A Prospective Study of Guillain-Barre Syndrome Cases Among Adults Admitted in Al Gamhouria Teaching Hospital and Private Hospital in Aden City - Yemen

MARWAN ABDULRAHMAN AHMED ABDULLAH, M.D. and OSAM SAEED ABDO GABALI, M.D.

The Department of Internal Medicine, Faculty of Medicine and Health Sciences, Aden University, Yemen

Abstract

Background: Guillain-Barre syndrome (GBS) is an acute autoimmune-mediated peripheral nervous system disease. Different studies from various geographical regions have reported considerable variability regarding its demographic data, clinical features, subtype and outcome.

Aim of Study: This study aimed to describe the demographics, clinical patterns, subtypes and outcome of patients with Guillain-Barre syndrome admitting in Al-Gamhouria Teaching Hospital and a private hospital in Aden City in Yemen.

Patients and Methods: It was a prospective study carried out in Al Gamhouria Teaching Hospital and a private hospital in Aden City Southwest of Yemen, over a period of three years, Between (February 2020 to February 2023), it included 30 patients >16 years of age diagnosed with with Guillain-Barre syndrome.

Results: During the study period, 30 patients were enrolled, mean age was (31 ± 6) years, (60%) of cases were male while (40%) being female, half of patients belonging to the age group of (16-39) years (50%), Prior history of infection was detected in (67%) of patients, sensorimotor form, was the most common form, it was (63%), demyelinating lesion subtype composed (57%) while axonal lesion subtype (43%), (20%) of the patients subjected to mechanically ventilated, while (80%) of patients have not been ventilated.

Conclusion: This study concluded that young, middle age male patients were the majority of cases, demographics, clinical, and eletrodiagnostic results similar to those reported in previous studies, majority of cases were not being ventilated.

Key Words: Guillain-Barre syndrome – Sensorimotor form – Demyelinating lesion.

Introduction

GUILLAIN-BARRE syndrome (GBS) is an acute immune mediated polyneuropathy characterized

by flaccid and rapidly progressive paresis that is symmetrical, ascending and reflexes are absent or diminished [1].

GBS is mediated by humeral and cellular responses that directly destroy the myelin sheath of axons of peripheral nerves [2]. Although GBS variants share immunomediated pathogenesis, they differ in their pathophysiology, clinical presentations and endpoints, and are classified into different subtypes. For example, immune reactions against epitopes of Schwann cell surface membranes or myelin result in demyelinating neuropathy, while those directed against axonal membrane antigens cause the acute axonal form of the syndrome [3].

It is a major cause of acute neuromuscular paralysis and causes respiratory failure requiring ventilator support in approximately 25% with a mortality rate of 4-15%. The annual incidence of GBS is 1.3-4 per 100,000 all over the world [4,5,6]. Men are approximately 1.5 times more affected than women [7].

The GBS is believed to include a variety of acute neuropathies with underlying immunemediated pathogenic mechanisms rather than a single disease; therefore, GBS recognized variants are considered as syndromes including (acute inflammatory demyelinating polyneuropathy) AIDP (acute motor axonal neuropathy) AMAN (acute motor-sensory axonal neuropathy) AMSAN and (Miller-Fisher syndrome). The most prevalent form of GBS reported is AIDP, which is responsible for 70-90% of cases [8,9]. In addition to history and clinical examination, confirming the diagnosis of GBS may include cerebrospinal fluid analysis (CSF) and electroneurodiagnostic testing, both of which can be normal in the early phase of the disease [10,11].

Correspondence to: Dr. Marwan Abdulrahman Ahmed Abdullah, The Department of Internal Medicine, Faculty of Medicine and Health Sciences, Aden University, Yemen

Patients and Methods

This was a prospective study carried out in Al Gamhouria Teaching Hospital and a private Hospital in Aden City Southwest of Yemen, 30 patients with Guillain-Barre syndrome enrolled in this study and admitted to Department of Internal Medicineand Intensive Care Unitsover a period of three years, Between (February 2020 to February 2023).

All patients were subjected to full medical history, neurological examination and routine laboratory investigations included Random blood sugar, complete blood picture, Serum urea, Serum creatinine, Serum electrolytes (sodium, potassium, and calcium), Erythrocyte sedimentation rate, and electro diagnostic studies.

Diagnosis of Guillain-Barre syndrome was assessed by Brighton criteria [12,13] which included (1) Acute or sub acute flaccid weakness involving lower and/or upper limbs; (2) Monophasic disease, reaching nadir of weakness between 12h and 4 weeks; and at least one of the following: (a) Hyporeflexia or areflexia in the weak limbs, (b) Cytoalbuminological dissociation defined as the combination of cerebrospinal fluid (CSF) protein level >0.45g/L and cell count <50 cells/µl, and (c) the reported electrophysiological features are compatible with a subtype of GBS.

Data was collected from patients using a predetermined questionnaire, particular emphasis being given to, sex, age, Prior history of infection (including diarrhoea, respiratory infections or unexplained fever, neurological symptoms and signs at time of arrival, subtypes of neuropathy by electrophysiological study and decision of mechanical ventilation.

Lumber puncture for cerebrospinal fluid analysis skipped due to patients rejection.

Patients grouped according to clinical form into sensorimotor form, pure motor form (caseswere not preceded or associated with sensory findings) and cranial nerve involvement in the two forms (Which included facial weakness, oropharyngeal weakness, and ophthalmoplegia).

A neuro muscular specialist performed electrophysiological examinations within 2 weeks of the onset of illness in all patients. Nerve conduction studies with evaluation of median, ulnar, common peroneal, tibia and surasal nerves were performed in all. Needle electromyogram (EMG) was done in at least two proximal and two distal limb muscles, for assessment of denervation and motor unit action potential changes, in all patients. Patients were classified as having axonal or demyelinating subtype based on the electro diagnostic criteria given by Hadden et al. [14].

Inclusion criteria included patientswithGBS diagnosis fit Brighton criteria, >16 years old, bothgender.

Exclusion criteria included patients with other causes of polyneuropathy such as diabetic, uremic, drug-related neuropathy, para neoplastic neuropathy or hereditary neuropathy, previous trauma leading to paresis, previous neuromuscular weakness, poliomyelitis, periodic paralysis, transverse myelitis, and diphtheria allwere excluded from the study.

The result was calculated manually, and presented as means, percentages and tables as appropriate.

Ethical consideration: Verbal informed consent was obtained from all participating subjects; the study design was approved by the research and ethics committee in the Faculty of Medicine University of Aden.

Results

A total of (30) subjects were included in this study All the patients had GBS, their age range from (17 to 69) with a mean value (31 ± 6) SD years

The sex distribution in Table (1) showed a prominence of male gender (60%) versus (40%) being female a male-to-female ratio of (1.5:1).

Table (2) showed maximum of patients belonging to the age group of (16-39) years (50%).

Table (3) revealed that (67%) of patients enrolled in the study had Prior history of infection included (respiratory tract infection, diarrhoea ornonspecific fever).

Table (4) demonstrated the Clinical forms where (63%) of patients presented with sensorimotor form, which was the most common form at time of arrival followed bypure motor form (37%), cranial nerve involvement was (10%) of cases included in this study.

Table (5) demonstrated the result of electro diagnostic study, where Demyelinating subtype composed (57%) while axonal subtype (43%).

Table (6) revealed the outcome of subjects enrolled in this study (20%) of the subjects mechanically ventilated, while (80%) of patients have not been ventilated.

Table (1): Distribution of patients according sex.

Sex	Number (%)
Male	18 (60%)
Female	12 (40%)

Table (2): Distribution of patients according age.

Age group	Number (%)	
16 - 39 years 40 - 59 years 60 - 70 years	15 (50%) 12 (40%) 3 (10%)	

Table (3): Distribution of patients according the presence of prior history infection.

Prior history infection	Number (%)
Present	20 (67%)
Absent	10 (33%)

Table (4): Clinical forms of GBS at time of arrival.

Clinical forms of GBS at time of arrival	Number (%)
Pure motor form Sensorimotor form	11 (37%) 19 (63%)
Cranial nerve involvement	3 (10%)

Table (5): Distribution of patients according the electrophysiological subtypes.

Electrophysiological subtypes	Number (%)
Demyelinating subtype	17 (57%)
Axonal subtype	13 (43%)

Table (6): Distribution of patients according to decision of mechanical ventilation.

Mechanical ventilation	Number (%)
Yes	6 (20%)
No	24 (80%)

Discussion

The majority of subjects in our study were male (60%) this study result was close with results found in Al Maawali et al., [15] study carried out in Oman in Khoula Hospital in Muscat, from 2016 to 2018 and included 44 patients, men constitute (63.6%), In contrast with the study carried out in eastern Nepal by Bhagat et al., [16] and included 31 patients from 2013 to 2017 and published in 2019. Where the majority of subjects were Female composed (51.6%).

The predominance of GBS in men is consistent with those in previous regional and international studies [17,18].

The majority of patients were related to the age group of (16-39) years (50%) Approximately near the result mentioned by Alanazy et al., [19] retrospective multicentre study conducted in Saudi Arabia and published in 2021, where approximately, half of the patients (51.9%) were aged 18-39 years.

Prior history of infection included (respiratory tract infection, diarrhoea and nonspecific fever) was presented in (67%) of cases recruited in this study, similar to the result reached by Chaudhuri et al., [20] study from Yashoda Hospital in Hyderabad South India published in 2014 included 37 patients where Prior history of infection constitutes (67%).

Literature review revealed that two-thirds of patients with GBS have an antecedent respiratory or gastrointestinal tract infection in the 6 weeks preceding the onset of GBS [21].

According to the clinical forms, sensorimotor form constitute (63%) of cases enrolled in this study closely related to findings obtained in Alloush et al., study [22] conducted at Ain Shams university hospitals and Kobri Elkoba Military Hospital including twenty patients with the diagnosis of GBS in the duration from 2016 to 2018 and published in 2019, and Alanazy et al., [19] retrospective multicentre study conducted in Saudi Arabia where sensorimotor form was (60.0%), (64.1 %) respectively.

While isolated pure motor form composed (37%), closeto Alanazy et al., [19] where pure motor forms was (34.6%).

Cranial nerve involvement was (10%) in this study near to (9.7%) reached by Bhagat et al., in eastern Nepal [16].

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype composed (57%) while axonal subtypes included acute motor axonal neuropathy (AMAN), and acute motor-sensory axonal neuropathy (AMSAN) composed (43%), close to results obtained by Sharma et al., study [23] conducted in India and published in 2011 and included 50 patients with GBS, 30 had AIDP (60%), 20 had axonal subtypes (40%).

In retrospective study from Northern China reported different frequencies of the electrophysiological subtypes of GBS, AMAN was the predominant subtype (55.8%) and AIDP occurred less frequently (21.2%) [24].

This variation in the distribution of subtypes between countries may be related to the exposure

A Prospective Study of Guillain-Barre Syndrome Cases

to different types of infections; however, the genetic characteristics of the population may also have an effect [25].

Only 6/30 patients (20.0%) needed mechanical ventilation, similar to the result present in the Egyptian Study of Alloush et al., [22] 4/20 (20%), higher than result of Al Maawali et al., in Oman [15] were 13.6% admitted to ICU for mechanical ventilation.

Recommendations:

Due to the very limited data, which is available in Yemen on this important issue, our results can be used as baseline data for understanding the characteristics of GBS in Yemen.

Further studies, which contain larger samples of GBS patients, from different healthcare centers and hospitals, needs to be carried out to achieve more reliable and representative data, which can lead to the best outcomes.

References

- HUGHES R.A. and CORNBLATH D.R.: Guillain-Barré syndrome. Lancet, Nov. 366 (9497): 1653-66. https://doi.org/10.1016/S01406736 (05)67665-9, 2005.
- KOSKI C.L.: Humoral mechanisms in immune neuropathies. Neurol. Clin., Aug. 10 (3): 629-49, 1992.
- 3- HAFER-MACKO C.E., SHEIKH K.A., LI C.Y., HO T.W., CORNBLATH D.R., MCKHANN G.M., et al.: Immune attack on the Schwann cell surface in acute inflammatory demyelinating polyneuropathy. Ann. Neurol., May 39 (5): 625-35. https://doi.org/10.1002/ana.410390512, 1996.
- 4- VAN KONINGSVELD R., VAN DOORN P.A., SCHMITZ P.I., ANG C.W. and VAN DER MECHE F.G.: Mild forms of GuillainBarre syndrome in an epidemiologic survey in The Netherlands. Neurology, 54: 620-5, 2000.
- 5- GOVONI V. and GRANIERI E.: Epidemiology of the Guillain-Barre syndrome. Curr. Opin. Neurol., 14: 605-13, 2001.
- 6- HUGHES R.A. and REES J.H.: Clinical and epidemiological features of Guillain- Barre syndrome. J. Infect Dis., 176: S92-S98, 1997.
- 7- BOGLIUN G. and BEGHI E.: Incidence and clinical features of acute inflammatory polyradiculoneuropathy in Lombardy, Italy, 1996. Acta. Neurol. Scand, 110: 100-6, 2004.
- 8- SEJVAR J.J., BAUGHMAN A.L., WISE M. and MOR-GAN O.W.: Population incidence of Guillain-Barré syndrome: A systematic review and meta-analysis. Neuroepidemiology, 36: 123-133, 2011.
- 9- ANSAR V. and VALADI N.: Guillain-Barré syndrome. Prim Care, 42: 189-193, 2015.
- 10- KALITA J., MISRA U.K., GOYAL G. and DAS M.: Guillain-Barré syndrome: Subtypes and predictors of outcome from India. J. Peripher Nerv. Syst., 19: 36-43, 2014.

- I-VAN DOORN P.A., RUTS L. and JACOBS B.C.: Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. Lancet Neurol., 7: 939-950, 2008.
- 12- SEJVAR J.J., KOHL K.S., GIDUDU J., AMATO A., BAKSHI N., BAXTER R., et al.: Guillain-Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine, 29: 599-612, 2011.
- 13- FOKKE C., VAN DEN BERG B., DRENTHEN J., WAL-GAARD C., VAN DOORN P.A. and JACOBS B.C.: Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain J. Neurol., 137: 33-43, 2014.
- 14- HADDEN R.D., CORNBLATH D.R., HUGHES R.A.C., et al.: Electrophysiological classification of Guillain-Barre syndrome: Clinical associations and outcome. Ann. Neurol., 44: 780-8, 1998.
- 15- SAID M. AL MAAWALI, M.D., OMSB, ALMQDAD Y. AL SHIBANI, MD, Ph.D., AHMED S. NADEEM, M.D., Ph.D. and ABDULLAH M. AL-SALTI, M.D.: Guillain-Barre syndrome: Demographics, clinical features, and outcome in a single tertiary care hospital, Oman, Neurosciences, Vol. 25 (5): 369-374, 2020.
- 16- SAROJ KUMAR BHAGAT, SHREYSIDHANT, MUKE-SH BHATTA, ASHISH GHIMIRE and BHUPENDRA SHAH: Clinical Profile, Functional Outcome, and Mortality of Guillain-Barre Syndrome: A Five-Year Tertiary Care Experience from Nepal, Neurology Research International Volume, Article ID 3867946, 5 pages, 2019.
- 17-DOETS A.Y., VERNON C., VAN DEN BERG B., HARPO T., CORNBLATH D.R., WILLISTON H.J., et al.: Regional variation of Guillain-Barré syndrome. Brain J. Neurol., 141: 2866-77, 2018.
- 18- ZENG Y., LIU Y., XIE Y., LIANG J., XIAO Z. and LU Z.: Clinical Features and the Validation of the Brighton Criteria in Guillain-Barré Syndrome: Retro-spective Analysis of 72 Hospitalized Patients in Three Years. Eur. Neurol., 81: 231-8, 2019.
- 19- MOHAMMED H. ALANAZY, SAWSAN S. BARKY, AFNAN ALQAHTANI, NORAH S. ALAKEEL, NAAEL ALAZWARY, AFAG M. OSMAN, RANIA A. MUSTA-FA, TALAL M. AL-HARBI, SAMEEH O. ABDULMA-NA, AIMEE C. AMPER, YOUSEF ALDUGHAYTHIR, ABDULRAHMAN S. ALI, SERAJ MAKKAWI, ALAA MAGLAN, LOUJEN ALAMOUDI, FERAS ALSULAIM-AN, MAJED ALABDALI, AYSHA A. ALSHAREEF, AHMAD R. ABUZINADAH and AHMED K. BAMAGA: Clinical features and outcome of Guillain- Barre syndrome in Saudi Arabia: A multicentre, retrospective study, BMC Neurol., 21: 275, 2021.
- 20- JAYDIP RAY CHAUDHURI MD DM, 2SUVARNA ALLADI DM, K. RUKMINI MRIDULA DNB DM, 2DEMUDU BABU BODDU MD DM, MV RAO MD, C. HEMANTH MD, V. DHANALAXMI MD DM, J MAY-URNATH REDDY MD, S. MANIMALA RAO MD, 3BANDA BALARAJU M.D. and VCS SRINIVASARAO BANDARU Ph.D.: Clinical outcome of Guillain-Barré syndrome with various treatment methods and cost effectiveness: A study from tertiary care centre in South India: Yashoda GBS Registry, Neurology Asia, 19 (3): 263-270, 2014.
- 21- LEONHARD S.E., MANDARAKAS M.R., GONDIM F.A.A., BATEMAN K., FERREIRA M.L.B., CORN-

BLATH D.R., et al.: Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat. Rev. Neurol., 15: 671, 2019.

- 22- ALLOUSH T., FAHMY N.A., FOUAD M.M., ALBA-ROUDY H.O., HAMDY M. and SALEM H.H.: Prediction of Outcome in Patients with Guillain Barre Syndrome-An Egyptian Study, Neuroscience & Medicine, 10: 232-246, 2019.
- 23- SHARMA A., LAL V., MODI M., VAISHNAVI C. and PRABHAKAR S.: Campylobacter jejuni infection in Guillain-Barré syndrome: A prospective case control

study in a tertiary care hospital. Neurol. India, 59: 71721, 2011.

- 24- TIAN J., CAO C., LI T., ZHANG K., LI P., LIU Y., et al.: Electrophysiological Subtypes and Prognostic Factors of Guillain-Barre Syndrome in Northern China. Front Neurol., 10: 714, 2019.
- 25- VAN DEN BERG B., WALGAARD C., DRENTHEN J., FOKKE C., JACOBS B.C. and VAN DOORN P.A.: Guillain-Barré syndrome: Pathogenesis, diagnosis, treatment and prognosis. Nature Reviews Neurology, 10: 469-482, 2014.

دراسة مستقبلية لحالات متلازمة غيلان بارى بين البالغين الودعين فى مستشفى الجمهورية التعليمى ومستشفى خاص فى مدينة عدن اليمن

المقدمة : تعتبر متلازمة غيلان بارى مرض حاد يصيب الجهاز العصبى الطرفى بوساطة مناعية، أشارت دراسات مختلفة من أمان جغرافية متعددة إلى تنوع واسع فى البيانات الديموغرافية، السمات السريرية، الأنواع الفرعية وحصيلة المخرجات.

الأهداف : قصدت الدراسة إلى وصف الديموغرافية، الصورة النمطية، الأنواع الفرعية وحصيلة المخرجات لمرضى متلازمة غيلان بارى المودعين في مستشفى الجمهورية التعليمي ومستشفى خاص في مدينة عدن اليمن.

الأساليب : دراسة مستقبلية أنجزت فى مستشفى الجمهورية التعليمى ومستشفى خاص فى مدينة عدن اليمن خلال ثلاثة سنوات من فبراير ٢٠٢٠ إلى فبراير ٢٠٢٣.

شملت الدراسة ثلاثين مريض تم تشخيصهم بمتلازمة غيلان بار، تجاوزت أعمارهم ١٦ سنة.

النتائج : تم ادراج ثلاثون مريضاً خلال فترة الدراسة، كان متوسط العمر (6±31) عاماً شكل الذكور ٦٠٪ بينما الاناث ٤٠٪، نصف المرضى كانوا من الفئة العمرية، (٢٩–١٦) لوحظ وجود تاريخ سابق لعدوى فى ٦٧٪ من المرضى، كان الشكل الحسى الحركى أكثر الأشكال شيوعاً ٣٢٪، شكل نوع الإصابة المزيلة للميلين ٥٧٪ شكل نوع الإصابة المحورية ٤٣٪. ٢٠٪ من المرضى خضعوا للتنفس الصناعى، بينما يخضعوا له.

الخلاصة : خلصت الدراسة إلى أن المرضى الذكور الصغار و متوسطى العمر شكلوا أغلبية الحالات، النتائج الديموغرافية، السريرية والكهربائية التشخيصية مشابهة للنتائج المشار لها في الأبحاث السابقة، أغلبية الحالات لم تخضع للتنفس الصناعي.