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Short report

Guillain-Barré syndrome after combined tetanus-diphtheria toxoid vaccination

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Abstract

Guillain-Barré syndrome (GBS), an acute inflammatory polyradiculoneuropathy, is often associated with an antecedent factor, such as an infection, surgery, systemic malignancy, or vaccination. The first case of GBS following a vaccination with combined tetanus-diphtheria toxoid is reported. © 1997 Elsevier Science B.V.

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1. Introduction

Guillain-Barré syndrome (GBS), an acute inflammatory polyradiculoneuropathy, is often associated with an antecedent factor, such as an infection, surgery, systemic malignancy, or vaccination (Ropper et al., 1991). We report the first case of GBS following a vaccination with combined tetanus-diphtheria toxoid.

2. Case report

A 22-year-old college student was admitted to the University of California Los Angeles Medical Center complaining of 3 days of paresthesias and leg weakness. Nine days prior to admission, he stepped on a 'rusty nail' while playing beach volleyball. Two days later, he received an intramuscular injection of absorbed tetanus-diphtheria toxoid into the left deltoid muscle at our institution (0.5 ml, lot# 4928024, Wyeth Co.). Four days after vaccination, he awoke with bilateral tingling of the fingertips and toes, which progressed to involve his lips and tongue by the next morning. He noted progressive proximal leg weakness over the next 2 days, which progressed to involve his hands by the day of admission. He had asthma as a child, but denied other previous medical problems, antecedent illness during the past 6 months, fever, chills, diarrhea, blood transfusions, substance abuse, homosexual contact, tick bites, or a significant family medical history. He was not taking any medications. The tetanus prophylaxis was the second he had received since his routine childhood series; the previous injections had been well tolerated.

Examination on admission, 7 days after vaccination, revealed a blood pressure of 150/100 mmHg, a regular pulse of 105 beats per min, unlabored respirations and euthermia. Forced vital capacity was 6.0 l. General physical examination was unremarkable, including no evidence for inflammation at the nail puncture site or the toxoid injection site. The patient was alert and fully oriented with normal speech, language and memory function. Cranial

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nerve functions were intact. He had bilateral symmetric moderate weakness of shoulder abduction, hip and knee flexion, ankle and great toe dorsiflexion. Position and vibration sense were profoundly diminished distally in all four limbs. Deep tendon reflexes were absent at the triceps, knees and ankles and diminished at the biceps. Plantar responses were flexor.

The cerebrospinal fluid contained 1 white blood cell per cubic ml, no red blood cells, an elevated protein of 70 mg/dl and a normal glucose of 60 mg/dl (serum: 100 mg/dl). Nerve conduction studies on hospital day 2 showed prolonged latencies of the left median, left common peroneal and left posterior tibial nerves and prolonged F-responses and abnormal axonal reflexes of the left ulnar and right common peroneal nerves. Electromyography showed abnormal recruitment patterns with a slight loss of motor units in muscles of the left leg. Serum liver function tests and vitamin B_{12} level, serologies for human immunodeficiency virus (types 1 and 2), *Mycoplasma pneumoniae* and Lyme disease were unremarkable. *Campylobacter jejuni* serology was not performed.

Intravenous immunoglobulin (0.4 g/kg per day) was given for 5 days, beginning on hospital day 1. During the first eight hospital days, weakness of his hip girdle continued to progress and he could not ambulate; he developed bilateral facial weakness and dysphagia. Forced vital capacity steadily decreased to 3.6 l. However, his deficits reached maximum and stabilized by hospital day 10. He minimally improved and was transferred to a rehabilitation facility on hospital day 15.

An outpatient evaluation 6 weeks after tetanus-diphtheria toxoid injection, revealed significant improvement. He could swallow, and walk up and down stairs without difficulty. He complained of residual fatiguability and painful twitching in the arms. He had only mild bifacial weakness. Deep tendon reflexes were normal in the lower extremities and diminished but present in the upper extremities. Otherwise, the neurological examination was normal, including sensation, gait and muscle power.

3. Discussion

Our patient met the clinical, laboratory and neurophysiologic diagnostic criteria for GBS (Ashbury and Cornblath, 1990). His symptoms developed 4 days after a tetanus-diphtheria toxoid vaccination. Two cases of GBS following administration of pure tetanus toxoid have been reported (Pollard and Selby, 1978; Newton and Janati, 1987); therefore, we suspect that the tetanus portion of the vaccination produced the GBS, but we cannot offer proof. In addition, we are unable to exclude that the GBS was secondary to the diphtheria portion of the vaccination or simply represented a coincidental occurrence. No additional antecedent factors were identified as potential triggers for the GBS.

Adverse neurological effects associated with pure tetanus toxoid also include transverse myelitis (Read et al., 1992), peripheral neuropathy (Blumstein and Kreithen, 1966), brachial plexitis (Tsairis et al., 1972) and encephalomyeloneuropathy (Schlenska, 1977). However, neurological complications following combined tetanus-diphtheria toxoid administration are only sparsely reported (Holliday and Bauer, 1983) and have an estimated incidence of only 0.04 per 100 000 doses (Immunization Practices Advisory Committee, Centers for Disease Control, 1981). In a large-scale adult immunization campaign, no neurological complications occurred after more than 220 000 doses (Middaugh, 1979). Thus, it remains likely that the benefits of prevention of tetanus and diphtheria infection far outweigh the risk of GBS.

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