



## Guillian-barre Syndrome as one of the Rare Manifestation of Systemic lupus Erythematosus- A Rare Case Report

Authors

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### Abstract

*Systemic lupus erythematosus (SLE) is an autoimmune disorder being mediated by immunocomplex and autoantibodies and INF-Alpha is the key mediator. Among neurological manifestations Guillian barre syndrome (GBS) is one of the rare presentation. We described a 35 year female who developed acute onset progressive ascending quadriparesis with tingling and numbness of bilateral lower limb. NCS showed acute motor sensory axonal polyneuropthy. The patient had diffuse non scarring alopecia with anaemia with proteinuria and both ANA and anti –dsDNA were reactive. The patient improved with pulse dose of methylprednisolone and i.v immunoglobulin (2gm/kg body weight) and discharged with hydroxychloroquinie and low dose of prednisolone.*

### Introduction

Systemic lupus erythematosus (SLE) is characterized by heterogeneous, multisystem involvement and the production of an array of autoantibodies, immune complex mediated disease. The complex process is triggered by genes, female hormones, environmental factors like UV light, smoking and infections like EBV. Neuropsychiatric systemic lupus erythematosus (NPSLE) as described by the American College of Rheumatology (ACR) research committee includes 19 neuropsychiatric syndromes divided into neurologic syndromes of the central, peripheral, and autonomic nervous system and the psychiatric syndromes observed in patients with SLE in which other causes have been excluded. These symptoms may precede the onset of SLE or can occur at any time during the course of SLE<sup>[1]</sup>. However, to the best of our knowledge, few cases

have been reported in the last 50 years in which the initial manifestation of SLE was Gullian–Barre syndrome (GBS)<sup>[2]</sup>

### Case Report

A 35-year-old female presented with tingling and numbness of both the lower limbs with weakness of both the limbs which was sudden onset progressive ascending type and gradually involved both the upper limbs in span of 2 days. She got bed ridden and came under Hugh score 3. There was no bladder and bowel involvement. There was no muscle fasciculation, no headache, nausea, vomiting, no blurred vision, no difficulty in swallowing and drinking. There was no history of vaccination. There was no antecedent fever, loose stool episodes in past 1 month. The patient is not a known case of diabetes mellitus, chronic kidney disease, any thyroid disorder.

On examination patient was afebrile, respiratory rate 18/min, blood pressure of 126/70 mm of Hg. The patient had diffuse non scarring alopecia. On neurological examination tone was diminished in both upper and lower limb with no atrophy. Power was 3/5 proximally and 4/5 distally in both upper and lower limbs. All the DTRs are diminished. Paresthesia was there in distal limbs in gloves and stoking patterns. There was also fine touch and vibration lost up to mid of b/l legs and hands. Lab data showed anemia was there with Hb 10.2 gm/dl. TLC and TPC were in normal limits. Serum urea and creatinine were normal. LFT was normal. There was hypoalbuminemia (albumin was 2.4gm/dl). Urinalysis revealed protein was 3+. 24 hr urine protein came out to be 12.4 gm. The ESR was 45. Serological tests for HIV,

hepatitis B/C were negative. The ANA was 8.7 units (>1.2 –Positive), anti-dsDNA was >150IU/ml(30 IU/ml-positive). Antiphospholipid antibodies were negative. CSF study revealed albumin-cytological dissociation (total protein: 153gm/dl, total cell count: 3/microlit). Contrast MRI was normal. Nerve conduction study revealed non of the Motor Nerve Conduction Velocity (MNCV) and Sensory Nerve Conduction Velocity(SNCV) was generated in any of the four limbs and also F-wave was not generated which was in more favour of motor sensory axonal polyneuroradiculopathy. The patient was improved with pulse dose of methylprednisolone 1gm daily for 5 days with IVIG 2gm/kg over 5 days. Then the patient was discharged with Hydroxychloroquine, and low dose prednisolone.

DEPARTMENT OF NEUROLOGY		RMS	
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BURLA, SAMBALPUR, ODISHA			
Patient ID	[Redacted]	Gender	: Female
Name		Age	: 45 Years 0 Months
Ref By Physician		Height/Weight	: 0 cms/0 Kg
		Date: 04-Oct-2021	
<p><i>MNCV - All limbs are not generated</i>  <i>SNCV - All limbs are not generated</i>  <i>F-wave - Lt. median Not generated</i></p> <p><i>Imp - Above exam study for motor sensory axonal polyneuroradiculopathy. Kindly correlate clinically.</i></p> <p style="text-align: right;"><i>[Signature]</i></p>			
<p><small>NOTE: THE RESULTS MAY BE CLINICALLY CORRELATED</small></p> <p style="text-align: right;"><small>Page: 1/1  <a href="http://www.rmsindia.com">http://www.rmsindia.com</a> © RMS EMG(Salus, *7.5.155)</small></p>			

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██████████ Date : 03.10.2021

TEST FOR ANTI-dsDNA (SCREENING)

Results : > 150.00 IU/ml ✓

N.B.

<20.0 IU/ml ..... Negative  
>30 IU/ml ..... Positive  
>20- 30 IU/ml ..... Equivocal

N.B. :

LIKE OTHER LAB REPORTS THIS IS TO BE CO-RELATED WITH OTHER CLINICAL FINDINGS

(DR. N. K. Jajodia)

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██████████ DATE: 02.10.2021

TEST FOR ANA (SCREENING)

Results : ↑ 8.7 Units

N.B.

<0.8 ..... Negative  
0.8-1.2 ..... Equivocal  
>1.2 ..... Positive

S.F. <del>12018</del> ADA - 12 U/L			
✓ Sugar	173	mg/dl	40 - 70
✓ Protein	153	mg/dl	10 - 40
Choloride (Total)		mg/dl	90 - 110
<b>RINE</b>			
Protein		mg/day	<150
Creatinine		mg/day	1.0-1.6
Microalbumin		mg/day	0-30
HbA1c		%	4.2%-6.2%
HS CRP		mg/L	≤ 3
D-Dimer		mg/L	0-0.5
CCP		IU/ml	≤ 30

Date: 5/10/2021

Signature: *AP*



**V.S.S. INSTITUTE OF MEDICAL SCIENCES & RESEARCH,  
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**REPORT**

Cell Count: 03 cells / Cmm  
 - Nbc Count: 25 / Cmm  
 D.C.C. - 03 mononuclear cells  
 Polymorphs - 0

Impression:- tralignant cell not seen

Advice:-

**Discussion**

The signs and symptoms in the presented patient, fulfilled the Asbury criteria<sup>[3]</sup> and ACR<sup>[1]</sup> case definitions for the diagnosis of GBS. Various forms of lupus related polyneuropathy have been reported in 10–20% of patients with SLE<sup>[4]</sup>. However, GBS which is a demyelinating polyneuropathy, is a rare complication in lupus<sup>[5,6]</sup>. The prevalence of SLE with GBS has been reported to be between 0.6% and 1.7%<sup>[7,8]</sup>. The pathogenesis of GBS as a manifestation of active SLE is not clear, but both cell-mediated and humoral processes may play a significant role<sup>[6]</sup>. There are currently four kinds of anti-neuronal antibodies: antilymphocytic antibodies, anti-phospholipid antibodies (including cardiolipin

antibodies and lupus anticoagulants), and anti-ribosomal P protein antibodies. These antibodies are often present in the plasma and cerebrospinal fluid, and they cause more extensive neurological damage. Corticosteroids, cyclophosphamide, plasmapheresis, and immunoglobulin have been used in AIDP or GBS with SLE according to the previous literature. The combination of corticosteroids and cyclophosphamide is considered the first-line treatment option in a review of the literature<sup>[9]</sup>.

**Conclusion**

Our case shows GBS can be one of the initial presentations of SLE which was successfully treated by IVIG and pulse dose of

methylprednisolone. Still more researches are required to find the pathogenesis and treatment options of both the disease.

**Conflict of Interests:** No

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