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Opinion Article

Note on Pathogenesis, Clinical features, Complications of *Corynebacterium diphtheriae*

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DESCRIPTION

Corynebacterium diphtheriae is an aerobic gram-positive bacillus. Toxigenicity occurs only when the bacillus is itself infected by a specific bacteriophage carrying the genetic information for the toxin. Only toxigenic strains can cause severe disease. Known as the "deadly scourge of childhood," diphtheria struck fear in the hearts of many and claimed the lives of innumerable children in the 19th and early 20th centuries. As far back as 1890, Emil von Behring, a German physician and scientist, found that certain diseases were the expression of the action of toxins which could be neutralized by antitoxins. Behring and his assistant Kitatso, a Japanese scientist, inoculated animals with modified, noninfectious (attenuated) forms of the infectious agents of diphtheria and tetanus, the animals produced neutralizing antitoxin serum. Serum from these animals was injected into nonimmunized animals that were previously infected with the fully virulent bacteria. The ill animals could be cured through the administration of the serum. They thus introduced passive immunization into modern medicine and for this; von Behring was awarded the French Legion of Honor and the Nobel Prize in 1901. Despite this, physicians and public health experts viewed diphtheria as one of the most difficult to treat and control of all childhood diseases. It was only in 1930, after the successful immunization of thousands of preschool and school-aged children in the USA, that the power of immunization to control this most deadly infectious disease was apparent. Disease can involve almost any mucous membrane. For clinical purpose, it is convenient to classify diphtheria into a number of manifestations, depending on the site of disease.

Since the introduction and widespread use of diphtheria toxoid; diphtheria toxin that has been treated to destroy its toxic properties without eliminating its capacity to stimulate the production of antitoxins by the immune system beginning in the 1920s, respiratory diphtheria has been well controlled in the most parts of the world. However, diphtheria remained endemic in some states through the 1970s, with reported incidence rates of greater than 1.0 per million populations. In recent years diphtheria has re-emerged as an important threat to health in many parts of the world. In the 1990s, a massive epidemic throughout the Newly Independent States of the former Soviet Union marked the re-emergence of epidemic diphtheria in industrialized countries. There were approximately more than a million cases and thousands of deaths reported since 1990. This epidemic, primarily affecting adults demonstrated that in a modern society, diphtheria can still spread explosively and cause extensive illness and death. Factors contributing to the epidemic included a large population of susceptible adults; decreased childhood immunization due to the breakdown of the public health infrastructure and high population movement. The epidemic also demonstrated that a concerted worldwide public health effort to vaccinate both children and adults could bring the epidemic under control within a few years.

Anterior nasal diphtheria

The onset is indistinguishable from that of the common cold and is usually characterized by a mucopurulent nasal discharge which may become blood-tinged. A white membrane usually forms on the nasal septum. The disease is usually fairly mild because of apparent poor systemic absorption of toxin in this location and can be terminated rapidly by antitoxin and antibiotic therapy.

Pharyngeal and tonsillar diphtheria

The most common sites of infection are the tonsils and the pharynx. Infection at these sites is usually associated with substantial systemic absorption of toxin. Often by the time a physician is contacted, the membrane is greyish-green in color or black of there has been bleeding. Extensive membrane formation may result in respiratory construction. The patient may recover at this point or if enough toxin is absorbed, develop sever prostration, striking pallor, rapid pulse, stupor, coma, and may even die within 6-10 days. Patients with severe disease may develop marked edema of the submandibular areas and the anterior neck along with lymphadenopathy fiving a characteristic bullneck appearence.

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Laryngeal diphtheria

Laryngeal diphtheria can be either an extension of the pharyngeal form or the only site involved. Symptoms include fever, hoarseness and a barking cough. The membrane can lead to airway obstruction, coma and death.

Cutaneous diphtheria

Skin infections are quite common in the tropics and are probably responsible for high levels of natural immunity found in these populations. Skin infections may be manifested by a scaling rash or by ulcers with clearly demarcated edges and membrane, but any chronic skin lesion may harbor *C. diphtheriae*, along with other organisms. The toxin, when absorbed affects organs and tissues distant from the site of invasion. The most frequent complications of diphtheria include Myocarditis; Abnormal cardiac rhythms can occur early in the course of the illness or weeks later, and can lead to heart failure. If myocarditis occurs early, it is often fatal. Neuritis is the complication most often affects motor nerves and usually clears completely. Widespread update of the vaccination has yielded interesting changes in the clinical and epidemiological presentation of the disease caused by these strains. Development of severe and sometimes recurrent sore throat is the primary presentation of infection with nontoxigenic strains.