https://doi.org/10.46389/rjd-2022-1109
www.rjdnmd.org

Rom J Diabetes Nutr Metab Dis
2022; volume 29, issue 4, pages 476-479

Case Report

A case report of Guillain-Barre syndrome with demyelinating polyradiculoneuropathy

Anuradha Ganesan [1], Santhini Gopalakrishnan [1]*, Durga Venkata Satyamurthy Golla [1]

[1] Department of Biochemistry, Chettinad Academy of Research and Education, Kelambakkam, India

* Correspondence to: Santhini Gopalakrishnan, Department of Biochemistry, Chettinad Academy of Research and Education, Kelambakkam, Chengalpattu District, Tamil Nadu 603103, India. Phone: 7299069636; E-mail: santhini.research@gmail.com

Received: 3 January 2022 / Accepted: 9 August 2022

Abstract

Guillain-Barre syndrome (GBS) is an autoimmune disorder with the most common clinical presentation of neuromuscular paralysis. Here we have reported a rare case of GBS in a 64-year-old patient who presented with chief complaints of generalized body aches and difficulty in walking and sitting for the past five days. Laboratory investigations showed an elevation of protein level in the cerebrospinal fluid (122.1 mg/dl) and CK total (24315 U/L). A nerve conduction study done by sampling from Median, Ulnar, Peroneal, Tibial and Sural nerves was suggestive of asymmetrical severe sensory demyelinating polyradiculoneuropathy more prevalent in the lower limb than in the upper limbs. The patient was admitted and treated with antibiotics, analgesics, muscle relaxants, multivitamins and other supportive measures. Given the progressive worsening of symptoms, intravenous immunoglobulin infusion began, after which the patient's condition gradually improved.

Keywords: Guillain-Barre syndrome, weakness, cerebrospinal fluid.

Introduction

Guillain-Barre syndrome (GBS) is a degenerative inflammatory condition with demyelinating polyneuropathy, which can be acute or chronic. GBS is a neurological disorder mainly affecting the peripheral nervous system, cranial nerves and spinal nerve roots with autonomic nervous system disturbances [1]. It involves both cell and humoral-mediated immune systems [2]. The most common clinical presentation in GBS is muscle weakness which is usually coupled with peripheral neuropathy [3]. We have reported a rare case of GBS in a 64-year-old man who presented with difficulty in walking and sitting for the past five days.

Case report

A 64-year-old man was referred to the casualty with complaints of generalized body pain with more involvement of the lower limb than the upper limb and difficulty walking and sitting over the past five days. Also, he has had difficulty opening his mouth for the past three days. The patient had no history of numbness, fever/cough/breathlessness or head injury. No chest pain/palpitations/sweating was noted. He denied recent contact/travel.

The patient is a known case of type 2 diabetes mellitus (under medication for 20 years), systemic hypertension (under medication for the past 10 years), and coronary artery disease for the past 2 years. An angiogram was performed 2 years ago. His personal history revealed that he has a mixed diet and that his bowel and bladder habits are normal. He occasionally drank alcohol over the past 10 years and no history of smoking was present.

On Examination, the patient was pallor, conscious, oriented and afebrile. His vital signs were heart rate – 60/min, blood pressure – 140/70 mm Hg, SpO_2 – 100% and respiratory rate – 18/min. Severe sensory loss is present. Both upper and lower limb muscle power are reduced (4/5). Deep tendon reflexes are reduced.

Table 1: Laboratory test results.

Complete blood count	
Total Leukocyte count	8100/CMM
RBC Count	4.39 Million/c.mm
Hemoglobin	12.8 g/dl
PCV	37.4%
MCV	85.2 FL
MCH	29.1 PG
MCHC	34.2 g/dl
RDW	14.6%
Platelet count	2.01 LAC/C.MM
Neutrophil	78.8%
Eosinophil	1.7%
Basophil	0.3%
Lymphocyte	12%
Monocyte	7.2%
Prothrombin time (PT)	
Control	11.7 sec
Test	**12.4 sec**
ISI	1.09
INR	1.05
Activated partial thromboplastin time (APTT)	
Control	29 sec
Test	**24.8 sec**
D-DIMER	**324.00 ng/mL**
Complete urinalysis	
Color	Straw
Appearance	**Turbid**
pH	6.0
Specific gravity	1.020
Albumin	**TRACE**
Sugar	++
Ketones	Negative
Bile salts	Negative
Bile pigments	Negative
Urobilinogen	Normal
Blood	**Positive**
Leukocyte Esterase	Negative
Nitrite	Negative
Deposits	Nil
Pus cells	5–7/HPF
Epithelial cells	2–5/HPF
RBC	**Plenty/HPF**
Casts	Nil
Crystals	Nil
Others	Nil
Hb A1c(Glycosylated HB)	**8.6%**
Blood Urea Nitrogen	**101 mg/dl**
Serum Creatinine	**3.76 mg/dl**
Serum Phosphorus	**5.6 mg/dl**
Serum CK Total	**24315 U/L**
Serum Calcium	**8.1 mg/dl**
Serum Vitamin B12	894 pg/mL
Serum Vitamin D	**17.15 ng/mL**
Serum liver function test (LFT)	
Total Protein	7.1 g/dl
Albumin	3.8 g/dl
Globulin	3.3 g/dl
A/G Ratio	1.2:1
Total Bilirubin	0.41 mg/dl
Direct Bilirubin	0.076 mg/dl
AST	587 U/L
ALT	210 U/L
Alkaline phosphatase	92 U/L
Gamma GT	75 U/L
Serum electrolytes	
Sodium	133 mEq/L
Potassium	5.5 mEq/L
Chloride	104 mEq/l
Bicarbonate	18 mEq/L
Cardio 3 panel	
BNP	45 pg/mL
CK-MB	**>292.0 ng/mL**
Troponin I	**61.0 pg/mL**
LDH	**1391 U/L**

Table 2: Microbiolgy report.

CRP	**Reactive (24.31 mg/L)**
Pro calcitonin	Negative

Table 3: Cerebrospinal fluid analysis.

CSF Fluid Report	
Color	Straw
Appearance	Clear
Total leukocyte count	No cells
Impression	Acellular smear
CSF Fluid Analysis	
CSF Chloride	125 mEq/L
CSF Protein	**122.1 mg/dl**
CSF Glucose	**91 mg/dl**

Laboratory examination reports

Table 1 shows the laboratory test results performed for the patient, while the microbiology report can be found in Table 2.

A lumbar puncture was performed and cerebrospinal fluid (CSF) analysis is shown in Table 3.

Magnetic resonance imaging (MRI) of the brain was performed as well. The results were suggestive of age-related cerebral atrophy.

Echocardiograpy was also performed and Table 4 shows the echocardiographic regional wall motion analysis report.

Nerve conduction studies of all four limbs were performed by sampling from the median, ulnar, peroneal, tibial and sural nerves, as seen in Table 5.

Discussion

Atypical presentation of GBS often serves as a challenge for the medical fraternity to arrive at the diagnosis. The factors that precipitates GBS include viral infection [4], mycoplasma infection [5], blood transfusion [6], surgery [7–9].

In this case, the patient clinically presents with muscle paresis and decreased deep tendon reflexes, similar to the study by Hund EF *et al.* [10]. Also, biochemical reports showing elevation in CSF protein and CK-Total levels and demyelinating polyradiculoneuropathy in electromyography and nerve conduction velocity test favors the diagnosis of GBS. The patient was treated with immunomodulators like intravenous

Table 4: Echocardiographic regional wall motion analysis.

Basal inferior - Mildly hypo kinetic
Impression
Normal left ventricle systolic function (EF-60%)
Grade I diastolic dysfunction
Concentric left ventricular hypertrophy
Sclerosed aortic valve
Mild aortic regurgitation
Mild Tricuspid regurgitation with normal pulmonary artery pressure (tricuspid regurgitation pressure gradient – 25 mm HG)

Table 5: Nerve conduction study results.

Motor nerve studies
The distal latencies are prolonged, with normal CMAPs amplitudes and reduced conduction velocities from both median and ulnar nerves.
F-wave studies
Late response F-wave latencies from both median nerves are prolonged;
Late responses F-wave from both ulnar nerves are absent;
Late responses F-wave from both peroneal and tibial nerves are prolonged.

https://doi.org/10.46389/rjd-2022-1109

Table 5: Continued.

Sensory nerve studies
Both median sensory nerves latencies are prolonged, with mildly reduced amplitude and reduced conduction velocities;
Both ulnar sensory latencies are prolonged, with reduced amplitude and reduced conduction velocities;
Both sural and superficial peroneal nerves' sensory potential could not be obtained.
H-reflex studies
Both H-reflexes latencies are prolonged.
Impression
This electrophysiological study suggests asymmetric severe sensory motor demyelinating polyradiculoneuropathy (Lower limb more than upper limbs).

immunoglobulin and plasma exchange, following which the patient recovered gradually.

Conclusion

GBS is a degenerative neurological disorder where muscle weakness and neurological manifestation predominate. The patient usually presents with symmetrical or asymmetrical muscle paresis and pain with or without cranial nerve involvement and autonomic disturbances. In this case, the clinical picture, CSF analysis, and electromyographic studies were diagnostic tools for GBS.

Conflict of interest

The authors declare no conflict of interest.

References

1. Van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014; 10(8): 469–82.
2. Murray DP. Impaired mobility: Gullain-Barre syndrome. J Neuroscience Nursing 1993; 25(2):100-104.
3. Miller RG. Guillain Barre syndrome. Postgraduate Medicine 1985; 77(7):57-64.
4. Ormerod IE, Ceekerell OC. Guillain-Barre syndrome after herpes zoster infection, a report of 2 cases. European Neurology 1993;33(2):136-318.
5. Nadkarni N, Lisak RP. Guillain-Barre syndrome (GBS) with bilateral optic neuritis and central white matter disease. Neurology 1993; 43(4):842-843.
6. Merelli E, Sola P, LaSpina I, Orlando A, Milanti G. Guillain-Barre polyradiculoneuritis after blood transfusion. Ital J Neuro Sci 1991; 12(3):313-315.
7. Stambough JL, Quinlan JG, Swanson JD. Guillain-Barre syndrome following spinal fusion for adult scoliosis. Spine 1990;15(1):45-46.
8. Ennis JH, Bednar DA. Lumbosacral fusion in a patient with recurring Guillain-Barre syndrome and acute brachial neuritis. J Spine Disord 1992; 5(2):217-218.
9. Ginn D. Guillain-Barre syndrome. An uncommon but severe illness. Postgrad Med 1991; 90(5):145-416, 153-156.
10. Hund EF, Borel CO, Comblath DR, Hanley DF, McKhann GM. Intensive management and treatment of severe Guillain-Barre syndrome. Critical Care Medicine 1993; 21(3):433-446.