European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 09, Issue 03, 2022

Original Research Paper

Electrophysiological study of Landry Guillain Barre syndrome at a Tertiary care hospital

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Abstract

A reasonable description of what we now call Guillain-Barre' Syndrome (GBS) was offered in 1892 by Osler. The critical features of the illness were not fully synthesized until after the advent of diagnostic lumbar puncture near the end of this century (Osler, 1892). In 1916, Guillain, G and Barre' JA, then French army neurologists and Strohl, Al from England, simultaneously published reports. All adult patients, diagnosed as Guillain-Barre Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study. Data collection was done according to the proforma attached. In retrospective cases, the information was collected from the patient's records. In the prospective group, a detailed history was taken, all patients were examined and then the data collected. Nerve conduction velocities (NCV) were studied in median, ulnar, common peroneal and posterior tibial nerves. Abnormal nerve conduction velocities were found in 46 (98%) of total. Distal latency was prolonged in 47 (100%) patients in the present study.

Keywords: Guillain-Barre 'syndrome, nerve conduction velocities, electrophysiological study

Introduction

Acute ascending paralytic illnesses have been recognised for centuries. The earliest report was by Landry in 1859 who described the presenting symptoms, course and outcome of this disease in 10 patients. His findings were those of a progressive paralysis beginning in the distal extremities, preceded by paresthesia and transitory muscle cramps. Two of his ten patients succumbed to respiratory failure. In those who recovered, the process was generally one of rapid recession in a descending pattern [1].

A reasonable description of what we now call Guilla in-Barre' Synd Rome (GBS) was offered in 1892 by Osler. The critical features of the illness were not fully synthesized until after the advent of diagnostic lumbar puncture near the end of this century (Osler, 1892). In 1916, Guillain, G and Barre' JA, then French army neurologists and Strohl, Al from England, simultaneously published reports. Their title can he translated as follows: "Concerning a syndrome of radiculoneuritis with increased albumin in the cerebrospinal fluid without

ISSN 2515-8260 Volume 09, Issue 03, 2022

cellular reaction: remarks on its clinical characteristics and description of tendon reflexes" [2]. Later in 1918 came the controversy when Bradford, Bashford and Wilson reported 30 patients with disorder in a report entitled 'Acute infective polyneuritis' (Bradford *et al*.). They claimed to have isolated an organism transmissible into other primates. However, their claim of infectivity was retracted the following year. An allergic basis for this disease was put forward sporadically (Bannwarth; Furtado; Asbury, 1990) [3, 4].

Methodology

All adult patients, diagnosed as Guillain-Barre. 'Syndrome, who strictly complied with the NINCDS criteria, admitted to medical college hospital were included in this study. This includes a retrospective study of four years and prospective study of one year. Ninety seven patients entered the study.

Data collection was done according to the proforma attached. In retrospective cases, the information was collected from the patient's records. In the prospective group, a detailed history was taken, all patients were examined and then the data collected.

All the Patients underwent lumbar puncture on the day of admission and the cerebrospinal fluid was sent for the cell count, culture, protein content and sugar analysis. Each one of them had a complete haemogram and a urine examination.

Urine was also examined for porphobilinogen. Serum electrolytes and serum calcium levels were estimated an admission to rule out hypokalemic paralysis and hypoglycemic states. Liver function tests and HBsAg and HIV tests were done on all the patients. A throat swab was taken in patients with sore throat to rule out diphtheria.

Results

Table 1: Nerve Conduction Velocities

NCV m/s	Median motor	Ulnar	Common peroneal	Post tibial
25-30	8	10	20	24
31-35	17	16	15	13
36-40	14	11	9	8
41-45	6	6	2	1
46-50	2	3	1	1
More than 50	0	1	0	0

Table 2: Distal Latency response

Nerve studied	Prolonged	Not prolonged
Median	47	0
Ulnar	47	0
Common peroneal	47	0
Post tibial	47	0

Table 3: Conduction block

Nerve studied	No. of patents studied	No. of patients with conduction block
Median	25	23
Ulnar	25	23
Common peroneal	25	23
Post tibial	25	23

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Table 4: Needle EMG

Abnormal	Present	Absent
Fibrillations	9	38
Positive sharp waves	8	39
Fasciculation	1	36

Table 5: Results of Electrophysiological study

Type of neuropathy	No. of patients	Percentage
Demyelinating	38	79.8
Mixed	5	10.5
Axonal	4	8.4

Discussion

Forty seven (48.4%) patients had electrophysiological studies. Majority of them were done during the period of recovery. One or more parameters were abnormal in all the patients.
Nerve conduction velocities (NCV) were studied in median, ulnar, common peroneal and posterior tibial nerves. Abnormal nerve conduction velocities were found in 46 (98%) of total patients in this study as compared to 73% in the study by Kur *et al*. and 24% by Cornblath *et al*. [5]
Distal latency was prolonged in 47 (100%) patients in the present study against 74% in the study by Ka11r and 40% in Cornblath et all group [6, 7].
F wave latency was prolonged in 60% of patients, absent in 18.9% of patients and normal in 18.9% of patients in median nerve. In ulnar nerve, F waves were prolonged in 52% of patients, absent in 27.3% and normal in 18.9% of patients. In the common nerve, 31% had prolonged, 51.2% had absent and 17.04% had normal F waves. In posterior tibial nerve, 48% had prolonged, 52% had absent F waves.

H reflex: On right side, 63% patients had absent 35% had normal H reflex. On left side, 67.2% had absent and 31 % had normal H reflex. 23 out of 25 patients in whom H reflex was studied, it was abnormal [8].
Abnormal sensory nerve conduction was seen in (R) median sensory (62%), (R) ulnar sensory (63%) and Sural Sensonry (67.5%) nerves.
Fibrillations were observed in 9 (19%) patients and absent in 38 (81%) of patients. Positive sharp waves were present in 39 (83.07%) patients and absent in 8 (16.9%) patients.

Conclusion

- Abnormal nerve conduction velocities were found in 46 (98%) of total patients.
- F wave latency was prolonged in 60% of patients, absent in 18.9% of patients and normal in 18.9% of patients in median nerve.
- Fibrillations were observed in 9 (19%) patients and absent in 38 (81%) of patients.

References

1. Markland LO, Riley HD. The Guillain-Barre' Syndrome in childhood. Clin Pediatr (Phila). 1967;6:162-170. In McKhan GM. Guillain-Barre' Syndrome-clinical and Therapeutic Observations. Ann Neurol. 1990;7:S13-S16.
2. Panosian MS, Quafela VC. Guilla in-Barre 'Syndrome prP.sPring as acute bilateraloe} cordpnralysi R. Otnlnryngol Head Neck Surg. 1993;108:17-173.
3. Kanda T, Hayash IH, Tanabe H, *et al*. A fulminant caRd of Guillain-Barre' Syndrome-

topographic and figure size related analysis of demyelination Engrenages. J Neurol. Neurosurg Psychiatry. 1989;52:587-588.

4. Kaur U, Chopra JS, Prabhaka RS, *et al*. Guillain-Brre' Syndrome. A clinical, electrophysiological and i or.hP. Medical study. Acta Neurol. Scand. 1986;73:394-402.
5. Kelly JJ. The electrodiagnostic findings in the peripheral neuropathy associated with monoclonal gammopathy. Muscle Nerve. 1983;6:504-509.
6. Chopra JS, Kaur U, Prabhaka RS, *et al*. Guillain-Barre' Syndrome. A clinical electrophysiological and biochemical study: Acta Neurol. Scand. 1986;73(4):394-402.
7. Dowling PC, Cook SD. Role of infection in Guillain-Barre' Syndrome Laboratory confirmation of herpes virus in 41 cases. Ann Neurol. 1981;9:44-55.
8. Gratta N CEH, Berman P. Chlamydial infection as a possible aetiological factor in the Guillain-Barre' Syndrome. Post-grad Med J. 1982;58:776-777.