

Successful surgical treatment of scoliosis secondary to Guillain–Barré syndrome

Case report

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Abstract

Guillain-Barré syndrome (GBS) is an acute autoimmune inflammatory demyelinating polyneuropathy that mostly affects the peripheral nervous system. Little is reported about spinal deformity associated with GBS. This study aims to present a case of scoliosis occurring in the setting of GBS.

Case report and literature review.

The patient was a 14-year-old male with scoliosis. His spinal plain radiographs showed that the Cobb angle of thoracic scoliosis was 114°. History review revealed that he developed profound lower extremity pain, weakness, and numbness after catching a cold 5 years ago. These symptoms progressed to unsteady gait and inability to stand up from squatting position. The diagnosis of GBS was confirmed based on these symptoms. He underwent a posterior correction at Thoracic 5–Lumbar 5 (T5–L12) levels using the (LEGACY, USA) spinal system. The Cobb angle was corrected from 114° to 45° (correction rate 60.5%). His follow-up was symptomatic, well balanced in the coronal planes, with solid fusion 12 months after the operation.

Neuromuscular scoliosis could develop secondary to GBS. When evaluating patients with acute inflammatory polyneuropathy, clinical examination of the spine is essential to identify patients with rare neuromuscular scoliosis.

Abbreviations: CT = computed tomography, GBS = Guillain–Barré syndrome, MRI = magnetic resonance imaging.

Keywords: Guillain-Barré syndrome, neuromuscular scoliosis, scoliosis

1. Introduction

Guillain–Barré syndrome (GBS) is an acute autoimmune inflammatory demyelinating polyneuropathy that mostly affected the peripheral nervous system.^[1–3] GBS is clinically characterized by progressive symmetrical weakness of limbs, with or without autonomic or sensory disturbances.^[4–6] The worldwide incidence of GBS is 1.2 to 3 cases per 100,000.^[7] GBS may be induced by various factors, such as immunization, infection, trauma, or surgery.^[8] The pathogenesis of GBS was that neural

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antigens were cross-reactivated with antibodies to infections including cytomegalovirus, *Hemophilus influenzae*, *Mycoplasma pneumonia*, and herpes simplex.^[9,10] In this study, we reported a case of GBS in a 14-year-old patient with scoliosis.

2. Consent

Written informed consent was obtained from the patient's parents on behalf of the child for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

3. Case report

We present a 14-year-old patient admitted for a correction of his progressive scoliosis deformity. His spinal plain radiographs showed that the Cobb angle of thoracic scoliosis was 114° (Fig. 1), suggesting the need for surgical correction.

His medical history included the diagnosis GBS at the age of 9. He complained about profound lower extremity weakness and numbness after catching a cold. One week later, pain and weakness in the lower limbs, unsteady gait, and the inability to stand up from squatting position unassisted, developed, and progressed. The diagnosis of GBS was confirmed in the local hospital. The patient received treatment with intravenous immunoglobulin and steroids. He showed significant recovery in strength in lower extremities after 2 weeks of treatment.

Two years later, the patient presented to the spinal service complaining of asymmetry of his shoulders. The deformity progressed over 1 year. His spinal plain radiographs showed that the Cobb angle of thoracic scoliosis was 114°, suggesting the need for surgical correction. Computed tomography revealed no

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cavernous angioma.



Figure 1. Standing anteroposterior and lateral radiographs of the preoperation.

Three months after the cavernous hemangioma resection

surgery, a posterior correction and fusion at T5 (Thoracic 5)-L5

(Lumbar 5) levels was performed, using the LEGACY spinal system (USA). The total operation time was about 4h. Total

amount of blood loss was 1200 mL and the amount of autologous

blood transfusion was 650 mL. During the operation, the signal



Figure 3. Standing anteroposterior and lateral radiographs of 4 d after operation.

of this patient was normal using intraoperative spinal cord

vertebral body deformities. Magnetic resonance imaging revealed that cavernous hemangioma in the T4 (Thoracic 4) level of the spinal cord (Fig. 2). Therefore, cavernous hemangioma resection was performed in the Department of Neurosurgery of our hospital and histological examination confirmed the diagnosis of

4. Discussion

GBS is an acute postinfectious autoimmune polyneuropathy, characterized by rapidly progressive, areflexia, and symmetrical limb weakness.^[11,12] The diagnosis of GBS depends on the clinical features including rapid development of areflexia, muscle paralysis, and albuminocytologic dissociation of cerebrospinal fluid.^[13,14] Limited reports are available on neuromuscular

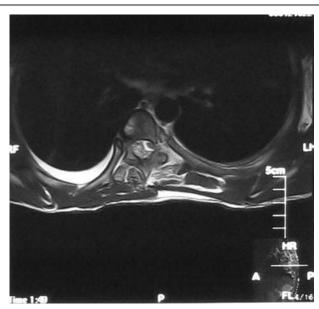


Figure 2. Magnetic resonance imaging revealed that spinal-cord cavernous hemangioma in the Thoracic 4 level of the spinal cord.



Figure 4. Standing anteroposterior and lateral radiographs of 12 mo after operation.

scoliosis.^[15–17] In this study, we reported this case of a 14-yearold GBS case with scoliosis.

In our case, lower limb pain occurred first, followed by weakness in the lower limbs, unsteady gait, and the inability to stand up from squatting position unassisted. These symptoms progressed, indicating a high possibility of GBS. The patient showed significant recovery in strength in lower extremities after treatment with intravenous immunoglobulin and steroids.

Some patients may develop neuromuscular complications such as neuromuscular scoliosis after acute onset of GBS.^[18] In this case, the patient presented to the spinal service with asymmetry of shoulders 2 years after the onset of GBS. The deformity progressed over 1 year. These clinical features suggested the necessary of surgical correction. As far as we know, there is no specific guideline for operations on patients with scoliosis secondary to GBS. The onset of GBS with scoliosis could follow elective spine surgery. The present case described a successful surgical management of a rapidly progressive scoliosis with contemporary posterior surgical instrumentation and fusion.

In conclusion, GBS is a relatively rare syndrome described in recent years. Doctors must keep in mind that neuromuscular scoliosis could develop secondary to GBS. When evaluating patients with acute inflammatory polyneuropathy, clinical examination of the spine is essential to identify patients with neuromuscular scoliosis.

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References

- Hiew FL, Rajabally YA. Sural sparing in Guillain–Barre syndrome subtypes: a reappraisal with historical and recent definitions. Clin Neurophysiol 2016;27:1683–8.
- [2] Samieirad S, Khajehahmadi S, Tohidi E, et al. Unusual presentation of Guillain–Barre syndrome after mandibular fracture treatment: a review

of the literature and a new case. J Oral Maxillofac Surg 2016;74: 129.e1-6.

- [3] Kalita J, Ranjan A, Misra UK. Outcome of Guillain–Barre syndrome patients with respiratory paralysis. QJM 2016;109:319–23.
- [4] Wang Y, Zhang HL, Wu X, et al. Complications of Guillain-Barre syndrome. Exp Rev Clin Immunol 2016;12:439–48.
- [5] Farbu E, Rudolph T, Stefansdottir S. Guillain–Barre syndrome-incidence and clinical course in Southern Rogaland, Norway. Clin Neurol Neurosurg 2015;141:33–7.
- [6] Munday WR, DiCapua D, Vortmeyer A, et al. Guillain–Barre syndrome mimics primary biliary cirrhosis-related myopathy. Oxf Med Case Reports 2015;2015:272–5.
- [7] Vellozzi C, Iqbal S, Broder K. Guillain–Barre syndrome, influenza, and influenza vaccination: the epidemiologic evidence. Clin Infect Dis 2014; 58:1149–55.
- [8] Santos L, Mesquita JR, Rocha Pereira N, et al. Acute hepatitis E complicated by Guillain–Barre syndrome in Portugal, December 2012 a case report. Euro Surveill 2013;18: pii: 20563.
- [9] Rosca EC, Rosca O, Simu M. Intravenous immunoglobulin treatment in a HIV-1 positive patient with Guillain–Barre syndrome. Int Immunopharmacol 2015.
- [10] Pyun SY, Jeong JH, Bae JS. Recurrent Guillain–Barre syndrome presenting stereotypic manifestations, positive antiganglioside antibodies, and rapid recovery. Clin Neurol Neurosurg 2015;139:230–3.
- [11] Wang Y, Sun S, Zhu J, et al. Biomarkers of Guillain–Barre syndrome: some recent progress, more still to be explored. Mediators Inflamm 2015;2015:564098.
- [12] Wu X, Zhang B, Li C, et al. Short-term prognosis of mechanically ventilated patients with Guillain–Barre syndrome is worsened by corticosteroids as an add-on therapy. Medicine 2015;94:e1898.
- [13] van den Berg B, Walgaard C, Drenthen J, et al. Guillain–Barre syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol 2014;10:469–82.
- [14] van Doorn PA. Diagnosis, treatment and prognosis of Guillain-Barre syndrome (GBS). Presse Med 2013;42:e193-201.
- [15] Edwards MR, Panteliadis P, Lucas JD. Neuromuscular scoliosis as a sequelae of Guillain-Barre syndrome. J Pediatr Orthop B 2010;19: 95-7.
- [16] Huang SL, Qi HG, Liu JJ, et al. A rare complication of spine surgery: Guillain–Barre syndrome. World Neurosurg 2015;84:697–701.
- [17] Jameson R, de Loubresse CG, Maqdes A. Spinal neuroarthropathy associated with Guillain–Barre syndrome. Eur Spine J 2010;19(suppl 2): S108–13.
- [18] Dua K, Banerjee A. Guillain-Barre syndrome: a review. Br J Hosp Med (Lond) 2010;71:495-8.