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GENERAL CONSIDERATION OF GUILLIAIN BARRE SYNDROME

Zeel A. Gandhi, Brijal A. Maisuria, Rajesh G. Patel and Bhoomi Arora*

Institute of Clinical Research India, Ahmedabad, Gujarat, India

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Correspondence to Author:

Zeel A. Gandhi

I-5, Bhalchandra Flats, Near Vasna bus stand, Vasna, Ahmedabad – 380007, Gujarat, India

E-mail: zgandhi3107@yahoo.com

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ABSTRACT

Guillain-Barré syndrome is a rare but important disease that can lead to life threatening respiratory failure. This review summarises the verified consideration of Guillain-Barré syndrome (GBS) which are thought to be caused by direct autoimmune attack on peripheral nerves. Aim- Nerve conduction study helps differentiate the heterogeneous subtypes of GBS. Patients exhibit a progressive paralysis that reaches a plateau phase. Treatment and Result- Treatment with both intravenous immunoglobulin and plasma exchange reduces the time taken for recovery to occur, although mortality remains around 8%, with about 20% of patients remaining disabled. Though no significant differences were found between patients with Guillain Barre syndrome treated with plasma exchange and patients treated with intravenous immune globulins either alone or in combination with high dose methylprednisolone. Conclusion- It can be concluded that tentative and the gap in current research should not be interpreted as proof that multidisciplinary care is ineffective. Further research is needed into appropriate study designs; outcome measurement; caregiver needs; and the evaluation of optimal settings, type, intensity or frequency and cost-effectiveness of multidisciplinary care in the Guillain-Barré syndrome population.

INTRODUCTION: Almost a century ago, the French neurologists Guillain, Barre, and Strohl described two soldiers who developed acute paralysis with areflexia that spontaneously recovered [1]. They reported the combination of increased protein concentration with a normal cell count in the CSF, or albuminocytological dissociation, which differentiated the condition from poliomyelitis [1]. Guillain-Barre' Syndrome (GBS) is the most common cause of acute flaccid paralysis [2]. GBS is an acute-onset, monophasic, immune-mediated, an autoimmune disorder of the peripheral nervous system (PNS) with a range of presentations from mild to life threatening paralysis. They reported the combination of increased protein concentration with a normal cell count in the CSF, or albuminocytological dissociation, which differentiated the condition from poliomyelitis [1]. Despite the fact that Landry had already reported similar cases in 1859 [3] the combination of these clinical and laboratory features became known as Guillain-Barre syndrome (GBS). In this review, we will focus on sub types, pathophysiology, trigger factor, symptoms, diagnosis and drug therapy of Guillain-Barre Syndrome**.**

GBS's peripheral nerve damage can be histopathologically classified into two main types: demyelination and axonal degeneration. Motor nerve fibers are more susceptible to the disease than sensory ones. In 1995, GBS was subdivided into four main distinct forms based on histopathological and neurophysiological properties [4, 5].

Acute Inflammatory Demyelinatin Polyneuropathy (AIDP): The most common and least well understood entity is acute inflammatory demyelinating polyneuropathy (AIDP) that probably constitutes about 75% of the syndrome. Careful neurophysiological assessment will usually show a demyelinating neuropathy. Although not usually required for diagnosis, histological study of nerve at biopsy or post mortem material reveals perivascular infiltrates and demyelination [6]. Motor and sensory fibers are usually affected simultaneously and produce corresponding neurological deficits.

Acute Motor Axonal Neuropathy (AMAN): Acute motor axonal neuropathy (AMAN) is an axonal, entirely motor disorder which is commonly associated with antibodies against gangliosides, especially GM1 [7]. Specific binding of antibodies to the axonal membrane of motor fibres, predominantly at the nodes of Ranvier, complement activation, and intrusion of macrophages into the periaxonal space result in destruction of motor axons while lymphocytic infiltration is rare. Gangliosides have been considered as the most promising candidate targets. *C. jejuni* is the commonest preceding infectious agent, and the increasing level of anti-ganglioside antibodies is usually found in this type of GBS.

Acute Motor and Sensory Axonal Neuropathy (AMSAN): AMSAN is an axonal disorder similar to AMAN with the exception that the sensory nerves are also involved. This subtype is very few (less than 10% of AMAN cases). Its pathological pattern closely resembles that in AMAN, including damage and degeneration of axons, except that sensory nerves are affected simultaneously AMSAN is usually associated with a more severe course and poorer prognosis [4, 8].

Miller Fisher Syndrome (MFS): MFS is an infrequent variant of GBS (around 5%).The involvement of the cranial nerves is very distinct in this syndrome, and ocular motor (oculomotor, trochlear and abducens) nerves are usually affected and produce typical clinical trial of ophthalmoplegia, ataxia, and areflexia. GQ1b is enriched in human ocular-muscle nerves, which may account for the vulnerability of these nerves to humoral immune mediated attack in the MFS patients apart from the involvement of some other factors in its pathogenesis [4].

Epidemiology: The annual incidence of GBS is reported to be 1.2 – 2.3 per 100 000 [9-15]. GBS occurs in all age groups with a slight increase in the young adult and elderly populations [16]. Joseph and Tsao [17] report the youngest recorded patient as four years old and the oldest patient as 95 years old. GBS affects both males and females, but males are about 1.5 times more likely to be affected than females [18]. GBS is found to be slightly more common in Caucasians than African-Americans in the United States. A recent epidemiological report from the USA indicated that the incidence of GBS among patients aged 18 years or older did not change over the period from 2000 to 2004 [12]. Reports on temporarily increased incidences of GBS are rare. In the West incidence increases with age, but in China the incidence of all forms across age groups is more uniform. Acute motor axonal neuropathy (AMAN) is the commonest form in China and shows a marked seasonal variation and paediatric predominance [19].

Pathogenesis: The major thrust in understanding the pathogenesis of inflammatory neuropathy has been the identification of antibodies to gangliosides that correlate with different clinical patterns of neuropathy [20]. The presence of antibodies and activated T-cells that react against the peripheral myelin gives rise to the theory that GBS is a type of autoimmune pathology. Macrophages invade the Schwann cell, strip myelin from the axon, and sometimes degeneration of the axon occurs, which may be secondary to an autoimmune attack on the axon or myelin. Autoreactive T-cells are thought to be “activated” and mediate this autoimmune response by initiating the cascade to inflammation [21]. There is a very close association between antibodies to ganglioside GQ1b and Fisher syndrome.

In the other conditions where these antibodies are found, there are clinical features, usually ophthalmoplegia, which form part of Fisher syndrome. There is still some disagreement between laboratories about the details. However sera from patients with Fisher syndrome do contain antibodies probably directed against ganglioside GQ1b that bind to terminal motor nerve fibres and induce conduction block [22, 23, 24].

Investigations continue to discover whether the best fit with axonal neuropathy is with antibodies to ganglioside GM1 or a related ganglioside such as GD1a, N-acetylgalactosaminyl GD1a, or GM1b [20, 25]. Interest in antibodies has deflected attention from T cell responses in the pathogenesis of inflammatory demyelinating polyradiculoneuropathy, probably inappropriately. Experimental autoimmune neuritis is an accurate model of the neurophysiological and pathological features of human inflammatory demyelinating polyradiculoneuropathy. It is clearly a primarily T cell mediated disease which can be induced by immunization with P0, P2 and now, PMP22 myelin proteins [26]. Although, each subtype of GBS presumably has a relatively independent immunopathogenesis.

Trigger Factor: Evidence of preceding *Campylobacter jejuni* infection is found in about 25% of GBS cases. Cytomegalovirus and Epstein–Barr virus occur in about 10% of cases [27]. The accurate etiology of GBS is not yet completely understood. The commonly identified preceding pathogens are as follows: *Campylobacter jejuni (C. jejuni), Haemophilus influenzae, Mycoplasma pneumonia, Herpes zoster*, cytomegalovirus and *Epstein Barr virus*.

Some other rare GBS associated antecedent events have been reported such as surgery, cancer, pregnancy, autoimmune diseases, use of drugs, spinal anesthesia, non-Hodgkin's lymphoma, insect stings, leigh syndrome, epidural-general anesthesia, surgery for obesity, olanzapine administration and transplantation operations. Several cases have been found to develop GBS after therapeutic injection of bovine brain ganglioside preparations [28].

Clinical features: The cardinal clinical features of Guillain-Barré syndrome (GBS) are progressive, fairly symmetric muscle weakness accompanied by absent or depressed deep tendon reflexes. Patients usually present a few days to a week after onset of symptoms. The weakness can vary from mild difficulty with walking to nearly complete paralysis of all extremity, facial, respiratory, and bulbar muscles.

Studies from the United States and Europe, reflecting primarily patients with acute inflammatory demyelinating polyneuropathy (AIDP), show that GBS is associated with the following clinical features [29]. Although the weakness usually starts in the legs, it begins in the arms or facial muscles in about 10 percent of patients. Severe respiratory muscle weakness necessitating ventilatory support develops in 10 to 30 percent [12]. Facial weakness occurs in more than 50 percent and oropharyngeal weakness eventually occurs in 50 percent. Oculomotor weakness occurs in about 15 percent of patients. Paresthesias in the hands and feet accompany the weakness in more than 80 percent of patients, but sensory abnormalities on examination are frequently mild.

Pain, typically located in the back and extremities, can be a presenting feature and is reported during the acute phase by 66 percent of patients with all forms of GBS [30, 31]. Dysautonomia occurs in 70 percent of patients and manifests as symptoms that include tachycardia (the most common), urinary retention, hypertension alternating with hypotension, orthostatic hypotension, bradycardia, other arrhythmias, ileus, and loss of sweating. Severe autonomic dysfunction is important to recognize since this is occasionally associated with sudden death [31].

Diagnosis: Cerebrospinal fluid (CSF) examination is needed largely to exclude alternative diagnoses, such as infectious (for example, *Borrelia* or poliomyelitis) or lymphomatous polyradiculitis. The CSF protein is classically elevated as a result of albumin leakage from the blood, but may be normal within the first week [33]. The risk of developing GBS after *C. jejuni* enteritis is less than 1 in 2500 [34].

Probably the diagnosis of GBS is based on typical clinical features; electrodiagnostic examination and examination of the cerebrospinal fluid [35, 36]. An elevated level of CSF protein without an increase in the number of cells, albominocytologic dissociation, is the cardinal laboratory finding in GBS [37], in case of childhood Guillain Barre Syndrome. Electrophysiological features differ according to the clinicopathological type (Box 1) [38, 39, 40]

Box 1: Electrophysiological features

- Acute inflammatory demyelinating polyneuropathy (AIDP) Reduced conduction velocity Conduction block or abnormal temporal dispersion Prolonged terminal latency Absent F wave or prolonged F wave latency.

- Acute motor axonal neuropathy (AMAN) absent or reduced compound muscle action potential (CMAP) amplitude Normal motor terminal latency and conduction velocity Normal sensory nerve action potential (SNAP).

- Acute motor and sensory axonal neuropathy (AMSAN) absent or reduced SNAP amplitude Absent or reduced CMAP amplitude Normal motor terminal latency and conduction velocity

An acute progressive symmetric weakness of the extremities with areflexia or hyporeflexia, CSF showing albuminocytological dissociation and electrophysiology revealing features of demyelinating/axonal neuropathy [41] is analyzed in diagnosing GBS. There may be signs of decreased breathing caused by paralysis of the breathing muscles. The following tests may be ordered: Cerebrospinal fluid sample ("spinal tap"), ECG, Electro-myography (EMG) tests the electrical activity in muscle, Nerve conduction velocity test, pulmonary function tests.

Treatment: The following points are important in the affective prognosis:

Plasma exchange (PE): It is the first treatment for GBS and is most beneficial when started within 7 days of disease onset and of some benefit if started within 30 days of the onset [42]. Plasmapheresis shortens the time a patient stays on respiratory support, the time required to achieve independent walking, and is associated with greater functional mobility at 6 months [43, 44] and reduces the amount of residual weakness compared with no Treatment [45].

PE has also been compared to CSF fluid filtration in a single randomized trial [46]. In this trial, 20 patients were assigned to PE (five or six sessions) and compared with 17 patients treated with CSF filtration. The CSF filtration consisted of five or six cycles of 30 to 50 ml of CSF filtered and reinstilled daily for 15 days. Median improvement of clinical grades was not significantly different at four weeks, nor was there any significant advantage to CSF filtration. The establishment of plasma exchange as the gold standard treatment for GBS, two large and some smaller trials have shown that intravenous immunoglobulin has equivalent efficacy [47].

The French studies used larger exchanges of 1.5 plasma volumes. Two trials have investigated the amount of PE. In one trial, patients who could not stand unaided and who did not need respiratory assistance were randomized to either two or four 1.5 plasma volume exchanges [48]. Significantly more participants (93/155, 64%) treated with four PEs recovered full muscle strength after a year than those treated with two PEs (67/149, 48%), RR 1.35 (95% CI 1.09–1.67, P¼0.006).

In a parallel trial, 161 ventilator-dependent GBS patients were randomized to receive either six PEs or four PEs. There was no significant difference between the two regimens in the same measure of recovery [49]. In most studies, the replacement fluid has been a mixture of albumen and saline. In one study, 57 patients were randomly allocated to receive PE with albumen and gelatine as replacement fluids, and 52 received PE with fresh frozen plasma as the replacement fluid. There was no significant difference between the two groups in any measure of recovery [48].

Intravenous Immunoglobulin (IVIg): Immunoglobulin infusion hastens recovery in GBS as much as plasmaphoresis [49]. Administration of IVIg after plasma exchange has no added advantage over plasma exchange alone [50]. Intravenous immunoglobulin (IVIg) was introduced for the treatment of auto-immune thrombocytopaenia [51] and tried for the treatment of chronic inflammatory demyelinating polyneuropathy [52].

A favourable response in patients with GBS was reported in 1988 [53] and led to the first randomized controlled trial. A meta-analysis of IVIg for GBS found three randomized trials that compared IVIg with PE [54, 55] and the only trial comparing IvIg with supportive treatment was considered inadequate to establish its value [56].

High-dose immunoglobulin: The empirical dose of IVIg generally used for the treatment of GBS is 0.4 g/kg per day for 5 days. There was a non-significant trend toward a better outcome noted in the group receiving longer treatment of 6 days, and this trend reached significance when only ventilated patients were considered, but the shorter course such as 3 days was proven to be significantly less effective [57, 58, 59].

In pediatric patients with GBS, IVIg significantly hastens the recovery of patients and has also been found to be effective and safe. The mechanisms of action of IVIg have not been fully understood, but it is known that IVIg has multiple functions including down regulation of antibody production, acceleration of antibody metabolism, neutralization of complement-mediated effects, interference with antibody-dependent cytotoxicity mediated by macrophages, modulation of nitric oxide production and microglial function, direct effects on T-cell activation, inhibition of cell adhesion, and induction of apoptosis. Any or all of these could be the predominant mechanisms of IVIg in the treatment of GBS [60, 61, 62].

Both plasma exchange and intravenous immunoglobulin are expensive, inconvenient and only effective in the short term. A continued search for non-toxic immunosuppressive agents for inflammatory neuropathies has embraced azathioprine, cyclophosphamide, cyclosporin, beta interferon and more recently mycophenolate mofetil. None have been rigorously demonstrated to be beneficial.

Immunomodulating treatment: Effective immuno-modulating treatment can lessen nerve damage, reduce progression, and shorten hospitalization. Plasmapheresis and IVIg is the mainstay of immunomodulatory treatment at present. Both treatments have proven to exhibit beneficial effects in various controlled trials by favourably altering the natural course of the disease. Their effectiveness is similar and both appear to be more effective than supportive treatment alone. Corticosteroids are still a doubtful topic in the treatment of GBS.

Steroids: Treatment of GBS with steroids was ineffective in a large prospective randomised study.

Research said use of methylprednisolone in dose of 500 mg [63], is effective in decreasing severity of illness [64]. A Dutch trial suggested the combination of intravenous methylprednisolone followed by IVIg hastens the recovery of GBS patients slightly more than IVIg alone. A single pilot study addressing combined treatment with methyl prednisolone and intravenous immunoglobulin was not included in the Cochrane analysis because it was not randomized, but suggested a possible advantage.

Other treatments: Pasin was reported in 89% of GBS patients; 75% of them additionally required oral or parenteral opioids and 30% were treated with intravenous infusion of morphine [65]. Ten percent of the patients received tricyclic antidepressants and a further 10% received carbamazepine as adjuvant treatment for neuropathic pain during the later phase of the illness. Carbamazepine and gabapentin may also be effective in the management of pain, and epidural infusion of morphine may be helpful in controlling intractable and severe pain [65].

Rehabilitation: Rehabilitation is necessary for the recovery of GBS patients. About 40% of all cases require inpatient rehabilitation as most patients are very disabled and will have required ventilator support during the acute stage. Patients are initially closely monitored in the rehabilitation setting for signs of respiratory distress. They require intubation when the vital capacity decreases to <18 mL/kg and are transferred back to hospital for medical stabilisation. The aim of rehabilitation is to restore and maintain a person's functional independence as soon as the patient is medically stable. Treatment in the acute phase should include an individual program of gentle exercises involving isometric, isotonic, isokinetic, and manual resistive and progressive resistive exercises. Rehabilitation should be focused on proper limb positioning, posture, orthotics, and nutrition [65].

CONCLUSION: The prognosis of GBS is dependent upon early diagnosis and intervention. CSF protein level might be found high in the first week of the disease in about one half of the patients, with a higher rate of morbidity and mortality in patients with axonal involvement than in those with AIDP. Although GBS is rare, further research of triggers is warranted. Much is still unknown about GBS (cause, pathophysiology, treatment, and recovery/prognosis) therefore, future research has endless limits.

REFERENCES:

1. Guillain G, Barre J, Strohl A. Sur un syndrome de radiculo-nevrite avec hyperalbuminose du liquide cephalorachidien sans reaction cellulaire. Remarques sur les caracteres cliniques et graphiques desm reflexes tendineux. *Bull Soc Med Hop Paris* 1916; 28: 1462–70.
2. Nachamkin, I., Allos, B. M., & Ho, T. (1998, July). Campylobacter Species and Guillain-Barre' Syndrome. Clinical Microbiology Reviews, 11, 3. Retrieved November 19, 2005, from http://www.ncbi.nlm.nih.gov/entrez

3. Landry O. Note sur la paralysie ascendante aigue. *Gazette Hebdomadaire Méd Chir* 1859; 6: 472–74,486–88.
4. 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; 30:131-156.
5. Hughes RA. The concept and classification of Guillain-Barre syndrome and related disorders. Rev Neurol 1995;151:291-294]
6. Asbury AK, Arnason BG, Adams RD. The inflammatory lesion in idiopathic polyneuritis: its role in pathogenesis. Medicine 1969; 48:173–215
7. Ho TW, Mishu B, Li CY, et al. Guillain-Barre´ syndrome in northern China: relationship to Campylobacter jejuni infection and anti-glycolipid antibodies. Brain 1995; 118:597–605
8. Schwerer B. Antibodies against gangliosides: a link between preceding infection and immunopathogenesis of Guillain- Barre syndrome. Microbes Infect 2002; 4:373-384.
9. Hahn AF. Guillain-Barre syndrome. *Lancet* 1998; 352: 635–41
10. Hughes RA, Cornblath DR. Guillain-Barre syndrome. *Lancet* 2005; 366: 1653–66.
11. van Koningsveld R, van Doorn PA, Schmitz PI, Ang CW, van der Meche FG. Mild forms of Guillain-Barre syndrome in an epidemiologic survey in The Netherlands. *Neurology* 2000; 54: 620–25.
12. Alshekhlee A, Hussain Z, Sultan B, Katirji B. Guillain-Barre syndrome: incidence and mortality rates in US hospitals. *Neurology* 2008; 70: 1608–13.
13. Bogliun G, Beghi E. Incidence and clinical features of acute infl ammatory polyradiculoneuropathy in Lombardy, Italy, 1996. *Acta Neurol Scand* 2004; 110: 100–06.
14. Hauck LJ, White C, Feasby TE, Zochodne DW, Svenson LW, Hill MD. Incidence of Guillain-Barre syndrome in Alberta, Canada: an administrative data study. *J Neurol Neurosurg Psychiatry* 2008; 79: 318–20.
15. Lehmann HC, Kohne A, zu Horste GM, Kieseier BC. Incidence of Guillain-Barre syndrome in Germany. *J Peripher Nerv Syst* 2007; 12: 285.
16. Seneviratne, U. (2000, April 19). Guillain-Barre' syndrome. Institute of Neurology, April, 2000. Retrieved November 20, 2005, from http://www.ncbi.nlm.nih.gov
17. Joseph, S. A., & Tsao, C. (2002, October). Guillain-Barre' syndrome. Adolescent Medicine, 13. Retrieved January 27, 2006, from http://home.mdconsult.com/das/article
18. Hughes, R. A., & Cornblath, D. R. (2005, November 5). Guillain-Barre' syndrome. Retrieved January 6, 2006, from www.thelancet.com
19. Ho TW, Li CY, Cornblath DR, Gao CY, Asbury AK, Griffin JW, et al. *Patterns of recovery in the Guillain-Barré syndromes.* Neurology 1997; 48(3):695-700.
20. Hughes R. A. C., Gregson N. A., Hadden R. D. M., Smith K. J. Pathogenesis of Guillain-Barré syndrome. J. Neuroimmunol., 1999, 100: 74-97.
21. Hadden, R. D., & Gregson, N. A. (2001). Guillain-Barre' syndrome and Campylobacter jejuni infection. Journal of Applied Microbiology, 90, 145S-154S.
22. Buchwald B., Weishaupt A., Toyka K. V., Dudel J. Immunoglobulin G from a patient with Miller-Fisher syndrome rapidly and reversibly depresses evoked quantal release at the neuromuscular junction of mice. *Neuroscience*, 1995, 201: 263-166.
23. BUCHWALD B., WEISHAUPT A., TOYKA K. V., DUDEL J. Preand postsynaptic blockade of neuromuscular transmission by Miller-Fisher syndrome IgG at mouse motor nerve terminals. Eur. J. Neurosci., 1998, 10: 281-290.
24. WILLISON H. J., O'HANLON G. M. The immunopathogenesis of Miller Fisher syndrome. J. Neuroimmunol., 1999, 100: 3-12.
25. YUKI N., ANG C. W., KOGA M. et al. Clinical features and response to treatment in Guillain-Barré syndrome associated with antibodies to GMlb ganglioside. Ann. Neurol., 2000, 47 : 314-32
26. Gabriel C. M., Hughes R. A. C., Moore S. E., Smith K. J., Walsh F. S. Induction of experimental neuritis with peripheral myelin protein 22. Brain, 1998, 121: 1895-1902.
27. Hadden RD, Karch H, Hartung HP, Zielasek J, Weissbrich B, Schubert J, et al. *Preceding infections, immune factors, and outcome in Guillain-Barré syndrome.* Neurology 2001; 56(6):758-65.
28. Govoni V, Granieri E. Epidemiology of the Guillain-Barre syndrome. Curr Opin Neurol 2001;14:605-613
29. Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992; 326:1130.
30. Moulin DE, Hagen N, Feasby TE, et al. Pain in Guillain-Barré syndrome. Neurology 1997; 48:328.
31. Ruts L, Drenthen J, Jongen JL, et al. Pain in Guillain-Barre syndrome: a long-term follow-up study. Neurology 2010; 75:1439.
32. Zochodne DW. Autonomic involvement in Guillain-Barré syndrome: a review. Muscle Nerve 1994; 17:1145.
33. Jacobs BC, Rothbarth PH, Van der Meché FGA, et al. The spectrum of antecedent infections in Guillain-Barré syndrome. A case control study. Neurology 1998; 51:1110–15.
34. Willison HJ, Yuki N. Peripheral neuropathies and antiglycolipid antibodies. Brain 2002; 125:2591–625.
35. Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992; 326:1130-6.
36. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990; 27(suppl):S21-4.
37. Bradshaw DY, Jones HK Jr. Guillian- Barre Syndrome in children: clinical course electrodiagnosis and prognosis. Muscle Nerve 1992; 15: 500- 6.
38. Rees JH, Soudain SE, Gregson NA, et al. (1995) Campylobacter jejuni infections and Guillain-Barré syndrome. N Engl J Med 333:1374–1379.
39. McKhann GM, Cornblath DR, Griffin JW, et al. (1993) Acute motor axonal neuropathy: a frequent cause of acute flaccid paralysis in China. Ann Neurol 33:333–342.
40. Asbury AK, Cornblath DR (1990) Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 27 (suppl) S21–S24.
41. Koul R, Chacko A, Ahmed R, Varghese T, Javed H, Al-Lamki Z. Ten-year prospective study (clinical spectrum) of childhood Guillain-Barré 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.
42. Raphael JC, Chevret S, Hughes RAC, Annane D. Plasma exchange for Guillain Barre Syndrome (Cochrane Review). In: The Cochrane Library, Issue 1, 2004. Chichester UK: John Wiley & Sons, Ltd.)
43. French Cooperative Group. Plasma exchange in Guillain Barre Syndrome: role of replacement fluids. Ann Neural 1987; 22:753–761.
44. Guillain Barre Study Group. Plasmapheresis and acute Guillain Bare syndrome. Neurology 1985; 35:1096–1104).
45. RAPHAEL, J.-C., CHEVRET, S, AND HUGHES, R. A. C. Plasma Exchange for Guillain-Barré Syndrome. Cochrane Library (Cochrane Protocol), 2000. Update Software.

46. Wollinsky KH, Hulser PJ, Brinkmeier H, Aulkemeyer P, Bossenecker W, Huber-Hartmann KH, et al. CSF filtration is an effective treatment of Guillain-Barre´ syndrome: a randomized clinical trial. Neurology 2001; 57: 774–80.
47. HUGHES R. A. C., RAPHAEL J.-C., SWAN A. V., VAN DOOM P. A. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Library (3), (Cochrane Protocol), 2000.
48. French Cooperative Group on Plasma Exchange in Guillain-Barre´ Syndrome. Appropriate number of plasma exchanges in Guillain-Barre´ syndrome. Ann Neurol 1997; 41: 298–306.
49. Van der Meche FGA, Schmitz PIM, the Dutch Guillain Barre Study Group. A randomised trial comparing intravenous immune globulin and plasma exchange in Guillain Barre Syndrome. N Engl J Med 1992; 326:1123–1129.
50. Hughes RAC, Raphael JC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain Barre Syndrome (Cochrane Review). In: The Cochrane Library, Issue 1, 2004. Chichester, UK: John Wiley & Sons, Ltd.
51. Imbach P, Barundun S, d'Apuzzo V, Baumgartner C, Hirt A, Morell A, et al. High dose intravenous gammaglobulin for idiopathic thrombocytopaenic purpura. Lancet 1981; 1:1228–31.
52. Vermeulin M, van der Meche´ FGA, Speelman JD, Weber A, Busch HFM. Plasma and gammaglobulin infusion in chronic inflammatory polyneuropathy. J Neurol Sci 1985; 70:317–26.
53. Kleyweg RP, van der Meche´ FGA. Treatment related fluctuations in Guillain-Barre´ syndrome after high-dose immunoglobulins or plasma-exchange. J Neurol Neurosurg Psychiat 1991; 54:957–60.
54. van der Meche´ FGA, Schmitz PI. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barre´ syndrome. Dutch Guillain-Barre´ Study Group. N Engl J Med 1992; 326:1123–9.
55. Anonymous. Randomised trial of plasma exchange, intravenous immunoglobulin, and combined treatments in Guillain-Barre´ syndrome: Plasma Exchange/Sandoglobulin Guillain-Barre´ Syndrome Trial Group. Lancet 1997; 349:225–30.
56. Gurses N, Uysal S, Cetinkaya F, Islek I, Kalayci AG. Intravenous immunoglobulin treatment in children with Guillain-Barre´ syndrome. Scand J Infect Dis 1995; 27:241–3.
57. Raphael JC, Chevret S, Harboun M, Jars-Guincestre MC; French Guillain-Barre Syndrome Cooperative Group. Intravenous immune globulins in patients with Guillain- Barre syndrome and contraindications to plasma exchange: 3 days versus 6 days. J Neurol Neurosurg Psychiatry 2001; 71:235-238.
58. Nobile-Orazio E, Terenghi F. IVIg in idiopathic autoimmune neuropathies: analysis in the light of the latest results. J Neurol 2005; 252(Suppl 1):I7-13.
59. Hadden RD, Hughes RA. Management of inflammatory neuropathies. J Neurol Neurosurg Psychiatry 2003; 74(Suppl 2): ii9-14.
60. Kuwabara S. Guillain-Barre syndrome: epidemiology, pathophysiology and management. Drugs 2004; 64:597-610.
61. 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; 30:131-156.
62. Dalakas MC. Mechanisms of action of IVIg and therapeutic considerations in the treatment of acute and chronic demyelinating neuropathies. Neurology 2002; 59(Suppl 6): S13-21.
63. Guillain Barre Syndrome Steroid Group. Double blind trial of intravenous methylprednisolone in Guillain Barre Syndrome. Lancet 1993; 341:586–590.
64. Feasby TE. Inflammatory demyelinating polyneuropathies. Neurologic Clin 1992; 10:651–670.
65. Hughes RA, Wijdicks EF, Benson E, Cornblath DR, Hahn AF, Meythaler JM, et al; Multidisciplinary Consensus Group. Supportive care for patients with guillain-barre syndrome. Arch Neurol 2005; 62:1194-1198.

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