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ORIGINAL ARTICLE

A Diagnostic Pattern in Guillain-Barré Syndrome (GBS) in Children a Multi-Center Study

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ABSTRACT

Objectives: Analyze how children with GBS manage in the clinic.

Materials and methods: From 2015 to 2020, researchers at the Department of Neurosurgery at LRH and HMC Hospital peshawar analyzed data from 67 children (younger than 14) hospitalized at the hospitals with AFP. the Multicenter study in All patients had standard examinations, with most also undergoing [CSF examination], nerve conduction studies (NCS), and electromyography (EMG), depending on available resources. Supportive care, intravenous immunoglobulin (IVIG), and plasmapheresis were employed as treatment options, with the choice of modality determined by clinical need and accessibility to the respective procedures.

Results: Sixty-nine percent (60) of the AFP cases were determined to be GBS. The average kid with GBS was six years old, and the male-to-female ratio was 1.06 to 1. Sixty-eight percent of patients had a previous medical history, seventy-two percent had sensory complaints, and sixty-two percent had autonomic dysfunction. There were ninety-three percent of patients with classical GBS (symmetrical ascending paralysis), two percent with descending paralysis, four percent with relapsing variation, and one with chronic inflammatory demyelinating polyneuropathy. A third of patients had the demyelinating type, another third had demyelinating with axonal involvement, and another twenty-one percent were classed as unclassified based on their electrophysiological investigations. Mechanical ventilation was necessary for 28 patients (44%). Mechanical breathing was necessary for 36% of those with axonal type and 24% with demyelinating type. Of the 28 patients given IVIG, 18 exhibited improvement, five did not, and plasmapheresis was performed on them.

Conclusion: The majority of cases of AFP can be attributed to exposure to GBS. IVIG is an effective therapeutic strategy for children with GBS, and GBS with axonal involvement is more severe than demyelinating kind.

Keywords: Children with Acute Flaccid Paralysis, Guillain-Barre Syndrome

INTRODUCTION

Acute acquired demyelinating polyneuropathy, often known as Guillain-Barre syndrome (GBS), is thought to be immune-mediated. It is the most prevalent cause of acute flaccid paralysis (AFP) after poliomyelitis has been eradicated1. Worldwide, an incidence of [0.05-05/100000] children per year has been reported GBS is distinguished by muscular weakness and areflexia. GBS paralysis is flaccid, ascending, and symmetrical, with absent or decreased deep tendon reflexes (DTRs)[01]. At the start of paralysis, pain and muscular discomfort are prevalent. Aside from flaccid paralysis, the patient may experience sensory problems and involvement of the cranial nerves, breathing muscles, and autonomic nervous system.GBS is often diagnosed clinically. Aside from regular examinations such as CSF and electrophysiological testing, nerve conduction studies and electromyography (NCS & EMG) can aid in diagnosing GBS. Administration of the kid with GBS comprises necessary interventions such as regular monitoring, airway maintenance, ventilatory assistance, feeding, urine and stool control, and frequent posture changes[02]. Intravenous immunoglobulins and plasmapheresis are used for specific management. Steroids have no part in the treatment of GBS. The prognosis for GBS patients is favorable, with full recovery in more than 95% of cases, but it usually takes weeks to months[03].

MATERIALS & METHODS

Department of Neurosurgery LRH and hmc Peshawar conducted a multi center study Between the years 2015 and 2020, researchers looked at. Patients with pseudoparalysis, central nervous system infections, encephalopathy, chronic flaccid paralysis, and stroke were not included in the research, which comprised 67 children and adolescents under the age of 14. The children were transferred to the PICU. The extent of the patient's weakness, cranial nerve involvement, sensory loss, and autonomic failure were all assessed by a comprehensive history and clinical examination. Patients were tracked to check for indicators of weakness, autonomic dysfunction, respiratory muscle involvement, and bulbar paralysis. Common diagnostic procedures such as complete blood count, erythrocyte sedimentation rate, serum electrolytes, and a random blood sugar were carried out on all patients. A cerebrospinal fluid analysis was done in the second week of illness. Electromyography and nerve conduction studies were conducted where possible. Supportive care, steroids, intravenous immunoglobulins (IVIG), and plasmapheresis were selected as therapeutic choices for individuals with GBS based on clinical reasoning and accessibility to these procedures. IVIG was indicated for patients with rapidly advancing illnesses, respiratory muscle paralysis, dysphagia, and autonomic nervous system dysfunction. Statistics were analysed using SPSS 24.0

RESULTS

67 AFP-diagnosed children were studied. Sixty-one (or 90%) had GBS. Three patients with transverse myelitis (0.04%), one with poliomyelitis (0.01%), two with traumatic neuritis (0.3%), and one with porphyria, periodic paralysis, and RTA had AFP (Table.1). The clinical course of GBS patients were investigated based on illness epidemiology, clinical features, clinical variations, and therapy alternatives.

The average GBS patient was 06.02 years old, ranging from 6 months to 14 years. Just 3% were babies, 39% were 1 to 6-year-olds, 32% were 6 to 11-year-olds, and 22% were 10 to 16-year-olds. (Fig.1). 1.06:1, with 38 (60%) male patients. GBS was usually diagnosed in July-September, and March-April .41 (or 69%) patients had a medical history. 20% of the patients had no history of infection or illness, 15% had a lung infection, 14% had a G.I. infection, 20% had a nonspecific fever, and 1% had a rash. Five patients (15%) reported symptoms less than two days before admission, whereas 44 (73%) had symptoms for 2-12 days. 100% of patients experienced generalized weakness and lower-extremity areflexia/hyporeflexia. 27 (85%) people had both arms and legs afflicted, 27 (42%) respiratory muscles, and 21 (35%) cranial nerves. 40 (65%) of 44 (72%) felt pain, paresthesias, and sensory loss. 16 (27%) autonomic dysfunction patients had hypertension

and tachycardia, whereas 14 (22%) had tachycardia. 17 (30%) reported diaphoresis, 4 (07%) bradycardia, and 15 (25%) bowel/bladder disturbance (Table 2).56 (91%) patients had typical GBS (symmetrical ascending paralysis), two had descended paralysis, 3 had relapsing variation, and 1 had CIDP (Table-3). Electrophysiologically tested GBS patients (n=52) were categorized into three categories: demyelinating (34%), demyelinating with axonal involvement (45%), and unclassified (21%). (Table 4). Fifty-one people's CSF fluid was evaluated, and 29 (58%) exhibited albuminocytological separation. Albuminocytological dissociation was determined by measuring CSF protein [>78 mg/dl and 10/cmm]. 27 out of 67 required supplementary breathing aids. Twenty-eight persons were analyzed; 4 had demyelinating, eight had demyelinating plus axonal, 9 were unclassified children, and electrophysiological tests could not be done on six due to a shortage of hospital equipment. 18 (65%) of the 28 patients who needed ventilators were successfully weaned off, 7 (27%) died, and 3 (13%) refused to remain against medical advice. (Table 5). Patients were treated with supportive care, mechanical ventilation, steroids, IVIG, and plasmapheresis. 29 (90%) of 33 supportive group patients improved, 3 (8%) died, and one did not. 28 patients (45%) received IVIG. 18 (65%) improved, 5 (15%) did not, 4 (13%) died, and 2 (7.5%) departed against medical advice. Three of four plasmapheresis patients improved, while two died (Table 6).

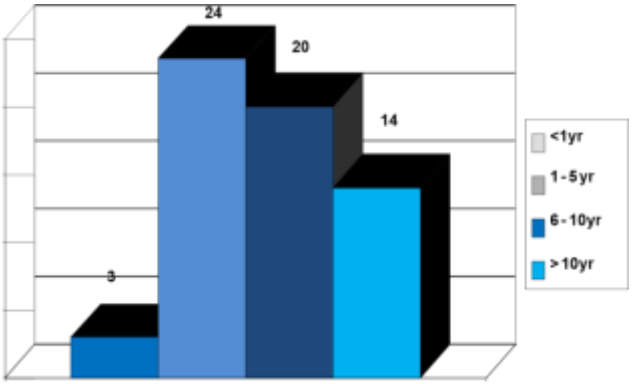

Figure 1: Patients diagnosed with Guillain-Barré syndrome (n = 67) and their ages.

Table 1:Acute flaccid paralysis (n=567)

GBS	60
Trasverse myelitis	3
Traumatic neuritis	1
Prophyria	0
Periodic paralysis	0
Renal tubular acidosis	0

Table 2: Clinical presentation of GBS

Preceding H/O infection	
No H/O any illness	20(30%)
Nonspecific fever	12(20%)
GIT infection	14(23%)
RTI	15(25%)
Rash	01(02%)
Duration of illness prior to admission	
<02 days	09(15%)
2-10 days	44(73%)
>10 days	08(12%)
Weakness	60(98%)
Legs	61(99%)
Arms & legs	53(88%)
Respiratory muscles	27(45%)
Cranial nerves	21(35%)
Areflexia/Hyporeflexia	60(98%)
Sensory symptoms	44(72%)
Pain	
Parenthesis	03(05%)
Sensory loss	1(01%)
Autonomic symptoms	38(62%)
Diaphoresis	17(29%)
Hypertension & Tachycardia	16(25%)
Tachycardia	14(24%)
Bradycardia	04(07%)
Bowel & or bladder	15(25%)

Table 3: Changes in the Clinic (GBS)

Variants	=n	%age
Classical GBS	56	93
Relapsing	03	04
Descending	02	03
Ch. Inflammatory demyelinating polyneuropathy	01	01

Table 4: Neuromuscular and Electromyographic Studies (n=51)

Types	=n
Demyelinating	17 (33%)
Demyelinating with axonal involvement	23 (45%)
Unclassified	12 (23%)

Table 5: Modalities of therapy comparison

Type	No.	Improved	Not improved	Expired/ LAMA
Supportive	33	29	01	03
M.G.	28	18	4	4+1
Plasmapheresis	05	03	01	02

DISCUSSION

In children younger than 14 year acute flaccid paralysis" refers to flaccid paralysis in one or more limbs or bulbar paralysis GBS causes most AFP[04]. Other causes include transverse myelitis, botulism, tick bite paralysis, and traumatic neuritis. Rare causes include polymyositis, diphtheria, porphyria, medications, and vitamin B12 deficiency. In earlier investigations, GBS, TM, polio, and traumatic neuritis were the most common causes of AFP. Porphyria, periodic paralysis and RTA are rare causes[05]. Acute flaccid paralysis (45%) was most commonly caused by Guillain-Barré syndrome (45%) and transverse myelitis (20%), followed by acute disseminated encephalomyelitis, traumatic neuritis, tic bite paralysis, and infantile botulism1. According to a Hong Kong study, GBS (40%) and T.M. (16%) induce AFP in children. Two U.S. studies8,14 concur that GBS causes AFP. Our findings match the referenced study[06]. Our analysis demonstrates that polio is still sometimes found in the U.S. We found no incidences of paralysis from tic bites or infantile botulism, unlike an Australian study. We may not have the target wood tic. 44% of GBS-affected children were younger than six. According to studies from Hong Kong and Central America, this may be connected to a high infection incidence in very young infants[07]. Our 1.06:1 male-to-female ratio is comparable with a Malaysian study that reported 1.04:1. (10). According to our study, GBS rises in the spring and autumn. Summer (July–September) and spring had the most outstanding results (March–April). Summer and winter are peak seasons for gut and respiratory infections, respectively. 46% of individuals suffered breathing-related issues. This number matches a Pakistani study of adults (56%). Three-fifths of the children exhibited cranial nerve involvement, compared to 45% and 51% in previous studies[08]. 71% of children experienced sensory complaints, and 60% had autonomic dysfunction. In a study of children with GBS7, 79% and 51% reported neuropathic pain and autonomic dysfunction. 58% of our patients demonstrated cerebrospinal fluid albuminocytological separation, compared to 98% in another study All patients received a CSF test during the second week of their illness, and protein [>78 mg/dl and cells 10/cmm] was utilized to dissociate. Timing, frequency, and depth of additional dissociation criteria and the CSF test may explain this difference[09]. Electrophysiological examinations categorized GBS children into three groups: demyelinating (33.3%), demyelinating with axonal involvement (42.3%), and unclassified (23.3%). 61% of GBS patients had acute inflammatory demyelinating neuropathy, 24% had acute motor axonal neuropathy, and 13% had Miller-

Fisher syndrome[10]. Variable immune responses to past diseases may explain this discrepancy. 36% (08/23) of individuals with axonal electrophysiology and 77% (09/12) of those not yet categorized required breathing. 32% of children with axonal involvement died, whereas 13% with demyelinating. These data suggest that axonal-type GBS is worse than demyelinating GBS. Patients who received IVIG could not be compared to those who had plasmapheresis since plasmapheresis was conducted on patients who had already received IVIG, nor to those who received merely supportive treatment because those receiving IVIG had more advanced disease. 12,4% of GBS patients survived. These people get mechanical ventilation with or without further treatment. Patients requiring mechanical ventilation had a poor prognosis. Infection and barotrauma may contribute to the high mortality rate in this group[11].

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