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Case Report

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GUILLIAN- BARRE SYNDROME: A CASE REPORT

Udaya lakshmi. K [1], Hema Latha. A [1], Bhavya. V [1], Divya. G [2], Ranganayakulu. D [3]

[1,2]Departmentof pharmacy practice, Sri Padmavati School of Pharmacy, Tiruchanoor, Tirupati, 517503, Andhra Pradesh, India.

[3]Principal, Sri Padmavati School of Pharmacy, Tiruchanoor, Tirupati, 517503, Andhra Pradesh, India.

***Corresponding Author: Udaya lakshmi K.**

India.

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ABSTRACT

Cerebral palsy is commonest motor disability affecting a large pediatric population worldwide. Children affected with the disease presents with developmental delay and motor deficits and may have one or more associated problems like mental retardation, seizures, feeding difficulties along with ophthalmologic and hearing impairments. In the ayurvedic literatures consumption of *ghrita* is highly recommended in the management of psychiatric disorders as it is having lipophilic action and it acts on brain so; it is well established that it can cross the Blood Brain Barrier (BBB). *Brahmi Ghrita* contains *Brahmi (*Bacopa monneri*), Vacha (*Acorus calamus*), Kushtha (*Saussurea lappa*), Shankhapushpi (*Convolvulus pluricalis*) and Purana Ghrita*, indicated for treatment of *Apasmara* (seizures) and *Graha* (syndromic disorders). This *ghrita* was first mentioned in *Charak Samhita* and in due course of time, this formulation was modified by various *Acharya* as per requirement of patients, nature of disease, *Desh, Kala*, etc. *Acharya Kashyapa* has mentioned *Lehanakarma* in *Lehadhyaya* in which *Brahmi Ghrita* is having *Medhajanana karma*. It is given with *Madhu* in unequal quantity in early morning before food. Multiple clinical and experimental studies have been conducted on *Brahmi Ghrita* which has shown its results in improved learning and memory, anticonvulsant action, CNS depressant activity, anti-amnestic actions, antinociceptive action, its effect on depression and in ADHD children and many are on its neurocognitive actions.

KEYWORDS: Cerebral palsy, *Brahmi ghrita*, blood brain barrier, *Lehankarma*, Neurocognitive action.

INTRODUCTION

Guillian – barre syndrome (GBS) is an acute inflammatory polyneuropathy characterized by rapidly progressive, essentially symmetric weaknesses and areflexia in a previously well child. Weaknesses and neuropathic pain are the most predominant symptoms of an affected children, and cranial nerve involvement is common in paediatric GBS. GBS is the most common cause of acute flaccid paralysis in childhood. The variable incidence of GBS in different populations may reflect differential genetic susceptibility or exposure to causative pathogens.

Vaccination against the flu, rabies and meningitis are also documented precipitating factors that have been reported.[1]

Rarely gullian barre syndrome can be fatal in 5-10 % of patients with respiratory failure and cardiac arrhythmia.[2]

Hyperreflexia is significantly associated with the presence of anti GM 1 antibodies.[3]

Epidemiology

Indian case – control study reported that 27.7 % of childhood GBS cases were associated with c-jejuni infection.[4]

Etilogy

Autoimmne – mediated disease.

Environmental triggers (Eg: pathogenic or streeful exposue).

Infections (Eg: Epstein – barr virus, cytomegalovirus, hepatitis, varicella others herpes viruses, mycoplasma pneumonia, c- jejuni) Sugery procedures.

Clinical Findings

Gastroenteric or respiratory illness or immunization can be developed 2-4 weeks after a prodromal of GBS. Childhood GBS had never been proven to result from vaccination against poliomyelitis, tetanus or measles, but several recent publications have suggested a weak association between GBS and immunization against influenza A (H1N1).

Children with GBS usually complain of weaknesses and fatigue.

Parasthesia and pain.
Difficulty in walking,
Arising from the floor or climbing stairs.

Weaknesses are generally symmetrical, starting in the lower extremities then ascending into the upper extremities over days to weeks.

Ataxia resulting from weaknesses and sensory loss is very common in children with GBS rather than from cerebellar involvement.

Respiratory muscle weaknesses in GBS is usually progressive and tends to correlate with the degree of limb muscle weaknesses, but rare cases of pediatric GBS present with acute-onset respiratory failure, which can be life-threatening.

Neuropathic pain and dysesthesias are common and this pain is often poorly localised and may cause markedly irritability, vomiting and headache, with meningism and apparent encephalopathy.

Sensory loss is rarely prominent in childhood GBS but can be identified on detailed examination in about 40% of cases.

Autonomic involvement – manifesting as blood pressure instability, sinus tachycardia, pupillary abnormalities and abnormal sweating – is common and often underrecognized in pediatric GBS.

Spinster dysfunction (urinary retention, urinary and faecal incontinence) is occasionally seen in children with GBS but is far more common in transverse myelitis, tumors and other spinal cord lesions.

Recent reports of headache and severe hypertension as initial manifestations of this disorder demonstrate the importance of considering GBS as part of the differential diagnosis of acute dysautonomia.

Complications
Weakness of respiratory muscles
Autonomic instability
Pneumonia
Adult respiratory distress syndrome
Septicaemia
Pressure sores
Pulmonary embolus
Ileus, constipation gastritis and dysesthesisas
Nephropathy in the paediatrics patients[5]

Clinical Variants of Guillian – Barre Syndrome In Childhood[6,7]

Clinical syndrome	Relative frequency	IgG antiganglioside antibody association
Acute inflammatory demyelinating polyneuropathy	Common	GM1
Acute motor axonal neuropathy	Common	GM1,GD1a
Acute motor and sensory axonal neuropathy	Uncommon	GM1,GD1a
Miller – fisher syndrome	Uncommon	GQ1b,GT1a
Pharyngeal – cervical – brachial variant	Rare	GT1a,GQ1b,GD1a
Polyneuritis cranialis	Rare	GQ1b,GT1a
Acute (ataxia) sensory neuropathy	Very rare	GQ1b,GT1a
Acute pandysautonomia	Very rare	
Acute opthalmoparesis	Very rare	GQ1b,GT1a

Electrophysiology shows reduction in muscle action potentials with relatively preserved motor nerve conduction velocity and normal sensory nerve action potentials and F waves.[6,8]

Pathophysiology
Infectious agents such as Epstein – Barr virus, cytomegalovirus, mycoplasma pneumoniae and jejunum, immunization or surgery which may leads to B-cell and T-cell activation causing antibody production finally immune mediated nerve injury will occur.

Investigations
Clinical examination.
Lumbar puncture.
Neurophysiology and radiologic investigations.
Spinal MRI.
Neuroimaging.
Anti ganglioside antibodies detection.

Differential Diagnosis
The differential diagnosis of childhood GBS includes disorders involving the central nervous system, peripheral nerves, neuromuscular junction, nerve and muscle.

Diagnosis criteria for Guillian – barre syndrome (GBS)
Development of essential symmetric limb Weaknesses
Loss or decrease in deep tendon reflexes within 1 week of onset.
Progression of the above features over several days to 4weeks.

Paresthesias of the hands and feet.
Features casting doubt on the diagnosis.
Persistent asymmetry of weaknesses.
Identifiable sensory level.
Prominent bladder or bowel dysfunction.
Greater than 50 mononuclear cells/mm3 in CSF.

Laboratory abnormalities which support the diagnosis:
Elevation of CSF protein >45 mg / DL within 3 weeks of onset.

Neurophysiologic abnormalities consistent with an acute inflammatory polyneuropathy in a least two limbs:

1. Slowing of motor and sensory nerve conduction
2. Conduction block or temporal dispersion of compound muscle action potentials.
3. Increased distal latencies.
4. Abnormalities of F waves.
5. Criteria for axonal forms include lack of neurophysiologic evidence of demyelination, with loss of amplitude of CMAP or sensory nerve action potentials to at least <80% of lower limit of normal values for age.

Treatment

Respiratory failure – intubation and mechanical ventilation.

Pain – nonsteroidal anti-inflammatory medications and agents used to treat neuropathic pain (such as gabaepntin and tricyclic anti depressents).

Corticosteroids.
Opioid analgesics.
Physiotherapy.
Immobilisation hypercalcaemia – Calcitonin Bisphosphonates.
Immunomodulation,[9]
Intravenous immuno globulin,[11,12]

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