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Mimicking Guillain-Barre Syndrome after Spinal Surgery: Report of 2 Cases and Literature Review

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Abstract

Objective: Guillain-Barre syndrome is considered to be an immune-mediated acute inflammatory peripheral neuropathy. Multiple neuropathy after decompression of the spinal canal has been reported in the previous literature. Is this a coincidence or a complication? It is still unclear now.

Methods: Two cases with similar GBS after spinal cord decompression were observed and analyzed, and the clinical characteristics were summarized in combination with the reported similar cases.

Results: Both cases showed electrophysiological and CSF changes similar to GBS, but the manifestations of spinal cord injury were obviously inconsistent with the clinical features of classic GBS. At the same time, all the 9 cases reported in the literature involved spinal canal surgery, and 2 cases (22.2%) suffered from surgical injury and dura/meningeal membrane and caused changes in cerebrospinal fluid pressure, and 3 cases (33.3%) suffered from postoperative paraplegia.

Conclusion: The change of brain/spinal pressure caused by spinal decompression is a possible cause of GBS-like syndrome, not a coincidence, but a surgical complication.

Keywords: Spinal canal decompression; Intracranial hypotension syndrome; Polyradiculoneuropathy; Guillain-Barre syndrome

Introduction

Spinal canal decompression is a common spinal surgery, which can effectively relieve spinal cord compression and save spinal cord function. Previous cases of acute quadriplegia have been reported in patients who briefly recovered from the original symptoms after surgery. This complication is rare but serious, and most patients will be left with severe functional disability. At present, the causes of polyradiculoneuropathy, in patients after spinal decompression are not clear, which also affects the treatment of patients. We present two cases of tetraplegia after spinal decompression. These two patients developed rapid quadriplegia about 1 week after spinal decompression. What is special is that the paralysis of the patients was characterized by both spinal cord injury and peripheral neuropathy, and the neurological function recovered slowly after receiving immunoglobulin therapy. We compared these two patients with previously reported cases in an attempt to identify the cause of acute paralysis in the patients after surgery.

Case Presentation

A 46-year-old female patient, because of schwannoma (L2-3) and the left leg pain and weakness for eight years, accept + cut lamina decompression + lumbar posterior internal fixation spinal neoplasm resection (preoperative spinal cord image is shown in Figure 1a), and incision drainage after surgery, postoperative three days every day about 500 ml, drain the late flow gradually reduce, pull out the tube 7 days postoperatively. Relevant preoperative physical findings included lumbar spinous process tenderness and limited motion, muscular atrophy of the left lower limb with weaker muscle strength than the healthy side (grade 4/5), negative straight leg raising test and pathological reflexes, and presence of bilateral knee jerk reflexes. Sense of postoperative patients with lower limb pain, in good condition, but on the third day postoperatively, the patient suddenly experienced numbness and weakness from far to near, symptom progression, appear urine retention at the same time, the physical examination found that limb muscle strength grade 1 to grade 2/5, hypoesthesia depth under T10, and knee jerk and tendon reflex can't elicit pathological character negatively, considering maybe GBS, IVIG (0.4 g/kg) immediately shock therapy, patients still have the double side slumped, dysarthria, dysphagia. On the 6th day after the operation, the patient had sudden sharp drop in blood pressure and respiratory muscle paralysis and received endotracheal intubation

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Table 1: Summary of published case reports of postoperative Guillain-Barré syndrome.

Author/Year	Operation	Age (years)/Sex	Factors affecting intracranial pressure	Time from Operation to Symptoms	Clinical presentation addition to limb numbness and fatigue	CSF protein (mmg/l)/ WBC Count	NCS and EMG	Treatment	Prognosis
Yui et al., 2019 [17]	Surgery for spinal stenosis at the level of L2/3	70/F	/	8d	Facial paralysis, Lost sphincter control, Bulbar paralysis	1216/6	Motor nerves showed prolonged latency, decreased conduction velocity, No sensory nerve action potentials were observed, F-wave showed increased latency	IVIG、MP	To move facial muscles and limbs quickly
Eric et al., 2017 [15]	L3-S1 instrumentation arthrodesis	57/M	/	8d	Facial diplegia, Paraplegia, Lost sphincter control, Respiratory paralysis, Autonomic nervous abnormality	/	/	IVIG、MP	Regained motor function and ambulation (6M)
Zain et al., 2015 [22]	L4-5 Laminectomy	58/M	Epidural fluid on the left Laminectomy site	3h	Respiratory failure	70/4	Sensory-motor axonal polyneuropathy	IVIG、Pph	Recovered full motor power(1Y)
	L3-4 Laminectomy	40/M	/	1h	/	225/4	F-wave showed decreased conduction velocity	IVIG、Pph	Recovered motor function (8M)
Massimo et al., 2012 [2]	C6-7 Laminectomy for chondroma Resection	55/M	/	36h	Paraplegia, Lost sphincter control, Respiratory paralysis	Protein increased	Intense denervation activity was evident in the 4 limbs, F-wave showed conduction blocks	IVIG	Seriously disabled (3Y)
F. et al., 2012 [1]	L1 kyphoplasty	73/F	/	14d	Facial diplegia, Bulbar paralysis	Albumin-cytological dissociation	Motor nerves showed prolonged latency, decreased conduction velocity, No sensory nerve action potentials were observed, F-wave showed increased latency	IVIG	Only peripheral facial palsy (3M)
Jocelyn et al., 2011 [23]	T1–3 Laminectomy for meningioma Resection	59/F	Perioperative lumbar cistern cerebrospinal fluid drainage	6h	Facial paralysis, Bulbar paralysis, respiratory paralysis, Autonomic nervous abnormality	Protein level persistently elevated	Intense denervation activity was evident in the 4 limbs, Sensory-motor axonal polyneuropathy	IVIG	Minimal improvement (7M)
Dong et al. 2011 [24]	T12 spinal fusion for Fracture	55/M	/	10d	Facial diplegia, respiratory paralysis	/	Motor nerves showed decreased conduction velocity, and conduction blocks	IVIG	Almost completely resolved (2M)
James et al. 2008 [4]	T4-T10 instrumentation and fusion	28/M	/	1w	Paraplegia, facial paralysis, Autonomic nervous abnormality	5030/1	Extensive denervation activity, Sensory-motor axonal polyneuropathy	IVIG	Extremities and face recovered significantly (6M)

Pph: Plasmapheresis; IVIG: Intravenous Immunoglobulin; MP: Methylprednisolone; NCS: Nerve Conduction Studies; EMG: Electromyography; CSF: Cerebrospinal Fluid; WBC: White Blood Cells; F: Female; M: Male, Month; Y: Years; H: Hours; d: Days; w: Week

and ventilator auxiliary support treatment. The reexamination of the spinal cord MRI (Figure 1b) showed that the mass had been removed, and postoperative hematoma compression or spinal cord damage was excluded. We continued to improve the relevant examinations, and our positive findings mainly included: 1. The protein level of cerebrospinal fluid (19328 mg/L) was significantly increased; 2. Weak positive serum peripheral nerve antibody GMI IgG; 3. Nerve Conduction Studies (NCS) and Electromyogram (EMG): lower limbs showed denervation activity; motor nerves showed prolonged latency, decreased conduction velocity and amplitude; sensory nerve showed decreased amplitude; F-wave disappeared. 4. MRI of the head and spinal cord showed little subdural bleeding in the right frontal-parietal occipital region; Subarachnoid hemorrhage (Figure 1c, 1d). Patients to continue in the next month successively IVIG shock therapy (0.4 g/kg * 5 days) and plasma exchange, but the effect is not obvious, after June, patients in stable condition did not continue to progress, basic recovery of respiratory function does not need auxiliary breathing machine, upper limb muscle strength back to 3/5 level, swallowing function and sphincter disturbances did not see recovery. A 60-year-old man presented with recurrent low back pain

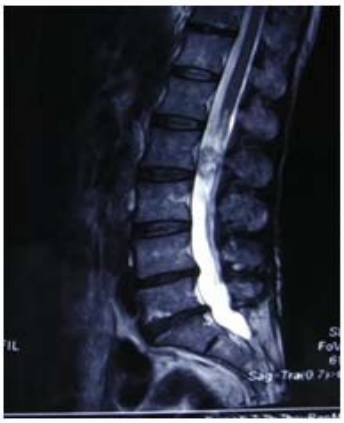

Figure 1a: Preoperative lumbar spine MR of case 1: L2-3 intravertebral canal space occupied, schwannoma considered.

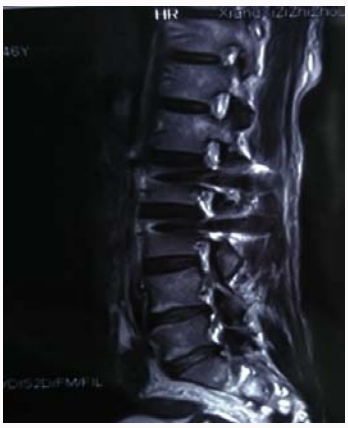

Figure 1b: Postoperative lumbar MR of case 1: Postoperative changes of L2-3 spinal canal (no hematoma or mass observed Signal).

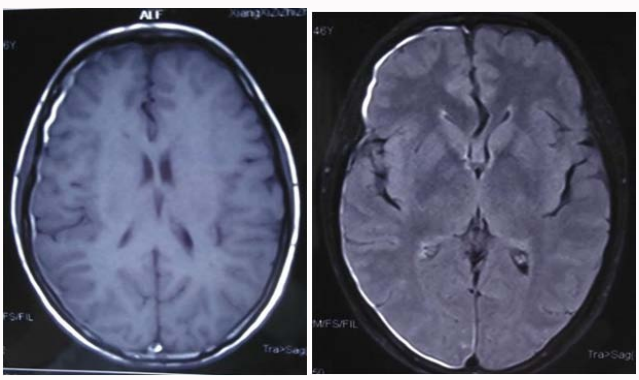

Figure 1c and d: subdural hemorrhage: Postoperative subdural hemorrhage occurred in the right frontal, temporal, parietal, occipital and lobes (short T1, long T2, FLAIR sequence high signal).

associated with radiating pain in both lower extremities for 10 years. He developed weakness and numbness in both lower extremities 2 years ago, and MR found lumbar and cervical stenosis (Figure 2a, 2b). This was the second time the patient had undergone spinal decompression surgery for cervical spinal stenosis. Preoperative-related physical examination included mild weakness of the left lower limb (muscle strength 5-/5), active tendon reflexes in all cases, and positive bilateral Babinski sign. The patient received L4 and L5 spinal

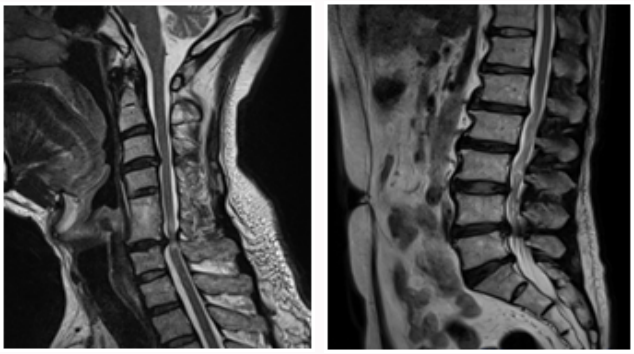

Figure 2a and b: Preoperative lumbar spine MR of case 2: C3-4 disc bulge, C6-7, L4-5, and L5-S1 spinal cord compression.

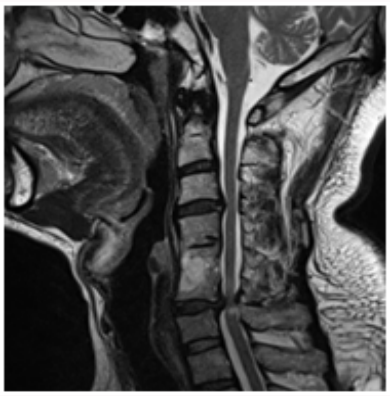

Figure 2c: Postoperative lumbar MR of case 2: The lesions after the first operation are the same as before.

stenosis total lamina decompression + artificial lamina implantation, and temporary postoperative drainage tube implantation. The drainage fluid in the drainage tube was always more, 400 ml on the first day, 300 ml on the second day; 150 ml on the third day, and about 1200 ml in total when the tube was pulled out on the sixth day. Postoperatively, the patient's nerve compression symptoms improved rapidly, pain relieved, and lower limb muscle strength returned to normal (5/5). However, from day 8, the patient gradually developed urine retention, weakness; and numbness of the limbs. Physical examination showed that the muscle strength of the patient's upper limbs was 3/5 and that of the lower limbs was 2-3/5. All deep reflexes disappeared, and sensory depth below C7 decreased. Babinski sign was positive in both lower extremities. The head MR was improved to exclude acute cerebrovascular accidents. The cervical MR suggested that cervical spinal cord compression was the same as before compared with the first MR (Figure 2c). The patient received a second posterior cervical laminectomy for decompression but the patient's symptoms were not significantly relieved after surgery. We continued to improve the relevant examinations, and our positive findings mainly included: 1. The protein of Cerebrospinal Fluid (CSF) 3.4 g/L was significantly increased; 2. NCS and EMG: Extensive denervation activity; motor nerves showed decreased conduction velocity, amplitude; Sensory nerve conduction showed generally normal; F-wave disappeared. The patient was then treated with gamma globulin impulse therapy (0.4g/kg * 5 days) and other conventional and rehabilitation treatments. The patient's symptoms recovered slowly, and after 6 months the tendon reflex recovered, she was able to stand, and after 18 months, she was able to walk almost independently.

Literature Review

We screened patients meeting the following criteria in PubMed and Elsevier: 1. Spinal or spinal surgery or manipulation; 2. Postoperative symptoms of multiple neurological deficits ;Including our case, there were a total of 11 cases, including 7 males and 4 females, with ages ranging from 28 to 73 years old. The onset time was concentrated within 2 weeks after the operation, and 1 case of the operation site included the cervical spine. Thoracic vertebrae in 3 cases; Lumbar spine 7 cases; the reason of operation was tumor in 3 cases. Fracture in 3 cases; Spinal canal stenosis: 5 cases; There were 4 cases with CSF leakage after operation, accounting for 36.4%. In addition to numbness and weakness of limbs, 5 patients with paraplegia (all excluded new spinal cord compression and damage), accounting for 45.6%; EMG axon demyelination in 8 cases. Six cases of abnormal F wave; The Cerebrospinal Fluid (CSF) protein was increased in all 8 patients. All patients received Immunoglobulin (IVIG) therapy and their neurological function recovered slowly.

Discussion

Acute quadriplegia is a rare complication after spinal cord surgery, but the consequences are serious. According to relevant reports, in addition to quadriplegia, such patients may also have dysphagia, facial diplegia, and respiratory muscle paralysis, deep and shallow sensory disorders below the corresponding spinal cord level, autonomic nervous symptoms such as severe hypotension, arrhythmia, urinary retention and other symptoms [1-5]. Patients pathogenesis is not very clear at present, the literature reports, described the postoperative multiple neuropathies as more Acute Motor and Sensory Axonal Neuropathy (AMSAN), it is a rare and serious GBS subtype, based on the diagnosis of patients usually accept gamma globulin or and plasma exchange treatment, but often less effective, behind are more serious in patients with functional disability [3-5]. GBS, also known as acute inflammatory demyelinating peripheral neuropathy, is an immune peripheral neuropathy [2,3,5]. It is self-limited and usually has an incubation period of 2 to 3 weeks, with symptoms reaching a peak within 2 to 4 weeks after onset and then disappearing within weeks to months [3,5]. Common clinical manifestations of GBS include the absence of tendon reflex [3,4], albumin cytological dissociation [3-20]; besides, early in the onset of the disease, it can be seen that EMG motor nerve conduction velocity slows down, the latency period is prolonged, the amplitude is reduced, and F-wave showed prolonged latency period or disappeared [14,20]. The previously reported cases and our two cases all have the above characteristics, which is the main reason why all patients were diagnosed with GBS. There are the following hypotheses for the causes of GBS in patients after spinal surgery: Surgery and anesthesia drugs damage nervous tissue, expose antigens (myelin, glycolipids, gangliosides), activate the endocrine stress system, change the balance of the immune system, and thus trigger autoimmune response [1]; or the patient may have a potential subclinical exogenous infection before surgery [15]. These claims lack further experimental and clinical evidence.

But in the two cases, patients with GBS unexplained phenomena emerge: 1. Spinal cord injury, the two have the performance of the spinal cord injury patients, urine retention and sensory plane (the first level of T10, the second is C7 level), and retrieved to literature has 3 patients with a similar situation [2,4,15], spinal cord involvement is rarely seen in GBS [20]. 2. Intracranial hemorrhage: Spontaneous subdural hemorrhage and subarachnoid hemorrhage occurred in the first patient, which could not be explained by GBS. 3. Short incubation period and long duration of disease progression. Tetraplegia occurred 3 and 8 days postoperatively in two patients and lasted for more than a month. This is also inconsistent with the natural course of GBS. Immunoglobulin and plasmapheresis are effective methods for the treatment of GBS, and a good prognosis can be obtained with early treatment [3-5,20]. Our case did not see an obvious recovery of neurological function after receiving immunoglobulin shock therapy. All of these characteristics are enough to make us question the diagnosis of GBS. Further investigation found that both patients had cerebrospinal fluid leakage, and the cerebrospinal fluid drainage volume was both large, reaching 400 ml to 500 ml/day at most. Cerebrospinal fluid leakage is a common complication of spinal surgery, which is usually seen after lumbar surgery [8]. If the amount of CSF collected in the postoperative drainage tube is between 221 and 250 mL/day, the possibility of postoperative CSF leakage should be suspected [9]. Rapid loss of cerebrospinal fluid can lead to the occurrence of low intracranial pressure syndrome, and patients generally appear with sudden acute symptoms within 5 days after surgery [11]. Intracranial Hypotension Syndrome (IHS) is the result of decreased pressure or volume of cerebrospinal fluid. The common clinical manifestations of IHS include headache related to changes in body position, accompanied by nausea and vomiting, tinnitus and photophobia, dizziness, unstable gait, palpitations, sweating, etc. [6,7]. Less common symptoms include numbness, weakness, facial paralysis, difficulty swallowing, and difficulty pronouncing words. Rare manifestations include radiculopathy, quadriplegia, epilepsy, and Parkinson's symptoms [21]. In severe cases, the level of consciousness drops, brain disease comas, and death may occur [7]. In addition, subarachnoid hemorrhage and spinal epidural hematoma have also been described as a result of low intracranial pressure, with an incidence of approximately 16% to 57% [10]. Typical manifestations of MR include subdural effusion or hematoma [6], diffuse dural enhancement, venous sinus dilatation, cerebral ptosis, and cerebellar tonsil hernia [2]. Does IHS cause multiple peripheral nerve damage? Through a literature search, we found no related reports, but there are pointed out in the literature of intracranial pressure can lead to a wide range of nerve root disease, can appear in patients with facial nerve paralysis, bulbar paralysis, legs, and shallow breathing, tendon reflex disappears, electromyography of F wave disappear or lengthen the incubation period, motor nerve demyelinating change [12,13]. This kind of change and the two patients and 1/4/5/6 patients were similar [1,2,17,22]. Therefore, we speculated that the sharp decrease of intracranial pressure in the patient led to spinal cord injury and nerve root injury, and the postoperative cerebrospinal fluid leakage was the reason for the quadriplegia of the patient. One patient was positive for ganglioside antibody GM1 IgG. GM1 is a peripheral nerve antibody associated with GBS, but it is also seen in a variety of immune neuropathy, such as Multifocal Motor Neuropathy (MMN), Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), and para-tumor associated peripheral neuropathy [19-24]. Other studies have shown that the positive rate of GM1 is related to the degree of an inflammatory response, the severity of the disease, and the prognosis of the disease, and a positive GM1 means that patients respond well to gamma globulin treatment [18]. However, our patients did not respond well to gamma globulin therapy. Therefore, we believe that the patient in this paper cannot be diagnosed as CBS because of GM1 positivity, which only indicates that there may be antigen exposure and activation of relevant antibodies in the course of the patient's disease. GBS is lack of effective means of prevention and treatment for complex, and cerebrospinal fluid leakage can prevent remedy,

found that the cerebrospinal fluid leakage after can be treated through blood patch, stay in bed, and rehydration methods such as improving symptoms [6-8], also can be repaired by epidural for example fat transplantation, fibrin glue, glycoprotein acid, such as bone wax prevention or repair of CSF leakage [7,8,11]. These two cases provide new ideas for the diagnosis and treatment of quadriplegic patients after spinal decompression and alert surgeons to the importance of cerebrospinal fluid leakage as a common complication.

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