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Guillain-Barre Syndrome in High Tetraplegia Following Spinal Cord Lesion

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Abstract

Study design: A case report of Guillain–Barre Syndrome (GBS) variant presenting in a patient with high tetraplegia following cervical spinal cord lesion (C3-C6).

Objective: To illustrate a clinical presentation of GBS in an individual with tetraplegia.

Setting: Zhongnan Hospital of Wuhan University, Wuhan, China.

Case presentation: A 55-year-old male with high spinal cord lesion at the level of C3-C6, following an emergency posterior cervical instrumentation and fusion with decompression of C3-C6 vertebral bodies under general anesthesia, developed urinary incontinence and weakness of the limbs, and was admitted to our facility for tetraplegia. Two months after admission, the patient had a sudden onset of fever (T40.0C) for which blood culture was done, and antibiotics were administered to sub side the fever. A few days later, the patient's previously noted weakness progressed. A nerve conduction study was performed, which revealed severe axonal polyneuropathy affecting motor and sensory nerve fibers, prompting a diagnosis of acute motor-sensory axonal neuropathy (a variant of Guillian-Barre syndrome). Electromyography (EMG) reports indicated abnormal spontaneous activity in all limb muscles. An emergency Lumbar Puncture (LP) was performed which revealed the classical sign of albumino-cytological disassociation of cerebrospinal fluid. GBS was diagnosed, but since the patient had pre-existing tetraplegia, autonomic dysfunction and was ventilated, the diagnosis was overshadowed and unfortunately delayed. Nevertheless, treatment modalities for both tetraplegia and GBS were initiated. There was a significant improvement in all extremities, but the bilateral decrease in the lower limbs muscle tone persisted. However, the patient refused to perform a repeat LP and was discharged three months later, under the requisite for regular follow-up.

Conclusion: A careful neurological assessment prompted the diagnosis of acute polyradiculoneuropathy in a chronic patient with tetraplegia. It demonstrates how, in this population, an otherwise uncomplicated diagnosis of GBS can easily be missed. A deeper understanding of the cause and necessity for a subsequent therapeutic intervention in potentially life-threatening autonomic instability was understood via these signs.

Keywords: Electromyography • Hypertension • Lumbar puncture

Introduction

Sneddon's Guillain–Barre Syndrome (GBS) is an acute inflammatory disorder of the peripheral nerves, often triggered by an acute infectious process in the weeks prior to the onset of GBS. Hence, GBS is also known as acute inflammatory demyelinating polyneuropathy (AIDP) [1]. GBS is characterized by the weakness that affects the lower limbs first, rapidly progressing to the arms, upper body, and face in an ascending manner. The hallmark features of GBS are bilateral, symmetric and ascending weakness with areflexia. The pathological organisms usually involved in subsequent GBS include Epstein-Barr virus, Mycoplasma pneumoniae, Campylobacter jejuni, and cytomegalovirus [1]. GBS has also been reported following surgery and head trauma [2-4]. Thus, due to its varying etiology, the diagnosis of GBS could be somewhat challenging.

We report an intriguing case of GBS presenting in a patient with high tetraplegia following cervical spinal cord lesion (C3-C6). The hallmark features of GBS were obscured by pre-existing tetraplegia. The patient was ventilated at the time and had a pre-existing condition of hypertension and bladder dysfunction. This made the complications of autonomic instability and respiratory failure, frequently seen in GBS, more diagnostically challenging. Patient consent for publication of this case report was obtained.

Case Presentation

A 55-year-old male was admitted to our facility for tetraplegia. Approximately two weeks before admission, the patient had undergone a DSA guided cervical radiofrequency ablation under local anesthesia in a nearby hospital. A few days later, he developed fever, pain in his right upper limb, and bilateral weakness in upper and lower limbs. Blood analysis revealed a bacterial infection for which levofloxacin and cefoxitin were administered as symptomatic supportive treatment. The patient later on developed urinary incontinence and his weakness of the limbs aggravated. MRI reports showed high spinal cord lesion at the level of C3-C6. An emergency posterior cervical instrumentation and fusion with decompression of C3-C6 vertebral bodies was performed under general anesthesia. The patient stabilized; however, his weakness of bilateral upper and lower limbs persisted. The patient was admitted to our facility for "Tetraplegia". The patient's initial muscle strength grading was: left upper arm graded 4/5, left forearm 4/5, left-hand grip 4/5, right upper arm 3/5, right forearm 3/5, right-hand grip 2/5, and lower extremities 0/5 bilaterally. The muscle tone for upper limbs was normal with a bilateral decrease in the lower limb muscle tone. Deep tendon reflexes, superficial reflexes were present and pathological reflexes absent. The patient underwent standard rehabilitative treatment for tetraplegia and the treatment modalities were adjusted per the patient's needs.

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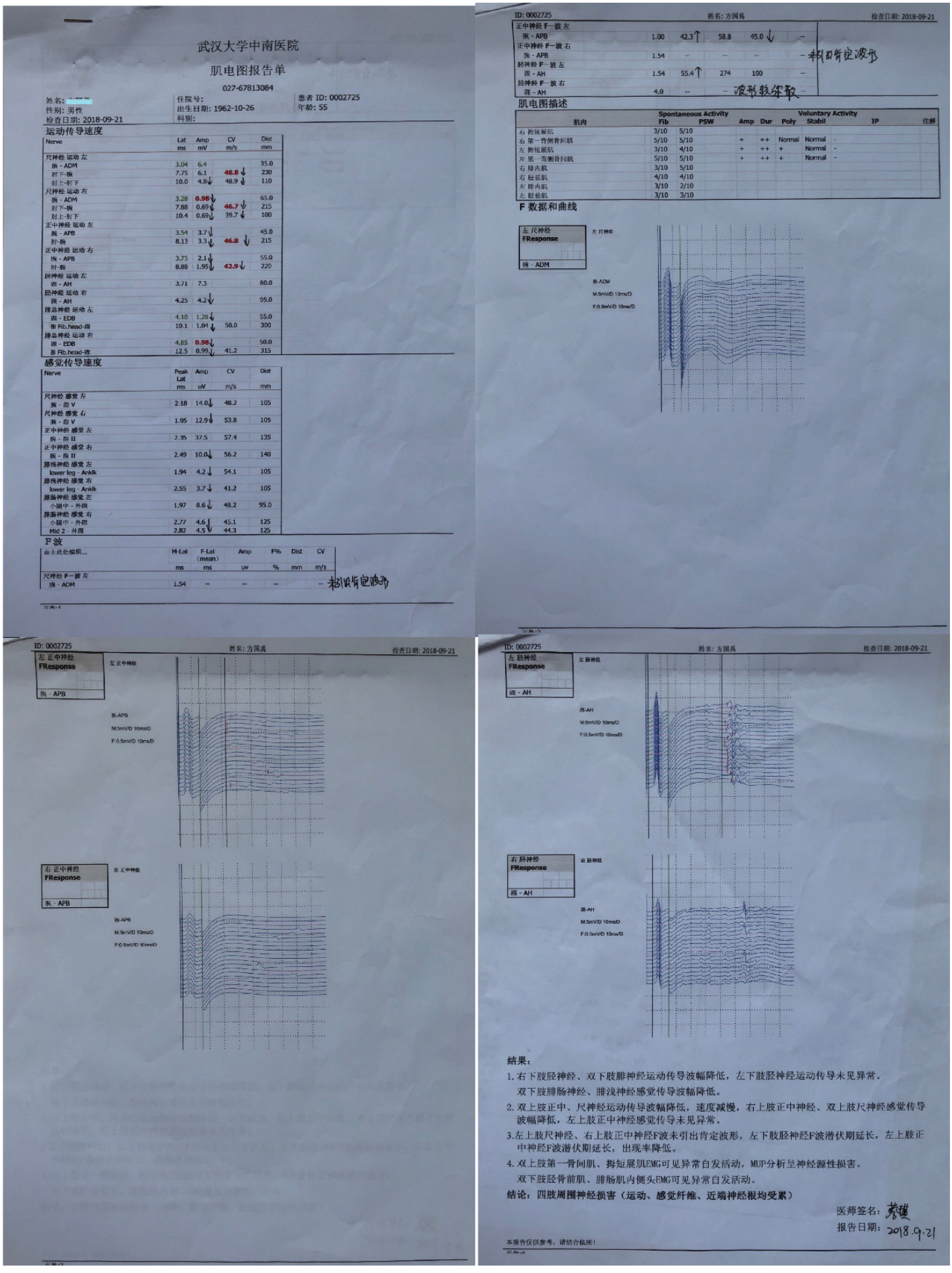

Figure 1. Abnormal spontaneous activity in all limb muscles, indicating acute motor-sensory axonal neuropathy.

A week after admission, tracheotomy was performed under local anesthesia. The tracheotomy was carried out smoothly and the patient's vitals were stable.

Approximately two months after admission, the patient had a sudden onset of fever (T40.0C). A blood culture was done, and antibiotics were administered to sub side the fever. A few days later, the patient's previously noted weakness progressed. A nerve conduction study was performed, which revealed severe axonal polyneuropathy affecting motor and sensory nerve fibers. Electromyography (EMG) reports indicated abnormal spontaneous activity in all limb muscles (Figure 1). An emergency Lumbar Puncture (LP) was performed which showed CSF Protein: 0.77 g/L↑, LDH:<43 U/L, ALB-CSF:450.0 mg/L↑, G:90.70 mg/L↑, SF-2I gM:3.9 mg. L↓. GBS was diagnosed, but since that the patient had pre-existing tetraplegia, autonomic dysfunction and was ventilated, the diagnosis was overshadowed and unfortunately delayed. Nevertheless, treatment modalities for both tetraplegia and GBS were initiated. The patient was discharged three months later. At this point, his strength had improved in all extremities, with left upper arm graded 5/5, left forearm 5/5, left-hand grip 4/5, right upper arm 3/5, right forearm 3/5, right-hand grip 3/5, and lower extremities 2/5 bilaterally. Although the bilateral decrease in the lower limbs muscle tone persisted, the patient refused to perform a repeat LP and was discharged under the requisite for regular follow-up.

Discussion

Although GBS following spinal cord injury and surgery have been described, only few cases of GBS in high tetraplegia following spinal cord lesion has been reported [2-5]. This case is interesting because many of the hallmark features that lead a clinician to a relatively straightforward diagnosis of GBS were shadowed by the patient's pre-existing condition of tetraplegia and autonomous dysfunction. The progression in the patient's previously noted weakness following fever, with reports of EMG, and lumbar puncture revealing albumino-cytological disassociation was an unmistakable sign in diagnosing GBS.

Conclusion

This case emphasizes the importance of early neurological assessment in a chronic patient with tetraplegia. It demonstrates how, in this population, an otherwise uncomplicated diagnosis of GBS can easily be missed. The degrading muscle strength despite proper treatments, along with EMG and LP reports were the key-points in diagnosing GBS. A deeper understanding of the cause and necessity for a subsequent therapeutic intervention in potentially life-threatening autonomic instability was understood via these signs.

References

1. Anand B. Pithadia and Nimisha Kakadia. "Guillain-Barré Syndrome (GBS)." Pharmacol Rep 62 (2010): 220-232.

2. Robert, Duncan and P. G. Kennedy. "Guillain-Barre Syndrome Following Acute Head Trauma." Postgrad Med J 63 (1987): 479-480.

3. Tsai-Ming Lin, Su-Shin Lee, Ruey-Tay Lin and Chung-Sheng Lai. "Guillain-Barré Syndrome Following Facial Bone Fracture." J Plast Reconstr Aesthetic Surg 59 (2006): 543-546.

4. Dong Wuk Son, Geun Sung Song, Sun Ki Sung and Sung Hoon Kim. "Guillain-Barre Syndrome Following Spinal Fusion for Thoracic Vertebral Fracture." J Korean Neurosurg Soc 50 (2011): 464.

5. James Scozzafava, Glen Jicking, Jack H. Jhamandas and Michael J. Jacka. "Guillain-Barre Syndrome Following Thoracic Spinal Cord Trauma." Can J Anesth 55 (2008): 441-446.

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