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Research Article

STUDY TO KNOW THE ACUTE FLACCID PARALYSIS CLINICAL PRESENTATION ELECTROPHYSIOLOGIC SUB TYPES OF GBS AND ITS SEASONAL VARIATIONS

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Abstract:

Acute flaccid paralysis (AFP) is a clinical syndrome determined by a prompt onset of weakness, often involving bulbar and respiratory weakness. Early and accurate diagnosis of the cause has a significant impact on the prognosis and management. Guillian Barre syndrome (GBS) is post-infectious polyradiculoneuropathy, which mostly affects the motor, but sometimes affects autonomic and sensory nervous system.

Objective: *To determine the acute flaccid paralysis clinical presentations, electrophysiologic subtypes of GBS, their outcome and seasonal variations in our setup.*

Study Design: *A Retrospective Study.*

Place And Duration: *In the Department of Neurology, Combined Military Hospital (CMH), Lahore for one year duration from September 2017 to September 2018.*

Methods: *Retrospective and hospital studies were performed in the department of Neurology to determine the clinical characteristics of GBS including current treatment methods and outcomes, and prognosis of disease severity. Diagnostic features include paresis, flaccid paralysis or weakness of limb with or without autonomic symptoms or sensory symptoms, albino-cytological dissociation, nerve conduction rate (NCV), laboratory properties such as ECG, serum electrolytes and MRI.*

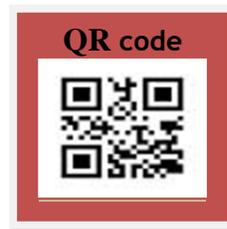
Results: *In this study; 55 patients were included for 1 year duration. Of these, 29 (53%) had GBS, hypokalemic periodic paralysis in 21 (38%) and idiopathic neuropathy in 5 (9%). NCV test was applied to all and categorized as 18 patients ha AIDP (acute inflammatory demyelinating polyneuropathy, 8 patients had AMAN (acute motor axonal neuropathy) and 3 patients had AMASAN (acute motor and sensory axonal neuropathy. Most of the patients presented symmetrically elevated paralysis, gradually progressing in all extremities. In our study, the rate of males was affected by more females: 1.63: 1 and in spring and winter season; 23 patients presented. During the 2nd and 3rd decade there was mild increase age range and in the 5th decade, the second peak was seen. Ten percent of the patients has recurrence in five years. URTI, pneumonia, sore throat, diarrhea in most of the related diseases. The most common cranial nerve involvement was the paralysis of the facial nerve. The majority improved only with supportive therapy, ventilator support was needed in 11% of patients and 22% indicated IVIG or plasmapheresis. Almost half of the patients completely recovered at 3 months of follow-up and improved at follow-up.*

Conclusion: *Timely treatment and diagnosis is necessary to support and manage these treatable diseases. Adequate training, psychological support and physiotherapy are mandatory.*

Key words: *Hypokalemic periodic paralysis, acute flaccid paralysis, GBS, AIDP, AMASAN, AMAN.*

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INTRODUCTION:

Acute flaccid paralysis (AFP) is a clinical appearance characterized by paralysis or weakness and decrease tone of muscles. If it affects the respiratory muscles, this can be fatal which cause death and choking [1-3]. Early and accurate diagnosis of the reason has a significant impact on prognosis and management [4]. In 1916, three French doctors (Strohl, Guillain and Barre) described two French soldiers having cerebrospinal fluid (CSF), areflexia, motor weakness, decreased deep tendon reflexes and albuminocytological decomposition [5]. Guillain-Barre syndrome can be defined as a group of clinical conditions that are seen as acute inflammatory polyradiculoneuropathy with emerging decreased reflexes and weakness [6]. Historically, GBS was a single disease; however, the present application recognizes various variable forms are AMAN, AIDP, Miller Fisher syndrome and AMSAN. Among the 2 dominant subtypes, a demyelinating subtype (AIDP) is the predominant form in the Europe and United States and in China it is the dominant form of axonal subtype (AMAN) [7]. Previous clinical studies have recommended that AMAN has also appeared in children of Mexico. The incidence in the US is 1.2 to 3 per lac people, the most usual cause of acute flaccid paralysis [8]. It is supposed that the disease is autoimmune and has been related with a previous infection, previously respiratory or gastrointestinal infections. In general, infections of microorganisms such as CMV, Campylobacter jejuni, influenza virus or Mycoplasma pneumonia exist some weeks before about 2/3rd of GBS cases [9]. Acute flaccid paralysis result in high death rate if not managed with time, so it can save lives by preventing complications such as autonomic dysfunction, respiratory failure, residual disability, pulmonary embolism and infection if timely diagnosis and rapid treatment given [10]. An epidemiological study in 2008 stated 2 to 12%

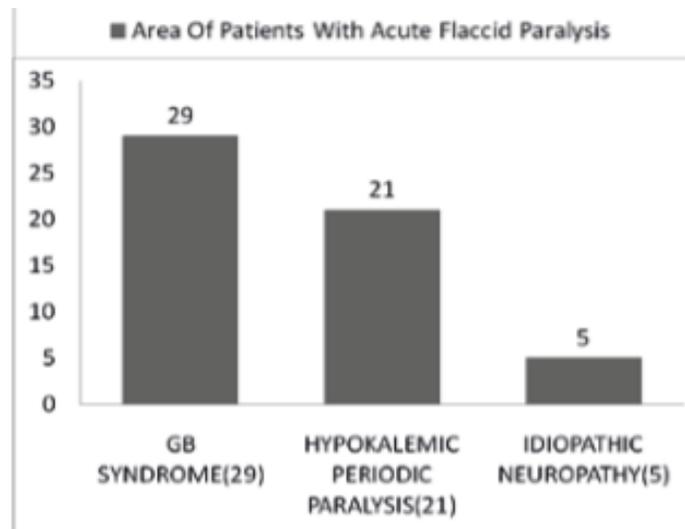
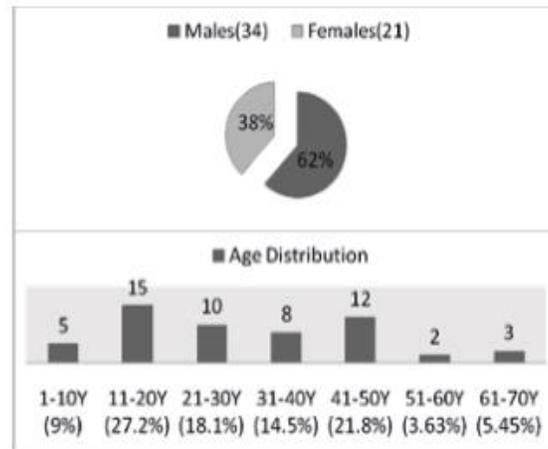
mortality rate despite treatment in the ICU. Death related causes of GBS include sepsis, acute respiratory distress syndrome (ARDS), venous thromboembolic disease cardiac arrest and pneumonia [11]. Mostly mortality is because of prolonged intubation, severe autonomic instability and paralysis complications.

MATERIALS AND METHODS:

Conditions meeting the criteria for acute flaccid paralysis were revised rendering to WHO. The rapid onset of GBS causing involving limbs weakness of the respiratory and swallowing muscles, progresses in maximum intensity from 1 to 10 days. The term "flaccid" refers to the lack of spasticity or an irregular motor pathway of the central nervous system (CNS) signs such as extensor plantar responses, hyperflexia or clonus. The data were collected by age, gender, distribution area, seasonal change, acute flaccid paralysis causes, electrophysiological GBS subtypes and their management. On OPD basis; cases were followed up. The data were collected by the data analysis and researcher and recorded in SPSS version 17. Patients with more than one limb progressive motor weakness who had symmetrical palsy with or without autonomic or sensory symptoms were included. Suspected myopathies and suspected spinal injuries were excluded from the study. Clinical examination, Clinical history, serum electrolytes, CSF analysis and nerve conduction studies were made and MRI was done when required.

RESULTS:

There were 55 patients in our study. Of these, 34 (61.8%) were male, 21 (38%) were female and 36 years was the mean age of patients. The majority of them were 15 (27.2%) in the second decade (12-20 years), the third decade 10 (18.1%) of patients and in the 5th decade 12 (21.8%) of patients.



Of these hypokalemic periodic paralysis was seen in 21 (38%), GB syndrome in 29 (53%) and idiopathic neuropathy in 5 (9%). After nerve conduction studies; GB syndrome variants were detected included classic GB syndrome AIDP 18 (62%), sensory and acute motor axonal neuropathy (11.1%) and acute motor axonal neuropathy 8 (27.5%). The seasonal spectrum of GBS in our study is shown below. Most cases were observed in spring and winter months. There were 15 GB syndrome cases in the winter and 8 hypokalemic periodic paralysis cases. Less cases are observed in autumn and summer. Associated diseases were pneumonia in 1 patient, upper respiratory tract infection in 3 patients, facial nerve palsy in 14 patients (25.4%) and diarrhoea in 6 patients. During treatment, with supportive treatment, 65% of the patients recovered and ventilation and intubation required in 11%. 22% were sent for immune modulation therapy (Plasmapheresis/ IVIG).

DISCUSSION:

Most of the cases belong to the Lahore district, which belongs to the age group of 11 to 20 years. GB syndrome was the most common variant of AIDP and the most usual cause of acute flaccid paralysis¹². When more cases were observed in winter and spring months, a seasonal change was observed. Enhanced majority with supportive management. The core mechanism may be the clarification of the pathophysiological bases at the top of the GB syndrome and the cessation of progression as the natural course of the disease. In our study, the maximum number of cases reported in spring and winter is same to reports from southern Kuwait and Iran [13]. Cases were informed throughout the year, the lowest in August (173) and the maximum were seen in January (261). There is a significant seasonal and monthly change in admission rates for patients with GBS in Shiraz (IRAN). Of the 389 GBS cases, 232 (59.6%) were male and 157 (40.4%) were female

[14]. There were seasonal change ($P = 0.004$) and monthly change ($P = 0.046$). Spring and winter had the highest number of patients and were accepted between February and June. Immunomodulatory therapy such as IVIG or plasmapheresis gives the best results¹⁵. On the other hand, hypokalemic periodic paralysis requires ECG for bedside and emergency potassium level and provides a striking response to oral / IV potassium. Almost half of the patients completely recovered during follow-up of 3 months in outpatient clinics. 10% of the patients recurred in 5 years, no death or residual deficiency was observed during the disease.

CONCLUSION:

GB syndrome is a major public health problem if it leads to high mortality and is not correctly diagnosed. Supportive treatment, timely diagnosis and timely treatment with progression observation are the key to increase the looseness of your feet. Adequate training, physiotherapy and psychological support are necessary for the treatment of these treatable diseases.

REFERENCES:

1. Park, Soo Jin, Jong Kuk Kim, Hyun-Hwi Kim, Byeol-A. Yoon, Dong Yoon Ji, Chang-Wan Lee, Ho Jin Kim et al. "Integrative metabolomics reveals unique metabolic traits in Guillain-Barré Syndrome and its variants." *Scientific reports* 9, no. 1 (2019): 1077.
2. Al-Hakem, Helle, Søren H. Sindrup, Henning Andersen, Charlotte Dornonville de la Cour, Lisbeth L. Lassen, Bianca van den Berg, Bart C. Jacobs, and Thomas Harbo. "Guillain-Barré syndrome in Denmark: a population-based study on epidemiology, diagnosis and clinical severity." *Journal of neurology* 266, no. 2 (2019): 440-449.
3. Sedano, María J., Pedro Orizaola, Elena Gallardo, Antonio García, Ana L. Pelayo-Negro, Pascual Sánchez-Juan, Jon Infante, and José Berciano. "A unicyclic, prospective study of Guillain-Barré syndrome in Spain." *Acta Neurologica Scandinavica* (2019).
4. Jain, Rajendra Singh, Jagdeesh Chandra Kookna, Trilochan Srivastva, and Rahul Jain. "Guillain-barre Syndrome in Indian Population: A Retrospective Study." *Journal of The Association of Physicians of India* 67 (2019): 56.
5. Yoon, Lira, Bo Ryung Kim, Hye Young Kim, Min Jung Kwak, Kyung Hee Park, Mi Hye Bae, Yunjin Lee, Sang Ook Nam, Hee Young Choi, and Young Mi Kim. "Clinical characterization of anti-GQ1b antibody syndrome in Korean children." *Journal of neuroimmunology* (2019).
6. Tan, Cheng-Yin, Siti Nur Omaira Razali, Khean-Jin Goh, and Nortina Shahrizaila. "The utility of Guillain-Barré syndrome prognostic models in Malaysian patients." *Journal of the Peripheral Nervous System* (2019).
7. Mombo, Landry Erik, Rajendranath Ramasawmy, Samia Zertal-Zidani, Dominique Charron, and Ryad Tamouza. "Ethnic differences in CD1E, but not CD1A, gene polymorphisms between Sub-Saharan Africans, West Asians and Europeans." *Human Immunology* 80, no. 3 (2019): 204-207.
8. Bölükbaşı, Feray, Gülsun Ersen, Ayşegül Gündüz, Feray Karaali-Savrun, Sinem Yazici, Nurten Uzun, Mehmet Ali Akalin, and Meral E. Kiziltan. "Guillain-Barré Syndrome and Its Variants: Clinical Course and Prognostic Factors." *Archives of Neuropsychiatry* 56, no. 1 (2019): 71.
9. Kumar, NAVSK Ravi, and Kanduri Soumya. "AN OVERVIEW OF GUILLAIN BARRE SYNDROME WITH REFERENCE TO CLINICAL FEATURES AND PROGNOSTIC OUTCOME IN PATIENTS ATTENDING A TERTIARY CARE HOSPITAL." *Paraplegia* 2 (2019): 5.
10. Samadi, Alireza, Fariborz Mansour-Ghanaei, Farahnaz Joukar, Sara Mavaddati, and Iman Sufi Afshar. "A 30-Year-Old Man with Acute Motor Axonal Neuropathy Subtype of Guillain-Barré Syndrome Having Hepatitis A Virus Infection." *Middle East Journal of Digestive Diseases (MEJDD)* 11, no. 2 (2019).
11. Bunschoten, Carina, Filip Eftimov, W-Ludo van der Pol, Bart C. Jacobs, ICOS Consortium, P. A. van Doorn, E. Brusse et al. "International chronic inflammatory demyelinating polyneuropathy outcome study (ICOS): Protocol of a prospective observational cohort study on clinical and biological predictors of disease course and outcome." *Journal of the Peripheral Nervous System* 24, no. 1 (2019): 34-38.
12. Grapperon, A-M., M. Berro, E. Salort-Campana, A. Verschuere, E. Delmont, and S. Attarian. "Guillain-Barré syndrome subtypes: A clinical electrophysiological study of 100 patients." *Revue neurologique* 175, no. 1-2 (2019): 73-80.
13. Leitzen, Eva, Barbara B. Raddatz, Wen Jin, Sandra Goebbels, Klaus-Armin Nave, Wolfgang Baumgärtner, and Florian Hansmann. "Virus-triggered spinal cord demyelination is followed by a peripheral neuropathy resembling features of Guillain-Barré syndrome." *Scientific reports* 9, no. 1 (2019): 4588.

14. Koga, Michiaki, Masahiko Kishi, Toshihiro Fukusako, Naomi Ikuta, Masayuki Kato, and Takashi Kanda. "Antecedent infections in Fisher syndrome: sources of variation in clinical characteristics." *Journal of neurology* (2019): 1-8.
15. Zhang, Da-Qi, Yu Deng, Lin-jie Zhang, Li-min Li, Yuan Qi, Jing Wang, Rong Wang, Hui Zhai, Peng Zhao, and Li Yang. "Elevated resistin levels may regulate high mobility group box 1 expression in Guillain-Barré syndrome." *Journal of neuroimmunology* 330 (2019): 59-66.