e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2022; 14(9); 201-210

Original Research Article

A Clinical and Demographic Profile of Tertiary Care Patients with Guillain-Barré Syndrome in Himachal Pradesh

Sanjeev Kumar Singh[1], Veena Gupta[2], Ashwani Kumar[3], Sudhir Sharma[4]

[1]Assistant ProfessorDepartment of Physiology, SLBSGMCH, Mandi
[2]Assistant ProfessorDepartment of Physiology, SLBSGMCH, Mandi
[3]Assistant ProfessorDepartment of Biochemistry, SLBSGMCH, Mandi
[4]ProfessorDepartment of Neurology, IGMCH, Shimla

Received: 25-07-2022 / Revised: 25-08-2022 / Accepted: 10-09-2022
Corresponding author: Dr. Sudhir Sharma
Conflict of interest: Nil

Abstract

Guillain-Barré syndrome (GBS) is a complex disorder characterized by immune-mediated, self-limiting, reactive peripheral neuropathies. GBS can produce life-threatening problems if it affects the breathing muscles or the autonomic nervous system. The present study was done to see the clinical profile and the epidemiological trends occurring among 30 diagnosed patients reported over a one-year duration during their hospital stay. Patients with Guillain-Barre Syndrome (GBS) were diagnosed using the Asbury and Cornblath criteria for case definition of GBS, as well as supporting laboratory and electrodiagnostic findings. A total of 30 cases were diagnosed, F: M ratio of the case was 1:1.3. The age range of the case was 19-80 years. The maximum number of cases was in the 31 to 40 years (n=7) age range. Maximum cases were reported from July to September (43.33 percent) during the summer and rainy seasons, whereas there were no reported cases from October to December. 80 percent (n=24) of cases were reported within the first week, 16.67 percent (n=5) during the second week, and only 3.33 percent (n=1) during the third week. There was a history of previous illness in the form of loose stools, fever, and respiratory tract infection, as well as other GI infections, in 46.67% of cases, the most prevalent of which was loose stools with fever in 30% of cases.In 24 cases, weakness was present in both the upper and lower limbs, whereas in 6 cases, weakness was predominant in the lower limb. In six cases, facial weakness was also present. The most frequently reported sensory symptom was numbness, followed by a tingling sensation and a burning sensation. Almost all the cases had areflexia, either total areflexia (14 out of 30 or 46.67%) or partial areflexia (16 out of 30 or 53.33%), with the majority lacking knee and ankle reflexes.

Keywords: Guillain-Barré syndrome (GBS), Acute Flaccid Paralysis (AFP),Areflexia, Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), Acute motor axonal neuropathy (AMAN), Acute motor and sensory axonal neuropathy (AMSAN).

Introduction

Guillain-Barré syndrome (GBS) is a complex disorder characterized by immune-mediated, self-limiting, reactive peripheral neuropathies. GBS can produce life-threatening problems if it affects the breathing muscles or the autonomic

nervous system. [1,2] GBS is the primary cause of flaccid paralysis in both developed and poor nations following the eradication of polio. [3,4,5]

Patients with Guillain-Barré syndrome (GBS) present with complaints of weakness and/or unsteadiness of gait. Weakness is a characteristic of GBS. Typically, it begins in the legs and progresses to the arms. This progression may occur over hours to days to weeks. The weakness is usually symmetric. [6] In 1916, Guillain, Barré, and Strohl broadened the clinical description and first reported the characteristic cerebrospinal fluid (CSF) finding of albumin cytologic dissociation (i.e., the elevation of CSF protein with normal CSF cell count). [7] This condition has been reported worldwide in all age groups. [8, 9] The peak incidences of GBS occur in late adolescence and in the elderly. Population-based surveys attempting to document the annual incidence of GBS have been conducted in various countries worldwide and generally agree on a rate of 1 to 3 per 100, 000 persons annually. [10,11,12] In 1995, GBS was subdivided into 4 distinct forms based on the histopathological and neurophysiological basis, they were acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and Miller-Fisher syndrome (MFS). Features that strongly support diagnosis are a progression of symptoms over days to four weeks, relative symmetry of symptoms, and mild sensory or signs. [13] Cranial nerve involvement may affect the airway and facial muscles, eye movements, and swallowing. Supportive criteria include relatively mild sensory signs and typical electrodiagnostic features like neurophysiological evidence of conduction block. [1,2] In India, there is limited information available on the relative burden of GBS, particularly at a population level. A small case series of patients with GBS in India suggest that it is an important cause of non-poliomyelitis Acute Flaccid Paralysis (AFP). [14] In light of these facts, we conducted the study of the clinical profile of 30 diagnosed cases with the intention of gaining a deeper understanding of the symptomatology and augmenting the existing epidemiological database.

Material and methods: This was a prospective hospital-based observational study of Guillain-Barré Syndrome (GBS) cases diagnosed using the Asbury and Cornblath case definition criteria. [18,13,15] Laboratory and electrodiagnostic characteristics were also considered. The study was conducted over the course of one year, from August 2014 to August 2015, and 30 GBS patients were included.

Patients' informed consent was obtained prior to the initiation of the study. The patients' clinical histories were meticulously recorded, and they underwent a comprehensive general physical examination and systemic examination. In addition, the cranial nerves and sensory system were examined. Each patient's musculoskeletal system was thoroughly examined.

Using the Medical Research Council (MRC) strength grading scale, the power and strength of the muscles of both limbs were evaluated clinically at all joints. [16]

Table 1: Medical Research Council scale. Examination aids for the peripheral nervous system.[16]

Score	Finding
0	No contraction
1	Flicker or trace contraction
2	Active movement, with gravity, eliminated

3	Active movements against gravity
4	The active movement against gravity and resistance
5	Normal power

Table 2: Range of motion (ROM) Deep tendon reflexes were elicited and graded according to the Medical Research Council (MRC) scale. [16]

Grade	Response, Significance
0	no response, always abnormal
1+	slight but present, the response may or may not be normal
2+	brisk response, normal
3+	very brisk response, may or may not be normal
4+	4+ clonus, always abnormal

Thirty patients with Guillain-Barré Syndrome were included, with a female to male ratio of 13:17 (1:1.3), all of whom were admitted to the Department of Neurology and met the Asbury & Cornblath diagnostic criteria for GBS.[13,15]

Inclusion criteria for the patients:

The patients' willingness - Those who met Asbury and Cornblath's Clinical diagnostic criteria for GBS

(Asbury and Cornblath) criteria[8,13,15]

- Progressive limb and arm weakness
- Areflexia

Clinical features supportive of the diagnosis

- Progression over days to weeks
- Relative symmetry of signs
- Mild sensory symptoms or signs
- Cranial nerve involvement (bifacial palsies)
- Recovery beginning two to four weeks after the cessation of progression
- Autonomic dysfunction
- Absence of fever at the onset.

Statistical analysis: The results obtained from the patients were analysed using SPSS 22. The clinical characteristics of the study population were expressed as a percentage. The normal range for electrophysiological variables was determined to be between two standard deviations (SD). The results were analysed with descriptive statistics.

Results

A total of 30 cases were taken, F: M ratio of the case was 1:1.3. The maximum number of cases was in the 31 to 40 (n=7) age range. The age range of the case was 19-80 years.

Table 3: Age and sex distribution of the cases

Age group	Male	Female	Total
19 to 30	4	2	6
31 to 40	3	4	7
41 to 50	2	3	5
51 to 60	5	0	5
61 to 70	2	1	3
71 to 80	1	3	4
Total	17	13	30

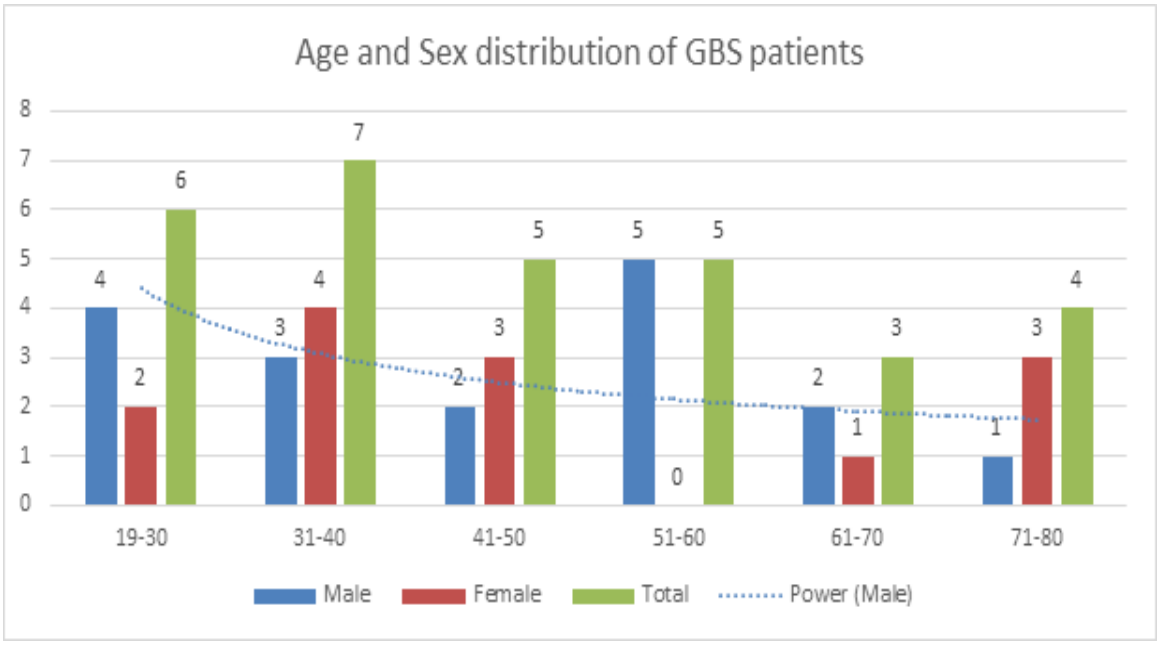

Figure 1: Most cases (n=7) fall between the ages of 31 and 40 according to a graphical representation of the cases' age and sex distribution. Case subjects ranged in age from 19 to 80 years old.

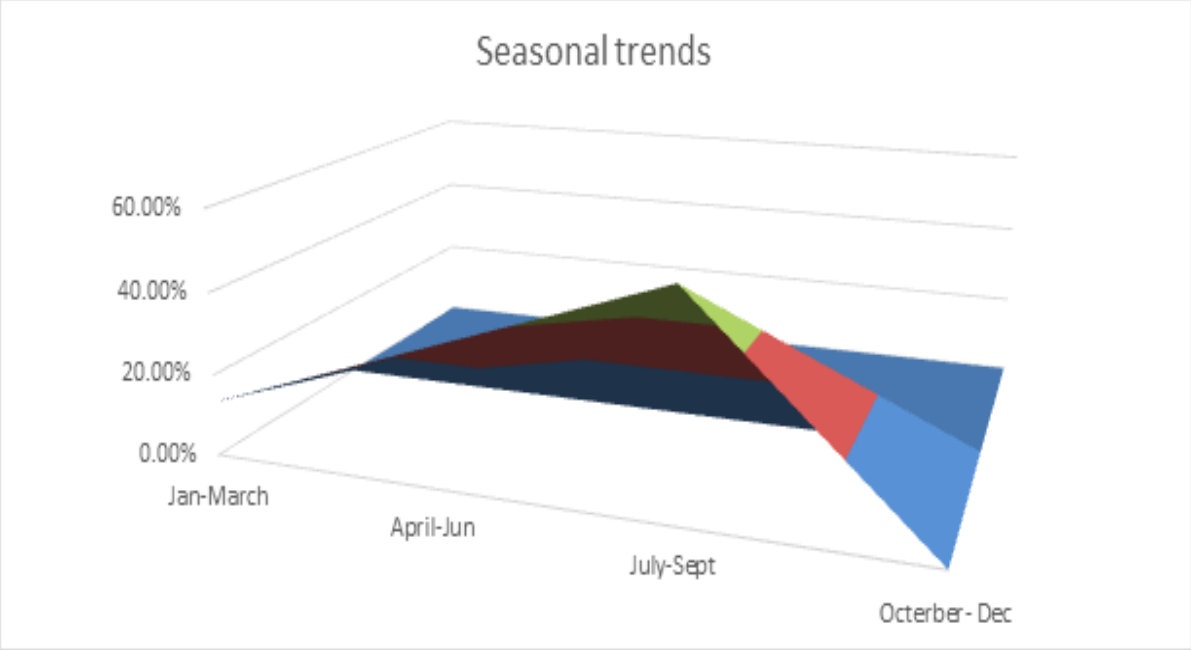

Figure 2: Maximum cases were presented during the summer and rainy season during July to September (43.33 percent), whereas there were no cases during the months of October to December.

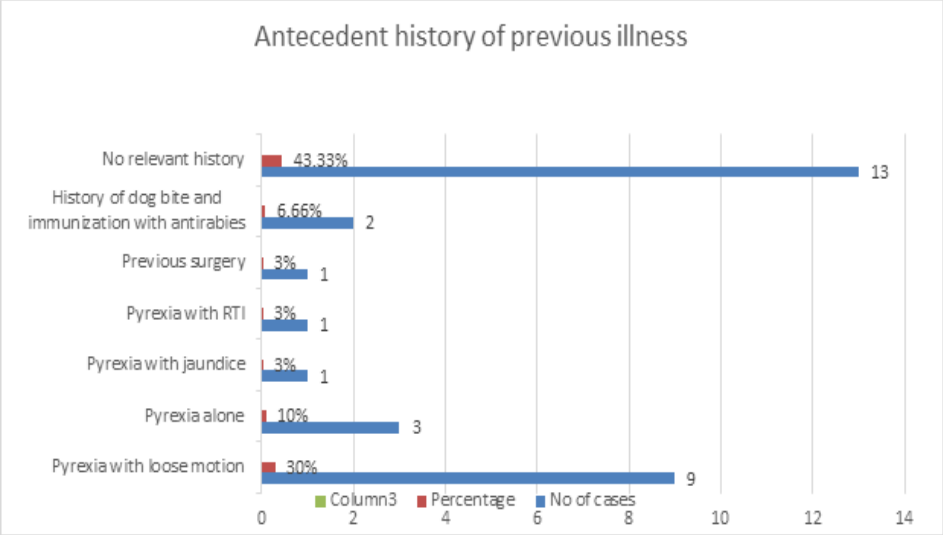

Figure 3: Antecedent infection in the cases of GBS, in 46.67% of cases, there was a history of previous illness in the form of diarrhoea, fever, and respiratory tract infection, as well as other GI infections; diarrhoea with fever was the most common.

There was an antecedent history of previous illness in form of loose motion, pyrexia, & respiratory tract infection, and other GI infections in 46.67% of the cases among which the most common was the loose motion with fever which was found in 30% of them, fever alone in 10% (n=3) of cases, 3.33 % (n=1) with fever along with jaundice, 6.66% (n=2) were having h/o dog bite and anti-rabies immunization 2 months back, & one patient i.e. (3.33%) was having h/o surgery 2 months back, there was no relevant history in 43.33% (n=13) of the cases.

Time to presentation: 80 percent (n=24) of cases were reported within the first week, 16.67 percent (n=5) during the second week, and only 3.33 percent (n=1) during the third week.

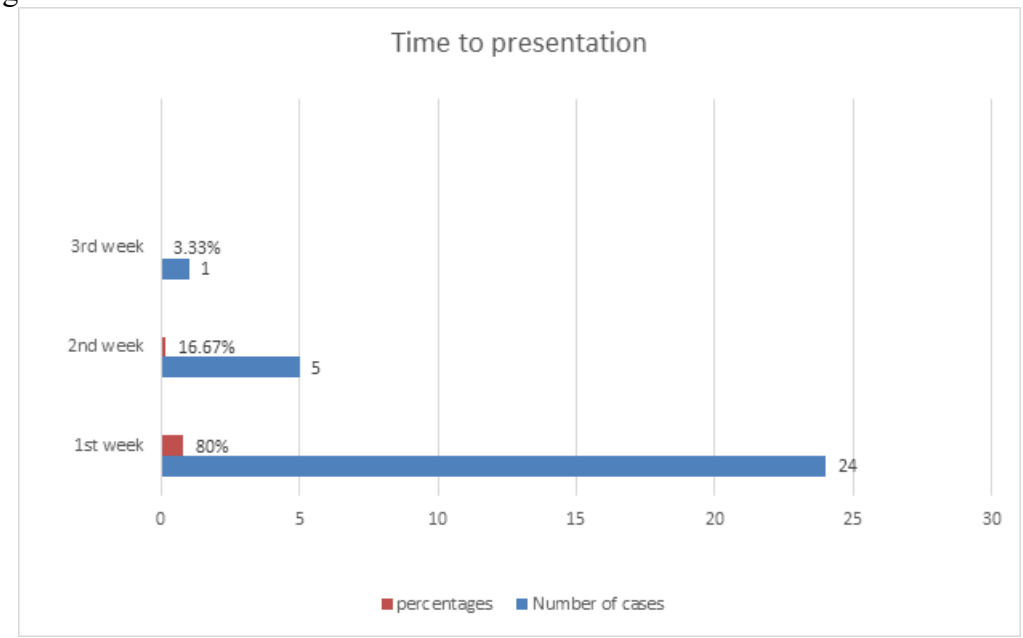

Figure 4: Eighty percent (n=24) of the cases were reported within the first weekin the third week, and only 3.33 percent (1 case) were reported, a weakness with areflexia was the most prevalent finding.

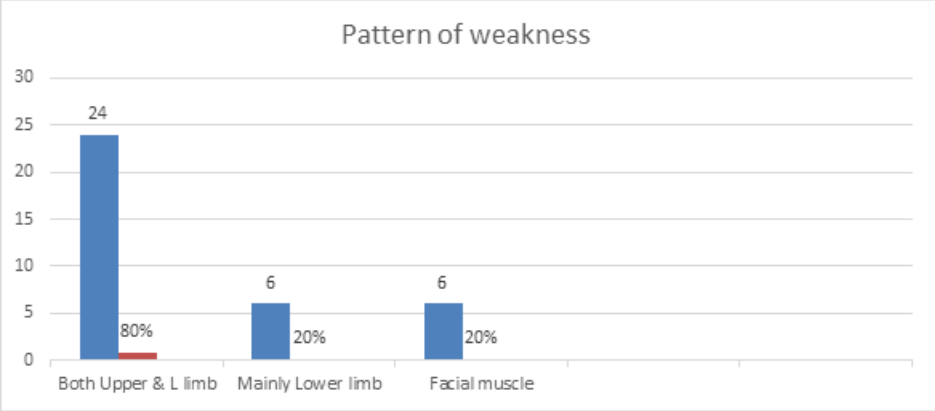

Figure 5: In 24 cases, the pattern of weakness involved both the upper and lower limbs, whereas in 6 cases, the lower limb was predominantly affected. In six cases, facial weakness was also present.

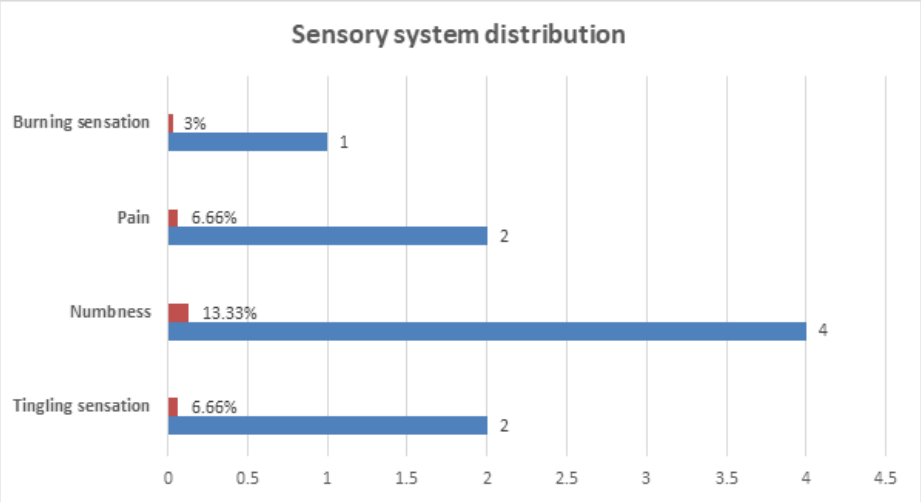

Figure 6: Numbness was the most frequently reported sensory symptom, followed by tingling sensation and burning sensation

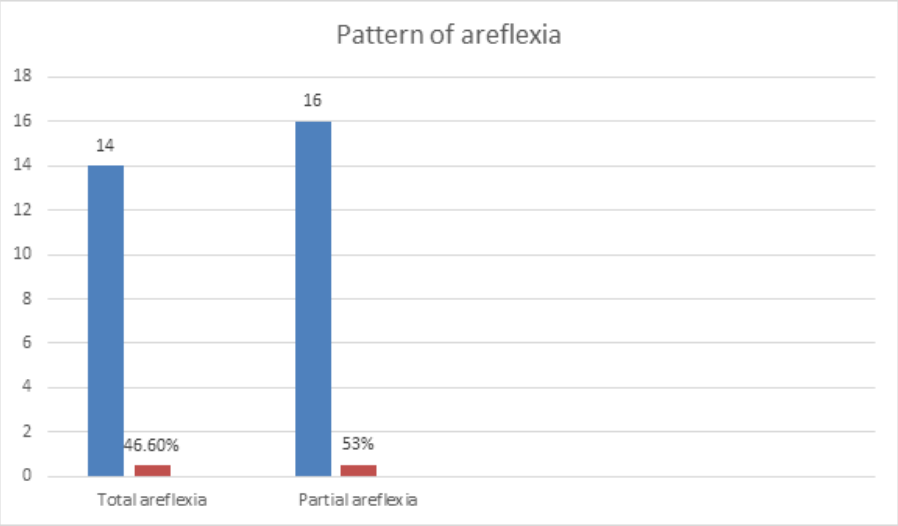

Figure 7: Almost all the cases had areflexia, either total areflexia (14 out of 30, or 46.67%) or partial areflexia (16 out of 30, or 53.33%), with the majority lacking knee and ankle reflexes.

At the time of writing this article, it was possible to contact a total of sixteen GBS patients, 13 of which were having no sequelae and leading a quite normal life, two complained of minor weakness in the grip and while walking, and one died two years later due to haematemesis, which was unrelated to the GBS.

Discussion

Our study aimed to determine the epidemiological pattern of GBS patients. The ratio of women to men among the 30 patients was 1:1.3. Consequently, the sex ratio revealed a slight male preponderance in comparison to females, which was consistent with previous research by Alter M in 1990 [17], who found a male preponderance among the hospitalized population. Kaur U., Chopra, et al. [18] (1986), Gupta S.K., and Taly A.B., et al. [19] (2008) also discovered similar outcomes. Similarly, studies conducted in 2013 by Geetanjali Sharma and Sushma Sood [20] revealed a sex ratio of 1:1.82. (F: M). In their study of 65 evaluated patients, 42 (64.61 percent) were male while 23 were female (35.38 percent).

Regarding the seasonal pattern, our study found that case clustering was greatest during the summer and the rainy season, i.e., July to September and April to June. Gupta S.K., Taly A.B., et al. [19] (2008) discovered comparable results. In September, the greatest number of cases (23.33 percent of the total) were reported. During the months of October to December, fewer cases were reported.

2013 Indian series studies by Geetanjali Sharma, Sushma Sood, et al. [20] was consistent with our findings. However, there were monthly variances in the incidence rates. According to their findings, the highest incidence of GBS (n = 27, 41.53 percent) was observed during the summer months of May to July; there were 19 cases (29.23 percent) during the spring months of February to April. 11 (16.92 percent) from November to January and 8 (12.30 percent) from August to October.

Like our findings, Gupta D et al.[19] reported a peak incidence between June–July, and September–October in 2008. A study by Akbayram et al. [21] in Turkey revealed a seasonal predominance in summer and spring, accounting for 41.6% and 22.2% of the episodes, respectively, which is consistent with our findings regarding summer predominance. Hughes et al. [22] reported peak summer clustering in a study based in Northern China. A Pakistan-based study by Zaheer et al. [23] found that the peak incidence of GBS during the summer months (April through September).

In our study there was the antecedent history of previous illness in form of loose motion with pyrexia in 30% (n=9) of the cases, Pyrexia alone in 10% (n=3) of the cases, respiratory tract infection in 3.33% (n=1) of the cases. There was a history of surgery 2 months back in 3.33% (n=1), and jaundice in 3.33% (n=1) of the cases. History of dog bite 2 months back was present in 6.66% (n=2) of the cases. These findings suggested the association of GBS with the previous infection & immunization, so our finding was in line with the previous study by Govoni & Granieri [24] in 2001 who reported that approximately two third of their patients were having preceding events of infection, most frequently an upper respiratory, GIT infection, surgery, immunization 1to 4 weeks before the onset of neurological symptoms. Our findings were also like the findings of Geetanjali Sharma, Sushma Sood et al [20] in 2013, In their study, as per the detailed history of each case, 5 patients (7.69%) had diarrhoea while 12 (18.46%) patients had flu-like syndrome 1 - 2 weeks before the onset of GBS.

In 1988, Winer JB et al. [25] published studies supporting the antecedent infection as the cause of GBS. they revealed serological evidence of recent infection in 31% of patients. Campylobacter

jejuni(14%) and cytomegalovirus (11%) were both significantly more frequently demonstrated in patients than in controls. Also, the previous study by Ho T W et al[26] in1995, Sinha S [27] showed both Campylobacter jejuni (14% of patients) and cytomegalovirus (11% of patients) were significantly more prevalent in patients than in controls.

As regards, the clinical finding weakness was an almost invariable feature among most of the cases, with 80% (n= 24) having weakness in both the upper limb and lower limb. The remaining 20% (n=6) were having weaknesses mainly in the lower limbs. Almost all the cases were having areflexia, in which 53.33% (n=16) had absent knee and ankle reflexes, and 46.67% (n=14) were having total areflexia in all 4 limbs. Cranial nerve with facial muscle weakness was observed in 20% (n=6) of cases. The most involved nerve was the facial nerve. Among the sensory symptoms, this was present in the form of tingling sensation in 6.67% (n=2) numbness in 13.33% (n=4) pain in 6.67%, (n=2), and burning sensation in 3.33% (n=1) of the cases. So, the clinical profile of the patients was conforming to the case definition of the GBS as per the Asbury and Cornblath criteria. [8,13] These were also in line with the clinical profile found by ChristiaanFokke, Bianca van der Berg et al, in their study "Diagnosis of GuillainBarre syndrome and validation of Brighton criteria" in 2014. [28]

Conclusion

GBS impacts the peripheral nervous system impairing nerve conduction and involves both its motor and sensory components. The disease has an acute onset with most of the patients presenting within the first week of illness. The most prevalent age range is between 31 and 40 years old. There is a preponderance of males with the antecedent illness being gastrointestinal infection. There is a relative case clustering in the summer and during the rainy season. The most encountered symptom is impaired gait with weakness in both upper and lower limbs, with lower limb involvement being more evident and frequent. Involvement of cranial nerve with facial muscle weakness, sensory and autonomic nervous system is encountered less frequently. Areflexia is a cardinal and hallmark clinical sign, with the absence of knee and ankle reflexes in most patients. GBS should be in a high index of suspicion in all cases presenting with acute flaccid paralysis.

Limitations: It involved a hospital-based observation. We were unable to follow up with all the patients. A large, multicentre study will provide a more accurate representation of Himachal Pradesh's tertiary care hospitals. We recommend that similar research be repeated in the future on a variety of neurological disorders.

Contribution of authors

Author 1 was responsible for drafting the main body of the manuscript, performing data analysis and interpretation, and writing the discussion and conclusion. Author 2 was responsible for proofreading and compiling the data of patients and revision of the manuscript. Author 3 was responsible for compiling and analysing the laboratory investigations. The corresponding author of this article was responsible for framing the core idea and technical guidance.

Acknowledgment

The authors are extremely appreciative of the patient's participation.

Informed consent Statement

Valid informed consent was obtained from the patients and their family members.

Sponsorship: Nil

References

1. Amato AA. Guillain Barre syndrome & related disorders. Rev Mex Neurosci. 2005; 6: 455- 469.
2. Haymaker W, Kernohan JW. The Landry-Guillain-Barré syndrome; a

clinicopathologic report of 50 fatal cases and a critique of the literature. Medicine. Baltimore. 1949;28: 59-141.
3. Khean Jin GOH, Wai Keong NG et al. A clinical and electrophysiological study of Guillain-Barré syndrome in Malaysia. Neurol J Southeast Asia 1999; 4: 67 – 72.
4. Griffin JW, Sheikh K. The Guillain-Barré syndromes. In: Dyck PJ, Thomas PK, editors. Peripheral neuropathy. Vol. 2, 4th ed. Philadelphia: W.B. Saunders; 2005; 2197-2199.
5. TS Saraswathy, H NorZahrin, MY Apandi et al. Acute Flaccid Paralysis Surveillance: Looking Beyond the Global Poliomyelitis Eradication Initiative. 2008.
6. Michael T Andary, Guillain-Barre Syndrome Clinical Presentation emedicine. Updated: Jul 01, 2015.
7. Guillain G, Barré J, Strohl A. Sur un syndrome de radiculo-nevrite avec hyperalbuminose du liquid cephalorachidien sans reaction cellulaire. Remarquessur les characters cliniqueetgraphique des reflexes tendinaux. Bulletins et Memories de la Societe Medicale des Hopitaux de Paris. 1916; 40:1462–1470.
8. Asbury AK. Diagnostic considerations in Guillain-Barre´ syndrome. Ann Neurol 1981;9(Suppl):1–5.
9. Kimoto K, Koga M, Odaka M, Hirata K, Takahashi M, Li J, et al. Relationship of bacterial strains to clinical syndromes of Campylobacter-associated neuropathies. Neurology. 2006 Nov 28; 67(10):1837-43.
10. DANA L. Newswanger, Bethesda, Maryland, et al. Guillain-Barré Syndrome Am Fam Physician. 2004 May 15;69(10):2405-2410.
11. Hahn AF. Guillain-Barré syndrome. Lancet. 1998; 352:635–41.
12. Seneviratne U. Guillain-Barré syndrome. Postgrad Med J. 2000; 76:774–82.
13. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990;27(suppl):S 21–24.
14. World Health Organization. Acute onset flaccid paralysis. WHO Press; 1993; 24–69.
15. Asbury AK, Arnason BG, Karp HR, et al. Criteria for the diagnosis of Guillain-Barré syndrome. Ann Neurol 1978; 3:565–566.
16. Medical Research Council. Aids to the examination of the peripheral nervous system. 1981; Memorandum no. 45, Her Majesty's Stationery Office, London.
17. Alter M. The epidemiology of Guillain–Barré syndrome. Ann Neurol. 1990; 27:7–12.
18. Kaur U, Chopra JS, Prabhakar S, Radhakrishnan K, Rana S. Guillain-Barré syndrome - a clinical electrophysiological and biochemical study. ActaNeurol Scand. 1986; 73:394–402.
19. Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A. Electrodiagnostic and clinical aspects of Guillain-Barré syndrome: An analysis of 142 cases. J Clin Neuromuscul Dis. 2008; 10:42–51.
20. Geetanjali Sharma, Sushma Sood, Sudhir Sharma: Seasonal, Age & Gender Variation of Guillain Barre Syndrome in a Tertiary Referral Center in India. Neuroscience & Medicine, 2013; 4, 23-28.
21. S. Akbayram, M. Dogan, C. Akgün, E. Peker, R. Sayin and F. Aktar, Clinical Features and Prognosis with Guillain-Barré Syndrome," Annals of Indian Academy of Neurology. 2011; 2;(14): 98-102.128.
22. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain–Barré syndrome. J Infect Dis. 1997; 176: S92–98.
23. M. Zaheer, M. Naeem, and M. Nasrullah, "Seasonal Variation and Sex Distribution in Patients with Guillain-Barre Syndrome, Pakistan

Journal of Neurological Sciences. 2008; 1;(3):6-8.
24. Govoni V, Granieri E. Epidemiology of the Guillain-Barré syndrome Curr Opin Neurol. Oct; 2001;14(5):605-613.
25. Winer JB, Hughes RAC, Anderson MJ, et al. A prospective study of acute idiopathic neuropathy. II. Antecedent events. J Neurol Neurosurg Psychiatry 1988; 51:613–618.
26. Ho TW, B. Mishu, C. Y. Li et al. Guillain-Barré Syndrome in Northern China. Relationship to Campylobacter jejuni Infection and Anti- Glycolipid Antibodies," Brain. 1995;118(3): 597-605.
27. Sinha S, Prasad KN, Jain D, Pandey CM, Jha S, Pradhan S. Preceding infections, and gangliosides antibodies in patients with Guillain–Barré syndrome: A single-center prospective case-control study. Clin Microbial Infect. 2007; 13:334–337.
28. Christiaan Fokke, Bianca van den Berg et al. Diagnosis of Guillain Barré Syndrome and Validation of Brighton Criteria Brain. 2014;137(1):33-43.