

**Time Frequency of Guillain-Barre Syndrome in Northwest of Iran**

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**Abstract:** Guillain-Barre syndrome (GBS) is an immune mediated polyradiculoneuropathy usually preceded with respiratory and gastrointestinal infections 3 to 5 days prior to neurologic signs appearance. It seems that GBS seasonally outbreaks in our region. This study was aimed to evaluate seasonal relationship of clinical and epidemiological findings of GBS in hospitalized patients in Imam Khomeini hospital in the period of 1992-2004. One hundred and seventy five cases of hospitalized patients diagnosed with GBS were studied considering following factors: age, sex, events prior to the onset, seasonal incidence, clinical patterns, and cerebrospinal fluid (CSF) protein, electrodiagnosis and mortality rate in patients. The age distribution was in the range of 2-83 years with the mean age of 38 years and male to female ratio was 1 to 2.3. Seasonal onset was spring in 21.1% of the cases, summer in 26.6%, fall in 28% and winter in 24.6%. There was no significant epidemic relation with the seasons of the year. The mortality rate was 8.6% during admission time. There was no significant relation between the season of onset of the disease and mortality rate.

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**1. Introduction**

In 1916, Guillain-Barre and Stroll reported a French soldier who had experienced motion weakness, areflexia and changes in CSF (Cerebro Spinal Fluid) protein. Later, they reported further cases with the same manifestations and these clinical conditions were called Guillain-Barre syndrome or GBS (Trojaborg, 1998; Seneviratne, 2000). GBS affects people from every race or age. This disease is non epidemic and females are more vulnerable. The age spectrum for being affected by this disease is from 8 months to 81 years. The most probable ages to experience the onset was 50-74 years. The annual incidence is 0.4 -1.7 in one hundred thousand (Victor and Roper, 2001; Aminoff, 2001). The main cause of GBS remains unknown and it is believed to have an immunologic etiology. Most of the cases have a history of an infection before the clinical onset. It is respiratory infection in 40% and gastroenteritis in 14-20% of cases (Jacobs et al., 1998; Hughes and Rees, 1997).

Considering our experiences with referred patients with GBS at neurology ward of Tabriz Imam Khomeini hospital, it seemed that the patients were

admitted periodically. As seasonal prevalence is confirmed in some of the articles that we reviewed (Cheng et al., 2003) and not confirmed in some (Hughes and Rees, 1997; Alter, 1990). Considering the controversy over this issue, we decided to study this subject in hospitalized patients with GBS in previous years and relation of it to short-term prognosis.

**2. Material and Methods**

In an analytic-descriptive and retrospective study, all the patients with GBS admitted to Imam Khomeini hospital from 1992-2004 were studied. This hospital was the biggest referral university medical center in north-west of Iran in that period. The following variables were studied: age, sex, season of onset, recent infection history, clinical and paraclinical findings, duration of admission and discharge situation. The collected data were analyzed using SPSS statistics software and the statistical analyses of the relation between variables were evaluated using chi square and T-test. A *P* value <0.05 was considered statistically significant.

### 3. Results

One hundred and seventy five patients (male:female ratio of 2.3:1) with GBS were admitted in the period of 1992 to 2004. The age range was 2-83 years with mean age of 68. Considering the onset season, 21.1% experienced the disease in spring, 26.3% in summer, 28% in fall and 24.6% in winter. The events prior to the weakness onset happened 2 to 4 weeks earlier including upper respiratory infections (59.4%), gastrointestinal infections (5.7%), and both (6%). Nearly one third of the cases had facial nerve palsies and 8.6% had other cranial nerves palsies.

Considering clinical pattern, almost 97.8% of the cases suffered from weakness in extremities including paraparesia (6.9%), quadriparesia and numbness (90.9%), and respiratory complaints (13.4%). Regarding loss of deep tendon reflexes (DTRs), 46.6% of the cases experienced loss of DTRs in lower extremities and 51.4% in both upper and lower extremities and decrease in reflexes was reported in 4% of the cases. Regarding sensory impairment in examination, 23.4% of the cases had pain and temperature impairment and 9.7% had deep sensory dysfunction. Autonomic dysfunction was present in 15.9% of cases, 5.7% of the cases had cardiovascular involvement, 9.1% bladder involvement and 1.1% had sweating dysfunction. Mechanical ventilation was required in 22.9% of the cases due to the weakness in respiratory muscles.

Electrodiagnostic tests showed GBS types as follows: acute inflammatory demyelinating polyneuropathy (AIDP) (67%), acute motor axonal neuropathy (17%), M. Fisher syndrome (4%), acute motor sensory axonal neuropathy (2%), and unclassified (10%). Hospitalization period lasted from 1 to 90 days. The mean admission days were 16 days. Mortality rate was 8.6% in admitted patients which was mostly due to the cardiopulmonary arrest.

Regarding the distribution of GBS in different seasons of the year, there was no significant relation between the disease and different seasons of the year ( $P=0.61$ ). Regarding the epidemiologic relation in different seasons of the year (e.g., 2002, 2003 and 2004), there was no significant relation. Regarding the mortality rates during admission period in different seasons of the year, 8.1% of the affected cases expired in spring ( $P=0.6$ ), 2.2 % in summer ( $P=0.57$ ), 12.2% in fall ( $P=0.21$ ), and 11.6% in winter ( $P=0.25$ ). Regarding the seasonal pattern, AIDP type was more common in winter but the relation was not significant ( $P=1.00$ ). There was no significant relation between history of respiratory infections and GBS in seasons of year ( $P=0.08$ ) and between mortality in GBS and previous respiratory infections ( $P=0.59$ ).

### 4. Discussions

This study reveals that there are no significantly seasonal outbreaks of GBS and no relation of it to preceding infections, GBS type and mortality. Regarding sex prevalence, Rowland (2005) and Aminoff (2001) reported an equal prevalence, whereas Victor and Roper (2001) and Alter (1990) reported a higher prevalence in females and other studies suggest higher prevalence in males (Hughes and Rees, 1997; Yuan et al., 2002; Rocha et al., 2004) which is in accordance with our study. These statistical differences are indicative of the inequality in sex distribution in different parts of the world.

The literature shows controversial results about seasonal distribution of GBS. Similar to our study, some researchers suggested no significant seasonal difference (Victor and Roper, 2001; Aminoff, 2001; Rowland, 2005; Hughes and Rees, 1997; Alter, 1990; Dyck, 1994; Walton, 1993; Hui et al., 2005; Soffer et al., 1978; Chroni et al., 2004; Sedano et al., 1994). In contrast, other studies showed a significant prevalence in spring (Olivé et al., 1997; Lyu et al., 1997), a significant prevalence in summer (Rocha et al., 2004; Van Koningsveld et al., 2000), and a higher prevalence in winter (Cheng et al., 2003; Cheng et al., 2000; Bogliun et al., 2004; Bahou et al., 1996). These statistics suggest that climatic diversity has been probably effective on chronological prevalence in different parts of the world. Regarding the preceding events in GBS, in our study respiratory infections were present in 59.4% of the cases and gastrointestinal infections in 5.6% and there was no seasonal relation. Pioneer events have been reported in 50-60% of the cases in the literature (Aminoff, 2001; Goldman and Bennett, 2004; Walton, 1993). In a Greek study, respiratory infections (28.8%) and gastrointestinal infections (7.5%) were reported as 50% of all pioneer events and there has also been a report of a significant relation between different seasons and affecting infections incidence (Chroni et al., 2004). In a Taiwanese study, there was a relation between respiratory infections incidence in spring with GBS (Cheng et al., 2003).

Regarding the different types of electrodiagnosis in our study, AIDP type was the most common (67%) and the incidence of any types did not have relation with different seasons. In the previous studies, AIDP was also the most common type (Bogliun et al., 2004; Kuwabara, 2004; Alam et al., 1998). However, AMAN type was more common in studies of northern China and Japan (Hiraga et al., 2003; Walton, 1993). Furthermore, Cheng et al. (2003) reported that AIDP was more common in spring compared to other seasons. In addition, mortality rate was 8.6% in our study and it had no

relation with seasons of the year and pioneer events. Mortality had been reported differently from 2-12% in the literature (Seneviratne, 2000; Victor and Roper, 2001; Cample, 1999).

In conclusion, in our region, there is no seasonal increase in incidence of GBS. Also, there is no significant relation between season of GBS occurrence with preceding infection, GBS pattern and mortality during a 12-year study period.

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#### References

1. Trojaborg W. Acute and chronic neuropathies: new aspects of Guillain-Barré syndrome and chronic inflammatory demyelinating polyneuropathy, an overview and an update. *Electroencephalogr Clin Neurophysiol*. 1998;107:303-316.
2. Seneviratne U. Guillain-Barré syndrome. *Postgrad Med J*. 2000;76:774-782.
3. Victor M, Roper A. Adams and Victor's Principle of Neurology, 7th ed. McGraw Hill, New York, 2001;1380-1387.
4. Aminoff M. Neurology and General Medicine, 3rd ed. Churchill Livingstone, USA, 2001;1011-1012.
5. Jacobs BC, Rothbarth PH, van der Meché FG, Herbrink P, Schmitz PI, de Klerk MA, van Doorn PA. The spectrum of antecedent infections in Guillain-Barré syndrome: a case-control study. *Neurology*. 1998;51:1110-1115.
6. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain-Barré syndrome. *J Infect Dis*. 1997;176 Suppl 2:S92-S98.
7. Cheng BC, Chang WN, Chang CS, Chee CY, Huang CR, Chen JB, Chang CJ, Hung PL, Wang KW, Chang HW, Lu CH. Guillain-Barré syndrome in southern Taiwan: clinical features, prognostic factors and therapeutic outcomes. *Eur J Neurol*. 2003;10:655-662.
8. Alter M. The epidemiology of Guillain-Barré syndrome. *Ann Neurol*. 1990;27 Suppl:S7-S12.
9. Rowland L. Merritt's Neurology, 11th ed. Lippincott Williams and Wilkins, USA, 2005;613-615.
10. Yuan CL, Tsou HK, Wang YJ, Tsai CP. Guillain-Barré syndrome: a retrospective, hospital-based study. *Zhonghua Yi Xue Za Zhi (Taipei)*. 2002;65:540-547.
11. Rocha MS, Brucki SM, Carvalho AA, Lima UW. Epidemiologic features of Guillain-Barré syndrome in São Paulo, Brazil. *Arq Neuropsiquiatr*. 2004;62:33-37.
12. Dyck PJ. *Peripheral Neuropathy*, 2nd ed. Saunders, Philadelphia, 1994;2050-2057.
13. Walton J. *Brain disease of the nervous system*. Oxford University Press, Oxford, 1993;599-603.
14. Hui AC, Chow KM, Tang AS, Fu M, Kay R, Wong KS. Electrophysiological, clinical and epidemiological study of Guillain-Barre Syndrome in Hong Kong Chinese. *J Clin Neurosci*. 2005;12:134-136.
15. Soffer D, Feldman S, Alter M. Epidemiology of Guillain-Barré syndrome. *Neurology*. 1978;28:686-690.
16. Chroni E, Papapetropoulos S, Gioldasis G, Ellul J, Diamadopoulos N, Papapetropoulos T. Guillain-Barré syndrome in Greece: seasonality and other clinico-epidemiological features. *Eur J Neurol*. 2004;11:383-388.
17. Sedano MJ, Calleja J, Canga E, Berciano J. Guillain-Barré syndrome in Cantabria, Spain. An epidemiological and clinical study. *Acta Neurol Scand*. 1994;89:287-292.
18. Olivé JM, Castillo C, Castro RG, de Quadros CA. Epidemiologic study of Guillain-Barré syndrome in children <15 years of age in Latin America. *J Infect Dis*. 1997;175 Suppl 1:S160-S164.
19. Lyu RK, Tang LM, Cheng SY, Hsu WC, Chen ST. Guillain-Barré syndrome in Taiwan: a clinical study of 167 patients. *J Neurol Neurosurg Psychiatry*. 1997;63:494-500.
20. Van Koningsveld R, Van Doorn PA, Schmitz PI, Ang CW, Van der Meché FG. Mild forms of Guillain-Barré syndrome in an epidemiologic survey in The Netherlands. *Neurology*. 2000;54:620-625.
21. Cheng Q, Jiang GX, Press R, Andersson M, Ekstedt B, Vrethem M, Liedholm LJ, Lindsten H, Brattström L, Fredrikson S, Link H, de Pedro-Cuesta J. Clinical epidemiology of Guillain-Barré syndrome in adults in Sweden 1996-97: a prospective study. *Eur J Neurol*. 2000;7:685-692.
22. Bogliun G, Beghi E; Italian GBS Registry Study Group. Incidence and clinical features of acute inflammatory polyradiculoneuropathy in Lombardy, Italy, 1996. *Acta Neurol Scand*. 2004;110:100-106.
23. Bahou YG, Biary N, al Deeb S. Guillain-Barre syndrome: a series observed at Riyadh Armed Forces Hospital January 1984--January 1994. *J Neurol*. 1996;243:147-152.
24. Goldman L, Bennett C. *Textbook of Cecil internal medicine*, Vol 2, 21st Ed. Saunders, New York, 2004;2193-2194.
25. Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. *Drugs*. 2004;64:597-610.
26. Alam TA, Chaudhry V, Cornblath DR. Electrophysiological studies in the Guillain-Barré syndrome: distinguishing subtypes by published criteria. *Muscle Nerve*. 1998;21:1275-1279.
27. Hiraga A, Mori M, Ogawara K, Hattori T, Kuwabara S. Differences in patterns of progression in demyelinating and axonal Guillain-Barré syndromes. *Neurology*. 2003;61:471-474.
28. Cample WI. *Essential of electro diagnostic medicine*. Lippincott Williams and Wilkins, USA, 1999;238-239.

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