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The Guillain-Barre Syndrome After Lumbar Disc Surgery: A Case Report

Lomber Disk Cerrahisi Sonrasında Guillain-Barre Sendromu: Olgu Sunumu

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ABSTRACT

The Guillain-Barre Syndrome (GBS) is an acute demyelinating polyneuropathy characterized by progressive muscle weakness and areflexia. Although its pathogenesis remains unknown, it is thought to be an autoimmune disease. Guillain-Barre syndrome following anesthesia or surgery is rare. In this case report, the patient in whom GBS developed following lumbar disc surgery is presented.

KEY WORDS: Disc herniation, Guillain-Barre, neurosurgery, spine

ÖZ

Guillain-Barre Sendromu (GBS) ilerleyici kas güçsüzlüğü ve refleks kaybı ile karakterize bir akut demiyelizan nöropatidir. Her ne kadar patogenezi bilinmemekteyse de otoimmün bir hastalık olduğu düşünülmektedir. Anestezi ya da cerrahiyi takiben ortaya çıkması nadirdir. Bu makalede, lomber disk cerrahisi sonrasında GBS gelişen bir hasta sunulmuştur.

ANAHTAR SÖZCÜKLER: Disk herniasyonu, Guillain-Barre, nöroşirürji, omurga

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INTRODUCTION

GBS is one of the most common reasons of acute polyneuropathy in adults. It is a rare disease although there are no definite statistics and its incidence is thought to be 1-2/100,000. It can be seen at any age and there is a slight male predominance (4). The classical sign of the disease is the stable neurological deficits which develop rapidly within two weeks after which there is a slow recovery period which goes on for months. While the weakness can develop acutely in few days, it can be also develop subacutely (3-4 weeks) (1). The pathogenesis of the disease is still unknown (3). GBS after major or minor surgical operations and anesthesia has recently been discussed more often. Reports are very rare and limited to case presentations in neurosurgery (5, 8, 9). We present a case where GBS developed following lumbar disc surgery.

CASE REPORT

A 41-year old male was operated for L5-S1 disc herniation. The patient's complaints resolved totally following standard discectomy and foraminatomy and he was discharged on third postoperative day. Two weeks later, he was readmitted to the hospital for progressive lower extremity weakness and sensory changes that subsequently involved his upper extremities. The patient was unable to stand or walk. Neurological examination revealed quadriparesis which was dominant in the legs with total loss of DTR. Cranial nerves and brain stem findings were normal. Blood test results including electrolytes and complete blood count were normal. CSF tests showed normal glucose and elevated protein levels (91.5 mg/dl). No cells were detected in the CSF. The EMG revealed prolonged motor distal latencies and reduced number of motor unit potentials as seen in polyradiculo-neuropathy. The patient was transferred to the neurology clinic where he received symptomatic treatment and supportive care for 10 days together with a rehabilitation program. He was able to walk with support at his discharge from the hospital. The patient's neurological status gradually improved and he was able to walk without support at the 8-week follow-up period. However, he continued to have a mild hypoesthesia in the lower extremities at the 2-year follow-up.

DISCUSSION

GBS is a complicated disease and there are no sufficient clinic or laboratory data to define it (3). It can cause rapidly advancing weakness of the extremities, abnormal nerve transmission, loss of DTR, increased CSF protein and autonomic dysfunction (1). The disease appears days or weeks after the event that suppresses immunity and shows slow improvement after reaching the highest point neurologically.

The muscle involvement includes both proximal and distal distributions. The most common presentation for the syndrome is a combination of sensory and motor involvement with the motor findings more prominent. Cranial nerves may be affected rarely in this syndrome. Prolonged motor distal latencies and reduced number of motor unit potentials on EMG are seen early in the course of the disease. Areflexia is the rule but hyporeflexia may be seen in some cases.

The presence of a bacterial or viral infection in the majority of cases suggests that it is triggered by a pathogen which cannot be isolated and an event which alters the immune system. An increasing number of GBS cases associated with surgery, transplantation procedures and anesthesia have been reported in the past years (3, 4, 8, 9). However, GBS is rare following neurosurgical intervention and has only been reported as small series (8-10). There is no information on the risk increasing with a specific type of surgery. Surgery, viral diseases, pregnancy, connective tissue disorders such as lupus, and malignancies may be responsible for the altered immune function that can result in GBS (1). GBS after transplant procedures is also thought to be due to the neurotoxicity of chemotherapeutic agents (3). Most patients with GBS have a respiratory-tract infection or gastrointestinal illness 1-3 weeks before the onset of symptoms (6).

Almost one-third of patients with GBS have a preceeding Campylobacter jejuni infection, with an associated high titer of anti-GM1 antibody titers which supports the notion that aberrant immunological mechanisms underlie this disorder (3). T-lymphocyte activation parallel to GBS activation has supported the notion of a primary lymphocytic T-cell mechanism in this inflammation. It has been experimentally shown that antimyelin

T-cell injection results in GBS in animals (3, 7). Perineural inflammation is either not found in pathology studies of GBS or only seen in the later stages. However, there is no lymphocyte infiltration in some GBS cases. Furthermore, GBS is seen in some diseases which are characterized by the impairment of T-lymphocyte function such as AIDS. It is therefore not possible to explain the pathophysiological mechanisms with a single factor. The occurrence of GBS in patients who have undergone organ or bone marrow transplantation indicates that iatrogenic immune suppression may be associated with GBS.

The pathogenesis of GBS is unknown but it is generally accepted that it results from an aberrant humoral and cellular immune response directed against components of the peripheral nervous system (6). The resolution of GBS correlates with the return of T-suppressor cell function. In spite of the GBS's appearance in immunosuppressive patients, immunosuppressive treatment is not a definite risk factor. In addition, most autoimmune neuromuscular diseases are treated with immunosuppressive agents (4).

Supportive treatment is the best treatment toward symptoms in GBS. Although there are questions which have not been answered yet experimentally and clinically, intravenous gamma-globulin and plasmapheresis may provide benefits in serious cases (10). Plasmapheresis is more effective in the first two weeks. The effect of the steroids unclear and they are generally found to be ineffective (2, 10). There can be recovery without any treatment in a few cases. While good functional recovery is seen in 85 percent of the patients, death is seen in 3 to 8 percent. However, it seems that immunotherapy will be more beneficial in the future (7).

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