Epiglottitis

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SUMMARY. A case of acute epiglottitis due to *Haemophilus influenzae* is described. The problems of making this diagnosis as early as possible are discussed, with a review of the literature.

Case report

A 20-month-old Down's syndrome baby had been well until he refused his evening meal and sneezed a few times. He went to sleep normally, but woke his parents at 03.00 with noisy breathing. His mother followed a regime she had been taught by doctors when, in the previous year, the child had had six bouts of so-called croup, from each of which he had recovered spontaneously. After one hour in a steamy atmosphere he was no better and was refusing to drink. In his mother's own words, "this was different from his previous attacks", so at 04.00 she called the general practitioner, who visited immediately.

The child did not appear very ill, but had a loud inspiratory stridor and a strange expiratory rattle. He was febrile. Examination of the ears and lungs was normal. The general practitioner did not examine the throat, but made a presumptive diagnosis of viral laryngitis, or croup, and advised fluids, aspirin and steam. He arranged to call back four hours later.

At 08.00 the picture had changed dramatically: the child was pale, restless and dehydrated with rib recession and stridor. The general practitioner arranged immediate admission to hospital, presuming this to be severe croup which might need intubation. It remained for the house physician to make the correct diagnosis and to initiate treatment with ampicillin (later changed to chloramphenicol) and dexamethasone. Two hours after admission, having continued to deteriorate, the child was intubated by an anaesthetist under general anaesthetic, when the characteristic swollen cherry-red epiglottis was clearly seen. One hour after this there was a marked improvement, and in two days the tube was removed. The child returned home one week after admission, fully recovered. *Haemophilus influenzae* type B was isolated from tracheal aspirates.

Comment

In retrospect, I should have made this diagnosis before the hospital admission, and I report this case to help others in recognizing epiglottitis.

The important clinical features of the condition have been well described (Jones and Camps, 1957; Addy *et al.*, 1972), and several were present in this case:

1. Stridor, of sudden onset, different in quality from croup, with a muffled as opposed to hoarse voice and an expiratory rattle (Illingworth, 1978).
2. Sore throat, dysphagia, drooling of saliva or refusal to drink.
3. Fever and signs of progressive systemic infection.

The age of the patient and any previous history of stridor are unreliable grounds for excluding this diagnosis. A high index of suspicion is needed in order to recognize this undoubtedly rare, but specifically treatable, serious disease.

Treatment

As soon as the general practitioner suspects the diagnosis, he should alert the nearest suitably equipped hospital and arrange to accompany the child there as quickly as possible. If the throat is examined, the obstruction may increase. The airway will be improved if the child is kept sitting up, but if respiratory arrest occurs it is well worth performing mouth-to-mouth ventilation, as at least one patient has survived as a result of this procedure (Johnson *et al.*, 1974).

Once in hospital, early establishment of an artificial airway, usually by endotracheal tube (Blanc *et al.*, 1977; Brevik and Klaastad, 1978) but previously by tracheostomy, results in a low mortality (less than 1 per cent) among cases reaching hospital alive. Chloramphenicol will usually be the antibiotic of choice in hospital because a significant proportion of *Haemophilus influenzae* are resistant to ampicillin (Zadik and Dadswell, 1981).

Conclusion

It will never be known how many children die from this condition before reaching hospital, but it seems clear that survival depends upon the general practitioner recognizing that a particular child with stridor might not have common croup, but instead might have the rare but treatable epiglottitis.

References

Addy, M. G., Ellis, P. D. M. & Turk, D. C. (1972). Haemophilus epiglottitis: nine recent cases in Oxford. *British Medical Journal,* **1**, 40-42.

Blanc, V. F., Weber, M. L., Leduc, C., *et al.* (1977). Acute epiglottitis in children: management of 27 consecutive cases with nasotracheal intubation with special emphasis on anaesthetic considerations. *Canadian Anaesthetists' Society Journal,* **24**, 1-11.

Brevik, H. & Klaastad, Ø. (1978). Acute epiglottitis in children, a review of 37 patients. *British Journal of Anaesthesia,* **50**, 505-510.

Illingworth, R. S. (1978). *Common Symptoms of Disease in Children.* 6th edition. Oxford: Blackwell Scientific Publications.

Johnson, G. K., Sullivan, J. L. & Bishop, L. A. (1974). Acute epiglottitis. A review of 55 cases and suggested protocol. *Archives of Otolaryngology,* **100**, 333-337.

Jones, H. M. & Camps, F. E. (1957). Acute epiglottitis. *Practitioner,* **178**, 223-229.

Zadik, P. M. & Dadswell, J. V. (1981). Stridor. Letter. *British Medical Journal,* **283**, 560.

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Digoxin inactivated by gut flora

In approximately 10 per cent of patients given digoxin, substantial conversion of the drug to cardioinactive, reduced metabolites (digoxin reduction products or DRPs) occurs. In four normal volunteers taking digoxin daily for four weeks, urinary excretion of DRPs was greatest after a poorly absorbed tablet was ingested, and least after intravenous administration. Stool cultures from subjects known to make DRPs *in vivo* ('excretors') converted digoxin to DRPs; cultures from non-excretors did not. Three excretors were given digoxin tablets for 22 to 29 days A five-day course of erythromycin or tetracycline, administered after a base-line period of 10–17 days, markedly reduced or eliminated DRP excretion in urine and stool. Serum digoxin concentrations rose as much as two-fold after antibiotics were given. We conclude that in some persons digoxin is inactivated by gastrointestinal bacteria. Changes in the enteric flora may markedly alter the state of digitalization.

Source: Lindenbaum, J., Rund, B., Butler, B. *et al.* (1981). Inactivation of digoxin by the gut flora: Reversal by antibiotic therapy. *New England Journal of Medicine,* **305**, 789-794.