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Clinical and Epidemiological Study on Inflammatory Polyneuropathy (Guillain-Barré Syndrome) Among Sudanese Cases

Mohamed N. Mohamed, Mohamed Salah M. Magzoub, Amani A. Osman, Nazar M. Abdalla

Department of Medical Microbiology & Parasitology, College of Medicine, King Khalid University, Abha, Aseer Kingdom of Saudi Arabia

Email address
uofgnazar@gmail.com (Nazar M. Abdalla)

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Abstract
Aim of the study: This study aiming to identify the acute inflammatory polyneuropathy; Guillain-Barré syndrome (GBS) and its pattern of Neurophysiologic disorders among Sudanese population. *Material and Methods:* Study design: This study is a retrospective analytical hospital base study. *Study area:* The study has been conducted in Khartoum neurological clinic. Population and sampling: Study population was all Sudanese patients suffering from Gillian Barry Syndrome (GBS) attended neurology clinic, during the period from January 2011 to December 2014. The sample size was 91 subjects. Neurophysiological data (Nerve Conduction Study, Electromyelogram and Evoked Potential tests Results) were considered in collection of data. The Data has been processed and analyzed using SPSS 20(statistical package for social sciences). *Results:* A review of 3years records of Khartoum neurology clinic revel 569 patients of different neurological problems, there were 91 patients with GBS, 88 were GBS and 3 were primary axonal loss. *Conclusion:* Acute axonal loss represent the least manifestation among GBS Sudanese patients were Mononeuropathies according to our study.

1. Introduction

Neuropathy is not a term that is reserved for a single disease – instead, the problem is seen with a number of different underlying medical conditions. It can also exist without the cause being possible to diagnose, when doctors called it "idiopathic. The term "neuropathy" simply means nerve damage - but it generally refers, to peripheral neuropathy - that is, the problem as it affects nerves outside of the central nervous system.

There are rarer neuropathies that specifically affect, the nerves leading directly from the brain. Cranial neuropathies include the one that affects the nerves in the eye which called retinopathy (it is known as microvascular cranial nerve palsy, happen as a complication of diabetes. Another example affects the face, known as Bell's Palsy) [1].

There are three main types of nerve can be involved in peripheral neuropathy Autonomic nerves, Motor nerves and Sensory nerves.

Autonomic nerves also called involuntary" nerve which regulate the automatic functions of the body - for example, control of the gut or the urinary bladder, heart rate, blood pressure, sweating, and so on. Motor nerves control the muscles of the body and are

under our conscious control. Sensory nerves pass sensations from a part of the body to the brain, including information about old, heat and pain. Peripheral neuropathy can result from physical trauma, repetitive injury, infection, metabolic problems and exposure to toxins and some drugs can all lead to neuropathy.

The commonest diseases that cause neuropathy is Diabetes, other diseases are Chronic liver disease, Chronic kidney disease, HIV infection and AIDS, Long-term excessive alcohol intake, Vitamin B deficiency and other nutritional deficiency, Cancer - lymphoma or multiple myeloma, Lyme disease, a tick-borne bacterial infection, Charcot-Marie-Tooth disease, a genetic cause of nerve damage, particularly in the lower limbs, Guillain-Barré syndrome (GBS).

GBS is considered to be an autoimmune disease triggered by a preceding bacterial or viral infection. Campylobacter jejuni, cytomegalovirus, Epstein-Barr virus and Mycoplasma pneumoniae are commonly identified antecedent pathogens. In the acute motor axonal neuropathy (AMAN) form of GBS, the infecting organisms probably share homologous epitopes to a component of the peripheral nerves (molecular mimicry) and, therefore, the immune responses cross-react with the nerves causing axonal degeneration; the target molecules in AMAN are likely to be gangliosides GM1, GM1b, GD1a and GalNAc-GD1a expressed on the motor axolemma. In the acute inflammatory demyelinating polyneuropathy (AIDP) form, immune system reactions against target epitopes in Schwann cells or myelin result in demyelination; however, the exact target molecules in the case of AIDP have not yet been identified. AIDP is by far the most common form of GBS in Europe and North America, whereas AMAN occurs more frequently in east Asia (China and Japan).

Electromyography (EMG) is used to diagnose muscle weakness caused by neuropathy, For the EMG test, a very thin needle electrode is inserted into the muscle through the skin. This picks up the electrical activity of muscles, which is shown on a monitor or heard through a speaker. Neuropathy may show a reduced response due to nerve damage. [2]. The EMG test is usually accompanied by a nerve conduction test. Nerve damage results in reduced conduction, and this electrical test measures the speed that impulses - messages - travel through a nerve. The diagnostic equipment for this involves placing electrode patches over the skin.[3]

As neuropathy is common condition all over the world but there is no enough information and researches done in Sudan. This study aimed to know about pattern of neuropathy in Sudan.

Aim of the study: This study aiming to identify the of acute inflammatory polyneuropathy GBS and its pattern of Neurophysiologic disorders among Sudanese population.

2. Material and Methods

Study design: This study is a retrospective analytical study

Study area: The study has been conducted in Khartoum neurological clinic, Khartoum state, Sudan.

Population and sampling: Study population was all Sudanese patients suffering from Gillian Barry Syndrome (GBS) attended neurology clinic, during the period from January 2011 to December 2014. The sample size was 91 subjects. Neurophysiological data (Nerve Conduction Study, Electromyelogram and Evoked Potential tests Results) were considered in collection of data. The Data has been processed and analyzed using SPSS 20(statistical package for social sciences).

Nerve conduction study:

Nerve conduction study (NCS) is a test to see how fast electrical signals move through a nerve. The test is performed by using patches called surface electrodes are placed on the skin over nerves at various locations. Each patch gives off a very mild electrical impulse, which stimulates the nerve. The nerve's resulting electrical activity is recorded by the other electrodes. The distance between electrodes and the time it takes for electrical impulses to travel between electrodes are used to determine the speed of the nerve signals. An NCS test shows the condition of the best surviving nerve fibers, so in some cases the results may be normal even if there is nerve damage. Patients should tell their doctor if they have a cardiac defibrillator or pacemaker (special steps will need to be taken before the test if he had one of these devices). During the test patient will feel the impulse it is like an electric shock. Test is almost always performed during the same visit as an EMG. [4]

Electromyography (EMG)**:**(recording from needles placed into the muscles) is often done at the same time as NCV test performed. [5]. To Prepare for this test: Patient must stay at a normal body temperature (Being too cold slows nerve conduction). Also avoid using any creams or lotions on the day of the test. If the patient taking blood thinners or anticoagulants, inform the person performing the test before it is done. In EMG test insertion of a very thin needle electrode through the skin into the muscle The electrode on the needle picks up the electrical activity given off by your muscles. This activity appears on a nearby monitor, and may be heard through a speaker. After placement of the electrodes, patient may be asked to contract the muscle. For example, by bending his arm. The electrical activity seen on the monitor provides information about his muscle's ability to respond when the nerves to his muscles are stimulated. This process can be uncomfortable during the test. The Risks of this test are bleeding (minimal) and infection at the electrode sites.

3. Results

A review of 3 years records of Khartoum neurology clinic revel 569 patients of different neurological problems, there were 91 patients with GBS, 88 were GBS and 3 were primary axonal loss, fig. 1.

Gender distribution:

Concerning gender distribution two third of the patients were male, all patients with primary axonal loss were male. The result shown in fig 2

Age Distribution

About age distribution of our study group, there were 6

age groups, The mean age of our study group is 38.74 years, the least number of patient fall in the age group less than 5years old and almost 60% of our sample (56) found in age group 25 to 50 and 50 to 70 years old. Other distribution shown in table (1)

Different Pattern of electrophysiological tests performed to all patients were shown in table (2). The commonest electrophysiological findings for patients with GBS was demyelination with secondary axonal degeneration and temporal dispersion with asymmetry (66case out of 88). While there was no severe sensorimotor axonal loss, but this severe sensorimotor axonal loss found in one third of patients with Primary axonal loss GBS.

Unfortunately, more than ninety percent of patients referred to the clinic without a documented clinical assessment, conversely, the remaining ten percent of the cases came with weakness and wasting of muscles. Different types of neuro-electrophysiological tests were done, where 67.4 % of the subjects performed the nerve conduction study only, On the other hand, 26.1% did electromyelography together with nerve conduction study test.

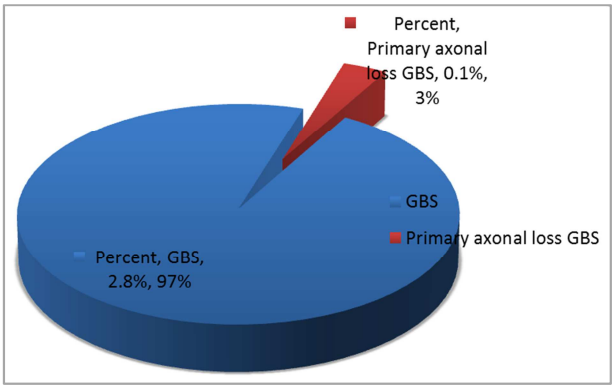

Fig. 1. *Classification of GBS.*

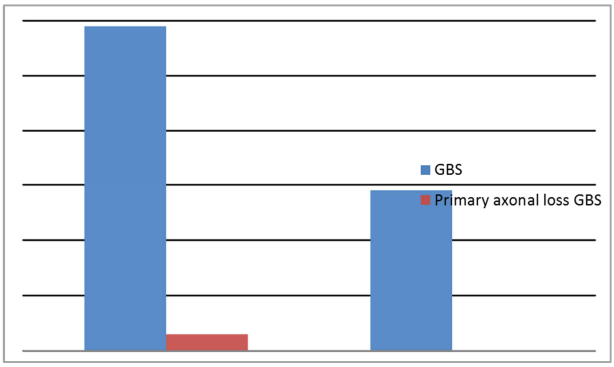

Fig. 2. *Gender distribution.*

Table 1. *Age Distribution.*

Age	GBS	Primary axonal loss GBS	Total
less than 5 years old	2	0	2
(5 - 15) years old	7	0	7
(15 - 25) years old	15	1	16
(25 - 50) years old	30	0	30
(50 - 70) years old	25	1	26
more than 70 years old	9	1	10
Total	88	3	91

Table 2. *The electrophysilogical findings for patients with GBS.*

	GBS	Primary axonal loss GBS	Total
Normal	1	0	1
Demyelination	1	0	1
severe sensorimotor axonal loss	0	1	1
Demyelination with secondry axonal degeneration and temporal dispersion with asymmetry	66	0	66
severe sensorimotor polyneuropathy primarly axonal and demyelination with temporal dispresion and sural sparing	11	1	12
sural sparing and temporal dispression with conduction block	3	1	4
sensorimotor polyradiculoneuropathy primary axonal with demyelination and conduction block with prolonged late responses	2	0	2
Conduction Block	1	0	1
Demyelination polyradiculoneuropathy and asymmetry	1	0	1
sensorimotor polyradiculoneuropathy demyelination secondary axonal and conduction block with prolonged late responses	2	0	2
Total	88	3	91

4. Discussion

Several studies were performed aiming to identify the pattern and variable modalities of neuropathy including the inflammatory neuropathy; Guillain-Barré syndrome (GBS).

Our study showed that acute inflammatory demyelinating polyneuropathy (Guillain-Barré syndrome) represented 16.21 % relatively similar to a study done in Mexico for a period of seven years where it represented 19.7 %.[6]

There were only two subtypes of Guillain-Barré syndrome in our study, the axonal type and AMAN type. Interestingly, we found one case of Bickerstaff's syndrome and it is presumed to be a sign of overlapping Guillain-Barre syndrome (GBS). Similar to other studies[7].

According to motor neuron diseases we only found 49 patients while study done in the United Kingdom in the period 1990-2005, 830 new cases of MND were identified[8]

Guillain-Barré syndrome (GBS) is clinically defined as an acute peripheral neuropathy causing limb weakness that progresses over a time period of days or, at the most, up to 4 weeks. GBS occurs throughout the world with a median annual incidence of 1.3 cases per population of 100 000, with men being more frequently affected than women. The prognosis of GBS is generally favorable, but it is a serious disease with a mortality of approximately 10% and approximately 20% of patients are left with severe disability. Treatment of GBS is subdivided into: (i) the management of severely paralysed patients with intensive care and ventilatory support; and (ii) specific immunomodulating treatments that shorten the progressive course of GBS, presumably by limiting nerve damage. High-dose intravenous immunoglobulin (IVIg) therapy and plasma exchange aid

more rapid resolution of the disease. The predominant mechanisms by which IVIg therapy exerts its action appear to be a combined effect of complement inactivation, neutralisation of idiotypic antibodies, cytokine inhibition and saturation of Fc receptors on macrophages. Corticosteroids alone do not alter the outcome of GBS. [9]

A study on Guillain-Barré syndrome clarified that, Guillain-Barré syndrome consists of at least four subtypes of acute peripheral neuropathy. Major advances have been made in understanding the mechanisms of some of the subtypes. The histological appearance of the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype resembles experimental autoimmune neuritis, which is predominantly caused by T cells directed against peptides from the myelin proteins P0, P2, and PMP22. The role of T-cell-mediated immunity in AIDP remains unclear and there is evidence for the involvement of antibodies and complement. Strong evidence now exists that axonal subtypes of Guillain-Barré syndrome, acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN), are caused by antibodies to gangliosides on the axolemma that target macrophages to invade the axon at the node of Ranvier. About a quarter of patients with Guillain-Barré syndrome have had a recent Campylobacter jejuni infection, and axonal forms of the disease are especially common in these people. The lipo-oligosaccharide from the C jejuni bacterial wall contains ganglioside-like structures and its injection into rabbits induces a neuropathy that resembles acute motor axonal neuropathy. Antibodies to GM1, GM1b, GD1a, and GalNac-GD1a are in particular implicated in acute motor axonal neuropathy and, with the exception of GalNacGD1a, in acute motor and sensory axonal neuropathy. The Fisher's syndrome subtype is especially associated with antibodies to GQ1b, and similar cross-reactivity with ganglioside structures in the wall of C jejuni has been discovered. Anti-GQ1b antibodies have been shown to damage the motor nerve terminal in vitro by a complement-mediated mechanism. Results of international randomized trials have shown equivalent efficacy of both plasma exchange and intravenous immunoglobulin, but not corticosteroids, in hastening recovery from Guillain-Barré syndrome. Further research is needed to discover treatments to prevent 20% of patients from being left with persistent and significant disability.[10]

Acute motor conduction block neuropathy pattern occurring in the course of an acute inflammatory demyelinating polyradiculoneuropathy. Was studied by Fernandez-Torre, J. L., J. Berciano, et al. in 2008. It described the case of a young woman with the diagnosis of acute inflammatory demyelinating polyradiculoneuropathy (AIDP), who during the course of the disease developed an electrophysiologic pattern of acute motor conduction block neuropathy (AMCBN). Electrophysiologic techniques including needle EMG, standard motor and sensory nerve conductions studies, and somatosensory evoked potentials were carried out over the four months after symptom onset. The results of four neurophysiological studies, performed on Days 14, 26, 35 and 125 after symptomatic onset were reported. All immunological determinations including antiganglioside antibodies (GM1, GM2, GM3, asialoGM1, GD1a, GD1b, GD3, GQ1b and GT1b) were negative. The patient had a favorable evolution following treatment with intravenous immunoglobulins (IVIg). They concluded that the electrophysiologic hallmark of AMCBN may occur in the course of AIDP. Serial investigation including proximal, intermediate and distal segments of all nerves from upper and lower limbs is essential for its detection. [11]

Neuropathy is a poorly studied subject in Sudanese medical literature, Few studies were conducted in Sudan. Neuropathy in Africa usually occur as complication of diabetes. Diabetes becoming a major chronic disease burden all over the world. [12-20]

5. Conclusion

Wide ranges of neurological problems were diagnosed via neuro electrophysiological tests.

Acute axonal loss represent the least manifestation among GBS Sudanese patients were Mononeuropathies according to our study. The commonest electrophysiological findings for patients with GBS was Demyelination with secondary axonal degeneration and temporal dispersion with asymmetry

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References

[1] Baltimore M. Multiple cranial neuropathies. Johns Hopkins Library, The Johns Hopkins University, The Johns Hopkins Hospital, and Johns Hopkins Health System. hopkinsmedicine.org. Information published online. 2015.

[2] National Institutes of Health.. Electromyography. Medline Plus, US National Library of Medicine. Published online. 29th September 2013. 2013.

[3] National Institutes of Health.. Nerve conduction velocity,. Medline Plus, US National Library of Medicine. Published online. 29th September 2013. 2013.

[4] Katirji B. Clinical neurophysiology: clinical electromyography. In: Daroff RB FG, Jankovic J, Mazziotta JC,. eds. Bradley's Neurology in Clinical Practice. Philadelphia, PA: Saunders Elsevier;. 2012; 6: chap 32B.

[5] Griggs RC JR, Aminoff MJ.. Approach to the patient with neurologic disease. In: Goldman L, Schafer AI, eds.Goldman's Cecil Medicine. U. S. National Library of Medicine. 2013.

[6] Velasquez-Perez L, Ramirez-Crescencio MA. [Neurological disease surveillance and mandatory reporting: a trend and outcome of the national neurological institute of health in mexico city from 2005 to 2011]. Gac Med Mex. Nov-Dec 2014; 150(6): 540-551.

[7] Rho YI. Overlapping Guillain-Barre syndrome and Bickerstaff's brainstem encephalitis associated with Epstein Barr virus. Korean J Pediatr. Oct 2014; 57(10): 457-460.

[8] Alonso A, Logroscino G, Jick SS, Hernan MA. Incidence and lifetime risk of motor neuron disease in the United Kingdom: a population-based study. Eur J Neurol. Jun 2009; 16(6): 745-751.

[9] Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. Drugs.. 2004; 64(6): 597-610.

[10] Hughes RA1 CD. Guillain-Barré syndrome. Lancet. 2005 Nov 5 2005 366(9497): 1653-1666.

[11] Fernandez-Torre JL, Berciano J, Garcia-Gorostiaga I, Calleja J. Acute motor conduction block neuropathy pattern occurring in the course of an acute inflammatory demyelinating polyradiculoneuropathy. Neurophysiol Clin. Aug 2008; 38(4): 211-216.

[12] Agyemang* MBaC. Prevalence and complications of diabetes mellitus in Northern Africa, a systematic review. BMC Public Health 2013; 13: 387.

[13] Wild S1 RG, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care.. 2004 May 2004; 27(5): 1047-1053.

[14] Victoria Hall RWT, Ole Henriksen, Nicolai Lohse. Diabetes in Sub Saharan Africa 1999-2011: Epidemiology and public health implications. a systematic review. BMC Public Health 2011; 11: 564.

[15] Elbagir MN EM, Elmahadi EM, Kadam IM, Berne C. A population-based study of the prevalence of diabetes and impaired glucose tolerance in adults in northern Sudan. Diabetes Care 1996 Oct, 1996; 19(10): 1126-1128.

[16] Ghannem H1 LK, Ben Abdelaziz A, Mtiraoui A, Hadj Fredj A, Marzouki M. [Risk factors in cardiovascular diseases in a semi-urban community of the Tunisian Sahel].[Article in French]. Rev Epidemiol Sante Publique.. 1992; 40(2): 108-112.

[17] Bouguerra R AH, Salem LB, Rayana CB, Atti JE, Gaigi S, Slama CB, Zouari B, Alberti K:, . The global diabetes pandemic: the Tunisian experience. Eur J Clin Nutr 2007 Feb, 2007; 61(2): 160-165.

[18] Herman WH AR, Engelgau MM, Thompson TJ, Ali MA, Sous ES, Hegazy M, Badran A, Kenny SJ, Gunter EW, Malarcher AM, Brechner RJ, Wetterhall SF, DeStefano F, Smith PJ, Habib M, Abd El Shakour S, Ibrahim AS, El Behairy EM,. Diabetes Mellitus in Egypt:Diabetes Mellitus in Egypt: glycaemic control and microvascular and neuropathic complications. Diabet Med. 1998; 15(12): 1045-1051.

[19] Saunderson P, Gebre S, Desta K, Byass P, Lockwood DN. The pattern of leprosy-related neuropathy in the AMFES patients in Ethiopia: definitions, incidence, risk factors and outcome. Lepr Rev. Sep 2000; 71(3): 285-308.

[20] Haimanot RT, Abdulkadir J. Neuropathy in Ethiopian diabetics: a correlation of clinical and nerve conduction studies. Trop Geogr Med. Mar 1985; 37(1): 62-68.