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CLINICAL FEATURES AND TREATMENT MODALITIES OF THE GUILLAIN-BARRE SYNDROME: A CASE REPORT

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Abstract

Guillain Barré syndrome refers to an inflammatory disorder that affects numerous spinal nerve roots (most commonly the anterior ones), often in conjunction with proximal segments of peripheral nerves. Guillain-Barré syndrome (GBS) and its variations are classified as immune-mediated post-infectious neurological conditions. The yearly incidence ranges between 0.5 and 2 cases per 100,000 people. It usually appears in the spring or fall.

The clinical examination results and history may strongly imply GBS. A lumbar puncture (LP) is commonly performed to look for albuminocytologic dissociation. However, this result should not be used to make a diagnosis of GBS; cerebrospinal fluid (CSF) protein levels are still normal in a considerable proportion of patients during the first week of symptoms, particularly in individuals with a Miller-Fisher variation.

We represent a case of A 65-year-old patient who was transported to the UC via ambulance due to bilateral limb weakness.First hospitalization was at university clinic for Neurology Skopje with symptomatology since 3 months ago. The patient tested positive for COVID-19,and his condition cleared up with just minor symptoms. After two weeks, the patient developed weakness in both legs and was unable to get out of bed. The patient eventually lost the ability to move all four limbs and experienced full loss of sensation in them.

According to the findings of the International Guillain-Barré Syndrome Outcome Study, there are regional differences in Guillain-Barré syndrome, including a lack of access to immunotherapy in low-income countries. There is a need to increase access to therapy for all Guillain-Barré syndrome patients, as well as to discover effective disease-modifying medicines that can decrease the degree of nerve destruction.

Key words: Guillain-Barré syndrome,COVID-19,bilateral limb weakness,lumbar puncture

Introduction

This phrase refers to an inflammatory condition that affects several spinal nerve roots (most typically the anterior ones), often with concurrent involvement of the proximal segments of peripheral nerves. Nerve root or nerve cell inflammation is frequently caused by immune-mediated processes, such as an autoallergic radiculitis or neuritis following a previous, perhaps asymptomatic viral or bacterial infection.

The degree of accuracy and major location of symptoms and signs characterize clinical types of polyradiculitis. The most frequent type is Guillain Barré syndrome, the chronic variant (CIDP = chronic inflammatory demyelinating polyradiculoneuropathy) is uncommon, as is localized polyradiculoneuritis, which affects just the cranial nerves or the nerves of the cauda equina.

Acute polyradiculitis is distinguished by quickly rising paresis and, at most, minor sensory abnormalities. In extreme situations, the cranial nerves and autonomic nervous system may be affected.Weakness normally heals on its own in all affected muscles (those that got weak earliest recover the most slowly).

This disease, also known as Guillain-Barré syndrome, can strike at any age. The yearly incidence ranges between 0.5 and 2 cases per 100,000 people. It usually appears in the spring or fall. [1].

Cranial neuropathies affect a tiny percentage of people. Patients with respiratory muscle weakness or lack of airway protecting reflexes may develop autonomic dysfunction or respiratory insufficiency/failure.

Approximately two-thirds of patients indicate an antecedent sickness during the previous three weeks. Symptoms worsen over days to weeks, with most patients reaching their maximum weakening after around 2 weeks. On examination, patients' affected limbs are areflexic or hyporeflexic [2].

Approximately two-thirds of patients would have been unwell a few weeks before the commencement of the illness (typically with an upper respiratory tract infection or diarrhea). Campylobacter jejuni infection (diarrhoea) will be responsible in 30% of cases, CMV infection in 15%, and Epstein-Barr virus, Mycoplasma (respiratory tract infection), or hepatitis E in the remaining instances.

It has been demonstrated that immunological cross-reactions can occur between microbe components and peripheral nervous system components.

The similarity between Campylobacter lipopolysaccharides and gangliosides found in peripheral nerves is a key element here [3].

The clinical examination results and history may strongly imply GBS. A lumbar puncture (LP) is commonly performed to look for albuminocytologic dissociation. However, this result should not be used to make a diagnosis of GBS, cerebrospinal fluid (CSF) protein levels are still normal in a considerable proportion of patients during the first week of symptoms, particularly in individuals with a Miller-Fisher variation.

About 15% of individuals have modest pleocytosis in their CSF; other diagnosis should be considered if the CSF white blood cell count exceeds 50 cells/L. Although MRI of the spine with contrast is not required, it can give further evidence if the MRI indicates enhancement and/or thickness of the thoracolumbar nerve roots and cauda equine. EMG/NCS are also not required for diagnosis and may be normal early on. EMG/NCS findings associated with GBS include delayed distal latencies, conduction block, prolonged or missing F waves or H reflexes, and temporal dispersion of waveforms. EMG/NCS can be done/repeated many weeks after the beginning of symptoms to look for signs of axonal damage vs demyelination for prognosis.

Because of the possibility of dysautonomia, all patients should have repeated pulmonary function tests (FVC and NIF) and cardiovascular surveillance while their symptoms are still progressing [2].

CASE REPORT

A 65-year-old patient was transported to the UC via ambulance due to bilateral limb weakness.First hospitalization was at university clinic for Neurology Skopje with symptomatology since 3 months ago.

The patient tested positive for COVID-19 in May 2023 (for the first time in 2021), and his condition cleared up with just minor symptoms.

After two weeks, the patient developed weakness in both legs and was unable to get out of bed. In the next week, the patient realized that, in addition to the motor weakness of his lower limbs, he also experienced a loss in sensation too, and decreased sensation expanded to both hands, more precisely the palms of his hands.

The patient eventually lost the ability to move all four limbs and experienced full loss of sensation in them.

Due of the patient's incapacity to move, a urine catheter was implanted over the next three months. A few weeks before admission, the symptomatology improved, and the upper limb sensitivity and motor strength was restored, but not completely. Hospitalization is required to perform diagnostic and therapeutic procedures.

It is basically a patient with hypertension,diabetes, on regular therapy with scopryl and insulin therapy.Neurological status: On examination, the patient was awake, aware, contactable, properly oriented with emphatic speech. Cranial nerves had a regular territorial innervation. Plantar-cutaneous reflex - an extensor response was obtained bilaterally.

Recommended therapy: Milgama (*benfotiamine,pyridoxine*) 100mg,Tiogamma (*thioctic acid*) 600mg,Nolpaza (*pantoprazole*) 20mg,Cardiopirin (*acetylsalicylic acid*) 100mg,Ospamox (*amoxicillin*) 1000mg,Fraxiparine (*nadroparin*) 0.4 ml.

Table 1.*Cerebrospinal fluid-lab report* **RBC** (reb blood cells normal ranges 2,70-4,10mmol\l),**WBC** (white blood cells normal ranges 12,00 10^6\l),**Mononuclear\Polymorphonuclear cells** (normal CSF cell counts are below 5 cells\ul consisting of mononuclear WBC with majority of lymphocytes and fewer monocytes,**IgG** normal ranges 3,00-30,00 10^3,**Chlorides** normal ranges (110,00-125,00 mmol\l) .

CSF	**Appearance**	Glucose	RBC	WBC	Mononuclear cells	Polymoropho-nuclear cells	IgG	Chlorides
	Bloody\clear	3,70	0,00	12,00	12,00	0,00	157,50	127,00

Table 2. *CSF protein profile and hemato-liquor barrier function,*the results shows dysfunction of blood brain barrier accompanied by intense intrathecal IgG synthesis and the **type of electrophoregram** is *transudative-gammaglobulin.*

	Results	***Normal Ranges***
Total proteins (g\L)	1,25	0.15-0.45
Albumins (mg\L)	642	50-250
IgG (mg\L)	157,5	3-30
Albumin-coefficient (10^3)	18,9	1.8-7.4
IgG-index (10^3)	0,76	< 0.7
IgG synthesis in CNS (mg\24h)	33,6	<0.5

Electromyography with neurography

Spontaneous denervation activity from the type of positive sharp waves and fibrillation potentials was obtained from m. tibialis ant dex I m.opp-policis dext.Neurographic findings: in the examined nerves, the right m. tibialis anterior dexter and m. opp. pollicis. dexter were absent.

When there was muscular activity in the examined muscles, indicated in the distal parts of the upper and lower muscle groups, on the right, a reduction of the neuropathic sample of severe degree was obtained.

EMNG - the findings indicate a generalized neurogenic lesion of the upper and lower extremities on the right side of a moderately-severe degree, especially in the distal muscle group of a severe degree, which according to the registered changes is *demyelination with subsequent axonal lesions.*

Evoked potential test was performed and:

In the median nerve there was a defect in conduction along the peripheral and central somatosensory pathways was obtained,whereas in the tibial nerve we found a severe defect in conduction along the peripheral and central somatosensory pathways. Changes were more expressed to the right side.

Discussion

Guillain-Barré syndrome (GBS) and its variations are classified as immune-mediated post-infectious neurological conditions. Animal models imply that molecular mimicry plays an important function. A lipooligosaccharide found in the outer membrane of Campylobacter jejuni bacteria is comparable to gangliosides, which are components of the peripheral nerves.

As a result, an immune response activated to combat infection may result in a cross-reaction on host nerves [4].

The diagnosis is based on symptoms and neurological examination results, such as decreased or absent deep-tendon reflexes. A lumbar puncture or electromyography (EMG) may be performed for informational purposes, but should not be used to postpone treatment. Anyone suspected of having GBS should be regularly examined for respiratory problems [5].

In GBS, antiganglioside antibodies are frequently examined, although their absence does not rule out the diagnosis. There is mounting evidence that they are pathogenic.Gangliosides are found throughout the neurological system and contribute to maintain cell membrane structure: Antiganglioside (typically GM1) antibodies are present in 25% of individuals with the acute inflammatory demyelinating (AIDP) GBS type.Anti-GQ1b antibodies are present in 95% of Miller-Fisher syndrome patients.Anti-GM1 antibodies are present in 50% of individuals with axonal variations (acute motor axonal neuropathy) [6].

A pandemic hit the whole planet with the emergence of coronavirus disease 2019 (COVID-19). The short-, medium-, and long-term consequences of this disease are continually being found as time passes and information of its dynamics and viral transmission grows. During this time, researchers discovered that several forms of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) might impair the brain system. A number of recent research and case reports have suggested a link between COVID-19 and Guillain-Barré syndrome (GBS) [7] .

Guillain-Barré syndrome (GBS) is considered a clinical diagnostic hence, in most cases, a diagnosis may be made confidently at the bedside. Ancillary testing can be beneficial in uncommon situations or rare subtypes.Nerve conduction studies (NCS) use technology to differentiate between demyelinating and axonal neuropathy [8].

IVIg (intravenous immunoglobulin) is usually the therapy of choice since it is easier to give and more readily accessible than plasma exchange. Aside from IVIg and plasma exchange, no other procedures or medicines have been shown to be useful in treating GBS. Although corticosteroids should be beneficial in reducing inflammation and thus disease progression in GBS, eight randomized controlled trials on the efficacy of corticosteroids for GBS found no significant benefit, and treatment with oral corticosteroids was even shown to have a negative effect on outcome.

Furthermore, plasma exchange following IVIg has not been more beneficial than either treatment alone, and there is inadequate data to support the efficacy of adding intravenous methylprednisolone to IVIg treatment in IVIg-treated patients. In individuals with GBS who have an ongoing infection, antimicrobial or antiviral therapy might be explored; however, previous infections normally cure before the onset of disability [9] .

Although they are the most often used therapies, many patients may not respond well to them, resulting in limited movement, discomfort, and exhaustion, and in rare cases, death, thus these treatments should be delivered as soon as possible to be more successful and avoid additional nerve damage [10] .

According to the findings of the International Guillain-Barré Syndrome Outcome Study, there are regional differences in Guillain-Barré syndrome, including a lack of access to immunotherapy in low-income countries. There is a need to increase access to therapy for all Guillain-Barré syndrome patients, as well as to discover effective disease-modifying medicines that can decrease the degree of nerve destruction [11].

Conclusion

Although the etiology of Guillain-Barré syndrome is uncertain, most instances develop after a respiratory or gastrointestinal illness.There is a need to increase access to therapy for all Guillain-Barré syndrome patients, as well as to discover effective therapeutic medication that can decrease the degree of nerve destruction.

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