

# PERCENTAGE OF PATIENTS WHO SHOW AGREEMENT BETWEEN EMG AND CSF FINDINGS FOR THE DIAGNOSIS OF GBS

FARYAD HUSSAIN, NASIR ABBAS BALOCH, ZAINAB FATIMA, ARIF ZAHEER,  
PROF AGHA SHABBIR ALI

## ABSTRACT

**Objective:** to determine the percentage of patients who show agreement between EMG and CSF findings for the diagnosis of GBS.

**Study Design:** Cross sectional survey.

**Setting:** This study was conducted in the Department of Neurology and Medical ICU, The Children Hospital Lahore, from 01-10-2011 to 31-03-2012.

**Methodology:** 120 patients fulfilled the inclusion criteria were enrolled in the study. EMG was conducted and conduction velocities of motor and sensory nerves of lower and upper limbs and amplitude of compound motor action potential (CMAP's) were checked. After ten days of start of disease lumbar puncture was done with aseptic measures by the researcher and was sent to the central laboratory of the hospital for analysis of CSF.

**Results:** The mean age of the patients was  $5.6 \pm 2.8$  years. There were 72 (60.0%) male patients and 48 (40.0%) female patients. 116 (96.7%) patients had positive EMG findings and 4 (3.3%) patients had negative EMG findings. 66 (55%) patients had positive CSF findings and 54 (45%) patients had negative CSF findings. Thus at least 64 (53.3%) patients had an agreement between CSF and EMG findings.

**Conclusion:** It is concluded from this study that there is strong agreement between EMG findings and CSF findings for the diagnosis of Guillain Barre Syndrome in children.

**Keywords:** Guillain Barre Syndrome, EMG findings, CSF findings, agreement

## INTRODUCTION

Guillain Barre Syndrome (GBS) is a post infectious polyneuropathy involving mainly motor nerves but sometimes also involves the sensory and autonomic nerves. This syndrome affects people of all ages and has been reported throughout the world.<sup>1</sup> Most of the GBS patients have a demyelinating neuropathy but primarily axonal degeneration is documented in some cases.<sup>1</sup>

Paralysis usually follow a nonspecific viral infection, 1-3 weeks prior to the onset of weakness.<sup>2,3</sup> The original infection may occur in gastrointestinal tract (GIT), (especially by campylobacter jejuni, helicobacter pylori) or in respiratory tract (Mycoplasma pneumoniae). Vaccines have also been linked to GBS.<sup>4</sup>

The onset is gradual and progresses over days or weeks. Weakness begins in the lower extremities and may involve the trunk, upper limb and finally the bulbar muscles. Initially weakness can cause inability to walk and later to flaccid tetraplegia. Tendon reflexes are lost usually early in the course. Dysphagia and facial weakness are impending signs of respiratory failure. Recovery usually begins 2-4 weeks after the progressive phase.<sup>5,6</sup>

CSF studies are helpful for the diagnosis. CSF proteins are elevated to more than twice the upper normal limits. Glucose level is normal and there is no pleocytosis. Fewer than 10 WBCs per cubic millimeter are found. There is relatively increased CSF protein (46-300mg/dl) and low cellular count (<10/cmm) in about 80% of patients with GBS. Elevated Cerebrospinal fluid protein is seen in most of the patients particularly after first week of illness due to breakdown of blood nerve barrier within the subarachnoid space.<sup>7,8</sup>

Motor nerve conduction velocities are greatly reduced and sensory nerve conduction time is often slow during second week of illness. Electromyography (EMG) shows evidence of acute denervation of muscles. EMG is comparable with CSF for diagnosis of GBS But EMG is costly and is not available in many places. On the other hand CSF is a cheaper and readily available modality so it can be used as alternating method where EMG studies can not be done. EMG shows positive findings in 95% of the patients while CSF abnormalities (cytoalbuminal dissociation) is noted in 52% of the patients.<sup>9</sup> No local studies are available in

the literature so I want to explore the use of CSF examination as an alternative to EMG studies for the diagnosis of GBS in our population.

**METHODOLOGY**

The calculated sample size is 120 cases with 95% confidence level, 9% margin of error and taking expected percentage of patients i.e. 52% between EMG & CSF findings for diagnosis of GBS. Patients included in study were between 1 to 10 years of age, both male and females and all suspected cases of GBS as per operational definition. Patients who were on artificial ventilation or having signs of meningeal irritation (Neck stiffness, Kernig’s sign, Brudzinski sign) were excluded from the study.

120 patients presenting to the Neurology Department and Medical ICU, The Children Hospital Lahore fulfilling the inclusion criteria were enrolled in the study, after taking an informed consent and biodata. Demographic profile including age, gender and address were recorded. EMG was conducted by a senior Neurologist in The Children’s Hospital in which conduction velocities of motor and sensory nerves of lower and upper limbs and amplitude of compound motor action potential (CAMP’s) were checked using a specific machine by electrical stimulation. After ten days of start of disease lumbar puncture was done with aseptic measures by the researcher and was sent to the central laboratory of the hospital for analysis of positive CSF. All data was entered on a pre-designed Proforma. Both EMG & CSF findings were interpreted as positive or negative (as per operational definition) for determination of agreement for the diagnosis of GBS.

The acquired data was entered and analyzed through SPSS Version 10.0. Variables studied included age and gender. Mean and Standard Deviation was calculated for quantitative variables like age. Frequencies and Percentages were calculated for qualitative variables like gender. Agreement between EMG findings and CSF findings were calculated as frequency and percentage. Kappa statistics was used to determine the strength of agreement between EMG and CSF findings for the diagnosis of GBS.

**RESULTS**

The mean age of the patients was 5.6±2.8 years. There were 20 (16.7%) patients in the age range of 1.0 to 2.0 years, 26 (21.7%) patients in the age range of 2.1-4.0 years, 26 (21.7%) patients in the age range of 4.1-6.0 years, 21 (17.5%) patients in the age range of 6.1-8.0 years, 27 (22.5%) patients in the age range of 8.1-10.0 years (Table 1). In the distribution of patients by sex, there were 72 (60.0%) male patients and 48 (40.0%)

female patients (Table 2). In the distribution of patients by EMG finding 116 (96.7%) patients had positive findings and 4 (3.3%) patients had negative findings (Table 3). In the distribution of patients by CSF finding 66 (55%) patients had positive findings and 54 (45%) patients had negative findings (Table 4). In the distribution of patients with regard to confirmation of agreement, 64 (53.3%) patients had been confirmed as having agreement and 56 (46.7%) patients did not have confirmation of agreement (Table 5). In the distribution of patients by agreement between EMG finding and CSF finding, 116 (96.7%) EMG positive patients and 66 (55%) patients were positive with CSF finding with significant p value of <0.001 (Table 6).

**Table 1:** Distribution of patients by age (n=120)

Age (Years)	No. of patients	Percentage (%)
1.0-2.0	20	16.7
2.1-4.0	26	21.7
4.1-6.0	26	21.7
6.1-8.0	21	17.5
8.1-10.0	27	22.5
Mean±SD	5.6±2.8	

Key:  
n Number of patients  
D Standard deviation

**Table 2:** Distribution of patients by sex (n=120)

Sex	No. of patients	Percentage (%)
Male	72	60.0
Female	48	40.0
Total	120	100.0

Key: n Number of patients

**Table 3:** Distribution of patients by EMG findings (n=120)

EMG finding	No. of patients	Percentage (%)
Positive	116	96.7
Negative	4	3.3
Total	120	100.0

Key:  
n Number of patients  
EMG Electromyography

**Table 4:** Distribution of patients by CSF findings (n=120)

CSF finding	No. of patients	Percentage (%)
Positive	66	55.0
Negative	54	45.0
Total	120	100.0

Key:  
n Number of patients

CSF Cerebrospinal fluid

**Table 5:** Distribution Of Patients By Final Assessment Of Agreement (N=120)

Final assessment of agreement	No. of patients	Percentage (%)
Yes	64	53.3
No	56	46.7
Total	120	100.0

Key:

n Number of patients

**Table 6:** Agreement between EMG finding and CSF finding (n=120)

Finding	EMG finding		CSF finding		P value
	No. of patients	Percent age	No. of patients	Percent age	
Positive	116	96.7	66	55.0	0.001
Negative	4	3.3	54	45.0	

Key:

n Number of patients

## DISCUSSION

Although the occurrence of GBS in children is relatively rare, it is the most common cause for the development of acute flaccid paralysis among infants and children.<sup>52</sup> Since the first report of GBS in childhood by Mannier-Vinard in 1925, showing an incidence of 0.24–1.26 per 100,000 children under 15 years of age.<sup>53</sup> GBS has had a worldwide distribution which has affected all races and all ages, including the newborn.<sup>54</sup>

In our study the mean age of the patients was 5.6±2.8 years. As compared with the study of Akbayram et al<sup>9</sup> the mean age of the patients was 5.9±3.8 years, which is comparable with our study.

Gender ratios in individual reports in the literature vary from 1.5 to 2.7 males for one female.<sup>54</sup> The gender ratio in our series was 1.3 in favour of males. The occurrence of GBS in children increases with age, and it is quite rare in children younger than 2 years of age.<sup>52</sup>

In our study there were 60% male and 40% female patients. As compared with the study of Akbayram et al<sup>9</sup> there were 55.5% male and 44.5% female patients, which is comparable with our study.

CSF is characteristically acellular. Protein levels may be normal during the first week of the illness, but the majority will have an increase in protein if measured 2 or 3 weeks later. Elevated CSF protein concentration in GBS has been mainly associated with increased permeability of the blood–CSF barrier.<sup>54,55</sup>

CSF studies are helpful for the diagnosis. CSF proteins are elevated to more than twice the upper normal limits. Glucose level is normal and there is no pleocytosis. Fewer than 10 WBCs per cubic millimeter are found. There is relatively increased CSF protein (46–300mg/dl) and low cellular count (<10/cmm) in about 80% of patients with GBS. Elevated Cerebrospinal fluid protein is seen in most of the patients particularly after first week of illness due to breakdown of blood nerve barrier within the subarachnoid space.<sup>7,8</sup>

Motor nerve conduction velocities are greatly reduced and sensory nerve conduction time is often slow during second week of illness. Electromyography (EMG) shows evidence of acute denervation of muscles. EMG is comparable with CSF for diagnosis of GBS But EMG is costly and is not available in many places. On the other hand CSF is cheaper and readily available so it can be used as alternating method where EMG studies are not available. EMG shows positive findings in 95% of the patients while CSF abnormalities (cytoalbuminal dissociation) is noted in 52% of the patients.<sup>9</sup>

In our study there were 96.7% patients had EMG positive finding. As compared with the study of Akbayram et al<sup>9</sup> there were 95% patients positive with EMG finding, which is comparable with our study.

In our study there were 55% patients had CSF positive finding. As compared with the study of Akbayram et al<sup>9</sup> there were 52% patients positive with CSF positive finding, which is comparable with our study.

The onset is gradual and progress over days or weeks. Weakness begins in the lower extremities and may involve the trunk, upper limb and finally the bulbar muscles. Initially weakness can cause inability to walk and later lead to flaccid tetraplegia. Tendon reflexes are lost usually early in the course. Dysphagia and facial weakness are impending signs of respiratory failure.<sup>5,6</sup>

Optimal management and treatment of GBS is critically important because the stakes are life or death. Although many patients with GBS are desperately ill and paralyzed, their chances of a full recovery are high if they can overcome the acute stages. Thus, an important aspect of treatment is to provide maximum supportive care during the acute stages. A recent large, multicenter, randomized trial made a comparison between plasma exchange, intravenous exchange and combined treatment. Its final analysis revealed that there was no significant difference in efficacy between these three therapeutic regimens.<sup>52</sup>

The only new observation with patients treated with IVIG was acute relapse in 11.9% of the patients. A

relapse rate ranging from 1.4 to 46.7 was reported with use of IVIG.<sup>56,57</sup>

Studies of GBS that focused on both children and adults together found that respiratory support was required in about 20–30% of the patients.<sup>58,59</sup> Case fatality rates requiring mechanical ventilation for respiratory failure were estimated to be 15–30%.<sup>52</sup> Childhood GBS in about one-third of all patients needed ventilatory support for respiratory muscle paralysis, and about 10% of the patients died of the disease and its complications.<sup>60</sup>

GBS in children has a shorter course and is associated with a more complete recovery than GBS in adult patients. Despite modern treatment regimens, about 10–20% of adult GBS patients continued to be disabled.<sup>61,62,63</sup> Moreover, older age at onset was significantly associated with a poorer outcome at 1 year.<sup>61</sup> In a retrospective study including adult patients in Taiwan, 12.5% of the patients remained at Hughes scale grade 4–6 after 1 year.<sup>64</sup> In contrast, although approximately 40% of the children became nonambulant during their illness and 15–20% required ventilatory support, more than 90% recovered fully, with a small minority showing minimal residual impairment, such as weakness of the ankle dorsiflexor 1–4 months after onset, but were able to walk unaided.<sup>65,66</sup> After 1 year, only 14.3% of the pediatric GBS patients needed assistance in walking.<sup>52</sup> Moreover, about 72% of the children with GBS could walk independently 1 year after onset, more than twice the percentage of adults.<sup>67</sup>

In our study, in the agreement between EMG findings and CSF findings, 53.3% patients showed agreement between EMG findings and CSF findings in the diagnosis of GBS. As compared with the study of Akbayram et al<sup>9</sup> 52% patients showed agreement between EMG finding and CSF finding in the diagnosis of GBS.

On the basis of above discussion it is concluded that there is a strong agreement between EMG and CSF findings for the diagnosis of Guillain Barre Syndrome in children.

## CONCLUSION

It is concluded from this study that there is a strong agreement between EMG findings and CSF findings for the diagnosis of Guillain Barre Syndrome in children.

## REFERENCES

1. Kushnir N, Kellan C, Pollak M. Evolving pattern of Guillen Barre Syndrome in community hospital. *Acta Neurol Scand* 2008;117:347-50.

2. Baravelli M, Fantoni C, Rossi A. Guillain-Barre syndrome as a neurological complication of infective endocarditis. Is it really so rare and how often do we recognise it?. *Int J Cardiol* 2009;133:104-105.
3. Nelson L, Gormley R, Riddle MS. The epidemiology of Guillain-Barre Syndrome in U.S. military personnel: a case-control study. *BMC Res Notes* 2009;2:171.
4. Souayah N, Nasar A, Suri MF. Guillain-Barre syndrome after vaccination in United States: data from the Centers for Disease Control and Prevention/Food and Drug Administration Vaccine Adverse Event Reporting System (1990-2005). *J Clin Neuromuscul Dis* 2009;11:1-6.
5. El Mhandi L, Calmels P, Camdessanche JP. Muscle strength recovery in treated Guillain-Barre syndrome: a prospective study for the first 18 months after onset. *Am J Phys Med Rehabil* 2007;86:716-24.
6. Shafqat S, Khealani BA, Awan F, Abedin SE. Guillain-Barre syndrome in Pakistan: similarity of demyelinating and axonal variants. *Eur J Neurol* javascript:AL\_get(this, 'jour', 'Eur J Neurol. '); 2006;13:662-5.
7. Sladky JT, Ashwal S. Guillain-Barre Syndrome with central nervous system manifestation. In: Kenneth F, Swaiman Q, Ashwal S, Donna M. *Pediatric Neurology: Principles and Practice*; 4<sup>th</sup> edition USA: Elsevier; 2006:1924-25.
8. Shields RW, JR. Asa J. Wilbourn-Acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barre syndrome). Christopher G. Goetz, *Text Book of Clinical Neurology* 3<sup>rd</sup> edition China: Elsevier; 2007:1137-42.
9. Akbayram S, Dogan M, Akgun C, Peker E, Sayin R, Aktar F, et al. Clinical features and prognosis with GBS. *Ann Indian Acad Neurol* 2011; 14:98-102.
10. Kuwabara S. Guillain-Barre syndrome: epidemiology, pathophysiology and management. *Drugs* 2004;64:597-610.
11. Lee JH, Sung IY, Rew IS. Clinical presentation and prognosis of childhood Guillain-Barre syndrome. *J Paediatr Child Health* 2008;44:449-54.
12. Seneviratne U. Guillain-Barre syndrome. *Postgrad Med J* 2000;76:774-82.
13. 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;67:1837-43.
14. Souayah N, Nasar A, Suri MF, Qureshi AI. Guillain-Barre syndrome after vaccination in United States: data from the Centers for Disease Control and Prevention/Food and Drug Administration Vaccine Adverse Event Reporting System (1990-2005). *J Clin Neuromuscul Dis* 2009;11:1-6.
  15. CDC. Estimating Deaths from Seasonal Influenza in the United States. Accessed Dec 22, 2009. Available at [http://www.cdc.gov/flu/about/disease/us\\_flu-related\\_deaths.htm](http://www.cdc.gov/flu/about/disease/us_flu-related_deaths.htm).
  16. Preliminary results: surveillance for Guillain-Barre syndrome after receipt of influenza A (H1N1) 2009 monovalent vaccine - United States, 2009-2010. *MMWR Morb Mortal Wkly Rep* 2010;59:657-61.
  17. World Health Organization. Programmes and Projects - Global Alert and Response (GAR). Pandemic (H1N1) 2009 briefing notes. World Health Organization. Available at [http://www.who.int/csr/disease/swineflu/notes/briefing\\_20091119/en/](http://www.who.int/csr/disease/swineflu/notes/briefing_20091119/en/). Accessed January 8, 2012.
  18. Tremblay ME, Closon A, D'Anjou G, Bussieres JF. Guillain-Barre syndrome following H1N1 immunization in a pediatric patient. *Ann Pharmacother* 2010;44:1330-3.
  19. da Silveira CM, Salisbury DM, de Quadros CA. Measles vaccination and Guillain-Barre syndrome. *Lancet* 1997;349:14-6.
  20. Slade BA, Leidel L, Vellozzi C, Woo EJ, Hua W, Sutherland A, et al. Postlicensure safety surveillance for quadrivalent human papillomavirus recombinant vaccine. *JAMA* 2009;302:750-7.
  21. Smith MJ. Meningococcal tetravalent conjugate vaccine. *Expert Opin Biol Ther* 2008;8:1941-6.
  22. Landaverde JM, Danovaro-Holliday MC, Trumbo SP, Pacis-Tirso CL, Ruiz-Matus C. Guillain-Barre syndrome in children aged < 15 years in Latin America and the Caribbean: baseline rates in the context of the influenza A (H1N1) pandemic. *J Infect Dis* 2010;201:746-50.
  23. Griffin JW, Li CY, Ho TW, Tian M, Gao CY, Xue P, et al. Pathology of the motor-sensory axonal Guillain-Barre syndrome. *Ann Neurol* 1996;39:17-28.
  24. Griffin JW, Li CY, Ho TW, Xue P, Macko C, Gao CY, et al. Guillain-Barre syndrome in northern China. The spectrum of neuropathological changes in clinically defined cases. *Brain* 1995;118:577-95.
  25. Nachamkin I, Arzarte-Barbosa P, Ung H, Lobato C, Gonzalez Rivera A, Rodriguez P, et al. Patterns of Guillain-Barre syndrome in children: results from a Mexican population. *Neurology* 2007;69:1665-71.
  26. Kalra V, Chaudhry R, Dua T, Dhawan B, Sahu JK, Mridula B. Association of Campylobacter jejuni infection with childhood Guillain-Barré syndrome: a case-control study. *J Child Neurol* 2009;24:664-8.
  27. Barzegar M, Alizadeh A, Toopchizadeh V, Dastgiri S, Majidi J. Association of Campylobacter jejuni infection and Guillain-Barré syndrome: a cohort study in the northwest of Iran. *Turk J Pediatr* 2008;50:443-8.
  28. Islam Z, Jacobs BC, Islam MB, Mohammad QD, Diorditsa S, Endtz HP. High incidence of Guillain-Barre syndrome in children, Bangladesh. *Emerg Infect Dis* 2011;17:1317-8.
  29. Korinthenberg R, Schessl J, Kirschner J. Clinical presentation and course of childhood Guillain-Barre syndrome: a prospective multicentre study. *Neuropediatrics* 2007;38:10-7.
  30. Shafqat S, Khealani BA, Awan F, Abedin SE. Guillain-Barre syndrome in Pakistan: similarity of demyelinating and axonal variants. *Eur J Neurol* 2006;13:662-5.
  31. Kalra V, Sankhyani N, Sharma S, Gulati S, Choudhry R, Dhawan B. Outcome in childhood Guillain-Barre syndrome. *Indian J Pediatr* 2009;76:795-9.
  32. Roodbol J, de Wit MC, Walgaard C, de Hoog M, Catsman-Berrevoets CE, Jacobs BC. Recognizing Guillain-Barre syndrome in preschool children. *Neurology* 2011;76:807-10.
  33. al-Qudah AA, Shahar E, Logan WJ, Murphy EG. Neonatal Guillain-Barre syndrome. *Pediatr Neurol* 1988;4:255-6.
  34. Kieseier BC, Kiefer R, Gold R, Hemmer B, Willison HJ, Hartung HP. Advances in understanding and treatment of immune-mediated disorders of the peripheral nervous system. *Muscle Nerve* 2004;30:131-56.
  35. Hughes RA. The concept and classification of Guillain-Barre syndrome and related disorders. *Rev Neurol (Paris)* 1995;151:291-4.
  36. Schwerer B. Antibodies against gangliosides: a link between preceding infection and immunopathogenesis of Guillain-Barre syndrome. *Microbes Infect* 2002;4:373-84.
  37. Ilyas M, Tolaymat A. Minimal change nephrotic syndrome with Guillain-Barre syndrome. *Pediatr Nephrol* 2004;19:105-6.

38. Heiner JD, Ball VL. A child with benign acute childhood myositis after influenza. *J Emerg Med* 2010;39:316-9.
39. Lacroix LE, Galetto A, Haenggeli CA, Gervaix A. Delayed recognition of Guillain-Barre syndrome in a child: a misleading respiratory distress. *J Emerg Med* 2010;38:e59-61.
40. Gorson KC, Ropper AH, Muriello MA, Blair R. Prospective evaluation of MRI lumbosacral nerve root enhancement in acute Guillain-Barre syndrome. *Neurology* 1996;47:813-7.
41. Nishimoto Y, Susuki K, Yuki N. Serologic marker of acute motor axonal neuropathy in childhood. *Pediatr Neurol* 2008;39:67-70.
42. Schessl J, Koga M, Funakoshi K, Kirschner J, Muellges W, Weishaupt A, et al. Prospective study on anti-ganglioside antibodies in childhood Guillain-Barre syndrome. *Arch Dis Child* 2007;92:48-52.
43. Hughes RA, Wijdicks EF, Barohn R, Benson E, Cornblath DR, Hahn AF, et al. Practice parameter: immunotherapy for Guillain-Barre syndrome: report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2003;61:736-40.
44. Yata J, Nihei K, Ohya T, Hirano Y, Momoi M, Maekawa K, et al. High-dose immunoglobulin therapy for Guillain-Barré syndrome in Japanese children. *Pediatr Int* 2003;45:543-9.
45. Korinthenberg R, Schessl J, Kirschner J, Monting JS. Intravenously administered immunoglobulin in the treatment of childhood Guillain-Barre syndrome: a randomized trial. *Pediatrics* 2005;116:8-14.
46. Shahar E. Current therapeutic options in severe Guillain-Barre syndrome. *Clin Neuropharmacol* 2006;29:45-51.
47. Baranwal AK, Ravi RN, Singh R. Exchange transfusion: a low-cost alternative for severe childhood Guillain-Barre syndrome. *J Child Neurol* 2006;21:960-65.
48. Centers for Disease Control and Prevention (CDC). Preliminary results: surveillance for Guillain-Barre syndrome after receipt of influenza A (H1N1) 2009 monovalent vaccine - United States, 2009-2010. *MMWR Morb Mortal Wkly Rep* 2010;59:657-61.
49. Yang YR, Liu SL, Qin ZY, Liu FJ, Qin YJ, Bai SM, et al. Comparative proteomics analysis of cerebrospinal fluid of patients with Guillain-Barre syndrome. *Cell Mol Neurobiol* 2008; 28:737-44.
50. Jin T, Hu LS, Chang M, Wu J, Winblad B, Zhu J. Proteomic identification of potential protein markers in cerebrospinal fluid of GBS patients. *Eur J Neurol* 2007;14:563-8.
51. Brettschneider J, Petzold A, Süßmuth S, Tumani H. Cerebrospinal fluid biomarkers in Guillain-Barré syndrome--where do we stand? *J Neurol* 2009; 256: 3-12.
52. Hung PL, Chang WN, Huang LT, Huang SC, Chang YC, Chang CJ, et al. A clinical and electrophysiologic survey of childhood Guillain-Barre syndrome. *Pediatr Neurol* 2004; 30:86-91.
53. Koul R, Chacko A, Ahmed R, Varghese T, Javed H, Al-Lamki Z. Ten-year prospective study (clinical spectrum) of childhood Guillain-Barre syndrome in the Arabian peninsula: Comparison of outcome in patients in the pre- and post-intravenous immunoglobulin eras. *J Child Neurol* 2003; 18:767-71.
54. Ammache Z, Afifi AK, Brown CK, Kimura J. Childhood Guillain-Barre syndrome: Clinical and electrophysiologic features predictive of outcome. *J Child Neurol* 2001;16:477-83.
55. Winer JB. Guillain-Barre syndrome. *J Clin Pathol Mol Pathol* 2001;54:381-5.
56. Vander-Meche FG, Schmitz PI. The Dutch Guillain-Barre syndrome study group. A randomised trial comparing intravenous immunoglobulins and plasma exchange in Guillain-Barre syndrome. *N Engl J Med* 1992;326:1123-9.
57. Castro LH, Ropper AH. Human immunoglobulin infusion in Guillain-Barre syndrome: Worsening during and after treatment. *Neurology* 1993;43:1034-6.
58. Feasby TE, Gilbert JJ, Brown WF, Bolton CF, Hahn AF, Koopman WF, et al. An acute axonal form of Guillain-Barre polyneuropathy. *Brain* 1986;109:1115-26.
59. Winer JB, Osmond C. A prospective study of acute idiopathic neuropathy: I: Clinical features and their prognostic value. *J Neurol Neurosurg Psychiatr* 1988;51:605-12.
60. Koul RL, Alfutaisi A. Prospective study of children with Guillain-Barre syndrome. *Indian J Pediatr* 2008;75:787-90.
61. Lee JH, Sung IY, Rew IS. Clinical presentation and prognosis of childhood Guillain-Barre syndrome. *J Paediatr Child Health* 2008;44:449-54.
62. Cosi V, Versino M. Guillain-Barre syndrome. *Neurol Sci* 2006;27:S47-51.
63. Bersano A, Carpo M, Allaria S, Franciotta D, Citterio A, Nobile-Orazio E. Long term disability

- and social status change after Guillain-Barre syndrome. *J Neurol* 2006;253:214–8.
64. Cheng BC, Chang WN, Chang CS, Chee CY, Huang CR, Chen JB, et al. Guillain-Barre syndrome in southern Taiwan: Clinical features, prognostic factors and therapeutic outcomes. *Eur J Neurol* 2003;10:655–62.
65. Bradshaw DY, Jones HR Jr. Guillain-Barre syndrome in children: Clinical course, electrodiagnosis, and prognosis. *Muscle Nerve* 1992;15:500–6.
66. Korinthenberg R, Monting JS. Natural history and treatment effects in Guillain-Barre syndrome: A multicentre study. *Arch Dis Child* 1996;74:281–7.
67. Sarada C, Tharakan JK, Nair M. Guillain-Barre syndrome: A prospective clinical study in 25 children and comparison with adults. *Ann Trop Paediatr* 1994;14:281–6.