

## ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY AFTER TETANOS VACCINATION

### Tetanos aşısı sonrası gelişen akut enflamatuvar demiyelinizan polinöropati

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**Abstract:** A 17-year-old girl presented with a 3-day history of weakness, tingling and numbness. Neurological examination demonstrated normal muscle bulk but severe weakness in distal muscles of extremities. Sensory examination revealed decreased vibration in lower extremities. Deep tendon reflexes were absent, except for bilaterally marked diminished biceps reflexes and normal right sided triceps reflexes. The patient had a history of tetanus vaccinations, one month and two days prior to admission. After laboratory examination and electrodiagnostic studies, diagnosis of acute inflammatory demyelinating polyneuropathy (GB) related to tetanose antitoxin was made.

**Key Words:** Acute inflammatory demyelinating polyradiculoneuropathy; Vaccine, tetanus

#### Case Report

A 17-year-old girl presented with a 3-day history of weakness, tingling and numbness which had started in both legs and eventually spread to her upper extremities. She was unable to walk but able to use her arms. She complained about shortness of breath, difficulty in swallowing, and problems with bowel and bladder functions. In physical examination, her temperature was 37° C, pulse rate was 70 / min with good peripheral pulses, respiration rate was 20 / min and blood pressure was 130/70 mmHg. In neurological examination, mental status, speech, and cranial nerves were normal. Motor examination showed normal muscle bulk and diminished muscle tone in all 4 extremities, severe weakness (grade 1–2 / 5) in

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**Özet:** Onyediyedi yaşında kız çocuğu üç günlük karıncalanma ve uyuşma şikayetleriyle başvurdu. Nörolojik muayenede normal kas kitlesi ve ekstremitelerde uylarındaki kaslarda güçsüzlük tespit edildi. Alt ekstremitelerde vibrasyon hissinde azalma bulundu. Sağ triceps refleksi normal ve iki taraflı olarak biceps refleksi azalmış olarak değerlendirilirken, diğer derin tendon refleksleri alınamadı. Şikayetlerin başlangıcından bir ay ve iki gün önce olmak üzere iki kez tetanos aşısı öyküsü mevcuttu. Laboratuvar araştırmaları ve elektrodiagnostik çalışmalar sonrasında, tetanos aşısı sonrası gelişen akut enflamatuvar demiyelinizan polinöropati tanısı kondu.

**Anahtar Kelimeler:** Akut enflamatuvar demiyelinizan polinöropati; Tetanos, aşısı

distal muscles of lower extremities and weakness (grade 3/5) in distal muscles of upper extremities, and proximal muscles strength grade 4/5. Diminished vibration was found in lower extremities in sensory examination. Deep tendon reflexes were absent, except for bilaterally marked diminished biceps reflexes and normal right sided triceps reflex. She was unable to walk independently, although not ataxic. Quadriplegia developed within one day after admission. She had a history of tetanus vaccinations one month and two days before admission. Laboratory findings revealed normal hemogram, liver function tests, electrolytes, urinalysis, and porphyria screen. Lung examination and pulmonary function tests were normal. Serologic studies for *Campylobacter jejuni* infection and Cytomegalovirus infection were normal. Her protein level was mildly increased (165 mg/dl), with normal opening pressure and 2-3/μL cell count in CSF examination. Erythrocyte sedimentation rate was 26 mm/hour. Nerve conduction studies showed prolongation of distal

latencies, small motor and sensory action potentials with temporal dispersion, absent F-waves, and slowing of conduction velocities, unequivocally revealing the dysfunction of the peripheral nerves as the cause of the patient's weakness and sensory findings (Table I). EMG revealed rare fibrillation potentials in both gastrocnemius muscles and markedly decreased motor unit recruitment in all muscles tested in both upper and lower extremities. A diagnosis of acute

inflammatory demyelinating polyneuropathy related to tetanose antitoxin was made with a history of tetanose injection two days before the onset of the symptoms. The patient was administered intravenous immunoglobuline (500 mg/kg/day for 10 days). She developed respiratory dysfunction; endotracheal intubation with ventilatory support was required on the fourth day of immunoglobuline therapy. She died on the 23rd day after admission.

**Table I.** Patient's electrodiagnostic findings (Nerve conduction study)

Nerve Stimulated	Stimulation site	Recording site	Amplitude (mV / microV)	Latency (msec)	Conduction Velocity (m/sec)	F-Wave Response
Median (m)	Wrist Elbow	APB	2.5 (>6) 2	5.2 (<4)	29 (>45)	No Response
Ulnar (m)	Wrist Below elbow Above elbow	ADM	3 (>4) 3 1.5	4.3 (<3.5)	35 (>50) 39 (>50)	No Response
Peroneal (m)	Ankle Below fibula Above fibula	EDB	1.5 (>2) 1 1	7.2 (<6)	30 (>40) 32 (>40)	No Response
Median (s)	Wrist	Index finger	4 (>15)	4.8 (<3.5)	35	
Sural (s)	Calf	Posterior Ankle	No response			

\* m: motor, s:sensory.

\*\* The normal values of nerve conduction studies were shown in parenthesis

## DISCUSSION

Guillian-Barré syndrome (GBS) is characterised by rapid symmetrical paralysis of limbs, abnormal sensations, loss of tendon reflexes and smooth muscle control. Sixty-six per cent of GBS patients report preceding infections (1,2). GBS is an auto-immune disease, whereby antibodies generated to combat the microorganisms cross react with components of peripheral nerves, including the

myelin sheath or axons of Schwann cells due to molecular "mimicking" (2,3). Antibody action on host nerve cells results in neural degradation and loss of muscle control. Paralysis usually progresses for up to 4 weeks before reaching a plateau (1) and is commonly triggered by gastrointestinal infection or respiratory infection, although other causes such as surgery and vaccination have been suggested.

Vaccination has often been cited as an antecedent to GBS (4). Oral Poliovirus Vaccine , Hepatitis B vaccine and Rabies vaccines may result in GBS (5,6). In a unique case, one patient developed 3 episodes of GBS following 3 separate tetanus toxoid vaccinations. The episodes were separated by 9 and 5 years, and the intervals between immunisation and onset of symptoms were 3 weeks, 2 weeks and 9 days (6). In 2 recent studies comparing over 200 GBS patients with healthy controls, no correlation was found between the onset of the disease and preceding immunisations for influenza, cholera, and Diphtheria-Tetanus-Pertussis (1). In a study conducted over 5 years in south America no increase was found in the rate of GBS among 70 million children immunised with measles vaccines compared with controls (7). In addition, a large survey of GBS among South American children found no temporal association or increased disease rate during mass immunisation with the polio virus vaccine (2). Although the rate of GBS has increased markedly with a small number of vaccines, including influenza and rabies , there is a large body of research that suggests there is no significant link between GBS and the current strains of influenza, polio, cholera and DTP vaccines (1,8,9). In the present case report, the patient had a history of tetanose injection just 2 days before the onset of symptoms. There was no history of preceding infectious illness including upper or lower respiratory infection and gastrointestinal dysfunction. Serologic evidence for infection with *Campylobacter jejuni* and cytomegalovirus were found to be negative.

GBS may lead to ventilatory failure, autonomic dysfunction, and also to many general medical problems that have great bearing on the outcome. Therefore, severe GBS patients need to be treated in an intensive care unit. Although in some cases a ventilatory paresis occurs, GBS is largely self limited and the outcome is excellent (>80% recovery) with modern intensive care support (8). The neurologist who plans to deal comprehensively with these patients must be familiar with therapy

for infections, nutrition, fluid management and selected aspects of pulmonary medicine, in addition to the indications for and complications of plasma exchange and gammaglobulin infusion (8). Dowling et al.(10) measured serum immunoglobulin concentrations and found significant elevations of IgM, A and G in the GBS group. In the severely affected group, serologic evidence for infection with *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, or *Mycoplasma pneumoniae* was found more frequently than in the mildly affected group (41% versus 16%,  $p = 0.001$ ) (9,10). GBS patients with high serum IgM were young and 68% showed serologic evidence of recent infection with DNA core agents, cytomegalovirus, Epstein-Barr virus, or *Mycoplasma pneumoniae* (9,10). In our patient, serology showed negative results of *Campylobacter jejuni* and cytomegalovirus with an immune component, although a severe form of GBS was seen at the end which resulted in mortality.

We believe there was a relation between tetanus injection and GBS in the present case and it must be considered that GBS may result in mortality despite modern intensive care support.

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