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Guillain – Barre Syndrome and its Ayurvedic Perspective

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Abstract: ***Guillain – Barre Syndrome is an acute demyelinating polyneuropathy which has Probably an autoimmune etiologies. It is a rare neurological disorder in which the Body's immune system mistakenly attack part of its peripheral nervous system which results in weakness of muscles , numbness , loss of reflexes , tingling in arm , leg and other parts of body In Guillain Barre Syndrome is inflammatory & destruction of Myelin sheath. It results in paralysis and other systemic illness. Direct Correlation of GBS with Ayurveda is difficult , Depending on presentation & dosha dooshya samoorchana is Considered 1st & then should proceed with the treatment. If dosha are in avarana then avarana should be removed then kevala vata vyadhi line of treatmemt should be adopted . If only dhatu kshaya brimhana line of treatment should be adopted.***

Keywords: dosha dooshya *samoorchana* , brimhana ,Guillain Barre Syndrome, avarana

1. Introduction

G B SYNDROME is an acute demyelinating polyneuropathy which has probably an autoimmune etiology .It is a rare neurological disorder in which the body's immune system mistakenly attack part of its peripheral nervous system – the network of nerves located outside the brain and spinal cord. There are 3 main types of degeneration processes in the peripheral nervous system – wallerian degeneration, axonal degeneration and segmental demyelination .In GB Syndrome -Inflammatory and destruction of myelin sheath. The peripheral nerves convey sensory information from the body to the brain and motor signals from the brain to body. Pathologically , polyneuropathy may be the result of axonal degeneration / segmental demyelination. In each type acute , sub acute and chronic forms are distinguished. As per *ayurveda* classics this condition can be considered as *sarvangavata* (vata affecting the whole body) . Depending on presentation, *dosha dooshya samoorchana* is considered first and then should proceed with the treatment protocol.

Definition-Guillain barre syndrome (GBS) is an acute , frequently severe , and fulminant polyradiculoneuropathy that is auto immune in nature .

Incidents-Its occurs at the rate of 1 to 4 cases per 100,000 annually . Males are at slightly higher risk for GBs than females . Western countries adults are more frequently affected than children[1].

Causative Factors - 70% of cases of GBs occur 1-3 weeks after an acute infectious process , usually respiratory or gastrointestinal infection. Infection or Reinfection with Campylobacter jejuni . Human herpes virus infection . Mycoplasma pneumoniae .(respiratory tract infection).Swine influenza vaccine .(recent immunization) . HIV – seropositive and systemic lupus erythematosus .GBS also occurs more frequently than can be attributed to chance alone in patients with lymphoma[2].

Physiology of Nerve Cells-*CELL BODY* (soma) is the spherical part of the neuron that contains the nucleus. A long projection called *AXON* carry electrical impulses to neuromuscular junction Transferred to muscles. Axon are wrapped in a sheath of *Schwann cells* that contains *Mylein sheath* . Between Schwann cells are gaps called as *Node of Ranvier* where axons are exposed.

Immunopathogenesis[3]-Several lines of evidence an autoimmune basis for acute inflammatory demyelinating polyneuropathy, most common and best studied types of GBS. Both cellular and humoral immune mechanism Contribute to tissue damage in AIDP . T cell activated is suggested by finding elevated level of cytokines and cytokine receptors are present in serum and in CSF .AIDP is also closely similar to an experimental T- cell mediated immunopathy designated experimental allergic neuritis. Experimental allergic neuritis is induced in laboratory animals by immune sensitization against protein fragments derived from peripheral nerve proteins and in particular against the p2 protein. Based on similar to EAN it was initially thought that AIDP was likely to be primarily a T cell mediated disorder. All GBS results from immune responses to self Antigens (infection agents , vaccines) that misdirected to host nerve tissue through a resemblance of molecular mimicry .Neural targets are Gangliosides . Gangliosides are present in large quantity in human nervous tissue such as Node of Ranvier .Anti Gangliosides antibodies- GM1 (20%-50% Cases of GBS). GBS have surface Glycolipid structures that Antigenically cross react with Ganglioside including GM1 , Concentrated in human nerve. Experimentally, ANTI-GM1 antibodies can trigger complement – mediated injury at Paranodal axon – Glial Junction , disrupting the clustering of sodium channels and contributing block.

Clinical Manifestation[4] - *Motor system* -Rapidly evolving areflexic motor paralysis with or without sensory disturbance. pattern → ascending paralysis that may be first noticed as rubbery legs. Weakness typically evolves over hour to a few days. Frequently accompanied by tingling

sensation in the extremities. Legs are usually affected than the arms. *Deeptendon reflexes*- Deeptendon reflexes disappears with in the 1st few days of onset. *Cranial nerves* -Facial diparesis is present in 50% of affected individuals. *Lower cranial nerve* →bulbar weakness with difficulty handling secretions & maintaining an airway. Pupillary paralysis ,Optic atrophy . *Sensory system* -Largely sensory fibres affected (myelinated), Proprioception is more affected than pain temperature Sensation .*Bladder*-Only in severe cases , If bladder dysfunction is a prominent features and Comes early in course , think other than GBS- spinal cord disease . *pain* - Early stage → pain in neck, shoulder, back/diffusely over the spine, Deep aching Pain may be present in weakened muscles ,Self limited ,Responds to analgesics . *Autonomic involvement* –Common, Seen even in mild cases, Wide fluctuation in blood pressure ,Postural hypotension ,Cardiac dysrryhthmias ,Close monitoring and management. Can be fatal , All require hospitalization,30% require ventilary support .

Laboratory Features[5] - *CSF* →Its often Normal when symptoms have been present for less than 48hours, by the end 1st week the level of protein is usually elevated. Elevated CSF protein level without accompanying plecocytosis, if sustained CSF plecocytosis increased suggests an alternative diagnosis such as HIV. *Electrodiagnostic finding*- In early stage of GBS – Normal Lags behind the clinical events demyelination -prolonged distal latencies ,slowing of conduction velocity , evidence of conduction block and terminal dispersion of compound action potential.

Subtypes Of Guillian – Barre Syndrome[6] **–**

Subtypes	Features	Electrodiagnosis	pathology
Acute Inflammatory Demyelinating polyneuropathy (aidp)	Adults affected more than children; 90% of cases in western world ; recovery rapid ; Anti- GM1 Antidodies 50%	Demyelinating	First attack on schwann cell surface ; widespread myelin demage , macrophages activation , and lymphocytic infiltration ; variable secondary axonal damage
Acute motor axonal neuropathy (AMAN)	Children and young adults; prevent in china and Mexico; may be seasonal; recovery rapid; anti- GD 1 a antibodies	Axonal	First attack at motor nodes of Ranvier; macrophage activation , axonal damage highly variable
Acute motor sensory axonal neuropathy (AMSAN)	Mostly adults; uncommon; recovery slow, often in complete; closely related to AMAN	Axonal	Same as AMAN, but also affects sensory nerves and roots; axonal damage usually severe
M. Fisher syndrome (MFS)	Adults and children; uncommon; ophthalmoplegia , ataxia and areflexia; anti GQ1b antibodies 90%	Demyelinating	Few cases examined; resembles AIDP

Diagnostic Criteria for Guillain-Barre Syndrome[7]- Required -Progressive weakness of 2 or more limbs due to neuropathy Areflexia .Disease course <4 weeks Exclusion of other causes [e.g. vasculitis (polyarteritis nodosa, systemic lupus erythematosus, Churg-Strauss syndrome), toxins (organophosphates, lead), Botulism, diphtheria, porphyria, localized spinal cord or cauda equina syndrome. Supportive -Relatively symmetric weakness, Mild sensory involvement ,Facial nerve or other cranial nerve involvement,Absence of fever, Typical CSF profile (acellular, increase in protein level) ,Electrophysiologic evidence of demyelination.

Differentail Diagnosios[8]- Acute myelopathies , Diphtheri ,Lyme polyradiculitis , Vasculitic neuropathy, Poliomyelitis CMV polyradiculitis , Myopathy, Neuromuscular Junction disorders : Myasthenia gravis ,Botulism ,Paralytic shellfish poisoning .

Treatment[9]- Initiate as soon as possible. Each day counts .2 weeks after the first motor symptoms – immunotherapy is no longer effective . Intravenous immune globulin and Plasmapheresis. Intravenous immune globulin- first choice, easy to administer . Five daily infusion for a total dosage 2g/kg body weight. Plasamapheresis 40 – 50 ml/kg four times a week. Treatment reduces the need for mechanical ventilation by nearly half, increase fully recovery 1year.Glucocorticoids are not effective in GBS. Severe cases worsening phase of GB syndrome – monitoring in a critical care setting. Attention to vital capacity, heart rhythm, blood pressure, nutrition, deep vein thrombosis prophylaxis, cardio vascular status, tracheotomy , chest physiotherapy , 30% requires ventilation

Ayurvedic Perspective of Gullain Barre Syndrome

Direct Correlation of GBS with Ayurveda terminology is difficult .hence it can be understood in various condition like

- Mamsagata Vata
- Kaphavrita Vyana
- Kaphavrita Prana
- Sarvanga Kupita Vata.

Vyadhi	Pathology	Symptoms :	Chikitsa[11]
MAMSAGATA / MEDOGATAVATA[10]	when morbid vata afflicts mamsa and medha	• Gurvangam- heaviness of the body • Ati toda – pricking / tingling sensation in the muscle • Dandamushti hatam yatha- feeling as if someone has hit with a closed fist / stick. • Saruk- pains • Shramitam – weakness	Vatashamana / Vataanuloma followed Brimhana , Virechana, Niruha basti , Shamana chikitsa .

Myeline sheath is a fatty white substance that surrounds the axon of some nerve cells , forming an electrically insulating layer. It is essential for the proper functioning of nervous system. This is damaged in GBS, this can be compared to medogata vata , that is vitiated vata owing to its property of dryness and roughness destroy the fat . This impair the conductivity of nerve signal at their synapse with muscle fibres.

Vyadhi	Pathology	Symptoms :	Chikitsa
Kaphavritavyana[11,12]	The vyana vata being covered by kapha	• Stambha- stiffness all over the body . • Dandaka- body becomes like sticks • Shoola- pain • Shotha- swelling / inflammation • (Ma.ni 22/26) • Gurutasarvagaatraanaam – heaviness of the whole body. • Sarva sandhi asthi ruja – pain in all the joints and bones. • Adhika gatisangha – severe restriction of movements. • (ch.chi. 28/228)	Avarana should be removed later kevala vata line of treatment should be adopted.

Shoola , sarva sandhi asthi ruja , adhika gatisangha this lakshana can be considered in GBsyndrome .

Vyadhi	*Pathology*	*Symptoms*	*Chikitsa*
Sarvangakupitavata[13]	The aggravated vata all over the body	• Gaatrasphurana – pulsation pain all over the body • Gatrabhanjana – breaking pain all over the body • Sandhi vedana – pain in all the joints • Sandhi sphota – curshing pain in all the joints (CH.CHI.28/25)	Abhyanga, Basti .
KAPHAVRITA PRANA[14]	The prana vata is covered by kapha	• Daurbalyam – weakness • Sadanam – fatigue • Tandra – stupor • vairasyam – manifestration of opposite tastes	

Pathya Ahara

- Ahara dravya which are having Properties like madhura , amla and lavana rasa , snigdha , Ushna guna and brimhana .
- In chakradutta , bhaishajya ratnavalli and yogarantnakara pathyapthaya is mentioned in detail.
- Shashtika Sali , kulatha , masha , dadhi , Gardabha Ksheera , patola , shigru , anupa mamsa , taila , yusha .
- In Ayurveda based on the symptoms it can be correlated with sarvangavata , mamsagata vata kaphavrita vyana , kaphavrita prana, sarvanga kupita vata.
- Ayurvedic treatment like panchakarma , shamanoushadhi , physiotherapy play a major role in improving the muscle tone and muscle strength .
- Immunoglobin treatment is costly alternative , cost effectiveness of the ayurvedic treatment seems promising.

2. Discussion

1) Myeline sheath is a fatty white substance that surrounds the axon of some nerve cells , forming an electrically insulating layer. It is essential for the proper functioning of nervous system. This is damaged in GBS, this can be compared to medogata vata , that is vitiated vata owing to its property of dryness and roughness destroy the fat . This impair the conductivity of nerve signal at their synapse with muscle fibres.
2) Shoola , sarva sandhi asthi ruja , adhika gatisangha this lakshana can be considered in GBsyndrome .
3) In Sarvanga vata & kapha vrita prana of the symptoms mimic the symptoms of GB syndrome.
4) Due to Agantuja leading to tridosa prakopa leading to affliction of dhatu causing dhatugata jwara can be considered in GB syndrome.
5) In Avarana 1st remove the Avarana the follow the vataja upakrama.

3. Conclusion

GBS is a rare form of damage to peripheral nerves that causes weakness of the limbs.

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