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AN EPITOME ON GUILLAIN BARRE SYNDROME AND ITS MANAGEMENT THROUGH AYURVEDA

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ABSTRACT

Guillain Barre Syndrome(GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature. An autoimmune reaction may damage the myelin sheath around the nerve. This damage is called demyelination. Damage parts of nerves cause tingling, muscle weakness, and paralysis. A feature common in all GBS variants is a rapidly evolving poly- radiculoneuropathy preceded by a triggering event, most often an infection. GBS generally manifests as a symmetric motor paralysis with or without sensory and autonomic disturbances. Population-based surveys attempting to document the annual incidence of GBS have been conducted in various countries worldwide and generally are in agreement on a rate of 1 to 3 per 100,000 persons annually. In classics of Ayurveda we get scattered references about Guillian Barre Syndrome by seeing its etiology, signs and symptoms we can correlate to ***Kapha vruta vyanavata, Sarvanga vata, Pranavrutha vyana, Nirupasthamba vatavyadhi, Anukta vata vyadhi***. *Ayurveda* stressed on the holistic approach to diseases, and hence, treatment is aimed at both the *dosha* (causal factor) and the *vyadhi* (disease). The present study deals with an overview of Guillian Barre Syndrome in *ayurveda* and its management.

KEYWORDS: Guillain Barre Syndrome(GBS), *Kapha vruta vyanavata*, *Sarvanga vata*, *Pranavrutha vyana*, *Nirupasthamba vatavyadhi*, *Anukta vata vyadhi*.

INTRODUCTION

Guillain- Barré syndrome (GBS) is a condition characterized by the acute or sub acute onset of varying degrees of weakness in limbs or cranial nerve-innervated muscles, associated decreased or absent deep tendon reflexes, and a characteristic profile in the cerebrospinal fluid and electro diagnostic studies.[1] Is a heterogeneous rapidly progressive disease. GBS has a monophasic disease course post infection and is usually non relapsing. Around 20-30% of patients may be associated with life threatening respiratory failures. Prevalence is 2.7 per 1,00,000 per year.[2] GBS occurs in all age groups, although rarely in infants, and the incidence varies. From birth to 30 years, the annual incidence is fairly uniform at 1.3 to 1.9 per 100,000. Peaks are noted in late adolescence and young adulthood, as well as in the elderly. The first peak likely correlates with increased risk of cytomegalovirus and *Campylobacter jejuni* infection. The reason for the peak in the elderly is unknown but is postulated to be caused by failing immune suppressor mechanisms.

In GBS rapid areflexic motor paralysis occurs, typically ascending paralysis, from lower limbs to upper limbs, evolves within hours to days. When it involves respiratory muscles (in around 20-30% patients)[3], it may affect life.

The underlying aetiology and Pathophysiology of GBS are not completely understood[4], but it is thought to be an immune-mediated process, resulting from the generation of autoimmune antibodies and inflammatory cells that cross-react with epitopes on peripheral nerves and roots, leading to demyelination, axonal damage or both.[5] This immune response is thought to be initiated in response to a variety of antigenic stimuli, such as viral or bacterial infection, particularly *Campylobacter jejuni*.[6] It is a post infectious poly neuropathy that causes demyelination in mainly motor but sometimes even in sensory nerves. It is not a hereditary disease. It affects people of all age groups.[7]

In ayurveda, *Vayusyantra tantra dhara*, *prana*, *udana*, *samana*, *vyana*, *apana*, *athmaha*, the *vata*, in its normal

state of functioning sustains all the organs of the body. It consists of *prana, udana, samana, apana and vyana.* It restrains and impels the mental activities. It co-ordinates all the sense faculties. In classics of *Ayurveda* we get scattered references about Gullian Barrie syndrome by seeing its etiology, signs and symptoms we can correlate to *Anukta vyadhi* having correlation with *pitta vikriti, kapha kshaya and vata prakopa Sarvanga vata, kapha vrutavyana vata. Shiras* is a seat of *kapha*. Any depletion resulting in the vitiation of *kapha* and ultimately resulting in the vitiation of *vayu*. Treatment regimens have been described to combat various symptoms seen in the course of the disease.

Table 1: Clinical Pathology and Clinical Feature of Guillian-Barrie Syndrome.

Acute Inflammatory Demyelinating Polyradiculoneuropathy (Aidp)	Acute Motor Axonal Neuropathy	Acute Motor Sensory Axonal Neuropathy	Miller Fisher Syndrome
Acute inflammatory demyelinating poly radiculoneuropathy (AIDP) is the commonest type of Guillain-Barré syndrome[8] Necropsy studies have shown lymphocytic infiltration of the peripheral nerves and macrophage mediated segmental demyelination.[9] Axonal loss may also occur, especially in severe cases as a secondary event. These pathological changes seem to be mediated by both humoral and cellular immunity in variable degrees. Characteristic Electro physiological features reflect segmental demyelination. Subsequent remyelination is associated with recovery	During summer epidemics of Guillain-Barré syndrome in northern China in 1991 and 1992, a majority of patients was found to have a pure motor axonal form of neuropathy, and the term "acute motor axonal neuropathy" (AMAN) was coined. Around 55–65% of the patients belonged to this category, of whom 76% were seropositive for *C jejuni*, compared with 42% in AIDP cases.[10] The earliest pathological changes are lengthening of the nodes of Ranvier, distortion of the paranodal myelin, and dissection of the axon from the adaxonal Schwann cell plasmalemma by extending macrophage processes.[11] Tendon reflexes could either be preserved[12] or exaggerated[13] the latter particularly in AMAN cases. Hyper reflexia is seen in about one third of patients, usually during the early recovery phase and occasionally in the acute phase. This finding is significantly associated with the presence of anti-GM1 antibodies and less severe disease[12] AMAN is characterised by rapidly progressive weakness, often with respiratory failure and usually good recovery[14]	The evidence of axonal degeneration in Guillain-Barré syndrome has been reported by some investigators in the past. In 1984, Brown and Feasby reported that the very low response amplitudes that can occur because of axonal degeneration in Guillain-Barré syndrome were correlated with subsequent denervation of muscles and a poor clinical outcome[15] In 1986, Feasby *et al* published observations on seven patients who had a very acute and severe illness with motor and sensory dysfunction, characterised by marked muscle wasting and poor recovery. Electrophysiology showed in excitable motor nerves and evidence of sensory and motor axonal dysfunction. Necropsy in one of the cases showed features of axonal degeneration with no demyelination or inflammation. They concluded that the features suggested a new clinico pathological entity[16] In AMSAN the disease course is typically fulminant, generally with slow and incomplete Recovery. This group probably has the most severe form of immune mediated axonal damage in Guillain-Barré syndrome.	In 1956, Fisher described three patients with ataxia, areflexia, and ophthalmoplegia (internal and external)—the classical triad of signs in the Miller Fisher syndrome Mild limb weakness, ptosis, facial palsy and bulbar palsy may also occur in Miller Fisher syndrome[17] This entity accounts for about 5% of patients with Guillain-Barré syndrome[18] Miller Fisher syndrome has been shown to be associated with preceding infections with two *C jejuni* strains, Penner serotype 2 and Lior serotype 4[19] Almost all patients have IgG autoantibodies against ganglioside GQ1b[20], which plays a key role in the pathogenesis. The pathogenesis of ataxia has been a focus of debate. Both peripheral and central mechanisms have been proposed. Some workers have suggested a peripheral mechanism, as a result of abnormalities of joint position sense and muscle spindle proprioception.[21] Motor and sensory nerve conduction velocities are either normal or minimally slowed. When slowed, the conduction velocity improves with clinical recovery.85 The tibial H reflex is

			usually absent. On needle electromyography, denervation changes are absent in limb muscles[22]

Clinical features are Symptoms are preceded by an antecedent event in about two thirds of patients.[23] Respiratory infections are the commonest, reported in about 40% of cases within one month before the onset of the disease.[24] About 20% experience gastroenteritis as the antecedent cause.[25] The commonest manifestation is limb weakness, more proximal than distal. Facial palsy is the commonest type of cranial nerve involvement involvement (in 53%), followed by bulbar weakness, ophthalmoplegia, and tongue weakness.16 In about half the cases the illness is heralded by sensory symptoms. Altogether about 80% have sensory symptoms.[26] Pain is a very common symptom, experienced by around 90%, and is often severe.[27] Autonomic dysfunction is seen in about two thirds of the cases, manifesting as either excess or reduced activity of the sympathetic or parasympathetic nervous system.[28]

Pulse and blood pressure changes are the commonest manifestations of dysautonomia (Table 2). The onset of symptoms can either be acute or subacute. Gradual recovery takes place after a plateau phase. In a large multicentre study, the mean time to reach nadir, improvement, and clinical recovery were 12, 28, and 200 days, respectively.[29] It was also found that 98% of patients achieved the plateau phase by four weeks from the onset. The mean duration of the plateau was found to be 12 days in another study.[30]

Table 2: Clinical features of Guillain-Barré syndrome in brief.[31,32]

• **Motor dysfunction**
Symmetrical limb weakness: proximal, distal or global
Neck muscle weakness
Respiratory muscle weakness
Cranial nerve palsies: III–VII, IX–XII
Areflexia
Wasting of limb muscles
• **Sensory dysfunction**
Pain
Numbness, paraesthesiae
Loss of joint position sense, vibration, touch and pain distally
Ataxia
• **Autonomic dysfunction**
Sinus tachycardia and bradycardia
Other cardiac arrhythmias (both tachy and brady)
Hypertension and postural Hypotension
Wide fluctuations of pulse and blood pressure
Tonic pupils
Hypersalivation
Anhydrosis or excessive sweating
Urinary sphincter disturbances
Constipation
Gastric dysmotility
Abnormal vasomotor tone causing venous pooling and facial flushing
• **Other**
Papilloedema

Antecedent events in Gullian- barrie Syndrome

INFECTIONS

Campylobacter jejuni Infections are well established as antecedent events of the Guillain-Barré syndrome. *Campylobacter jejuni* is the commonest pathogen identified. About 20% of patients report a preceding diarrhoeal illness.[33] Patients tend to develop acute motor axonal neuropathy or acute motor–sensory axonal neuropathy more often, and acute inflammatory demyelinating polyradiculoneuropathy less often, in association with *C jejuni* (24% *v* 2% and 40% *v* 76%, respectively).[34] Guillain-Barré syndrome following *C jejuni* infection has been shown to be associated with slower recovery, severe residual disability, and axonal degeneration.[35] ***Cytomegalovirus*** is the second commonest infection reported. The evidence of

preceding cytomegalovirus infection was present in 5% of patients with Guillain-Barré syndrome.[36] It is found more commonly in females and young age groups. Affected individuals tend to have a severe course initially, with respiratory difficulties. They often develop cranial nerve palsies (usually bilateral facial palsy) and severe sensory loss Reduced sensory nerve action potentials are found more often in this group, but nerve conduction velocity, percentage of conduction blocks, and denervation activity do not differ significantly from other groups. Their recovery is usually delayed[37], *Other infections* associations with **Epstein-Barr virus** and ***Mycoplasma pneumoniae*** are more often found in Guillain-Barré syndrome than in control patients (10% and 5%, respectively). Serological evidence of infections with *Haemophilus influenzae*, parainfluenza type 1 virus, influenza A and B viruses, adenovirus, varicella zoster virus, and parvovirus B 19 is not more common than in controls[38] The numerous other preceding infections that have been cited—such as hepatitis A, B, C, and D, typhoid, and falciparum malaria are confined to anecdotal case reports.

Vaccine Isolated case reports and epidemiological studies have drawn attention to a possible association of Guillain-Barré syndrome with several vaccines including Semple rabies[39] oral polio[40] influenza,[41] measles, measles/ mumps/rubella (MMR),[42] tetanus toxoid containing vaccines,[43] and hepatitis B.[44] However, a temporal association between the two events does not necessarily mean there is a cause– effect relation. One needs to evaluate the available epidemiological data to decide whether the alleged association is statistically significant.

Concepts of Ayurveda in Degenerative disorder-Guillain Barrie Syndrome

Ayurveda classics had explain about the Degenerative disorder in detail with scattered references in different condition i.e., *vataprakopa lakshana, Dhatukshaya lakshana, vatavyadhi, nirupasthambha, Anukta, avarana, kevalavataja, anonya avarana, gata vata.*

Niruasthamba vatavyadhi is the disease caused by *vata* exclusively, since it is neurodegenerative disorder caused due to degenerative changes, this can be consider as *dhatukshayajanya – nirupastambhit vaatvyadhi*.

Hetusevana (prayaha vataprakopaka)
↓
Rukshata, kharata, parushata produced in different srotas
↓
Vayupuran at riktasthana i.e. prakupita vayu 'kha' vaigunyasthanashrita
↓
Dhatukshayajanita i.e. Nirupastambhita Vatavyadhi

Pranavata controls *prayatna* action of *udana* and in turn *gati* induced by *vyana vata* becomes impaired. Due to decreased *prayatna* (action potential), there is less *urja karma (*ATP production) leading to decreased *bala* (cellular metabolism) and there is *ama dravya* produced at cellular level. This *ama* leads to cellular *strotorodha* (obstruction) and *vimargagamana* of iron which then reacts with oxygen to produce free radicals **(***ama visha*) and destroy the cell. At the end, *vata prakopa* occurs and manifest the features of GBS. *Prana* as *avaraka* (which obstructs) and *vyana* as *avarya* (which is getting obstructed) participate in pathogenesis. *Avarana* initiates pathogenesis followed by dhatu (body elements) *kshaya* (diminution) and *vata vyadhi.*

As in normal state, *Vyana Vayu* performs its *Rasa-Rakta Vikshepana Karma* normally. But when it gets vitiated, *Dhamani Sankocha* occurs due to its *Ruksha, Shita* and *Khara Gunas*, resulting in the reduction of *Srotovivar* (lumen of the Channels). Due to this there will be narrowed pathway, *Avarodha* (obstruction) occurs in *Rasa-Rakta-Vikshepana Karma*, causing decreased functioning in the particular site. The *vata*-specific nervous tissue, known as *prana vayu*, includes the movement of impulses along the nerves, as well as neuro transmitter movements across the synaptic space. Most of the western nervous system ailments arise from dysfunctional *vata dosha*.

Vataja Ahara& Vihara, abhigata
↓
Vata Prakopa
↓
This prakupita vata fills the Rikta srotas
↓
Leads to vata vikara (vata guna like laghu, chala guna vrudhi)

Based on some signs and symptoms it can be correlated to Sarvanga vata, *kapha vruta vyanavata, Pranavrutha vyana, Nirupasthamba vatavyadhi, Anukta vatavyadhi*. Here the *nidanas* of *Vata vyadhi* can be considered. *Vayu* gets aggravated by the following *nidanas* such as Intake of *Ruksha* (unctuous), *sheeta* (cold), *alpa* (scanty) and *laghu* (light) food. *Ati vyavaya* (Excessive sexual indulgence), *Ati prajagara* (remaining awake at night). *Vishama upachara*(inappropriattherapeuticmeasures); *Ati Doshaasruksravana*(administration of the therapies which cause excessive elimination of *Dosha* and blood); *langhana*(keeping fast in excess); *plavana*(swimming in excess); *Ati adhva*(walking excess); *Ativyayama*(excessive exercise); *vichesta*(other physical activities in excess); *dhatu kshaya*(loss of dhatu); excessive emaciation due to *chinta*(worry), *shoka*(grief) and due to *vyadhi*(affliction by diseases). *Dukha shayya asana*(sleeping and sitting over uncomfortable bed); *krodha*(anger); *divaswapna*(sleeping during day time); *vegasandharanat* (suppression of natural urges); due to formation of *ama*; *abhigata*(trauma); *abhojana*(abstention from food); *marmabhighata* (injuries to *marmas*). Riding over *gaja*(elephant), *ustra*(camel), *ashwa*(horse) or *sheegrayana*(fast moving

vehicle) and *patamsanat* (falling down from the seats on these animals and vehicles)[45] (Compression injuries) and *Virudha Sevana* (Intake of incompatible foods).[46]

If there is symptoms are seen in whole body then we can correlate to *sarvanga vata* where its *lakshanas* like *Gaatra supura*(twitching sensation- Give a short sudden jerking or convulsive movement), *Gatra Banjana*(affliction of the entire body with different types of pain), *Sandhi sputana and Sandhi vedana* (feeling as if the joints are getting cracked). If *vyana vata* is occluded by *kapha*, then there will be *Guruta*(heaviness) in *Sarvagatranam* (all over the body), Ruja (pain) in *Sarvasandhyaasthi* (all the joints and bones), and *Gatisanghasthata adhika* (excessive loss of mobility). Injury to *shiras* give rises to *manya-stambha*(torticollis), *Ardhita*(facial paralysis), *Chakshu-vibhrama*(agitation of eyes), *moha* (unconsciousness), *udvestana*(cramps), *ceshta-nasha*(loss of motor activities), *kasa*(cough), *shvasa*(dyspnea), *hanu-graha*(lock-jaw), *mukatva*(dumbness) *gadgadatva* (lulling speech), *akshi-nimilana*(closure of eye lids), *ganda-spandana*(twitching of cheeks), *jrumbana* (yawning), *lala srava*(excessive salivation), *svara hani*(aphasia), *vadana jihmatva*(twisting of face).[47]

If *Vyana vayu* is occluded by *pranavayu* then there will be symptoms like *sarvendriyanam shoonyatvam* (there will be loss of the functions of all the senses), *gnatva smrithibalakshayam* (there will be loss of memory as well as strength)[48] *Nirupasthambha vata vyadhi* is the disease caused by the *vata* exclusively and there is no any other occlusion present with it.[49] Guillain Barre Syndrome commonly occurs post viral or any other infections. Seeing the pathophysiology of GBS we can say that it is *Vata* dominating disorder with association of *Pitta* and *Kapha dosha*. Due to *Kapha Avaranum*, Vitiated *Vata* and *Pitta* accumulated in the *Majja Dhatu* and cause *Dhatu Kshaya* and *Oja Kshya*. Here is *Mandagni* play an important role as a precipitating factor.[50] In the demyelinating forms of GBS, the basis for flaccid paralysis and sensory disturbance is conduction block. First attack on schwann cell surface, widespread myelin damage, macrophage activation, and lymphocytic infiltration. If the axonal connection remains intact the recovery will be faster as rapidly as remyelination occurs. Circumstantial evidences suggests that all GBS results from immune responses to nonself antigens (infectious agents /vaccines).[51] By analysing the *Vyadhivruthanta* (history of illness*)*, *Nidana* (etiology), *Lakshanas* (symptoms) presented here we have taken in consideration of (pathology)and *Avaranajanya-vatavyadhisamprapthi* we can diagnose as *Sarvangavata, pranavruta vyana vata, kaphavruta vyana vata, anukta vata vyadhi, nirupasthamba vata vyadhi.*

SAMPRAPTHI GHATA

***Dosha*:** *vata pradhana (prana vata, udana vata & Vyana vata)*

Dushya: *Rasa, Rakta, Mamsa, Meda, Asthi, Majja, Sira, Snayu, Kandara*

Agni*-Jataragni* and *Dhatwagnimandya Adhistana: sarvasharira*

Aama*-Jataragni* and *Dhatwagnimandyajanya*

Srothas*- Rasavaha, Raktavaha, Mamsavaha, Medovaha, Ashtivaha, Majjavaha*

Srothodushtiprakara*-Sanga*

Udbhavasthana*-Amashaya, Pakwashaya*

Sancharasthana*– Sarvashareera*

Vyaktasthana*-Ubhayashakha*

Ragamarga*– Madhyama*

Sadhya asadhyata*- Sadhya/ krichrasadhya*

INVESTIGATION

Diagnostic criteria for Guillain-Barré syndrome have been laid down, based on clinical, laboratory, and electrophysiological features.[52] Progressive motor weakness and areflexia are prime requirements for diagnosis. Cerebrospinal fluid analysis is the only laboratory criterion. However, other laboratory tests provide corroborative evidence for diagnosis and are useful in the management (Table 3). In CSF, an elevated or rising protein level on serial lumber punctures and 10 or fewer mononuclear cells/mm3 strongly support the diagnosis. CSF protein level may be normal during the first week. In one of the studies 12% of patients were found to have > 5 cells/µl in the CSF.[53] CSF protein level may be normal during the first week. In one of the studies 12% of patients were found to have > 5 cells/µl in the CSF.[54] The presence of more than 50 mononuclear cells raises doubts about the diagnosis. CSF pleocytosis is well recognised in HIV associated Guillain-Barré syndrome[55] Electro physiological features differ according to the clinicopathological type Magnetic resonance imaging can be useful in diagnosis, especially when the electrophysiological findings are equivocal. It is a sensitive but unfortunately non-specific test. Spinal nerve root enhancement with gadolinium on MRI is a non-specific feature seen in inflammatory conditions and caused by disruption of the blood–nerve barrier. Selective anterior root enhancement appears to be strongly suggestive of Guillain-Barré syndrome[56] A study showed that 83% of patients had enhancement of the cauda equina nerve roots[57] Prominent nerve root enhancement was found to correlate with pain, disability grade, and time for recovery.[57]

Table 3: Investigation.

Investigations
• Cerebrospinal fluid
• Antiganglioside antibodies
• Stool culture for *C jejuni*
• Antibodies to *C jejuni*, cytomegalovirus, EBV, HSV, HIV,*M pneumoniae*
• Biochemical screening: urea, electrolytes, liver enzymes
• Full blood count
• Erythrocyte sedimentation rate
• ECG
• Autonomic function tests
• Electrophysiology
Electrophysiological features
• **AIDP**
Reduced conduction velocity Conduction block or abnormal temporal dispersion Prolonged terminal latency Absent F wave or prolonged F wave latency
• **AMAN**
Absent or reduced compound muscle action potential (CMAP) amplitude Normal motor terminal latency and conduction velocity Normal sensory nerve action potential (SNAP)
• **AMSAN**
Absent or reduced SNAP amplitude Absent or reduced CMAP amplitude Normal motor terminal latency and conduction velocity

MANAGEMENT OF GUILLAN BARRIE SYNDROME

In contemporary science the treatment of GBS has two components: supportive care and specific therapy.

Supportive care remains the cornerstone of therapy. If patients advance past the acute phase of illness, most with recover function. However, the neuropathy can advance so rapidly that endotracheal intubation and mechanical ventilation may be necessary within 24 hours of symptom onset[58] For this reason, all patients who have GBS should be admitted to a hospital for close observation for respiratory compromise, cranial nerve dysfunction, and autonomic instability. Autonomic nervous system dysfunction may manifest as fluctuations in blood pressure, cardiac dysrhythmias, gastrointestinal pseudo-obstruction, and urinary retention.[59] Prophylaxis for deep venous thrombosis should be provided because patients frequendy are immobilized for many weeks. Pain and psychologic stress should be treated. Narcotics should be used with caution because risk of Ueus is already increased. Physical therapy, including gentle massage, passive range-of-motion exercises, and frequent position changes may provide pain relief. Carbamazepine (Tegretol) and gabapentin[60] (Neurontin) have been used as adjuncts in pain management in GBS. Patients who were treated with these medications required less narcotic analgesia with fewer narcotic side effects and minimal sedation compared with those who received placebo. Patients are paralyzed by the Illness, but mentally alert and fearful. Reassurance and discussion about the phases of illness and recovery can help reduce psychologic stress.

Specific treatment should be initiated soon after diagnosis. High-dose intravenous immurioglobulin (IVIg; 400 mg per kg daily for five days) or plasmapheresis (five exchanges over five to eight days) can be initiated. To determine whether IVIg was as effective as plasma exchange in treating patients with GBS, a large multicenter trial was designed to compare plasma exchange and IVIg and the combination of both treatments for GBS. The study followed 150 patients over four weeks. There were no statistically significant differences in the disability rating between the two treatment groups. IVIg and plasmapheresis were found to be equally effective therapies[61]

In Ayurveda, it can be consider under the *sadhya* and also *Krichra sadhya* so a great deal can be done to ease symptoms and improve the quality of life of the patient. Treatment for the GBS usually involves the use of adaptive devices that help the patient attain as much independence as possible. The most important goal in the management of patients with GBS is to identify treatable entities like as lesion must be recognized promptly and treated appropriately.

Antah Parimarjana Chikitsa

There are three types of Antah parimarjana chikitsa, they are a) Shodhana, b) Shamana and c) Rasayana.

Role of *Shodhana*

Virechana

Mrudu Shodana by giving *snehapana* (arohanartha) with *Ashwagandha Ghrita, Moorchitha Goghrita, Kalyanaka gritha, Ksheerabala Taila(7,21 times avartita), Indhukanta gritha, Moorchitha taila.* By *snehana doshas* will achieve *utklishta avastha*, where in this state doshas will detach from *shaka* to *koshta* which is devoid of *leena avasta* and is ready to eliminate[63] *Mrudu virechana*- **Gandharvahastadi ErandaTaila –** Is the *Taila yoga containing Chirbilva, Chitraka, Shunthi, Haritaki, Punarnava, Yavakshara, Musali*. The *guna karma* of these drugs have *Usnavirya*, they showed their *Dipana*(appetizer), *Pachana* (Digestant), antiinflammatory, purgative, *malashodhaka*, analgesic *vedanasthapana, vatakaphahara* properties on this patient. *Shunthi* stimulates nerves and improves impulse transmission. *Haritaki* acts as *anulomaka* and gives strength. Thus *Erandataila* showed its best act as *Vatashamana, anulomaka*. Its processing with *Gandharvahastadi* gives stimulation and strength to nerves. The *virechana* eradicates the *Ama dosha* and clears the *Avarana*

Basti: *Basti* is considered as *ardhachikitsa* in *kayachikitsa vyadhis*, which is a form of Bio purifactory process administered into the rectum through anal route. *Vata* is responsible for the cognitive and neo-cognitive functions of the brain and secretion of various chemical neurotransmitters and hormones. *Vata prakopa* is mainly due to *marga avarna* and *strotavarodha*. For *Strotavarodha, strotoshodhana* should be done, which can be done with *Tikta rasa*. If the body is so weak that we cannot perform the classical *shodhana* procedures, as they will further raise the level of *vikrit vata*. So, we can easily administer *basti*. This basti karma exertes more systemic action besides exerting local action probably entering local action probably entering through large intestines involving enteric nervous system, it also helps to balance the gut brain axis. As *Yapana basti* is considered as *ayusho yapanam* we can understand this statement as regeneration of nerve, *deerghakalanuvartana* that is administered for long time without any adverse effect and also has *Rasayana* effect. *Kala Basti* is best for *Vata* Management. It brings all *Doshas* from whole body into *Koshtha* and expulsion of the *Doshas*.*Niruha* was given with *Mustadi Yapana Basti*[62] also called as *Rajayapana Basti*, as this is *Yapana Basti*, it is safe for children without side effects and can be given in any season. In the *PoorvaKarma* of *Basti, Snehana* and *Swedana* should be done on *Kati-Pradesha*, to make *Kala Basti* more effective in *Vata Shamana* and giving the patient *Balya* and *Brimhana* Effect.

Nasya prayoga: *Nasya karma* helps in acting on CNS and gives strength to *twacha, skandha, griva, aasya, vaksha*. In *Nasya*, the drug is administered through nasal route, by administering to nostrils this reaches the *Shringhataka (shiro marma)* through *nasa strotas* which scrapes the morbid *doshas* in supraclavicular region and extracts them from the *uttamanga*. So with *Ksheera bala taila, Maha masha taila* one can administered the *nasya* this will acts as *Vatahara, Balya and Brihmana* which gives strength and stability to patient.

Shamanoushadhi in Guillian Barre Syndrome

Some of the commonly used *shamanoushadhis* in all kind of vata rogas are *Bhadradaru, kushta, meshashringi, bala, atibala, sahachara, agnimantha, Yastimadhu, Manjistha, Mandukaparni, Nirgundi,* and *Dashamula*

Herbomineral preparation

Yogendra rasa, Brihat vata chintamani Rasa, Brahmi vati, Ashwagandha Ghrita, Brahmi Ghrita, Kalyanaka Ghrita, Mahakalyanaka Ghrita, Vidaryadi Kashaya, Sahacharadi Kashaya. Yograj Guggulu, Kaisore Guggulu, Trayodasanga Guggulu, Panchtiktagrita Guggulu, Mahavatavidhvansani Rasa, Vata Gajankusha Rasa, Lashunadi Vati, Chitrakadi Vati, Hinguvadi Vati, Kupiluhinguvadi Vati, Kapikacchu Churna, Ashvagandha Churna and *Dashamularista* are usually treat nerve damage.

Rasayana

Ashwagandha Rasayana, Ajamamsa Rasayana, Brihat Chagaladi Ghrita, Chyvanaprasha

Bahirparimarjana Chikitsa

It can be classified into 3 types.

- **Abhyanga**

Whole body massage with medicated oil which relieves the symptoms. The general line of treatment for *nirupastambhita vatavyadhi* was taken into consideration, *Sarvanga abhyanga, padabhynga* are *balya* and *vatahara karma*. *Abhyaṇga* (oleation therapy) mitigates *Vātadoṣa* act gives *Puṣṭi* (promotes strength). *Doṣa* involved is *Vāta* and the disease is caused due to the reduction in its *Chalaguṇa* causing inability to transmit nerve impulses, this helps in opening up of blocks in nerve conduction and facilitates remyelinating of nerves; thereby helps to transmit nerve impulses.

These *karmas* enhance the muscle power and thus helps in attaining the maximum balance of body. For *abhyanga* we can use *Dhanwantari Taila* there will be excruciating pain it is believed that by its mode of action pain subsides by this *taila*, *Balaashwagandha lakshadi taila* can also to be used. Based on the patient we can choose the *abhyanga taila*. Other tailas like *Mahamasha taila, ksheera bala taila, mahanarayana taila, sahacharadi taila, moorchitha tila taila*.

- **Udwartana**

It is a process where massage is done with some pressure and in upward direction (*Pratilom Gati*). *Kolakulatthadi Churna* was taken as this is *Ushna* and *Vata Shamaka*. The purpose to start the *Panchakarma* Procedures with *Udwartana* was that, in neurodegenerative or demyelinating disorders there is always some involvement of *Aama*. To get *Nirama Avastha, Udwaratan* is very useful as friction while scrubbing produces heat*(Ushnata*), the powder we took is *Rukhsa*(dry) and *Laghu* and was heated, these *Gunas* are opposite to the *Gunas* of *Aama* (*Picchila,Guru* and *Sheeta)*.

Thus helps in getting *NiramaAwastha*, along with this, due to *Ushnata Guna* initiation of *Vata*pacification process starts. When we start the therapy with *Udwartana* we are preparing the body for other procedures by giving the whole body a messageto be prepare through touch therapy. Here *Kolakulatthadi churna* was used for *Udwartana* which has *badar, kulathi* etc. ingredients which are *Vata Shamaka*.

- **Swedana**

Swedana therapy in the form of *Sankara Sweda* again and again told directly in *Indriyagata vata*. In *Swedana-shastikashali pinda Sweda, patrapinda sweda, nadi sweda* can be done, *swedana* helps in the nourishment of muscles and peripheral nerve. *Shastikashalipindasweda*. All ingredients of the *Shashtikashalipindasweda* such as *Kshira (milk), Shashtikashali* (type of red rice with 60 days old), and *Balamoola* possess *Santarpana* (nourishing)qualities with *Prithwi* and *Ap Mahabhuta* and is indicated for *Balya, Bruhmana*, and strengthening *Dhatus* and *Vata* pacification. *Ruksha Churna Pinda Sweda* was applied with *Kottamchukkadi Churna*[64], to get *NiraamAwastha*. After seven days next step was *Pitta Samshodhana*.

- **Mastishkya:** *Shirodhara, shiropichu, shiroabhyanga* and *shirobasti* are catagorised under Mastishkya. Among which *Shirodhara* also helps in reduction of anxiety along with marked reduction in disturbed sleep pattern. As *Shirah* is the placement of all *Indriya*, that *Shirobasti*, are essential.

These *karmas* enhance the muscle power and thus help in attaining the maximum balance of body.

Satwavajaya chikitsa

Satva referes to Mind; Avajaya refers to bringing the mind under the control.

The aim of this therapy is to restrain mind from the unwanted thought process, replacing the negative ideas, proper channelling of presumptions and proper advise through *jnanam*(knowledge), *Vijnanam*(analytical thinking), *Dhairya*(courage), *Smrithi*(memory), *Samadhi*(concentration).

Different therapies and counselling can be adopted such as–

Symptoms like Difficulty in walking, stiffness, spasticity, sleep disorders, muscle weakness, depression (or frustration, sadness, and anger) can be addressed with targeted medications, treatments, different therapies and counselling.

- Occupational therapy: This can help the person manage better around the house and at work. It may involve some home adaptations, wheelchair assessment, and making the kitchen more practical.
- Speech therapy: This can help with swallowing, coughing, choking, and speech problems. If speech becomes very difficult, the speech therapist can help the person learn how to use speech aids.
- Orthopedic care: This can help treat curvature of the spine.
- Physical therapy: This can help maintain strength and improve mobility. Physiotherapies like passive exercises, passive assisted exercises and resistive exercises were started when patient was in complete bedridden condition. Later on strengthening exercises for quadriceps, hamstrings, deltoid and biceps muscles along with calf muscle stretching exercises were given. After gaining muscle strength of lower limbs and when patient started to stand with support, co-ordination exercises, knee balancing and ankle balancing exercises started. Studies have shown that physical fitness can positively influence not only outcomes such as mobility and fatigue levels in GBS patients (GBSPs) but also mental functioning. Along with this Electrical nerve stimulation for lower back, upper and lower limb was started which helps in reducing pain.
- Counseling: Sessions can help the person manage frustration and depression that may arise when symptoms affect physical mobility and coordination.

DISCUSSION

Guillain-Barre syndrome (GBS) is an eponym for a heterogeneous group of immune-mediated peripheral neuropathies. A feature common in all GBS variants is a rapidly evolving polyradiculoneuropathy preceded by a triggering event, most often an infection. GBS generally manifests as a symmetric motor paralysis with or without sensory and autonomic disturbances. The patient with GBS typically presents with weakness accompanied by tingling dysesthesias in the extremities. This weakness is prominent in the proximal muscles; legs are more often affected than arms. Paresthesias occur, spreading proximally but seldom extending past the wrists and ankles. Deep tendon reflexes disappear within the first few days of symptom onset.

The progressive phase of the syndrome lasts from a few days to four weeks. About 73 percent of patients reach a nadir of clinical function at one week and 98 percent at four weeks. The progressive phase is followed by a

plateau phase of persistent, unchanging symptoms. Improvement will begin within days of the plateau. The time to resolution of symptoms varies among patients. Cranial nerve involvement may affect airway maintenance, facial muscles, eye movements, and swallowing. Poor outcomes primarily are associated with the increasing severity of disease, with a mortality rate as high as 20 percent occurring primarily in patients who require mechanical ventilation.

Pain, another common feature of GBS, is seen in approximately one half of all patients and is sometimes described as severe, occur- ring with even the slightest of movements. Pain is most severe in the shoulder girdle, back, and posterior thighs. Patients complain of a deep aching pain in the weakened muscles that is similar to the muscular discomfort experienced following exercise. Pain may be accompanied by muscle cramps, and it is most severe at night.

As the natural course of the pathology cannot be altered by the modern medicine, the scope of Ayurveda interventions in such nervous disorders has been increasing because of its holistic approach. Understanding such disorders in Ayurveda perspective is need of the hour. Nervous system and its disorders are described under the heading of *vata vyadhi* in *Ayurveda.* In *Ayurveda, vatavyadhis* are divided into two broad categories namely diseases due to *Vishudha vata(*vitiation of vata alone) *and* diseases due to *Avarana(*due to blockade in the natural flow of vata by its types or by other dosa or dhathu or mala).

Proper knowledge of the types of Proper knowledge of the types of *vata(prana, udana, samana, vyana, apana* their normal functions, categorizing the disorder either as *vishuddha vata janya* or *avarana janya* helps in understanding the diagnosis, prognosis and how to plan the treatment protocol. Ayurveda by seeing etiology and symptoms it *kapha vruta vyanavata, Sarvanga vata, Pranavrutha vyana, Nirupasthamba vatavyadhi, Anukta vata vyadhi.* Due to indulgence in vataja ahara, vihara and abhighata, there is vata and rakta pradushana leading to vata vyadhi.

There is no direct reference of this disease in our classics. But based on symptoms, the Dosha and Dushyas involved can be assessed and accordingly treatment can be provided. In this particular disease, predominance of Vata dosha is very much appreciated. The definition of Vata is "Vaagati gandhanayoh". Where in Gati is interpreted as motor and Gandhana is interpreted as sensory functions of Nervous System by various Ayurvedic scholars. It is also interpreted that Vata is the prime Dosha that governs the Nervous system. Manifestation of Vata vyadhi is of two types, Upastambhita and Nirupastambhita, by analyzing above pathology and symptoms most of which can be compared to *Kaphavruta vyana, Sarvanga vata, Pranavrutha vyana, Nirupasthamba vatavyadhi, Anukta vata vyadhi.*

Based on this the treatment protocol is selected. Mainly in Avarana conditions Avaraka dosha is treated first for example, Kapha dosha which is done by Shamanoushadhis and then treatment for Avruta dosha i.e., Vata dosha, for Vatavyadhi, Brimhana among shad Upakramas is highly indicated. Bastikarma has been doing wonders in the treatments of Vata vyadhi. From the above description it is understood that Brimhana type of Basti along with Shasthtika shali pinda sweda and Brimhana nasya plays major role. The drugs present in Raja Yapana Basti are very cost effective, easily available and without any known side effects. From the above description it is understood that Brimhana chikitsa is the requirement for the management of Gillian Barré syndrome.

CONCLUSION

- For all the activities, movements Balance are all control by vatadosha, its normal function of vata dosha.
- Symptoms are preceded by an antecedent event in about two thirds of patients. Respiratory infections are the commonest, reported in about 40% of cases within one month before the onset of the disease. About 20% experience gastroenteritis as the antecedent cause.
- The onset of symptoms can either be acute or subacute. Gradual recovery takes place after a plateau phase. In a large multicentre study, the mean time to reach nadir, improvement, and clinical recovery were 12, 28, and 200 days, respectively. It was also found that 98% of patients achieved the plateau phase by four weeks from the onset. The mean duration of the plateau was found to be 12 days in another study.
- An autoimmune reaction may damage the myelin sheath around the nerve. This damage is called demyelination. Damage parts of nerves cause tingling, muscle weakness, and paralysis. It causes nerve signals to move slowly. Damage to other parts of the nerve, can cause, the nerves to stop working altogether.
- In classical texts of ayurveda there is no separate description for Guillain Baree sundrome but we get scattered references, this scattered references of anukta vyadhi is collected from samhithas. By seeing their etiology, signs and symptoms ***kapha vruta vyanavata, Sarvanga vata, Pranavrutha vyana, Nirupasthamba vatavyadhi, anukta vyadhi.*** *Hetu* & *Samprapti* plays important role in the treatment of neurological disorders.
- The holistic approach by *Ayurveda Shodhana* and *Shamana Chikitsa* is helpful in the treatment of GBS.
- Significant changes were observed on both subjective and objective parameters after the course

of treatment. Hence, Guillain Barre Syndrome can be effectively managed in Ayurveda with the proper understanding of *Nidana, Samprapti and Dhatu dushti lakshanas.*

- Along with the Ayurvedic *panchakarma Chikitsa* as well as *Shamanoushadhis*, physiotherapy played a major role in improving the muscle tone, muscle strength and reflexes.
- As immunoglobin treatment is a costly alternative, cost effectiveness of the Ayurvedic treatment seems promising.

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