GUEST EDITORIAL

Diphtheria

A diphtheria-like illness was recorded as early as the second century AD. Croup has been known for centuries. Pierre Bretonneau (Semple, 1859) emphasised the differences between croup and membranous croup in 1821, and published his classical monograph in 1826. Klebs (1883) reported the morphological appearance of Corynebacterium Diphtheria. Loeffler isolated the organism in pure culture in 1884. The toxin was described by Roux & Yersin (1888). The antitoxin was produced by Von Behring (1893). The skin test for susceptibility to diphtheria was introduced by Schick (1913) and the toxoid was developed by Ramon (1924).

The incidence of diphtheria was high between the two world wars, mainly affecting non-immunised children under the age of 5 years. Cases were reported during the 1939–45 war, particularly in France and Holland. In the USA, outbreaks were reported in 1952 and 1959–70 (Cecil & Loeb, 1955; Doege et al., 1962; McCloskey et al., 1971; Brooks, 1974). Sporadic cases have been reported from time to time in adults and children. The disease occurs mainly in Autumn and Winter months, usually in temperate climates. Two recent outbreaks have been reported in Sweden (Malmvall et al., 1986): 13% of clinical cases were alcoholics and 66% of the carriers were alcoholics.

I saw my first case of diphtheria when, as a boy in the thirties, I occasionally accompanied my father, a Manchester General Practitioner, on his domiciliary visits and to the Public Health Laboratory handing in five or six swabs at a time. In those days, the swabs were placed in glass tubes, packed between two pieces of grooved wood, covered with a pale blue wrapper, on which was the patient's name, age and address, and secured with an elastic band. The patients were admitted to Monsall, the local fever hospital, and there, as a medical student, I witnessed my first tracheostomy on a child with laryngeal diphtheria. The doctors placed great emphasis on the bed charts, in particular, the pulse chart. The tachycardia in diphtheria is out of proportion to the pyrexia. Indeed, the temperature may only be slightly elevated in the early stages (Bristowe, 1909). Students were taught to listen for nasal speech and the pitch of the cough. Convergence accommodation was tested and recorded. There was always a careful watch for cardiac and neurological complications.

The onset of the disease is usually insidious with a mild sore throat and swallowing is relatively painless. The appearance of the throat is unlike that of streptococcal tonsillitis. The pharynx is dull red with moderate injection and oedema. The membrane is dirty white or greyish, and has the appearance of wash leather, hence the name diphtheria. The membrane is adherent and, on swabbing, bleeds revealing a raw surface over which the membrane rapidly grows. There is a typical foetor unlike that of Vincent's or streptococcal tonsillitis. Tachycardia is marked.

In streptococcal tonsillitis, the onset is acute, swallowing is painful and the temperature high. The fauces are fiery red and follicles are usually present. A thin white loose exudate is often present which does not bleed on swabbing.
The clinical course of diphtheria depends on the site and extent of the membrane, the amount of toxin absorbed, and the early administration of antitoxin. The disease may rapidly progress, the patient being gravely ill and toxic, often with a blood-stained nasal discharge, husky voice and high-pitched cough. Laryngeal diphtheria is particularly serious. The membrane and the oedema are responsible for the respiratory obstruction and the classical diphtheritic croup. Intubation, tracheostomy or a cricothyroid stab is mandatory.

Neurological complications may occur with palatal palsy, paralysis of accommodation and peripheral neuropathy (Scheid, 1948).

Myocarditis occurs in two thirds of patients (Gore, 1948; Boyer & Weinstein, 1948; Good, 1948). The majority of patients developing atrial fibrillation, ventricular tachycardia or complete heart block die. Congestive heart failure is revealed by sudden pain in the right upper quadrant of the abdomen as a result of rapid enlargement of the liver.

The diagnosis of diphtheria should be made on clinical grounds and the antitoxin, administered without waiting for the swab report. The antitoxin is the only specific treatment (Harmisch, 1983). There is evidence that, after 48 h, the administration of antitoxin may have little effect on altering the severity of the complications (Eller, 1982).

The amount of antitoxin administered depends on the extent of the membrane. It may be given in a dose of 40,000 units intramuscularly if the membrane is small and is confined to the anterior nares or tonsils. If the membrane is more extensive and involving the pharynx, then 80,000 units should be given intravenously in 200 ml of isotonic saline after testing for horse serum sensitivity. In severe pharyngeal and laryngeal diphtheria, 120,000 units should be given intravenously (Harmisch, 1983).

The isolated case of diphtheria in an accident and emergency department reported by Dr R. F. Walters and published in this journal has, once again, brought our attention to this neglected and often forgotten serious, yet preventable, disease.

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REFERENCES

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