Facing a diphtheria outbreak

This patient was photographed in 1995 during a Médecins Sans Frontières / Doctors Without Borders (MSF) mission in the Republic of Georgia. He had presented initially to a rural general physician 6 days after the onset of a sore throat, malaise and low-grade fever. Diphtheria was suspected, and the man was sent to the infectious disease hospital in Batumi, 60 km away, where a throat culture confirmed the diagnosis.

At hospital the patient had marked swelling of the neck and grey tonsillar-pharyngeal exudates, shown here with a hospital spoon being used as a tongue depressor (Fig. 1). Because of the late presentation and the changes on electrocardiography indicating left bundle branch block, the man was given the maximum dose of antitoxin (100 000 units) and penicillin G (600 000 units intramuscularly every 12 hours). He remained stable for 5 days but then began to have shortness of breath; he died of heart failure on the eighth day after admission.

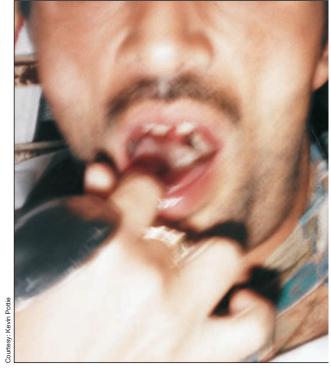
The infectious disease hospital in Batumi, with the assistance of MSF, treated over 300 cases of diphtheria from 1993 to 1995 and recorded a case-fatality rate of about 8%.

Diphtheria is most common in areas where there is overcrowding, poor hygiene and low immunity owing to waning immunization in adults and gaps in universal vaccination coverage in children. Corynebacterium diphtheriae is transmitted directly or indirectly from a case or carrier through respiratory droplets or skin infections involving C. diphtheriae.2 Patients typically present, after a brief incubation period of 1 to 7 days, with a mild sore throat and lowgrade fever. The characteristic thick, adherent, green-black pharyngeal or laryngeal membrane spreads rapidly, and the microorganisms invade the neck tissues, producing marked swelling and a "bullneck" appearance.3 In laryngeal diphtheria, primarily seen in children, the membrane may spread over the airway and result in severe respiratory obstruction. In addition, an exotoxin is produced that can cause cardiac arrhythmias, myocarditis and, rarely, heart failure, as well as nervous tissue damage with peripheral neuritis and paralysis.² The casefatality rate, usually between 3.5% and 12%,⁴ increases with late presentation. The antitoxin has little effect when given more than 48 hours after clinical diphtheria begins.

In the early 20th century diphtheria outbreaks evoked fear around the world. Until the 1930s it was one of the most common killers of children in Canada⁵ and the United States. Vaccination programs have since nearly eliminated diphtheria in the industrialized world, with the developing world accounting for 80% to

90% of reported cases over the last 20 years.1 In the early 1990s, however, a major resurgence of diphtheria was seen across the former Soviet Union,7 with more than 75 000 cases reported from 1991 to 1995. In Russia sporadic cases of diphtheria began to emerge after a drop in vaccination coverage in the 1980s. This drop in coverage was due to the loss of confidence in vaccination among health workers and the general public, increasing anti-vaccination propaganda and an irregular supply of vaccines.1 The economic collapse in the 1990s and the decline of social conditions, coupled with poor immunization rates, provided fertile ground for outbreaks across the Former Soviet Republics.

In the pre-vaccine era diphtheria was usually of the laryngeal type and affected predominantly children. The outbreaks in the Former Soviet Republics, consistent with other recent outbreaks in countries with faltering vaccination programs, have shown an epidemiologic shift toward pharyngeal diphtheria and involvement of people



over 14 years of age. The case described here, typical of many in the Former Soviet Republics, serves to remind us of the present-day risk of diphtheria and the importance of primary vaccination and booster doses.

Kevin C. Pottie

Family Medicine Centre University of Ottawa Ottawa, Ont.

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