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Guillain-Barré Syndrome as a Primary Manifestation of Systemic Lupus Erythematosus: A Comprehensive Case Report

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Abstract:
Guillain-Barré Syndrome (GBS) is an exceedingly rare presentation in patients with Systemic Lupus Erythematosus (SLE). This case report describes a 35-year-old female patient who presented with classical GBS symptoms and was subsequently diagnosed with SLE. We explore the diagnostic challenges, treatment approaches, and outcomes of this rare association, comparing it with other cases from the literature to provide insights into managing similar clinical scenarios.

Introduction:
Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease characterized by multi-systemic involvement, including the skin, joints, kidneys, and nervous system. Neuropsychiatric manifestations of SLE (NPSLE) occur in approximately 10-80% of patients, with peripheral nervous system (PNS) involvement accounting for less than 10% of cases. Guillain-Barré Syndrome (GBS), an acute inflammatory polyradiculoneuropathy, is an extremely rare manifestation of SLE, particularly as the initial presentation of the disease. The association between SLE and GBS is estimated to occur in only 0.6-1.7% of cases (1,2). This case report details a rare instance of GBS presenting as the first symptom of SLE, discusses the diagnostic process, and compares it with similar cases reported in the literature.

Case Presentation:
35-year old female patient with no previously diagnosed comorbidity was referred to us from another hospital with complaints of weakness of bilateral lower limb(patient had difficulty in getting up from squatting position) for 5 days and bilateral upper limb (difficulty in combing hair and holding of objects)for 3 days. There is no history of fever, diarrhea, vomiting, sore throat, recent vaccination, bowel/ bladder incontinence, change in speech, or difficulty swallowing. In past history, the patient had a history of recurrent oral ulcers for 1 year and was following up in dental opd for the same and was given tab vitamin b complex but she had no relief.
On examination the patient had BP-126/70 mm hg, Pulse-78 beats/min, spo2-98% . there was no significant abnormality on general physical examination. On central nervous system examination - there was hypotonia in all the limbs, power was 3/5 proximally in bilateral upper and lower limbs and 4/5 distally in bilateral lower limb. All the Deep tendon reflexes were absent and bilateral planters were flexor. Sensory and Cerebellar examination was normal. All the meningeal signs were negative. Lab data done showed normal CBC-10.9 gram/dl, TLC-3600/cu-mm, platelet-3.2lakh, MCV-78.4fl, and normal liver and

kidney function tests. ECG and chest x-ray were normal. NCCT head and CE-MRI brain were normal. CSF studies done showed albumino-cytological dissociation with less than 5 cells all of which were mononuclear, glucose -61.2 (spot blood sugar-121), protein-1.78 gram/dl, ADA-1(neg). Nerve conduction studies done showed axonal symmetric motor predominant polyneuropathy affecting all four limbs, the details of which are shown in table.1 and 2. Further autoimmune profile was sent and ANA by IF was 3+, showing speckled pattern and anti dsDNA was positive. Urine routine microscopy and 24-hour urine protein were normal. The patient was treated with IVIG 20 grams/ day for 5 days and at the end patient had a full recovery and was discharged.

Nerve	Latency (ms)		Amplitude (mV)			Duration (ms)			Distance (mm)	NCV (m/s)	F-Min (ms)	F-Max (ms)
	D	P	D	P	%Dec	D	P	%Inc				
Rt. Median	4.19	7.12	3.78	2.68	29.1	11.12	12.31	10.7	260	88.74	-	-
Rt. Ulnar	3.44	6.94	1.77	1.43	19.21	12	14.56	21.33	240	68.57	-	-
Lt. Median	3.81	7.69	2.56	3.09	20.7	10.69	12.56	16.93	240	61.86	-	-
Lt. Ulnar	2.53	4.56	4.57	2.67	41.58	12.94	15.75	21.72	250	128.87	-	-
Lt. CPN	5.44	11.56	1.48	0.79	46.62	39.56	32.19	18.63	380	62.09	-	-
Rt. CPN	-	-	-	-	-	-	-	-	-	-	-	-
Lt. PTN	5.5	13.44	0.79	1.19	50.63	10.62	19.44	83.05	410	51.64	-	-
Rt. PTN	6.12	14.87	2.87	2.62	8.71	8.44	14.31	69.55	380	43.43	-	-

Table 1. MNCV (motor nerve conduction velocity) showing no response in right CPN, reduced CMAP amplitude with normal DL (distal latency) and CV in bilateral median, ulnar and left PTN nerve, increased DL with reduced CMAP in right PTN and left CPN.

Nerve	Latency (ms)	Amplitude (μV)	Distance (mm)	NCV (m/s)
Rt. Ulnar	1.95	12.72	110.00	56.41
Rt. Median	2.17	24.50	140.00	64.52
Lt. Ulnar	1.39	23.97	110.00	79.14
Lt. Median	2.10	29.78	140.00	66.67
Lt. Sural	0.86	112.30	130.00	127.91
Rt. Sural	1.69	150.80	130.00	65.09

Table 2. SNCV (sensory nerve conduction velocity) showing normal peak latency, SNAP amplitude and CV in all tested nerves.

Discussion:

The coexistence of GBS and SLE poses significant diagnostic and therapeutic challenges due to the overlapping clinical manifestations and the rarity of their association. GBS in the context of SLE is believed to result from an autoimmune response, wherein antibodies against neuronal components, such as myelin, are produced. Additionally, vasculitis, immune complex deposition, and complement activation may contribute to peripheral nerve damage (2).

Diagnostic confirmation relies on a combination of clinical presentation, electrophysiological studies, and serological markers. In this case, the diagnosis of GBS was supported by the patient's acute onset of ascending muscle weakness, areflexia, and CSF findings of albumino-cytological dissociation. The concurrent presence of positive ANA and anti-dsDNA antibodies pointed towards underlying SLE. The response to IVIG therapy aligns with the standard treatment approach for GBS; however, SLE-associated GBS may require additional immunosuppressive therapies, such as corticosteroids or cyclophosphamide, particularly in cases with a suboptimal response to IVIG alone (1,2).

Reviewing the cases in the literature reveals a range of therapeutic strategies, from IVIG and corticosteroids to more aggressive regimens including rituximab and plasma exchange. The choice of therapy should be individualized, considering the severity of neurological impairment, the extent of systemic involvement, and the patient's overall clinical condition (1,3,4).

Several other cases have reported GBS as a primary manifestation of SLE, each illustrating different aspects of this rare presentation. Gao et al. described a 30-year-old female who presented with GBS as the first symptom of SLE. The patient was treated with glucocorticoids and cyclophosphamide, resulting in complete recovery after two months (3). Another case reported by Beshir et al. involved a 14-year-old girl who developed GBS with ascending bilateral muscle weakness and respiratory insufficiency. Despite aggressive therapy, including IVIG, methylprednisolone pulses, and rituximab, the patient required prolonged mechanical ventilation but eventually achieved full recovery after 14 plasma exchange (PLEX) treatments (4).

Saremi et al. documented a 39-year-old female initially presenting with acute peripheral neuropathy and Bell's palsy, who was eventually diagnosed with SLE. The patient demonstrated a significant neurological recovery following treatment with IVIG, azathioprine, and prednisolone (1). Similarly, Althagafi et al. reported a case of a 20-year-old female presenting with both GBS and diffuse alveolar haemorrhage (DAH) as initial manifestations of SLE. This patient was managed with a combination of corticosteroids, cyclophosphamide, and IVIG, resulting in substantial clinical improvement (2).

Conclusion:

This case emphasizes the importance of considering SLE in the differential diagnosis of GBS, particularly in young women presenting with unexplained neurological symptoms. Early identification and treatment of the underlying autoimmune condition are crucial for optimal patient outcomes. Further research is needed to establish the most effective treatment strategies for this complex clinical presentation.

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