Original Article

Epidemiologic and Clinical Characteristics of Guillain-Barré Syndrome in Patients Referred to Sina Hospital in Hamadan in 2018

Mojtaba Khazaei[1*] , Fatemeh Ghasemi[2] , Mehrdokht Mazdeh[3] , Elham Khanlarzadeh[4] , Masoud Ghiasian[1]

[1]Department of Neurology, Hamadan University of Medical Sciences, Hamadan, Iran
[2]Hamadan University of Medical Sciences, Hamadan, Iran
[3]Hearing Disorder Research Center, Sina (Farshchian) Educational and Medical Center, Hamadan University of Medical Sciences, Hamadan, Iran
[4]Department of Community Medicine, School of Medicine, Hamadan University of Medical Sciences, Hamadan, Iran

Abstract

Background and aims: Guillain-Barré syndrome (GBS) has several types, some of which damage myelin and some others cause axonal damage. Detecting the type of GBS is important in determining the type of treatment and its prognosis. This study was conducted to investigate the epidemiological characteristics of GBS and its variants in patients referred to Sina hospital in Hamadan, Iran, in 2018.

Methods: In this cross-sectional study, 51 patients who were admitted to Sina hospital and diagnosed with GBS in 2018 were examined. Demographic data, GBS type, disease outcomes, and pre-clinical and clinical findings of patients were collected. Data were then analyzed using the Stata software version 12, and P value < 0.05 was considered statistically significant.

Results: Of the 51 investigated patients, 34 (66.66%) were male. The most common variant type was acute inflammatory demyelinating polyneuropathy (AIDP) with 27.45% of cases followed by acute motor axonal neuropathy (AMAN) with 19.61% of cases. Further, the highest average hospitalization days (11.1 ± 11.7 days) were for chronic inflammatory demyelinating polyneuropathy (CIDP) patients, while the lowest (6.85 ± 1.91 days) was for AIDP patients (P < 0.001). All CIDP cases occurred in spring, and 71.43% of AIDP cases occurred in summer. Moreover, all 7 cases with acute motor and sensory axonal neuropathy (AMSAN) syndrome and the only case with the miller-fisher syndrome (MFS) occurred in fall (P < 0.001).

Conclusion: According to the results of this study, most variants of GBS in Hamadan province were AIDP in demyelinating form and AMAN variant in the axonal deterioration form. However, studies with a larger sample size are recommended in the west of Iran to better understand the epidemiology and to ensure common types of GBS.

Keywords: Guillain-Barré syndrome, Epidemiology, Sub-type, Iran

Introduction

Guillain-Barré syndrome (GBS) is an autoimmune neurological disease characterized by rapidly progressive symmetrical weakness of the extremities.[1] This post-infectious disease is considered the most severe acute paralytic neuropathy so that annually nearly 100 000 people can develop this disorder worldwide.[2] With regard to age distribution, the annual incidence rate of GBS ranges from 0.6 per 100 000 in children to 2.7 per 100 000 in people over 80 years old, and the male-to-female ratio is slightly greater than one.[3]

GBS is characterized by an acute onset and rapid progression. Symptoms begin with symmetric muscle weakness, bilateral limb weakness, and areflexia.[4,5] The paralysis is ascending in GBS, starting with weakness in the legs and spreading to the upper limbs.[6] The disease can progress for up to six weeks after onset, and this time

some patients may develop a few complications such as respiratory failure, sepsis, and cardiac arrhythmia.[6] The case fatality of GBS is 3%-7%.[7]

Similar to other autoimmune diseases, the etiology of GBS is multifactorial involving genetic and environmental factors.[8] It has been previously found that GBS is associated with chikungunya virus,[9] influenza,[10,11] *Campylobacter jejuni*,[12] poliovirus,[13] and hepatitis E.[14] In their study, Liu et al showed a strong association (OR = 9.5) between *C. jejuni* infection and GBS.[12] This strong association indicates that some infections as strong environmental agents are involved in the onset of GBS through several mechanisms. Regarding the relation between vaccinations and an increase in the risk of GBS, the results of most studies indicated no significant causal association[15,16]; however, Melnick et al reported a strong association (OR = 7.27) between polio vaccination and the development of GBS.[17]

Likewise, De Wals et al in their study in Canada found that the influenza A (H1N1) vaccine is associated with a small but significant risk of GBS.[18]

According to the clinical and pathologic spectrum, GBS has various clinical manifestations ranging from classic acute inflammatory demyelinating polyneuropathy (AIDP) and axonal variants to several clinical variants.[19] Determining the type of GBS is very important in allocating the proper treatment and predicting its prognosis. Failure to recognize GBS variants can lead to erroneous diagnoses and consequently inappropriate treatment. Given the superiority of some variants in each region, it is necessary to take medical actions and make economic decisions. Since epidemiological studies are the basis for choosing the type of treatment, this epidemiological study was conducted on GBS to determine its dominant variants in Hamadan, Iran.

Material and Methods
Study Design and Setting
This register-based cross-sectional study was conducted on all GBS patients referred to Sina hospital affiliated with Hamadan University of Medical Sciences as a referral center in Hamadan province for one year in 2018.

Eligibility Criteria
Only patients who were compatible with defined criteria according to the published international research diagnostic criteria by Asbury et al were included in this study.[20] These criteria were the presence of roughly symmetrical motor weakness over a period ranging from days to 4 weeks and a decrease or disappearance in deep tendon reflexes. Patients with diabetes mellitus or HIV/AIDS were excluded from the study because these diseases were related to AIDP, thus increasing the possibility of detection bias

Data Collection Tool
A researcher-developed checklist was used for gathering the data. Based on this checklist, patients' information, including age, sex, age of morbidity, length of hospitalization, clinical symptoms such as initial symptoms, neural findings, and laboratory and para-clinic findings. Other medical conditions were also collected, including familial history, underlying diseases, smoking, vaccination, complications, and special treatments (e.g., plasma purification and intravenous immunoglobulin). Further, the clinical data were collected from medical records of the hospitalized GBS patients during the study period. According to the telephone number in the medical records, patients were contacted to complete the incomplete information as well as disease progression.

Statistical Analysis
Descriptive analysis was used, including a frequency table, chart, mean, and standard deviation (SD) to describe and summarize data. Statistical analysis was run using the student's t test and one-way ANOVA for continuous variables, whereas the chi-square and Fisher's exact tests were used for categorical variables as appropriate. A level of 0.05 was considered significant for all statistical tests. Then, Stata software version 12 (StataCorp, College Station, TX, USA) was used to perform all the analytical operations.

Results
During one year, 51 cases of GBS were examined. The mean age of the patients was 54 ± 16.7 years (Ranges: 22-84 year), and 34 (66.67%) of them were male with a 54.29 ± 17.49 morbidity age. The most common variant type was AIDP with 27.45% of cases followed by acute motor axonal neuropathy (AMAN) with 19.61% of cases. Further, the clinical type was unknown in 10 patients. Patients with AMAN type had the lowest morbidity age (45.6 ± 13.5). Respiratory and gastrointestinal infections with 35.29% and 25.49% were the major etiology of GBS, respectively (Table 1).

As shown in Figure 1, none of the patients mentioned the family history of GBS in their first-degree relatives. Regarding risk factors and underlying disorders, 15.68% of them had a history of smoking, 23.52% had high blood pressure, 17.64% had lipid disorder, and 13.72% had cardiovascular disease.

According to Table 2, the average days of hospitalization in patients with determined variant type were 9.61+5.97 days with a range of 4-45 days. The highest average hospitalization days were for chronic inflammatory demyelinating polyneuropathy (CIDP) patients (11.1±11.7 days), and the lowest average hospitalization

Table 1. Gender and Age Distribution, Variant Type, and Etiology of GBS in Studied Patients

Variable		No. (%)	Morbidity Age (SD)
Gender	Male	34 (66.67)	54.29 (17.49)
	Female	17 (33.33)	57.06 (16.98)
Variant type	CIDP	9 (17.65)	49.22 (19.84)
	AIDP	14 (27.45)	54.43 (16.65)
	AMAN	10 (19.61)	45.6 (13.5)
	AMSAN	7 (13.73)	64.42 (12.31)
	MFS	1 (1.96)	82
	No data	10 (19.61)	62.2(16.44)
Etiology	Without history	14 (27.45)	53.5 (12.44)
	Vaccinating	1 (1.96)	54
	Gastrointestinal infections	13 (25.49)	53.54 (16.5)
	Respiratory infections	18 (35.29)	57.22 (21.34)
	Chemotherapy	3 (5.88)	53.67 (26.41)
	Surgery	1 (1.96)	70
	Delivery	1 (1.96)	44

Note. GBS: Guillain-Barré syndrome; SD: Standard deviation; CIDP: Chronic inflammatory demyelinating polyneuropathy; AIDP: Acute inflammatory demyelinating polyneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory axonal neuropathy; MFS: Miller-fisher syndrome.
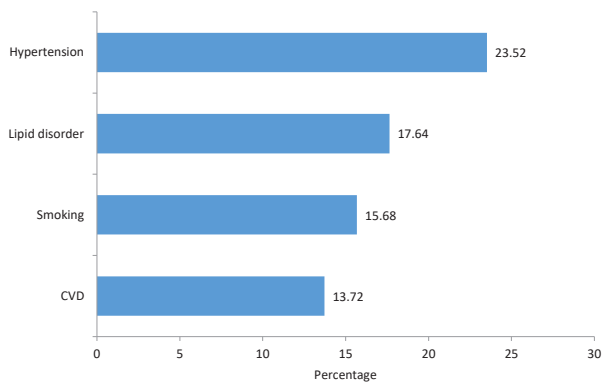
Figure 1. Risk Factors and Underlying Disorders Related to Investigated Patients.

days (6.85 ± 1.91 days) were for AIDP patients ($P < 0.001$). Moreover, there was no significant difference between age group and gender with syndrome type ($P > 0.05$). In 41 patients with determined syndrome type, 36.58% occurred in summer followed by 31.71% in spring. All CIDP cases occurred in spring and 71.43% of AIDP cases occurred in summer. All 7 cases with acute motor and sensory axonal neuropathy (AMSAN) syndrome and only one case with the miller-fisher syndrome (MFS) occurred in fall ($P < 0.001$).

The outcome of GBS according to GBS disorder type (demyelinating-axonal) is presented in Table 3.

As it can be observed, from 40 cases with known GBS disorder, 90% were recovered, of which 86.96% were in demyelinating and 94.12% in the axonal disorder type ($P = 0.62$). Further, 25% of cases had partial recovery, and 10% were completely disabled. Overall, patients with the demyelinating disorder had a better prognosis; that is, 65.22% had complete recovery in these patients compared to 41.18% in the patients with axonal disorder ($P = 0.38$). The most commonly observed complication was autonomic impairment, accounting for 67.5% of the cases, 10% had pulmonary infections, and 10% had respiratory and ventilation disorders.

Discussion

The present study was conducted to investigate the epidemiological characteristics of GBS and its variants in Hamadan province, Iran. According to the findings, AIDP in demyelinating form and AMAN variant in the axonal deterioration form were the most common GBS variants in Hamadan.

In other studies conducted in different geographical areas, different incidence of GBS has been reported. In Europe and North America, more than 90% of reported cases were AIDP type,[21] while in Asian countries, the type of axonal with 40-60% of cases is more prevalent.[22] In North America, MFS was slightly prevalent, accounting for 1% to

Table 2. Relation Between Variant Type and Hospitalization Duration, Age Group, Gender and Season

Variable		CIDP	AIDP	AMAN	AMSAN	MFS	Total	P Value
Duration (day)		11.7 (11.1)	6.85 (1.91)	11.5 (3.9)	10 (3.36)	8	9.61 (5.97)	<0.001
Age group (y)	22-30	1 (16.67)	2 (33.33)	2 (33.33)	1 (16.67)	0 (0)	6 (14.63)	
	31-50	4 (36.36)	2 (18.18)	4 (36.36)	1 (9.09)	0 (0)	11 (26.83)	0.72
	51-72	4 (16.67)	10 (41.67)	4 (16.67)	5 (20.83)	1 (4.17)	24 (58.54)	
Gender	Male	6 (21.43)	10 (35.71)	7 (25.0)	4 (14.29)	1 (3.57)	28 (68.29)	
	Female	3 (23.08)	4 (30.77)	3 (23.08)	3 (23.08)	0 (0)	13 (31.71)	0.92
Season	Spring	9 (100.0)	4 (28.57)	0 (0)	0 (0)	0 (0)	13 (31.71)	
	Summer	0 (0)	10 (71.43)	5 (50.0)	0 (0)	0 (0)	15 (36.58)	<0.001
	Fall	0 (0)	0 (0)	5 (50.0)	7 (100.0)	1 (100.0)	13 (31.71)	

Note. CIDP: Chronic inflammatory demyelinating polyneuropathy; AIDP: Acute inflammatory demyelinating polyneuropathy; AMAN: Acute motor axonal neuropathy; AMSAN: Acute motor and sensory axonal neuropathy; MFS: Miller-fisher syndrome.

Table 3. The Outcome of GBS According to GBS Disorder Type

Variable		Demyelinating	Axonal	Total	P Value
Survival status	Alive	20 (86.96)	16 (94.12)	36 (90.0)	
	Expire	3 (13.04)	1 (5.88)	4 (10.0)	0.62
Response	Complete recovery	15 (65.22)	7 (41.18)	22 (55.0)	
	Complete disable	2 (8.7)	2 (11.76)	4 (10.0)	
	Partial recovery	3 (13.04)	7 (41.18)	10 (25.0)	0.17
	Expire	3 (13.04)	1 (5.88)	4 (10.0)	
Complication	Autonomic impairment	15 (65.22)	12 (70.59)	27 (67.5)	
	Respiratory	1 (4.35)	3 (17.65)	4 (10.0)	
	UTI	1 (4.35)	1 (5.88)	2 (5.0)	0.38
	Pulmonary infection	3 (13.04)	1 (5.88)	4 (10.0)	
	Ventilator dependent	3 (13.04)	0 (0)	3 (7.5)	

Note. GBS: Guillain-Barré syndrome; UTI: Urinary tract infections.

7% of the GBS cases,[23] while in Japan and Taiwan, MFS accounted for more than 19% of GBS cases.[24] In this study, only 2% of patients had MFS. In Iran, adequate studies have not been conducted to address the epidemiology of this syndrome, especially in adults. In the conducted study by Ansari et al[25] in Isfahan, unlike the results of the present study, the AMSAN variant with 26% of cases was the most reported form. Consistent with the present findings, in the study by Yadegari et al[26] in Tehran, the AIDP variant was the most common form in 55.3% of cases. In another study by Salehiomran et al[27] in Babol, the results revealed that 17.6%, 12.6%, and 11.8% of cases were AMAN, AIDP, and AMSAN, respectively. Considering the high mean age of patients in the present study, the findings cannot be compared with Salehiomran et al. It should be noted that in the present study, the syndrome of about 20% of patients was not identified; therefore, the epidemiology of this self-immune syndrome cannot be adequately judged. The reason for these differences in the different regions of the world is still not well-known, but genetic factors, risk factors, and infections may play a role in this regard.[28]

GBS is more common in men than in women and has a lower rate in children than in adults. In the current study, the proportion of males to females was two, and the mean age of patients was 54 years. In most studies, the prevalence of the disease is more common in males than in females. The reason for this increased incidence with increasing age and high incidence in men is not yet clear. In autoimmune diseases, the incidence of the disease in women is higher than that of men, which is not the case with GBS. In this study, none of the patients reported a family history of GBS in their first-degree relatives.

Consistent with the results of the current study, in the study by Mazaheri et al[29] in Hamadan province in 2007, from 51 cases, 70% of them were male. Likewise, in the present study, 66% of patients were male. In the cross-sectional study conducted by Rahimi Jaberi et al[30] in 2013 in Shiraz, 214 patients were studied of whom 119 (55.6%) were male. In a study in Spain in 2015, of 45 patients with GBS, the male/female ratio was 1.4, and the mean age of patients was 48 years old, which was in agreement with the findings of this study.[31]

The findings of the current study indicated that among 51 patients, 37 (72.54%) had a history of a precursor event before the onset of the disease, 18 (48.64%) had respiratory infections, and 13 (35.13%) reported gastrointestinal infections. Based on the results of previous studies, in two-thirds of the patients before the onset of GBS, a precursor event (often an infectious disease) occurs, especially respiratory infections and gastrointestinal. In the present study, more than 70% of patients had a prognosis, nearly half of them had respiratory infections, and about 35% of them had gastrointestinal diseases. In a study by de la O-Peña et al[31] in Spain in 2015, 45 patients were diagnosed with GBS, 40% had respiratory infections, and 24.4% had a gastrointestinal infection, which is consistent with the findings of this study. One possibility is that

the mechanisms of infectious diseases by stimulating the immune system damage the myelin or the axons. Campylobacter is the most important microorganism detected in these patients and is detected in more than 30% of patients. Other factors include the Epstein-Barr virus, mycoplasma, para-influenza, HIV, and Zica virus.[32] Consistent with the results of the present study, in a study in the United States in 2009, respiratory and gastrointestinal infections were the most reported cases in GBS patients. In the study by Yazdchi et al on 43 patients diagnosed with GBS in Tabriz in 2005, 60% of patients had autonomic system involvement.[33] In the present study, the most common observed complications were autonomic dysfunctions (64.70%), and 19.60% of cases needed ventilation. In de la O-Peña and colleagues' study[31] in Spain, the highest number of cases were diagnosed in the summer as in the present study, and the most common episodes of symptoms were in the summer and autumn.

The present study suffered from a few limitations. Firstly, the collected data are based on registered medical records, and question from patients for incomplete variables is prone to information bias. Secondly, due to the small sample size, the generalizability of data should be made with caution.

Conclusion

According to the study, the most variants of GBS in Hamadan province were AIDP in demyelinating form and AMAN variant in the axonal deterioration form. Further, there is a seasonal variation in variant types of GBS in Hamadan. However, studies with a larger sample size are recommended in different parts of Iran to better understand the epidemiology and to ensure common types of GBS.

References
1. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. Lancet. 2021;397(10280):1214-28. doi: 10.1016/s0140-6736(21)00517-1.
2. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet. 2016;388(10045):717-27. doi: 10.1016/s0140-6736(16)00339-1.
3. Webb AJ, Brain SA, Wood R, Rinaldi S, Turner MR. Seasonal variation in Guillain-Barré syndrome: a systematic review, meta-analysis and Oxfordshire cohort study. J Neurol Neurosurg Psychiatry. 2015;86(11):1196-201. doi: 10.1136/jnnp-2014-309056.

4. Schessl J, Luther B, Kirschner J, Mauff G, Korinthenberg R. Infections and vaccinations preceding childhood Guillain-Barré syndrome: a prospective study. Eur J Pediatr. 2006;165(9):605-12. doi: 10.1007/s00431-006-0140-1.

5. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671-83. doi: 10.1038/s41582-019-0250-9.

6. Esposito S, Longo MR. Guillain-Barré syndrome. Autoimmun Rev. 2017;16(1):96-101. doi: 10.1016/j.autrev.2016.09.022.

7. Živković S. Intravenous immunoglobulin in the treatment of neurologic disorders. Acta Neurol Scand. 2016;133(2):84-96. doi: 10.1111/ane.12444.

8. Israeli E, Agmon-Levin N, Blank M, Chapman J, Shoenfeld Y. Guillain-Barré syndrome--a classical autoimmune disease triggered by infection or vaccination. Clin Rev Allergy Immunol. 2012;42(2):121-30. doi: 10.1007/s12016-010-8213-3.

9. Lebrun G, Chadda K, Reboux AH, Martinet O, Gaüzère BA. Guillain-Barré syndrome after chikungunya infection. Emerg Infect Dis. 2009;15(3):495-6. doi: 10.3201/eid1503.071482.

10. Stowe J, Andrews N, Wise L, Miller E. Investigation of the temporal association of Guillain-Barré syndrome with influenza vaccine and influenzalike illness using the United Kingdom General Practice Research Database. Am J Epidemiol. 2009;169(3):382-8. doi: 10.1093/aje/kwn310.

11. Sanz Fadrique R, Martín Arias L, Molina-Guarneros JA, Jimeno Bulnes N, García Ortega P. Guillain-Barré syndrome and influenza vaccines: current evidence. Rev Esp Quimioter. 2019;32(4):288-95.

12. Liu GF, Wu ZL, Wu HS, Wang QY, Zhao-Ri GT, Wang CY, et al. A case-control study on children with Guillain-Barré syndrome in North China. Biomed Environ Sci. 2003;16(2):105-11.

13. Kinnunen E, Junttila O, Haukka J, Hovi T. Nationwide oral poliovirus vaccination campaign and the incidence of Guillain-Barré Syndrome. Am J Epidemiol. 1998;147(1):69-73. doi: 10.1093/oxfordjournals.aje.a009369.

14. Loly JP, Rikir E, Seivert M, Legros E, Defrance P, Belaiche J, et al. Guillain-Barré syndrome following hepatitis E. World J Gastroenterol. 2009;15(13):1645-7. doi: 10.3748/wjg.15.1645.

15. Morris K, Rylance G. Guillain-Barré syndrome after measles, mumps, and rubella vaccine. Lancet. 1994;343(8888):60. doi: 10.1016/s0140-6736(94)90917-2.

16. Gervaix A, Caflisch M, Suter S, Haenggeli CA. Guillain-Barré syndrome following immunisation with Haemophilus influenzae type b conjugate vaccine. Eur J Pediatr. 1993;152(7):613-4. doi: 10.1007/bf01954093.

17. Melnick SC, Flewett TH. Role of infection in the Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry. 1964;27(5):395-407. doi: 10.1136/jnnp.27.5.395.

18. De Wals P, Deceuninck G, Toth E, Boulianne N, Brunet D, Boucher RM, et al. Risk of Guillain-Barré syndrome following H1N1 influenza vaccination in Quebec. JAMA. 2012;308(2):175-81. doi: 10.1001/jama.2012.7342.

19. Lin JJ, Hsia SH, Wang HS, Lyu RK, Chou ML, Hung PC, et al. Clinical variants of Guillain-Barré syndrome in children. Pediatr Neurol. 2012;47(2):91-6. doi: 10.1016/j.pediatrneurol.2012.05.011.

20. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol. 1990;27 Suppl:S21-4. doi: 10.1002/ana.410270707.

21. Hadden RD, Cornblath DR, Hughes RA, Zielasek J, Hartung HP, Toyka KV, et al. Electrophysiological classification of Guillain-Barré syndrome: clinical associations and outcome. Plasma Exchange/Sandoglobulin Guillain-Barré Syndrome Trial Group. Ann Neurol. 1998;44(5):780-8. doi: 10.1002/ana.410440512.

22. Ropper AH. Unusual clinical variants and signs in Guillain-Barré syndrome. Arch Neurol. 1986;43(11):1150-2. doi: 10.1001/archneur.1986.00520110044012.

23. Emilia-Romagna Study Group on Clinical and Epidemiological Problems in Neurology. Guillain-Barré syndrome variants in Emilia-Romagna, Italy, 1992-3: incidence, clinical features, and prognosis. J Neurol Neurosurg Psychiatry. 1998;65(2):218-24. doi: 10.1136/jnnp.65.2.218.

24. Yuan CL, Wang YJ, Tsai CP. Miller fisher syndrome: a hospital-based retrospective study. Eur Neurol. 2000;44(2):79-85. doi: 10.1159/000008201.

25. Ansari B, Basiri K, Derakhshan Y, Kadkhodaei F, Okhovat AA. Epidemiology and clinical features of Guillain-Barré syndrome in Isfahan, Iran. Adv Biomed Res. 2018;7:87. doi: 10.4103/abr.abr_50_17.

26. Yadegari S, Kazemi N, Nafissi S. Clinical and electrophysiological features of Guillain-Barré syndrome in Iran. J Clin Neurosci. 2014;21(9):1554-7. doi: 10.1016/j.jocn.2013.11.041.

27. Salehiomran MR, Nikkhah A, Mahdavi M. Prognosis of Guillain-Barré syndrome in children. Iran J Child Neurol. 2016;10(2):38-41.

28. Nagasawa K, Kuwabara S, Misawa S, Fujii K, Tanabe Y, Yuki N, et al. Electrophysiological subtypes and prognosis of childhood Guillain-Barré syndrome in Japan. Muscle Nerve. 2006;33(6):766-70. doi: 10.1002/mus.20520.

29. Mazaheri S, Rezaie AA, Hossein Zadeh A. The ten years survey on clinical and epidemiologic features of Guillain-Barré syndrome in Sina hospital, Hamadan, Iran. Avicenna J Clin Med. 2007;14(2):56-60. [Persian].

30. Rahimi Jaberi A, Manafi A, Mossallaiepoor A, Ebrahimi MM, Khazforoosh S, Shirazizadeh Mehraban SH, et al. The epidemiologic, clinical and laboratory findings of patients with Guillain-Barré syndrome in Southern Iran since 2007 to 2012. J Fasa Univ Med Sci. 2014;3(4):343-7. [Persian].

31. de la O-Peña D, Robles-Figueroa M, Chávez-Peña Q, Bedolla-Barajas M. [Features of Guillain-Barré syndrome in adults: results of a university hospital]. Rev Med Inst Mex Seguro Soc. 2015;53(6):678-85.

32. Blaser MJ, Olivares A, Taylor DN, Cornblath DR, McKhann GM. Campylobacter serology in patients with Chinese paralytic syndrome. Lancet. 1991;338(8762):308. doi: 10.1016/0140-6736(91)90447-w.

33. Yazdchi M, Arami M A, Najmi S, Mansoorpoor L. Evaluation of Heart Rate Variation In the Acute Phase of Guilain-Barre Syndrome And Its Relation to Disease Severity. Feyz 2005; 9 (2) :11-14