the Bradford-Leeds area, and he calculated that epiglottitis accounted for over 7% of the children admitted to the Bradford Children's Hospital between 1954 and 1969 with the diagnosis of laryngotracheitis. These facts suggest that the American pattern has existed in Britain but has gone unrecognized except in the Bradford-Leeds area. On the other hand, Russell,¹⁴ who based his account of epiglottitis on experience gained in the U.S.A. in 1965-7 and on many cases seen in Aberdeen since his return there in 1967, believes that the disease was not occurring in that area before 1965 (personal communication).

We are not concerned here to give a detailed description of yet another series of patients with epiglottitis. The following brief account of our local experience is intended to emphasize some points implied in the previous paragraph—that the frequency with which epiglottitis is recognized depends on local and personal awareness of its existence, and that family doctors and casualty officers in Britain need to be on the look-out for it and to know how to treat it. We also draw attention, in the Discussion, to some aspects of the pathogenesis and anatomy of epiglottitis which make for clearer understanding of its differential diagnosis and treatment.

Case Reports

FATAL CASE AND CONSEQUENT AWARENESS

In September 1969 a 2½-year-old boy died shortly after admission to the Radcliffe Infirmary. The diagnosis of epiglottitis, made clinically just before he died, was confirmed by necropsy findings and by culture of *H. influenzae* type b from his epiglottis and heart blood. So far as we can determine this was the first time that the diagnosis had been made in this hospital on a live patient, though it had occasionally been made here at necropsy on children who had died before admission. This first case provoked discussion on epiglottitis, with a view to its rapid recognition and treatment, at two meetings involving local family doctors and the departments of paediatrics, otolaryngology, anaesthetics, and bacteriology.

FOUR SUCCESSFUL CASES

Three children with clinical features of epiglottitis were admitted during November and December 1969. In all three the clinical diagnosis of epiglottitis was confirmed by laryngoscopy. They all had tracheostomies, received chloramphenicol, and made good recoveries. *H. influenzae* type b was recovered from the upper respiratory tracts and from blood cultures of the first two, a boy and a girl both aged 3 years. The third child was the 2-year-old sister of the second child and was admitted three weeks after her sister. No causative organism was isolated but she had received intramuscular ampicillin before coming to hospital. Nine days after admission her blood had a raised antibody titre for the capsular antigen of *H. influenzae* type b.¹⁵ These two children appear to provide an example of case-to-case transmission of epiglottitis, which so far as we know has not previously been described.

In September 1970 a 1½-year-old boy was admitted with similar features, was treated in the same way, and made a good recovery. Again the diagnosis was corroborated by isolation of *H. influenzae* type b from his blood.

TWO FURTHER DEATHS

In October 1970 the family doctor who had sent in the two sisters in December 1969 was called to see a 5-year-old girl; she had complained of severe sore throat since walking three hours earlier and stridor had been present for one hour. The doctor diagnosed epiglottitis and gave intramuscular ampicillin immediately, but the

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child stopped breathing as she was being carried out of the house and could not be revived. Postmortem appearances were typical of epiglottitis, and a heavy pure growth of *H. influenzae* type b was obtained from fluid aspirated from inside the swollen epiglottis.

In December 1970 a 22-month-old boy was seen one evening by his doctor with apparently mild croup. The doctor called again the next morning, found the child in a collapsed state, and sent him to hospital immediately with the diagnosis of bronchopneumonia. He died within a few minutes of reaching hospital. Heart blood and a throat swab taken immediately after death grew *H. influenzae* type b, and necropsy confirmed the diagnosis of epiglottitis.

TWO SUCCESSFUL CASES WITH ASSOCIATED FAMILY INFECTIONS

In March 1971, within a period of 12 hours, two boys aged 2½ and 3½ years were admitted to the Infirmary from homes several miles apart. In the first case the diagnosis was not at first clear on clinical grounds but it was established by the finding of epiglottic and aryepiglottic swelling in a soft-tissue x-ray picture (Dr. G. M. Ardran). While being carried to the operating theatre the child had a respiratory arrest and was given mouth-to-mouth respiration. A bronchoscope was then passed and tracheostomy performed. He received chloramphenicol and made a good recovery.

The second boy was rapidly diagnosed on clinical grounds and he also had a tracheostomy, received chloramphenicol, and made a good recovery. *H. influenzae* type b was grown from the blood of the first boy and from the nasopharynx (but not the blood) of the second.

Each of these two boys had one younger brother, and both of these developed interesting infections. Two days after the boys with epiglottitis were admitted the 11-month-old brother of the first of them developed an extensive purple bruise-like swelling of his cheek. This lesion conformed closely to the description of haemophilus cellulitis of the face given by Green and Fousek¹⁶ and other North American authors. *H. influenzae* type b was isolated from his nasopharynx but not from a blood culture; no attempt was made to isolate it from the lesion. He was given chloramphenicol and the lesion disappeared within a few days. Two months later the 1½-year-old brother of the other epiglottitis patient was brought urgently to hospital with symptoms closely resembling those of his brother—notably fever, apparent soreness of the throat, and profuse drooling. After preliminary examination, which did not include inspection of his throat (for reasons that will be made clear below), he was taken to the operating theatre, where laryngoscopy showed that the infection was not epiglottitis but follicular tonsillitis. He made a rapid recovery on treatment with benzylpenicillin, but no pathogens were isolated from his throat. Because of his brother's history, however, his initial investigations had included blood culture, and this gave a growth of *H. influenzae* type b. We wonder how often blood cultures from young children with tonsillitis not due to streptococci might give similar results.

Discussion

We would stress that despite its name this disease is not merely a local inflammation but also a septicaemic condition. Because of this, after an onset suggesting only a mild respiratory tract infection the severity of the illness may increase very rapidly and out of proportion to the degree of airway obstruction. Inspiratory stridor decreases as respiratory effort weakens. Both the septicaemia and the anoxia resulting from respiratory obstruction play a part in this weakening. Failure to deal immediately with both components has undoubtedly been responsible for fatalities. The child urgently requires both effective antibacterial therapy and relief of the airway obstruction. For the former purpose, when the child is in hospital we prefer chloramphenicol to ampicillin because of reported ampicillin failures in treatment of haemophilus meningitis.¹⁷ These failures have been attributed by some to inadequate dosage,¹⁸ but we know of no reports of comparable chloramphenicol failures. The family doctor is more likely to have ampicillin to hand and should use it immediately if he suspects epiglottitis even though this will spoil the chances of bacteriological con-

firmation of his suspicions; he will need to give it intramuscularly because of the dysphagia. His suspicion of epiglottitis is also a compelling reason for urgent admission of the patient to hospital so that both the septicaemia and the airway obstruction can be adequately treated.

The name epiglottitis is now firmly established in the literature, but as well as distracting attention from the septicaemic element it gives a misleading idea of the local inflammatory lesion. This involves not only the epiglottis, with its consequent, much-described "red cherry" appearance, but also the aryepiglottic and interarytenoid folds. There is thus a ring of inflamed oedematous tissue surrounding the supraglottic laryngeal space; "supraglottic laryngitis" is a more accurately descriptive name. This supraglottic localization has important implications for diagnosis and treatment. The child has intense sore throat and dysphagia (often indicated by drooling) and may also have cervical adenitis (principally "tonsillar") and some consequent neck swelling. All of this suggests severe pharyngitis, yet the pharynx itself may be virtually normal. (Suspicion of epiglottitis is in any case a contraindication to examining the pharynx—see below.)

Anatomical considerations also throw light on some important differences between virus laryngitis and bacterial epiglottitis. In virus infections involving the vocal cords the voice is hoarse and there is stridor due to airway narrowing, but the structure of the cords does not permit their rapid or gross distension by oedema fluid. Obstruction does not increase rapidly and is unlikely to become severe, and so there is no urgent indication for surgery. In contrast, supraglottic inflammation leads to muffling of the voice rather than true hoarseness. Oedema fluid collects easily and rapidly in the lax vascular supraglottic tissues, but because of the relatively large size of the supraglottic space the extent of this swelling may at first not be apparent from the relatively slight respiratory obstruction. Establishment of an artificial airway, however, is urgently required because complete obstruction is likely to develop and may do so suddenly and without warning. Such a crisis may be precipitated by an attempt to examine the throat, and therefore no such attempt should be made until the means of dealing with the crisis are to hand.

On arrival at hospital the child should be taken to an operating theatre where there are facilities for bronchoscopy and tracheostomy. If direct laryngoscopy confirms the diagnosis of epiglottitis a bronchoscope should be passed through the laryngoscope and tracheostomy should be performed. The experience of Berenberg and Kevy⁷ reinforced the advice of Alexander *et al.*² that tracheostomy should not be deferred in the hope that it will not be necessary. Nasotracheal intubation as an alternative to tracheostomy has its advocates;⁸ we have no experience of its use in the management of this disease.

In a disease of such rapid tempo the diagnosis must be based almost entirely on clinical criteria, but the finding of a greatly increased neutrophil polymorph count in the peripheral blood can be a useful means of differentiation from a virus infection. Bacteriological investigations are too slow to be the basis for treatment, but blood culture is nevertheless an important investigation because it may provide unequivocal retrospective evidence about the nature of the infection. Isolation of an untyped *H. influenzae* strain from the throat or epiglottis is of no significance, because non-capsulated strains of this species can be recovered from such sites in 50% or more of healthy children. The isolation of a type b strain is a different matter; this type is carried by only a few members of most healthy communities (of the order of 2-5%¹⁹) but has been found responsible for nearly all cases of epiglottitis in which careful and critical bacteriological investigations have been carried out. Claims that some cases are due to staphylococci, streptococci, or pneumococci⁷ are open to question when based on swab cultures without confirmation from blood culture because such organisms are commonly found in healthy upper respiratory tracts.

In conclusion we reiterate some of the main clinical features of epiglottitis. (1) In general the patients are children aged 2-7 years. (2) There is rapid progression from the first mild respiratory symptoms to severe illness, with pronounced dysphagia and drooling, stridor, muffling of the voice, bull neck (in some cases), and such deterioration of the general condition that the terms "shock," "prostration," and "collapse" have been used by various authors. (3) There is no time to spare, and the giving of parenteral antibiotics, admission to hospital, and the establishment of an artificial airway are urgently needed life-saving measures.

ADDENDUM

Three more children with haemophilus epiglottitis were admitted to the Infirmary in July and September 1971. Their ages were 3, 2, and 1½ years. Two were treated successfully. One died following a period of apnoea which occurred very soon after his admission, before the right diagnosis had been made.

For permission to report these cases we are grateful to Dr. H. L. Ellis, Dr. B. D. Bower, Mr. B. H. Colman, and Mr. W. S. Lund, the consultants under whose care the patients were admitted. We are also indebted to other colleagues in the departments of paediatrics and otolaryngology, to Dr. R. H. Cowdell and Dr. D. J. de Sa for the necropsy reports, and to the patients' family doctors.

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Measurement in Medicine

Experiences with Outpatient Medical Questionnaires

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Five years ago I began to use a questionnaire in the medical outpatient department. The original objectives were to reduce the time spent in routine questioning and to spend more time on analysis of the primary symptoms. Since then I have modified the questionnaire several times in the light of experience and have come to use the forms as guides to diagnosis. This paper gives an account of my experiences.

Material and Methods

There are two clinics a week, each lasting about three hours. About 650 new patients attend each year. The crude diagnostic breakdown is shown in Table I. The waiting time for a new patient is three to four weeks. The questionnaire is sent to the patient with his appointment and he is asked to complete it before coming to the clinic. Before seeing the patient I read the referral letter (usually from a general practitioner, occasionally from fellow specialists) and glance at the questionnaire. I then discuss the main complaint with the patient and

deal with affirmative answers on the form. The completed form is filed with the case notes. Recently I have adopted a similar routine with my private patients.

TABLE I—*Diagnostic Analysis of 631 new Outpatients seen during March 1970 to March 1971*

Gastrointestinal disorder ..	124	Thyroid abnormality ..	34
Diabetes mellitus ..	104	C.N.S. disorder ..	27
Rheumatism ..	64	Blood disorder ..	26
Vascular abnormality ..	62	Respiratory disorder ..	20
Urogenital system ..	56	Endocrine/metabolic disorder ..	17
Neurosis ..	46	Skin disorder ..	11
Heart disorder ..	40		

Results

The first form was modelled on a rubber stamp I had previously been using, and the answers were filled in by me. A brief explanation of the purposes of the form was followed by basic data and a series of questions with alternative possible answers. The appropriate answer was to be underlined. Spaces were left for remarks and past history and a table for family history, based on that used by insurance companies, was included.

I soon found that no objection was raised by the patients to the completion of these forms and that relatively many of them were able to answer the questions sensibly. I was spared the time and tedium of routine questioning, and the way in