Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/jptcp.v30i18.3138

CLINICAL AND AETIOLOGICAL SPECTRUM OF HYPOKALEMIC FLACCID PARALYSIS IN TERTIARY CARE HOSPITAL

Muhammad Abid[1], Hamza Shoaib[2*], Asrar Haider[3], Safi Ullah Khan[4], Tazeen Nazar[5], Taleah Tahir[6]

[1]Medical Officer, Medical Emergency, Tehsil Headquarters Hospital, Ahmedpur East - Pakistan
[2*]Junior Specialty Doctor, Department of Cardiology, Sandwell and West Birmingham Hospitals NHS Trust - UK
[3]Medical Officer, Allama Iqbal Memorial Teaching Hospital, Sialkot - Pakistan
[4]Medical Officer, District Health Office, Bannu, Health Department, Khyber PakhtunKhwa - Pakistan
[5]Assistant Professor of Medicine, Mayo Hospital, Lahore - Pakistan
[6]Senior Registrar, Department of Nephrology, Fauji Foundation Hospital Rawalpindi - Pakistan

[*]**Corresponding Author:** Hamza Shoaib
[*]Junior Specialty Doctor, Department of Cardiology, Sandwell and West Birmingham Hospitals NHS Trust – UK, Email: hmz71shoaib@gmail.com

ABSTRACT

Background and Aim: Hypokalemic flaccid paralysis refers to sudden muscle weakness caused by low levels of potassium in the body. This weakness affects respiratory and pharyngeal muscles and reaches its peak severity within a matter of days to weeks. The current study investigated the clinical and etiological spectrum of hypokalemic flaccid paralysis.

Patients and Methods: This cross-sectional study was conducted on 136 hypokalemic flaccid paralysis patients in the Department of Medicine and Nephrology, Jinnah Hospital, Lahore from January 2022 to December 2022. Patient's clinical presentation includes symptoms such as weakness, fever, difficulty in breathing, cough, heart beat variation, and vomiting etc. Improvement in clinical symptoms, complete improvement, and mortality rate were different outcomes measured.

Results: The overall mean age was 12.46±3.82 years with an age range 1 year to 20 years. There were 86 (63.2%) male and 50 (36.8%) females. Guillain bare syndrome was the most prevalent disease 52 (38.2%) followed by viral myositis 37 (27.2%) at presentation. The incidence of various symptoms such as weakness, fever, headache, vomiting, and difficulty in breathing was 100% (n=136), 44.1% (n=60), 42.6% (n=58), 17.6% (n=24), and 13.2% (n=18) respectively. Following the treatment, 90 cases (66.2%) showed some improvement, whereas 24 patients (17.6%) did not experience any improvement. About 16 (11.8%) cases developed residual neurological disability whereas the mortality rate was 2.9% (n=4) during follow-up.

Conclusion: The present study found that Guillain bare syndrome was the most prevalent disease followed by viral myositis. Treatment can improve acute flaccid paralysis in over 60% of patients.

Keywords: Hypokalemic flaccid paralysis, Clinical spectrum, etiological spectrum

INTRODUCTION

Hypokalemic paralysis is the lower levels of potassium causing sudden weakness in the limp muscle. This condition can arise due to an increased movement of potassium ions into cells or significant potassium loss through the kidneys or gastrointestinal system. In some cases, hypokalemic paralysis can lead to serious complications like irregular heartbeats and respiratory issues, although these occurrences are rare [1-3]. Periodic paralysis of familial hypokalemic is associated with genetic and comes from an affected parent due to autosomal dominant manner. Thyrotoxicosis is the second most prevalent cause of hypokalemic paralysis. The occurrence of secondary hypokalemic paralysis comes from substantial amount of potassium loss through renal and gastrointestinal systems. Patients experiencing hypokalemic paralysis require a thorough investigation to identify the possible underlying cause and may need potassium replacement therapy [4, 5]. Fortunately, the majority of individuals with hypokalemic paralysis tend to fully recover without any lasting disabilities [6].

Acute flaccid myelitis is a sporadic syndrome characterized by sudden muscle weakness, specifically involving spinal motor neurons. This condition is distinct clinically and idiopathically, setting it apart from other forms of acute flaccid paralysis [7]. Hypokalemic paralysis, on the other hand, is a type of metabolic myopathy that falls under a diverse group of disorders marked by low potassium levels, sudden muscle weakness, and potentially life-threatening episodes involving respiratory muscles and cardiac arrhythmias [8]. There are two main causes of hypokalemic flaccid paralysis (HKFP). One cause is the movement of potassium from the extracellular to intracellular space, observed in conditions such as hypokalemic periodic paralysis (HPP), which can be familial, sporadic, or associated with thyroid disorders. In this group, there is no excess potassium excretion through urine. The other cause involves increased renal loss of potassium, as seen in conditions like distal renal tubular acidosis, Gitelman's syndrome, primary hyperaldosteronism, and non-renal causes like diarrhea, vomiting, and prolonged sweating [9].

METHODOLOGY

This cross-sectional study was conducted on 136 hypokalemic flaccid paralysis patients in the Department of Medicine and Nephrology, Jinnah Hospital, Lahore from January 2022 to December 2022. Acute flaccid paralysis children aged 1-20 years, irrespective of their gender were enrolled. Patients with cerebral palsy, congenital myopathy, and underlying neuromuscular and neurometabolic diseases were excluded. The sample size was determined based on the percentage of Guillain-Barre syndrome, which is 9.8% in children diagnosed with acute flaccid paralysis. Patient's clinical presentation includes symptoms such as weakness, fever, difficulty in breathing, cough, heart beat variation, and vomiting etc. Improvement in clinical symptoms, complete improvement, and mortality rate were different outcomes measured. Guillain-Barre syndrome, viral myositis, and acute flaccid myelitis etc. were different causes of hypokalemic paralysis.

SPSS version 27 was used for descriptive statistics. Numerical parameters were expressed as mean and standard deviation whereas categorical parameters were described as frequencies and percentages. Inferential statistics were explored using the Chi-square test by taking 95% confidence interval and 5% level of confidence.

RESULTS

The overall mean age was 12.46±3.82 years with an age range 1 year to 20 years. There were 86 (63.2%) male and 50 (36.8%) females. Guillain bare syndrome was the most prevalent disease 52 (38.2%) followed by viral myositis 37 (27.2%) at presentation. The incidence of various symptoms such as weakness, fever, headache, vomiting, and difficulty in breathing was 100% (n=136), 44.1% (n=60), 42.6% (n=58), 17.6% (n=24), and 13.2% (n=18) respectively. Following the treatment, 90 cases (66.2%) showed some improvement, whereas 24 patients (17.6%) did not experience any improvement. About 16 (11.8%) cases developed residual neurological disability whereas the mortality rate was 2.9% (n=4) during follow-up. Demographic details and baseline characteristics are shown in Table-I. The incidence of different diseases are illustrated in Figure-1. Figure-2 depicts

the incidence of different symptoms of hypokalemic flaccid paralysis. Comparison of outcomes concerning age and gender are shown in Table-II.

Table-I demographic data and baseline characteristics (N=136)

Variables	Value (Mean ± SD) N (%)
Age (years)	12.46±3.82
Age groups (years)	
1-5	32 (23.5%)
6-10	46 (33.8%)
11-15	22 (16.2%)
16-20	36 (26.5%)
Gender	
Male	86 (63.2%)
Female	50 (36.8%)
Status of respiratory function	
Normal	114 (83.8%)
Need ventilation	22 (16.2%)
Post-treatment normal respiratory function	132 (97.1%)
Mortality	4 (2.9%)

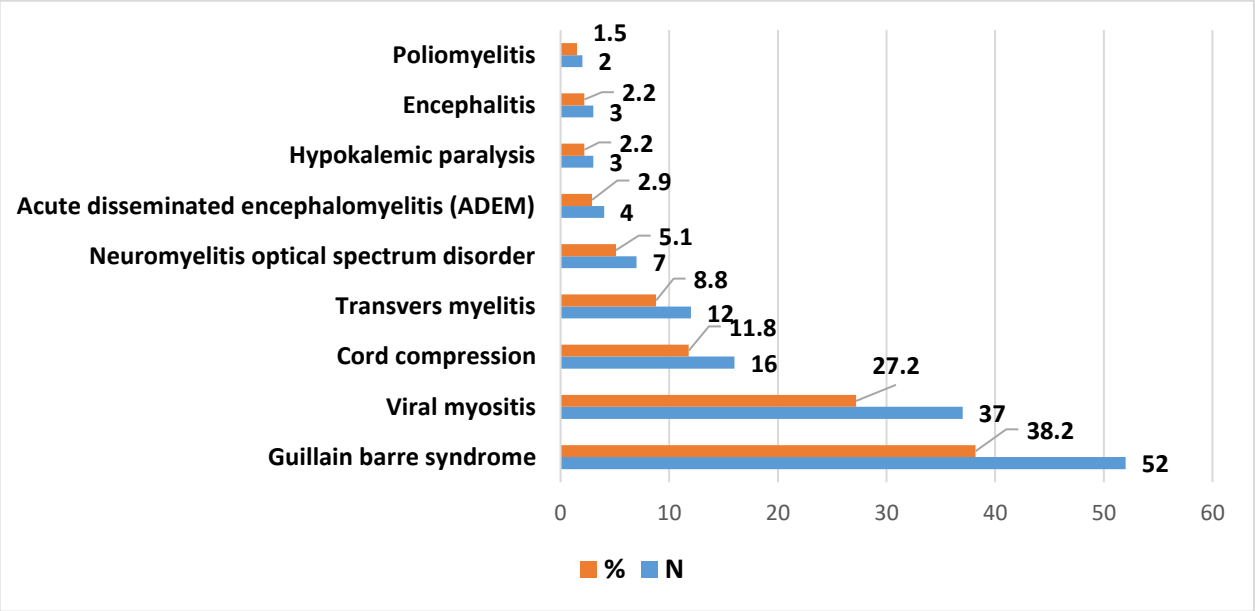

Figure-1 incidence of different diseases (N=136)

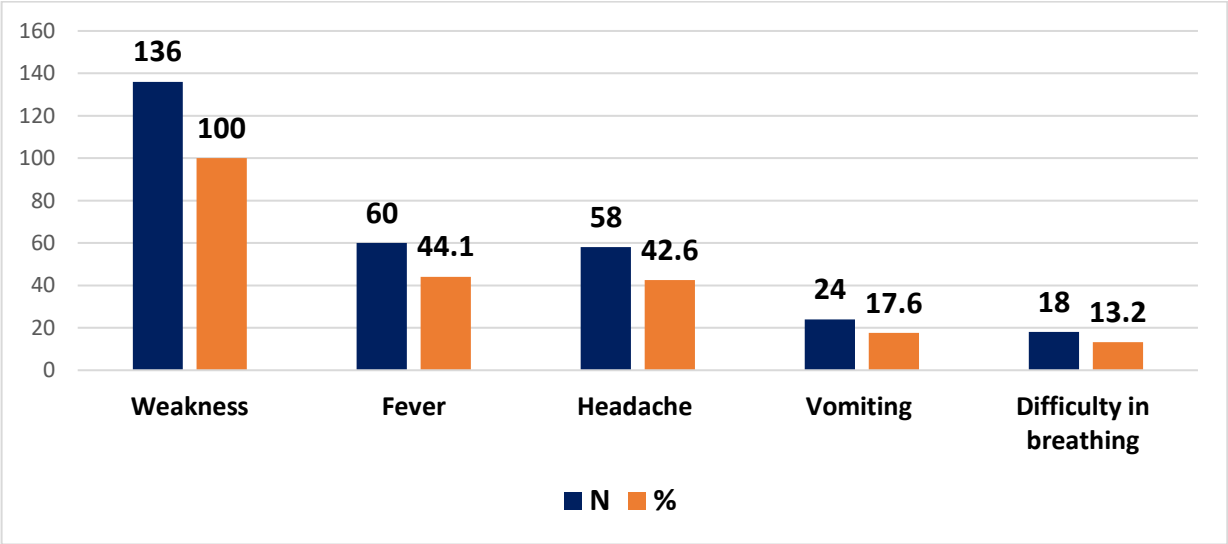

Figure-2 symptoms of hypokalemic flaccid paralysis (N=136)

Table-II Comparison of outcomes concerning age and gender.

	Improvement cases (N=90)	No Improvement cases (N=24)	Residual neurological disability (N=16)	P-value
Age Groups				0.021
1-5	14 (15.6%)	5 (20.8%)	2 (12.5%)	
6-10	18 (20%)	8 (33.3%)	3 (18.8%)	
11-15	26 (28.9%)	7 (29.2%)	8 (50%)	
16-20	38 (42.2%)	4 (16.7%)	3 (18.8%)	
Gender				0.746
Male	64 (71.1%)	15 (62.5%)	10 (62.5%)	
Female	26 (28.9%)	9 (37.5%)	6 (37.5%)	

DISCUSSION

Acute flaccid paralysis (AFP) is a condition marked by sudden weakness in children below the age of 15 years. The precise cause of this condition in this particular age group remains unclear and requires appropriate management and investigation. AFP is significantly caused by hypokalemic paralysis and it typically shows remarkable improvement with potassium supplements. In cases where it is secondary to an underlying condition, addressing the root cause is necessary. Numerous studies from various countries have reported comparable epidemiological patterns [10–13]. Patients having secondary causes of hypokalemic paralysis, including conditions such as distal renal tubular acidosis (RTA), Gitelman syndrome, thyrotoxicosis, Conn's syndrome, and dengue viral infection. A similar investigation reported almost identical findings, with 94% of patients experiencing secondary hypokalemic paralysis [14].

The chronic inflammatory polyneuropathy usually referred as Guillain-Barré syndrome is the leading cause for pediatric patients suffering from AFP. This might be considered as a sole reason for chronic neurological disorders. An earlier study reported that AFP children was mostly suffering from comorbid syndrome caused by Guillain-Barré syndrome followed by myelitis, neuritis, and central nerve system associated hypotonia, infections [15]. Another study showed that the incidence of GBS was 94% followed by numerous factors such as dysphagia (13%), sensory symptoms (23%), and respiratory failure (16%) [16].

Surveillance of acute flaccid paralysis plays a pivotal role in polio eradication monitoring process. It serves as a sensitive indicator for detecting potential cases of poliomyelitis and poliovirus infections. These indicators are used to assess and confirm the proper continuation of acute flaccid paralysis surveillance, ensuring its accuracy and efficiency in detecting potential cases of polio and related infections [17].

Hypokalemia is frequently observed in patients with fever. This decrease in potassium levels during fever could be attributed to factors such as insufficient caloric intake, excessive vomiting, profuse sweating, the use of certain drugs like chloroquine, and heightened adrenergic activity [18]. Additionally, various other factors can contribute to this condition, including exposure to cold, sleep deprivation, upper respiratory tract infections, consumption of specific foods like Chinese cuisine, fatigue, dehydration, sudden surprises or startling events, certain medications, and menstrual cycles [19].

Consuming carbohydrates leads to insulin release from the pancreas, causing shifts of potassium within cells [20]. Additionally, another significant source of carbohydrates [21]. Carbohydrate higher intake increase the susceptibility of AFP attack. The potassium balance of the body adversely affected in secondary hypokalemic paralysis patients [22]. Conversely, potassium intracellular transition was seen in primary hypokalemic paralysis [23]. As a result, a minute amount of potassium and limited recovery time is required for primary hypokalemic [24].

CONCLUSION

The present study found that Guillain bare syndrome was the most prevalent disease followed by viral myositis. Treatment can improve acute flaccid paralysis in over 60% of patients. Hypokalemia can lead to significant respiratory and cardiac issues, potentially resulting in the death of patients, as

indicated in our study. Prompt treatment is essential and satisfying due to the rapid recovery it enables.

REFERENCES

1. Bhagat SK, Sidhant S, Bhatta M, Ghimire A, Shah B. Clinical profile, functional outcome, and mortality of guillain-barre syndrome: A five-year tertiary care experience from Nepal. Neurol Res Int 2019; 2019(1): 3867946. https://doi: 10.1155/2019/3867946.
2. Li X, Xiao J, Ding Y, Xu J, Li C, He Y, et al. Clinical and electrophysiological features of post-traumatic Guillain-Barré syndrome. BMC Neurol 2017; 17(1): 142. https://doi.org/10.1186%2Fs12883-017-0919-x.
3. Ali S, Zia ur Rehman M, Sultan T. Spectrum of acute flaccid paralysis in children. Pak J Neurol Sci 2016; 11(4): 8-11.
4. Aliabadi N, Messacar K, Pastula DM, Robinson CC, Leshem E, Sejvar JJ, et al. Enterovirus D68 infection in children with acute flaccid myelitis, Colorado, USA, 2014. Emerg Infect Dis 2016; 22(8): 1387-1394. https://doi.org/10.3201%2Feid2208.151949.
5. Nelson GR, Bonkowsky JL, Doll E, Green M, Hedlund GL, Moore KR, et al. Recognition and Management of Acute Flaccid Myelitis in Children. Pediatr Neurol 2016; 55(1): 17-21.https://doi: 10.1016/j.pediatrneurol.2015.10.007.
6. Sejvar JJ, Lopez AS, Cortese MM, Leshem E, Pastula DM. Acute flaccid myelitis in the United States, August–December 2014: results of nationwide surveillance. Clin Infect Dis 2016; 63(6):737-745. https://doi.org/10.1093%2Fcid%2Fciw 372.
7. Chong PF, Kira R, Mori H, Okumura A. Clinical features of acute flaccid myelitis temporally associ-ated with an enterovirus d68 outbreak: results of a nationwide survey of acute flaccid paralysis in Japan, August-December 2015. Clin Infect Dis 2018; 66(5): 653-664. https://doi.org/10. 1093%2Fcid%2Fcix860.
8. Wang Q, Zhao Z, Shen H, Bing Q, Li N, Hu J. The clinical and genetic heterogeneity analysis of fve families with primary periodic paralysis. Channels Austin Tex. 2021;15(1):20–30.
9. Farooque U, Cheema AY, Kumar R, Saini G, Kataria S. Primary periodic paralyses: a review of etiologies and their pathogeneses. Cureus.2020;12(8): e10112.
10. Sardar Z, Waheed KAF, Javed MA, Akhtar F, Bokhari SRA. Clinical and etiological spectrum of hypokalemic periodic paralysis in a tertiary care hospital in Pakistan. Cureus. 2019;11(1): e3921.
11. Patra S, Chakraborty PP, Biswas SN, Barman H. Etiological search and epidemiological profle in patients presenting with hypokalemic paresis:an observational study. Indian J Endocrinol Metab. 2018;22(3):397–404.
12. Mohapatra BN, Lenka SK, Acharya M, Majhi C, Oram G, Tudu KM. Clinical and aetiological spectrum of hypokalemic faccid paralysis in Western Odisha. J Assoc Physicians India. 2016;64(5):52–8.
13. Garg RK, Malhotra HS, Verma R, Sharma P, Singh MK. Etiological spectrum of hypokalemic paralysis: a retrospective analysis of 29 patients. Ann Indian Acad Neurol. 2013;16(3):365–70.
14. Messacar K, Schreiner TL, Van Haren K et al.. Acute flaccid myelitis: a clinical review of US cases 2012–2015. Ann Neurol 2016; 80:326–38.
15. Mori S, Endo M, Uchida Y et al.. Six cases of enterovirus D68 infection including acute flaccid paralysis (in Japanese). J Jpn Pediatr Soc 2016; 120:1495–501.
16. Kimura K, Fukushima T, Katada N et al.. Adult case of acute flaccid paralysis with enterovirus D68 detected in the CSF. Neurol Clin Prac 2016. doi:10.1212/CPJ.311.
17. Gunde R, Jayashankar CA, Bhat N, Bhat V, Kodapala S. Gitelman Syndrome Manifesting With Acute Hypokalemic Paralysis: A Case Report. Cureus. 2023 Sep 26;15(9).
18. Gupta M, Daid S. Etiology and Outcomes of Acute Flaccid Paralysis in Adults: A Study in Tertiary Care Center. medRxiv. 2020 May 29:2020-05.
19. Kunju M, Eapen M. ACUTE FLACCID PARALYSIS BEYOND POLIO-A CASE BASED APPROACH. Indian Journal of Practical Pediatrics. 2020;22(1):12.

20. Chukwuedozie VC, Garg T, Chaudhry HA, Shawl SH, Mishra P, Adaralegbe NJ, Khan A, CHUKWUEDOZIE VC. Late-Onset Hypokalemic Periodic Paralysis in an Adult Female With Type 2 Renal Tubular Acidosis: A Case Report. Cureus. 2022 Aug 5;14(8).
21. Ahmed SF, Prathyusha M, Kumar KK, Ara OT. A Study of Clinical and Etiological Spectrum of Hypokalemic Flaccid Paralysis. European Journal of Molecular & Clinical Medicine (EJMCM).;9(03):2022.
22. Paz-Ibarra JL, Sáenz-Bustamante SM, Ildefonso-Najarro SP, Portillo-Flores K, Quispe-Flores MA, Plasencia-Dueñas EA, Concepción-Zavaleta MJ. Clinical-epidemiological profile of patients with thyrotoxic periodic paralysis in two peruvian hospitals. Revista Medica del Instituto Mexicano del Seguro Social. 2022 Jul 4;60(4):379-87.
23. Yuan JH, Higuchi Y, Hashiguchi A, Ando M, Yoshimura A, Nakamura T, Hiramatsu Y, Sakiyama Y, Takashima H. Gene panel analysis of 119 index patients with suspected periodic paralysis in Japan. Frontiers in Neurology. 2023 Jan 26;14:1078195.
24. Radhakrishan S, Sundari M, Dev A, Arun K. A Case Series Of Periodic Approach To Hypokalemic Periodic Paralysis-The Paralysis By The Potassium. Journal of Pharmaceutical Negative Results. 2022 Nov 13:1640-5.