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(CASE REPORT)

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Guillain barre syndrome: A comprehensive review and a case report

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Abstract

A 58-year-old male patient who had been complaining of weakness in his upper and lower limbs for three days was taken to the hospital. On examination, he had decreased muscle strength in both upper and lower limbs (4/5). MRI brain [plain] reveals acute CVA, left hemiparesis, small vessel changes in bilateral centrum semiovale, chronic lacunar infarct, left lentiform nucleus, and left corona radiata. The patient's complete blood count [CBC] revealed abnormal RBC, haemoglobin, PCV, MCH, and RDW levels. The patient has abnormal CRP and serum potassium levels. And the patient was diagnosed with Gullain barre syndrome. He received intravenous immunoglobulin [IVIG], plasma exchange, nutritional supplements, NSAIDs, anticonvulsants, and corticosteroid treatment. . Your immune system may unintentionally attack a component of your peripheral nervous system—the system of neurons that are not an element of your cerebral cortex or vertebral cord— It may result in Guillain-Barre syndrome (GBS), an uncommon neurological condition. Guillain-Barre syndrome is an illness that can be lethal. The patients require close monitoring and the quickest possible treatment. GBS consequences can result in mortality in just a few percent of individuals, even under the best of circumstances.

Keywords: Gullain Barre Syndrome; Upper and Lower Limbs; Left Hemiparesis; Corticosteroid Treatment

1. Introduction

1.1. Definition

An unusual neurologic condition known as Guillain-Barre syndrome (GBS) occurs when your immune system unintentionally targets a portion of your peripheral nervous system, which is a collection of nerves that are not part of your brain or the spinal cord.

Discovered less than a century ago, Guillain-Barré syndrome, also known as GBS, is a sharp, monophasic, inflammatory polyradiculoneuropathy that continues to be a major cause of muscular paralysis around the world. GBS can manifest clinically in a variety of ways, from a life-threatening quadriplegia requiring mechanical breathing to a modest, self-limiting muscular weakness. The wide range of clinical, electrophysiological, and autoantibody antibody profiles that define GBS are becoming more well recognized, indicating that this is not a single illness but rather a group of connected conditions. Any area other than nerve tissue outside of the spinal cord and brain can be impacted by GBS, an uncommon and dangerous inflammatory disease. The nervous system in the peripheral regions is the term for this. The injury stops nerve cells from delivering specific.

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1.2. Types

There are various kinds of GBS. The most prevalent kind of GBS in western countries is an acute inflammatory demyelinating polyradiculon europathy usually, a weakness starts in the lower body and works its way up to other parts of the body.

A kind of GBS known as Miller-Fisher syndrome is an acquired nerve illness. According to a trustworthy source, it accounts for between 5 and 10% of GBS cases in the US. Walking and balance issues may result from this condition. It has an impact on tendon reflexes and the muscles of the eyes.

A short motor axonal neuropathy is another uncommon type of GBS that can result in abrupt limb paralysis and occasionally respiratory difficulties. It could affect the head's nerves. Acute motor-sensory axonal neuropathy is comparable to GBS in that it begins with sensory abnormalities such as tingling or numbness.

Certain kinds have slower started points and persistent symptoms. Weakness episodes repeat over several years in chronic inflammatory demyelinating polyneuropathy. Multiple muscles in a particular location of one or both arms or legs become weak when someone has multifocal motor neuropathy. [1]

1.3. Disease statistics

GBS impacts people of every stage of life, ethnicities, and nations; it is a sudden monophasic immune-related polyradiculoneuropathy with an average age of beginning 40 years. Men are affected somewhat more often than women. Globally, the prevalence of GBS varies between 0.6 and 4.0 per 100,000 individuals. The general prevalence of GBS was determined as 1.1 to 1.8/100,000 in an exhaustive literature analysis of the epidemiology of the illness; however, in children, the prevalence was less, at 0.34 to 1.34/100,000. At age 50, the rate of GBS rises from 1.7/100,000 to 3.3/100,000, compared with patients that are younger. [2] Guillain-Barré syndrome (GBS) has rough mean yearly incidence rates ranging from 0.4 to 1.7 per 100,000 persons, according to population-based research. [3] GBS is a widespread illness throughout the world, affecting 1.3 cases per 100,000 people on average (0.4–4.0). Men are more likely than women to be affected by the illness, which peaks in youth and old age. There is no clear seasonal link in the West. [4]

1.4. Etiology and pathogenesis

Guillain-Barre syndrome's precise cause is unknown. After an upper respiratory or gastrointestinal infection, the disorder typically manifests itself days or weeks later. The condition known as Guillain-Barre syndrome can sporadically be brought on by a recent surgery or vaccine. After contracting the virus known as Zika, certain cases have been documented. It is possible for the COVID-19 virus to cause the condition known as Guillain-Barre syndrome. AstraZeneca or Johnson & Johnson COVID-19 vaccination recipients also experience this infrequently. Your immune system starts attacking your nerves when you have Guillain-Barre syndrome, even though it normally exclusively targets foreign threats. The myelin sheath, which covers and protects nerves, is involved with acute inflammatory demyelinating polyradiculoneuropathy. The injury results in feeling weak, numb, or paralyzed by stopping nerves from sending information to your brain. While its precise causes are yet unknown, infections are frequently the first step towards GBS development. A vaccine has occasionally caused it in rare instances, according to Trusted Source. Any age group can be affected; however, older people seem to be at a larger risk (reliable source). Campylobacter jejuni, which can induce diarrhoea and a colon infection, has been linked to GBS in humans. Glandular a high temperature, or mononucleosis with infection, is brought on by the Epstein-Barr virus. Both the Mycoplasma pneumoniae bacterium and the cytomegalovirus, which can infect the airways but may not show any symptoms, could be the virus known as Zika, which scientists are currently looking at.

According to one concept, viral and bacterial infections change how the immune system reacts to peripheral nerves in the body (refer to the reliable source). Thus, myelin and axons become targets of the immune system's misinterpretation of them as alien materials. [5] Precursor infections to GBS have been linked to numerous microbiological sources. Some of them include the Epstein-Barr virus, Hepatitis E virus, Mycoplasma pneumoniae, Influenza A virus, and, lately, the Zika virus. All the same, C. jejuni is the most common antecedent infectious aetiology and has been associated with the axonal type of GBS. The reason behind the low incidence of GBS in fewer than 0.1% of individuals with C. jejuni diarrhoea during the next two-month period is not well understood. This could result from being infected with strains of the virus that are specific to a given subtype and a mix of the host's vulnerability to genetic factors. Antibodies interact with specific ganglioside antigens concentrated on axonal membranes, such as GD1a or GM1, in the case of GBS after C. jejuni

infection, producing the AMAN GBS variation. Peripheral nerve damage is accelerated by the activation of complement, which is also linked to the development of anti-ganglioside antibodies. Complement inhibition has been shown to be beneficial in GBS-affected mice. [2]

1.5. Case study

A 58-years old male patient was admitted with the chief complaints of weakness of both upper limbs and lower limbs for 3 days. On examination he had decreased muscle strength in both upper limbs and lower limbs (4/5).

1.6. Personal history

Patient had reduced sleep, normal appetite and normal bowel and bladder sounds were observed on examination.

Table 1 Physical examination

S. No	Vitals	Normal values	Observed values
1.	Blood pressure	120/80 mmhg	130/90 mmhg
2.	Pulse rate	60 – 100 bpm	90 bpm
3.	Respiratory rate	12–20 breaths/min	18/min
4.	SPO2	95 – 100 %	98%
5.	Temperature	97.8°F – 99.1° F	Normal

The patient blood pressure was 120/64 mm/hg, pulse rate was 95/bpm, respiratory rate was 18/min, temperature was normal, blood sugar levels were normal.

Table 2 Systemic examination

S.no	Constituents	Observed
1.	CVS	S1 S2+
2.	RS	B/L AE+
3.	CNS	Rt – UL and LL (4/5) Lt – UL and LL (4/5)
4.	P/A	Soft

2. Diagnostic tests performed

2.1. Real time ultrasonography of the abdomen was performed

- Borderline hepatomegaly with grade I fatty changes.
- Mild irregular urinary bladder wall thickening noted – likely cystitis changes.

2.1.1. MRI brain (plain)

- Clinical details: Acute CVA, Left hemiparesis.
- Small vessel changes in bilateral centrum semiovale.
- Chronic lacunar infarct left lentiform nucleus and left corona radiata.
- No Acute infarcts

Table 3 Laboratory investigations

Laboratory parameters	Observed values	Normal values	Indication
HAEMATOLOGY			
RBC COUNT	3.93 million/cu.mm	4.50 – 6.50 million/cu.mm	Indicates anaemia
Haemoglobin (Hb)	12.8 g/dl	14-18 g/dl	Indicates anaemia
Haematocrit (PCV)	37.2 %	42 – 54 %	Indicates anaemia
MCH	32.6 pg	27 – 31 pg	MCH increased due to deficiency in nutrients such as vitB12 Or folic acid
RDW (CV)	14.3 %	11.6 – 14 %	Increased RDW is a sign of anemia.
MICROBIOLOGY			
CRP result	79.74 mg/dl	<5.0 mg/dl	A high CRP level indicates the presence of inflammation in the body.
BIOCHEMISTRY			
Serum. Potassium	3.4mmol/l	3.5-5.1 mmol/l	Low k^+ is due to excessive loss of potassium through the digestive tract, and also due to kidney problem.

The patient's complete blood count [CBC] revealed abnormal RBC, hemoglobin, PCV, MCH, and RDW levels. The patient has abnormal CRP and serum potassium levels (Table 3). On the day of admission, the patient complained of a cough with sputum. Doctor prescribed different medications which includes 1 vial IVIG @20ml/hour slow IV for 5 hours, Tab. Montair - lc [montelukast + levocetirizine] and Tab.Acebrophylline 100 mg P/O OD, On 2nd day of admission patient complained severe body pains, then patient was prescribed with inj.Tramadol 1 Amp IV and general medicines include IVIG, Tab. Montair - lc [montelukast + levocetirizine],Tab.Acebrophylline 100 mg P/O OD, Inj.Pan [pantoprazole] 40 mg IV OD, Inj. Optineuron [THIAMINE(VitB1) (100mg) +PYRIDOXINE(VitB6) (100mcg) + D-PANTHENOL(50mg)] 1 Cap IV BD, On 3rd day of admission patient complained Headache, numbness of limbs, cough with sputum and wheezing and patient was prescribed with Tab.Naxdom [Naproxen and Domperidone] 500mg P/O SOS, and Tab.Dolo 650 mg for headache and NEB. FORACORT[BUDESONIDE and FORMOTEROL] 1 Resp P/N BD, Neb. Duolin [Salbutamol Sulphate] 1 Resp P/N TID, Inj. Methylpred [Methylprednisolone Acetate] 40 mg IV BD for airway inflammation and inj.Piptaz [Piperacillin/Tazobactum]BD, TAB.GABAPENTIN [Neurontin] 300 mg P/O BD were added (Table 4).

Table 4 Drug chart

S. No	Brand name	Generic name	Dose	Route of administration	Frequency
1.	INJ. PAN	PANTOPRAZOLE	40mg	IV	OD
2.	INJ. OPTINEURON	THIAMINE(VitB1) (100mg) +PYRIDOXINE(VitB6) (100mcg) + D-PANTHENOL(50mg)	1cap	IV	BD
3.	T. MONTAIR - LC	MONTELUKAST + LEVOCETIRIZINE	1tab	P/O	HS-OD
4.	T. ACEBROPHYLLINE	ACEBROPHYLLINE	100mg	P/O	OD
5.	TAB. GABAPENTIN	NEURONTIN	300mg	P/O	BD

6.	INJ. PIPTAZ	PIPERACILLIN/TAZOBACTAM	4.5gm	IV	BD
7.	NEB. FORACORT	BUDESONIDE and FORMOTEROL	1 resp	P/N	BD
8.	NEB. DUOLIN	SALBUTAMOL SULPHATE	1 resp	P/N	TID
9.	INJ. METHYLPRED	METHYLPREDNISOLONE ACETATE	40mg	IV	BD
10.	T. NAXDOM	NAPROXEN and DOMPERIDONE	500mg	P/O	SOS

3. Discussion

The Guillain-Barre syndrome (GBS) is a varied condition with an average yearly prevalence of 1.55/100,000 in India, which increases to 4.67/100,000 for those over 75. Males are impacted 1.5 times more frequently than females [6], and the global incidence is 1–2 per one million person-years. [7] Although the exact cause of GBS is yet unknown, an infection is usually followed by GBS. Rarely, after receiving a vaccination, some people have reported having it. Although anybody could be affected, people older than 50 years seem to be at a higher risk. Campylobacter jejuni bacteria, Epstein-Barr virus, cytomegalovirus without any symptoms, the airway system can become infected with the bacteria Mycoplasma pneumonia. Zika virus, though researchers are still looking into this. A single hypothesis: The immune system's response to peripheral nerve injuries is altered by bacterial and viral infections, according to a reliable source. As an outcome, the immune system targets myelin and axons because it believes they are foreign substances. [8] High body temperature, cough, sore throat, and other upper respiratory symptoms are frequently reported prior to the onset of GBS. [9] Milder variants of GBS have been connected to Epstein-Barr virus infection. [10] The usual course of GBS therapy is intravenous immunoglobulin (IVIG) or plasma exchange (PLEX) and numerous randomized controlled trials (RCTs) have shown its comparable long- and short-term advantages against morbidity. [11-14] when treating GBS, oral corticosteroids or injections of methylprednisolone do not improve long-term results or speed up recovery. [15] Patients with GBS who cannot walk 10 meters without assistance (GBS mobility scale score ≥ 3) should start IVIG or PLEX as soon as possible after symptoms appear. [11] If administered within a shorter duration from onset of symptoms, IVIG expedites the process of recovering from GBS just as much as PLEX. Moreover, randomized controlled trials indicate that the IVIG is the one of the most likely to be to be finished than PLEX; probably because it is more convenient for the patient (the overall rates of side events are comparable in both groups). [13] The overall dosage of 2g/kg of IVIG, spread out over 2 or 5 days; however, it is not yet known which length is better. After the initial round of IVIG or PLEX treatment, 10% of patients may experience an episode of stabilization followed by a clinical deterioration; this condition is known as treatment-related fluctuation (TRF). [16] In this condition, Patient have increased MCH and CRP levels and decreased RBC, Hb, PCV, RDW, and serum potassium levels. The pathophysiology of GBS is complicated. Immune system assaults on the body begin. It is true that brain tissue and the immune system are related. Concurrent with the breakdown of myelin, inflammation takes place. A few days after symptoms start to show, these acute indications of inflammation become visible. It appears that nerve conduction is being slowed down or blocked. While the Schwann cells that make myelin in the peripheral parts of the nervous system are destroyed, in only the most extreme cases, the axons survive. Following two to three weeks of demyelination, remyelination starts, and inflammation goes down, and Schwann cells start to multiply, and this condition gets worse. GBS is characterized by a classic pathological characteristic of prominent demyelination and a range of inflammation invasion in the PNS. Although axonal degeneration has been noted by numerous writers, frequently at the advanced stages of demyelinated lesions, these alterations have traditionally been attributed to subsequent Wallerian degeneration. [17] GBS is rare. The majority of adult GBS cases are associated with co-occurring medical disorders. One of the most frequent risk factors for GBS is infection with Campylobacter jejuni, bacteria that causes gastroenteritis, which includes symptoms of nausea, vomiting, and diarrhoea. Infections with the cytomegalovirus and Epstein-Barr virus, as well as the flu, can also cause GBS in people. [18] A potentially fatal condition is Guillain-Barre syndrome. The fastest feasible treatment and observation are necessary for those who have Guillain-Barre syndrome. A tiny number of cases with Guillain-Barre syndrome complications can result in death, even under the best of conditions. Blood clots, infections, respiratory muscle paralysis, and cardiac arrest are a few examples of these outcomes. [19]

4. Conclusion

This case report describes a male patient, age 58, who had presented with sudden onset weakness in both upper and lower limbs, suggesting that he may have Guillain-Barré Syndrome (GBS). After a recent cerebrovascular accident, the clinical examination revealed decreased muscle strength (4/5) consistent with left hemiparesis. Neuroimaging revealed persistent lacunar infarcts and tiny vessel alterations. The multisystem nature of this GBS presentation is highlighted by concurrent abnormalities in the CRP, serum potassium levels, and total blood count. For the best possible care for patients, it is crucial to identify and treat the many clinical symptoms of GBS, taking into account both neurological and

systemic factors. This thorough analysis emphasizes this point. The majority of adult GBS cases are associated with co-occurring medical disorders.

Compliance with ethical standards

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Disclosure of conflict of interest

Authors declare that there is no conflict of Interest.

Statement of ethical approval

It does not need ethical approval because it is a case report.

Statement of informed consent

The privacy rights of human subjects must always be observed.

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