Section: General Medicine

Original Research Article

AN OVERVIEW OF GUILLAIN-BARRE SYNDROME WITH REFERENCE TO CLINICAL FEATURES AND PROGNOSTIC OUTCOME.

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ABSTRACT

Background: Guillain-Barré Syndrome (GBS) is the most common acquired demyelinating disorder of the peripheral nervous system worldwide. It is an acute, immune-mediated polyradiculoneuropathy with a wide spectrum of clinical manifestations ranging from mild weakness to severe paralysis and respiratory failure. Early recognition and appropriate management are essential to reduce morbidity and mortality.

Materials and Methods: A prospective observational study was conducted at Government Medical College and Hospital, Nizamabad, from October 2012 to September 2014. Fifty patients fulfilling the diagnostic criteria for GBS were included. Detailed clinical evaluation, neurological examination, respiratory assessment, routine laboratory investigations, cerebrospinal fluid analysis, and electrodiagnostic studies were performed. Patients with alternative causes of acute flaccid paralysis and those with disease progression exceeding four weeks were excluded.

Results: Among the 50 patients studied, 30 (60%) were males and 20 (40%) were females, indicating a male predominance. The majority of patients (52%) were below 40 years of age. Antecedent events were identified in 30% of cases, with upper respiratory tract infection (14%) being the most common, followed by gastrointestinal infection (10%). Clinically, 84% of patients presented with weakness involving both upper and lower limbs. Neck muscle weakness was observed in 30%, bulbar weakness in 16%, facial nerve involvement in 14%, sensory symptoms in 30%, and ocular muscle involvement in 4%. Respiratory muscle weakness occurred in 30% of patients, while 10% required mechanical ventilation. Autonomic dysfunction was noted in 40% of cases. Elevated CSF protein levels and severe abnormalities on electrodiagnostic studies were associated with delayed recovery and poorer outcomes. Conduction block was associated with slower recovery compared to isolated slowing of motor nerve conduction velocity. The overall mortality rate was 10%.

Conclusion: Guillain-Barré Syndrome predominantly affects males and commonly occurs in younger adults. Most patients recover without significant disability; however, respiratory involvement, bulbar weakness, autonomic dysfunction, advanced age, and associated comorbidities such as diabetes mellitus and ischemic heart disease are linked to poorer outcomes. Electrodiagnostic studies and CSF analysis are valuable tools for assessing disease severity and predicting prognosis. Early diagnosis and timely management are crucial for improving clinical outcomes in patients with Guillain-Barré Syndrome.

Keywords: Guillain-Barré Syndrome, Acute Inflammatory Demyelinating Polyneuropathy, Electrodiagnostic Studies, Cerebrospinal Fluid Analysis, Autonomic Dysfunction, Prognosis, Respiratory Failure.

INTRODUCTION

The most prevalent cause of acquired demyelinating disorders affecting the peripheral nerve system worldwide is Guillain-Barre Syndrome. It is a range of diseases with different causes that share a pathological mechanism. People of all ages are affected by this non-seasonal sickness. Guillain-Barre Syndrome can range in severity from modest weakness to complete paralysis and respiratory failure, which can occasionally be fatal. These patients may be spared death and severe morbidity if pathology, clinical presentation, appropriate investigations, and therapies are properly understood.[1-5]

The majority of Telangana's rural population in Nizamabad, is served by Government Medical College and Hospital. It provides medical care to people of all ages, all religions, and all economic backgrounds. Conducting research at this university is more ideal. Guillain-Barre Syndrome is mostly diagnosed by clinical examination, with the help of tests such as electrodiagnostic examinations and cerebrospinal fluid analysis.[6-10]

Aims of the study

1. To assess Guillain-Barre Syndrome kinds and antecedent events
2. To examine the illness's clinical characteristics and temporal profile
3. To evaluate the degree of sickness using electrodiagnostic tests and cerebrospinal fluid analysis
4. To use electrodiagnostic investigations to evaluate the prognostic outcome.

MATERIALS AND METHODS

Guillain-Barre Syndrome is a monophasic sickness that frequently resolves on its own. Clinical history, a thorough neurological examination, regular tests, and specialised tests such cerebrospinal fluid analysis and electrodiagnostic studies served as the foundation for the initial evaluation.

Inclusion Criteria

1. Any patient admitted with features suggestive of flaccid progressive weakness affecting all the four limbs were included
2. Any patient admitted with progression of weakness of less than 4 weeks duration were included.
3. Any patient admitted with reduced or absent deep tendon reflexes were included

Exclusion Criteria

1. Any patient admitted with features of hypokalemic periodic paralysis.
2. Any patient admitted with features of upper motor neuron signs and symptoms
3. Any patient admitted with severe protopathic sensory symptoms
4. Any patient admitted with history of bite preceding the illness
5. Anypatient admitted with history of exposure to toxins like organophosphates
6. Any patient with severe terminal illness
7. Patients admitted with history of suspected food poisoning
8. Patients in whom the weakness progressed for more than 4 weeks

Method

Number of cases studied: 50

Duration of study: October 2012–September 2014

Detailed neurological examination including higher mental functions, cranial nerves, motor system, sensory system and autonomic system was done for all 50 patients. Motor power in these patients were assessed according to Medical Research Council grading. Autonomic dysfunction was looked for in all these patients. History of dryness of mouth, postural giddiness and defective sweating over the body were specifically asked for. Blood pressure was routinely taken in lying and sitting posture and if possible in standing posture to bring out orthostatic hypotension. Sympathetic skin response was not done due to technical problem. Respiratory function tests were done in all patients, everyday during hospitalization, including breath-holding time, single breath count, blowing candle at one arm length, chest expansion, Litten's phenomenon. Likewise, basic investigations like complete blood count, peripheral smear, blood sugar and urea, serum creatinine and electrolytes, erythrocyte sedimentation rate, daily electrocardiogram, chest x-ray were done for all the 50 patients.

RESULTS

In the 50 patients studied, 30 were male and 20 were female.

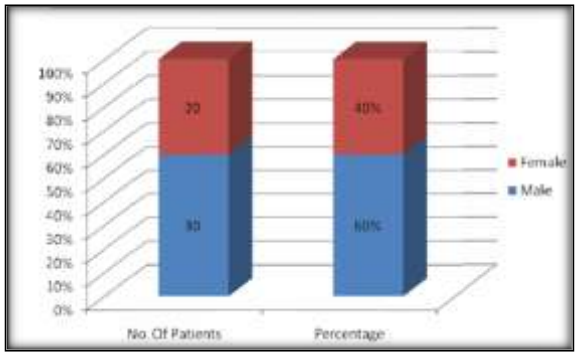


Table 1

Sex	No. Of Patients	Percentage
Male	30	60%
Female	20	40%

Age Incidence: All the 50 patients were above the age of 15; among which 26 (52%) patients were below the age of 40, 15 patients (30%) were between 40-60 years, 9 patients (8%) were above the age of 60.

Table 2

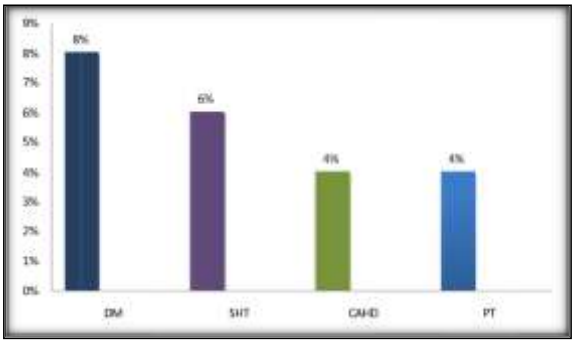
AGE IN YEARS	NO.OF PATIENTS	Percentage
4<0	26	52%
40-60	15	30%
>0	9	18%

Tables 3

Si.no	Illness	No. of patients	Percentage
1	DM	4	8%
2	SH	3	6%
3	CAHD	2	4%
4	PT	2	4%

Antecedent Events: 7 patients gave history of upper respiratory tract infection preceding the neurological illness. 5 patients gave history of gastroenteritis preceding the illness. 1 patient gave history of vaccination for dog bite preceding the illness.

1 patient was reactive for HIV. 1 patient was found to have generalised lymphadenopathy and mild hepatosplenomegaly, and Fine Needle Aspiration Cytology proved to be Hodgkin's lymphoma. Remaining patients had no antecedent event.



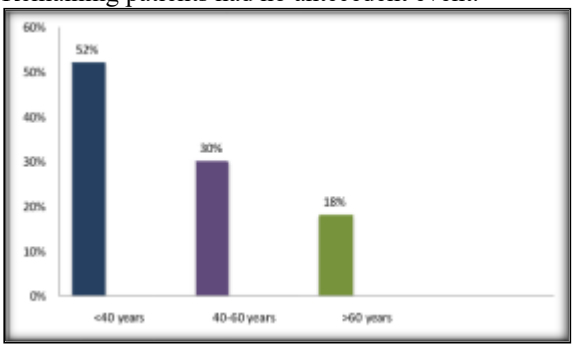


Table 4

Si. No.	Antecedent Events	No. Of Patients	Percentage
1	RESPIRATORY ILLNESS	7	14%
2	GASTROINTESTINAL ILLNESS	5	10%
3	VACCINATION	1	2%
4	HODKIN'S LYMPHOMA	1	2%
5	HIV	1	2%

Clinical Presentation: In 50 patients studied, 42 patients had weakness of both upper and lower limbs; 15 patients had neck muscle weakness; 8 patients had features suggestive of bulbar weakness; 2 patients had ocular muscle involvement.

15 patients gave history suggestive of sensory disturbance and 12 patients had electrophysiological evidence of sensory involvement. 7 patients had bilateral lower motor neuron type of facial weakness. 4 patients developed altered sensorium for a short period with complete recovery within days.

DISCUSSION

Our prospective study included fifty participants. Age Distribution All age groups appear to be impacted by GBS. Studies do, however, point to a more precise age distribution. For GBS, Adams, Victor, and Ropper1 have quoted ages ranging from 8 months to 80 years. Two peaks, one between the ages of 15 and 35 and the other between the ages of 50 and 75, were documented by Kaplan et al.In their Swedish investigation, Jiang-Guoxin et al. 60 found two peaks: one between the ages of 20 and 24 and the other between the ages of 70 and 75. Of the 50 patients in our study, 52% (26) were under 40, 30% (15) were between 40 and 60, and 18% (9) were

above 60. Our consecutive patients have ranged in age from 16 to 78, with those under 50 having the highest attack rates.[11-15]

Prevalence of Sex: In our study, the sex prevalence was 40% (20) for women and 60% (30) for men. Our investigation shows a male preponderance, which is consistent with the Robert M. et al. report. Males and females had an equal incidence, according to Peter C. Dowlin's research of 176 individuals.

Table 5: Comparison of Age Prevalance

Name of the study	Age peak in years
Jiang –guoxin et al	15-35(1st peak), 50-75(2nd peak)
Kaplan et al	20 -24(1st peak), 70-75 (2nd peak)
Present Study	20- 40 years(1st peak) Above 40-60 years(2nd peak)

Seasonal Differences In line with most research in the literature, no seasonal fluctuation in the incidence of GBS could be deduced from this study. A seasonal clustering of instances has been observed in a few investigations, nevertheless. Summertime and fall were shown to have higher incidences, according to Kauret al. Summer also saw an uptick, according to Peter C. Dowling.[16-18]

Preceding Infection An antecedent event was reported by almost one-third of the patients. More than half of GBS patients had signs of viral respiratory or gastrointestinal illnesses, according to Winter et al. A high prevalence of 73% was also found by Ropperet al. On the other hand, a study conducted by Kauret al. revealed a lower incidence of 32%.[19-21]

Failure to breathe Thirty percent of our patients had respiratory failure. According to Allan H. Ropper's meta-analysis, 10% of patients experience respiratory failure. A 23% incidence of respiratory failure was reported by Winer et al. Our patients were on mechanical ventilation for an average of 16.12 days. Respiratory muscular weakness occurred in 30% (15) of the patients. Litten's phenomenon was used to measure diaphragmatic movement. The second week saw an increase in diaphragmatic weakness, which started to improve during the third week.[22-24]

The presence of central cyanosis, oxygen saturation, chest expansion, respiratory rate, and single breath count were used to determine if patients experiencing respiratory distress needed ventilatory support. In a 2003 study by Sharshar et al., 85% of patients were anticipated to require mechanical ventilation due to short disease duration, incapacity to move the head, and a vital capacity of less than 60%. As a result, 10% (5) of our patients were placed on a ventilator.[25]

Autonomic dysfunction Forty percent (20) of the patients had autonomic dysfunction. Eight patients had orthostatic hypotension; the trunk and limbs were examined for abnormal perspiration, either increased or decreased. Seven patients had arrhythmias. Five patients experienced benign arrhythmias that lasted less than a week, including as ventricular and atrial ectopics, non-specific ST-T abnormalities, sinus tachycardia, and less frequently, sinus bradycardia. Two patients had fatal ventricular arrhythmias.[26]

Bulbar paralysis is also linked to a poor prognosis, according to a study by NK Singh et al. Bulbar weakness was also associated with a poor prognosis in our study. In line with the prognosis studies of NK Singh et al. and Winer et al, our investigation also indicated that the need for assisted ventilation was a significant prognostic factor. Additionally, our research revealed that the prognosis for individuals using mechanical ventilators is dismal. Patients with Guillain-Barré Syndrome who exhibited elevated protein linked with significant demyelination in electrodiagnostic testing experienced delayed recovery, according to cerebrospinal fluid research.

CONCLUSION

In our study, there was a male preponderance. Eighty percent of patients with Guillain-Barre Syndrome recovered without experiencing any problems. 10% of patients experienced varied degrees of bulbar weakness. Thirty percent of individuals experienced varied degrees of neck muscle weakness. Thirty percent of patients with Guillain-Barre Syndrome experienced variable degrees of respiratory muscle impairment. In order to maintain oxygen saturation, 10% of patients required ventilatory support. Autonomic disturbance symptoms of varied degrees were seen in 40% of patients. Two patients showed signs of ocular muscle involvement, while one patient demonstrated signs of coordination. Our study's prognostic results are not very good as people age.10. When diabetes mellitus or ischaemic heart disease coexist, the prognosis is not good. Increased protein in the cerebrospinal fluid of Guillain-Barre Syndrome patients was associated with severe demyelination in electrodiagnostic studies and delayed recovery. The prognosis of Guillain-Barre Syndrome patients varies linearly with the severity of electrodiagnostic investigations. All of the patients who were part of the trial consistently had no h-reflex. 70% of patients in the upper limbs and 90% of patients in the lower limbs had no F-response. All of these individuals showed a decrease in motor nerve conduction velocity, with various degrees of severity. Patients with extreme weakness had a conduction block characteristic. Patients with conduction block recovered more slowly than those with delayed motor nerve conduction velocity alone. Patients treated with or without steroids did not significantly differ in their outcomes in our study. In our investigation, the mortality rate was about 10%.

REFERENCES

1. RopperAh, Brown rh. ADAMS and VICTOR'S - Principles of Neurology - Ninth Edition, P-1322-1330.
2. Yuki, N. &Hartung, H. P. Guillain–Barré syndrome. N. Engl. J. Med.366, 2294–2304 (2012)
3. Winer, J. B. Guillain–Barré syndrome: clinical variants and their pathogenesis. J. Neuroimmunol.231, 70–72 (2011)
4. Kuwabara, S. & Yuki, N. Axonal Guillain–Barré syndrome: concepts and controversies. Lancet Neurol.12, 1180–1188 (2013)
5. Wakerley, B. R. & Yuki, N. Pharyngeal–cervical–brachial variant of Guillain–Barré syndrome. J. Neurol. Neurosurg. Psychiatry85, 339–344 (2014).
6. Asbury, A. K. &Cornblath, D. R. Assessment of currentdiagnostic criteria for Guillain–Barré syndrome. Ann. Neurol.27(Suppl.), S21–S24 (1990).
7. Willison, H. J. & Yuki, N. Peripheral neuropathies and anti-glycolipid antibodies. Brain125, 2591–2625 (2002).
8. vanDoorn, P. A. Diagnosis, treatment and prognosis of Guillain–Barré syndrome (GBS). Presse Med.42, e193–e201 (2013)
9. Hauser SL, Asbury AK. Guillain-Barre Syndrome andother immune- related neuropathies. In Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo D, Jameson JL et al. Harrison's Principles of Internal Medicine. 18th edition,vol-II. New York: McGraw-Hill, 2008;2667 -7
10. Willison, H. J. The immunobiology of Guillain–Barré syndromes. J. Peripher. Nerv. Syst.10, 94–112 (2005).
11. Griffin, J. W. et al. Guillain–Barré syndrome in northern China. The spectrum of neuropathological changes in clinically defined cases
12. Kuwabara, S. & Yuki, N. Axonal Guillain–Barré syndrome: concepts and controversies. Lancet Neurol.12, 1180–1188 (2013)
13. Mori, M. &Kuwabara, S. Fisher syndrome. Curr. Treat. Options Neurol.13, 71–78 (2011)
14. Koga M, Gilbert M, Li J, et al. Ante- cedent infections in Fisher syndrome: a common pathogenesis of molecular mim-icry. Neurology 2005;64:1605-11
15. Mori, M., Kuwabara, S. & Yuki, N. Fisher syndrome: clinical features, immunopathogenesis and management. Expert Rev. Neurother.12, 39– 51 (2012)
16. Hiraga, A. et al. Recovery patterns and long term prognosis for axonal Guillain–Barré syndrome. J. Neurol. Neurosurg. Psychiatry76, 719–722 (2005)
17. Willison, H. J. & Yuki, N. Peripheral neuropathies and anti-glycolipid antibodies. Brain125, 2591–2625 (2002).
18. Kaida, K. &Kusunoki, S. Antibodies to gangliosides and ganglioside complexes in Guillain–Barré syndrome and Fisher syndrome: mini- review. J. Neuroimmunol.223, 5–12 (2010).
19. Yuki, N. Guillain–Barré syndrome and anti-ganglioside antibodies: a clinician–scientist's journey. Proc. Jpn Acad. Ser. B Phys. Biol. Sci.88, 299–326 (2012)
20. Kusunoki, S., Kaida, K. & Ueda, M. Antibodies against gangliosides and ganglioside complexes in Guillain–Barré syndrome: new aspects of research. Biochim. Biophys. Acta1780, 441–444 (2008)
21. Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. Arch Neurol 1986;43:1150-2.
22. Winer, J. B. Guillain–Barré syndrome: clinical variants and their pathogenesis. J. Neuroimmunol.231, 70–72 (2011).
23. Kokubun, N. et al. Conduction block in acute motor axonal neuropathy. Brain133, 2897–2908 (2010)
24. Kuijf, M. L. et al. Diagnostic value of anti-GM1 ganglioside serology and validation of the INCAT-ELISA. J. Neurol. Sci.239, 37–44 (2005).
25. Rinaldi, S. Update on Guillain–Barré syndrome. J. Peripher. Nerv. Syst.18, 99–112 (2013)
26. Vanhoutte, E. K. et al. Modifying the Medical Research Council grading system through Rasch analyses. Brain135, 1639–1649 (2012).