CASE REPORT

Guillain-Barre Syndrome Following Spinal Anaesthesia
Refah Sayin1, Ismail Kati2, Yasemin Isik2 and Mustafa Günes3

ABSTRACT
Guillain-Barre Syndrome (GBS) is the most common disease resulting in acute diffuse flaccid paralysis. It is an autoimmune disease that can occur at any age. The clinical course is characterized by weakness in the arms and legs, areflexia and the progression of muscle weakness from the lower limbs to the upper limbs. The most common causes of GBS include infections, vaccinations, surgery and some medicines. We present the case of a 48 years old male patient, who developed GBS after undergoing surgery for renal calculus, under spinal anaesthesia. In this case report, we presented a rather rare case of GBS occurring following spinal anaesthesia.

Key words: Guillain-Barre syndrome, Spinal anaesthesia, Electromyography.

INTRODUCTION
Guillain-Barre Syndrome (GBS) is an acute and severe polyneuropathy characterized by inflammation of the peripheral nerves, which is considered to be an autoimmune phenomenon.1 The most common cause of GBS is infection 1 – 4 weeks before the onset of the disease at a rate of 90%. Other reasons include vaccinations, lymphomas (particularly Hodkin's lymphoma), surgery, and the use of some drugs (thromboembolic agents).2 Cases of GBS after epidural and general anaesthesia has been reported.3,4 However, in the literature, no cases of GBS are reported, which developed after spinal anaesthesia. This case was reported because it is the first case of GBS developed after spinal anaesthesia.

CASE REPORT
A 48-year-old male detected on pre-operative history had previously undergone renal calculus surgery under spinal anaesthesia. The calculus was removed during ureterorenoscopic surgery by placement of a double J catheter. Four to five days after the operation, weakness started in the feet with numbness in the arms and he presented to the urology outpatient clinic approximately 9 days after the onset of these complaints. The urologists did not attribute these complaints to the surgery and referred the patient to the anaesthesia outpatient clinic. The physical examination was normal. Upon detection of loss of strength in the lower extremities and numbness in the upper extremities, the patient was referred to the neurology clinic.

The neurologic examination showed that he was conscious, cooperative, oriented with normal speech, pupils were isochoric, direct and indirect light reflexes were +/+; and the fundus was normal; there were no findings of neck stiffness or meningeal irritation, the cranial nerves were normal, the deep tendon reflexes (DTRs) having areflexia on the lower extremities and hypoactive on the upper extremities; in the legs' strengths were at a level of 2/5 and while there was a glove-stocking sensory loss, the cerebellar tests were normal. Romberg and tandem-walk tests were impaired and ataxic walk was detected. The patient underwent electromyography (EMG) under the preliminary diagnosis of GBS. The sensory conduction investigations revealed peroneal superficial and sural sensory fiber conduction velocities that were compliant with the patient's age but peroneal superficial sensory response amplitude was detected as low. Motor conduction investigations revealed normal conduction velocities relative to the patient's age but motor action potential values were detected as low (Table I). The patient was hospitalized under the diagnosis of acute polyneuropathy. The patient did not agree to undergo lumbar puncture. The patient had received 3 cc of heavy marcaine from the L3-L4 space via single access for operation 9 days before. No complication occurred during the surgery. No blood or blood products were administered.

The patient, who exhibited normal routine blood investigations during the hospitalization process, was administered intravenous immunoglobulin (IVIG) treatment for 5 days under the diagnosis of acute sensorimotor polyneuropathy. Following treatment, the neurologic examinations revealed a strength of +4/5 and 3/5, on the proximal and distal of the lower limbs, respectively, and a reduction of the numbness in the hands was detected. The patient was advised to take 30 grams of IVIG once monthly for 6 months and discharged.

The patient underwent a follow-up EMG after 3 months. During sensory conduction investigations, the peroneal
superficial fibers could not be stimulated; the sensory conduction velocities and the sensory response amplitudes of the other nerves were within normal limits. During the motor conduction investigations, the peroneal motor fibers could not be stimulated; the conduction velocities of the other nerves were normal relative to the patient’s age, however, the motor response amplitudes were normal (Table II). The follow-up neurological examination revealed a strength of +4/5 and 4/5 on the proximal and distal of the lower limbs, respectively. DTRs were hypoactive in the patella and areflexia in the Achilles tendon on the lower limbs.

**DISCUSSION**

Several surgical treatments can be employed for the patients with neurological disorders, such as multiple sclerosis, GBS, Parkinson’s disease, amyotrophic lateral sclerosis, Alzheimer’s disease and spinal cord injury. It is possible that anaesthesia related complications are induced in these neurologically complicated patients in the perioperative period. GBS may very rarely represent the cause of (< 5%) postoperative unexplained diffuse peripheral neuropathy. The pathogenesis may involve the immune response to peripheral nerve components (triggered by the previous infection). In addition, postoperative GBS may occur secondarily to the metabolic response induced by surgery or anaesthesia. GBS occurring following pancreatic cancer surgery under epidural-general anaesthesia occurred 2 hours after transfer to the post-anaesthetic care unit and began in the upper limbs. The lower limbs could not be completely assessed due to epidural anaesthesia. GBS was identified in a patient presenting with acute respiratory distress and progressive muscle weakness occurring 7 days after gastroesophagectomy-splenectomy under general anaesthesia. In this case, the symptoms started in the lower extremity 4 – 5 days after surgery under spinal anaesthesia and progressed to the upper extremities. As far as we know, this is the first case of GBS diagnosed after spinal anaesthesia. These manifestations include botulism, toxic neuropathies, poliomyelitis, spinal mass, amyotrophic lateral sclerosis and in particular, neuropathies occurring after surgery.

Postoperative peripheral neuropathy may occur due to various reasons. These include surgical trauma and traction, tourniquet, deterioration of the existing neuropathy and compression of the nerve due to position failures. In addition, the risk factors may involve male gender, obesity, previous asymptomatic nerve dysfunction and prolonged hospitalization. In GBS treatment, the patients are administered plasmapheresis 4 – 6 times every other day or intravenous immunoglobulin (IVIG) at a dose of 0.4 g/kg/day (5 days). This patient was administered IVIG treatment, and he was discharged upon improvement of the complaints. He was put under follow-up with once monthly doses given for 6 months.

**REFERENCES**

3. Papantoni E, Sakorafas GH, Zouros E, Peros G. Guillain-Barre syndrome following total gastrectomy/ esophagectomy: a very...

