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A CASE STUDY TO EVALUATE "EFFECTIVENESS OF AYURVEDA CHIKITSHA IN CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY"

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ABSTRACT

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an uncommon, debilitating and degenerative neurological disorder characterized by progressive weakness due to peripheral nerve damage, with chronicity of more than 9 weeks. Clinical manifestation is bilateral symmetrical proximal and distal weakness of muscles of extremities, tingling, numbness, fatigue, gait imbalance with loss of tendon reflexes. It is also termed as an autoimmune disorder for which no specific and effective treatment is available, though steroid therapy, immunotherapy, stem cell therapy, plasmapheresis etc. have been tried with variable success rates. Here is a case study that war treated by Modern medicine by steroid and plasmapheresis for than 3 month with not improvement but case was only deteriorated. Such type of case came to Government Ayurvedic College and Hospital in wheel chair. With initiation of ayurvedic treatment, there was marked improvement in his condition, and that patient began to walk. This case study offered a ray of hope to thousands of such poor sufferers of CIPD and demonstrating that Ayurveda has potential and strength to effectively treat this type of progressive neurological disorder.

KEYWORDS: – Chronic inflammatory demyelinating disorder, Autoimmune disease, Shodhana and shamana chikitsha of Ayurveda, Guillain- barre syndrome, Aam vata vyadhi.

INTRODUCTION

Chronic inflammatory demyelinating polyneuropathy (CIDP) is a type of acquired immune-mediated disorder that affects the peripheral nervous system. Although it has diverse clinical presentations, the classical presentation includes symmetric proximal and distal sensory and motor involvement. CIDP can be monophasic, relapsing, or progressive, developing over eight weeks. The time course of 8 weeks and the duration to reach nadir help distinguish CIDP from Guillain-Barre syndrome (GBS) or other acute inflammatory demyelinating polyneuropathies (AIDP).[1] It is rare, progressive neurological disorder that targets the myelin sheath of nerve fibres and nerve roots by causing inflammation. This slows down the ability of nerves to send signals, causing weakness in muscles of extremities, tingling and numbness, fatigue, painful parasthesias, loss of reflexes etc. The weakness in the limbs is usually symmetrical.[2]

The first case was described by Eichhorst Burns in 1890. About 16% of the patients present with acute GBS.[3] CIDP is distinguished from GBS by its chronic course and this neuropathy shares many features with common demyelinating from GBS, including elevated CSF protein levels.[4] Most cases occur in adults, and males are affected slightly more often than female. A recent meta-analysis showed a crude incidence rate of 0.33 per 100,000 patients. Overall prevalence has been reported around 0.8 to 8.9 per 100,000 people and increases with advancing age, with a peak incidence of 40 to 60 years. Due to diverse clinical presentations and diagnostic criteria used worldwide, the incidence and prevalence rates also vary. CIDP predominantly affects males more than females, with a ratio of 2:1.[5]

CIDP is autoimmune disorder involving both T cell-mediated and humoral immune mechanisms by targeting myelin components of the peripheral nervous system. Classical CIDP is idiopathic. However, it has variants associated with a neoplastic process (e.g. osteosclerosis myeloma, Waldenstrom macroglobulinemia, lymphoma, monoclonal gammopathy of undetermined significance), HIV infections, and chronic history of diabetes mellitus type II. History of anteceding diseases have been commonly reported with AIDP/GBS; however, they are rare with CIDP.

Approximately 25% of patients with clinical features of CIDP also have a monoclonal gammopathy of undetermined significance (MGUS). Cases associated with monoclonal IgA or IgG kappa usually respond to treatment as favorably as cases without a monoclonal gammopathy. Patients with IgM monoclonal gammopathy and anti- bodies directed against

myelin-associated glycoprotein (MAG) have a distinct polyneuropathy, tend to have more sensory findings and a more protracted course, and usually have a less satisfactory response to treatment.[6]

There is no specific treatment but glucocorticosteroids are commonly used with encouraging results, but cannot be used for longer period due to its side effects. Plasma Exchange (Plasmapheresis) is tried with encouraging results. Stem cell therapy", and Immunotherapy by Intravenous Immunoglobulins has been tried but with variable results.[7,8]

AIM

To evaluate the effect of Ayurvedic Chikitsa in Chronic Inflammatory Demyelinating polyneuropathy.

OBJECTIVES

1. To gather information about the concept of chronic inflammatory demyelinating polyneuropathy as per modern and ayurvedic perspective.
2. To analysis the effect of ayurvedic chikitsa in CIDP.

Case discription

A 39 years old male patient was reported at Kayachikitsa OPD, Govt. Ayurvedic College And Hospital Guwahati with the symtoms - weakness in muscles of extremities, mainly in lower limbs symmetrical, tingling and numbness, fatigue, painful paresthesia, loss of reflexes, since last 6 to 8 month. Also patient has been suffering from constipation. He was diagnosed as a case of C.I.D.P. NCS/EMG & MRI report - Motor and sensory nerve conduction studies were carried out in both upper and lower extremities. Distal Latency prolonged in all motor nerves and reduction of amplitude of Compound Muscle. Action Potential in lower limbs. F waves were prolonged in upper limb and not recordable in lower limbs. Motor nerve conduction velocities were reduced to 70% of normal. Sensory Nerve Action Potential (SNAP) were absent in lower limbs.

History of present illness

The patient has been suffering from weakness in muscles of extremities, mainly in lower limbs symmetrically, unable to walk, tingling and numbness over limbs, fatigue, painful paresthesia, loss of reflexes and reduced movement, since last 6 to 8 month. Also patient has been suffering from constipation.

Past history

No history of Diabetes, Hypertension.

Family history

None of the family members have CIDP, GBS.

General examination

Local and systemic examination of patient

Bowel - Not clear.

Micturition - Normal

Appetite - Low.

BP-130/80 mmHg

PR-78/min

Urine culture and sensitivity - Shows no growth after 2 days of aerobic incubation at 37°c

Urea- 13 mg/dl

Creatinine-0.7 mg/dl

Sodium – 136 mg/dl

Potassium- 3.7 mEq/L

Chloride- 98 mmol/L

Calcium- 9.2 mg/dl

Phosphorus- 3.5 mg/dl

Astavidha pariksha

Nadi-, Vatajkaphaj	Mala - Malavastambha
Mutra-Samyak	Jivha - saam
Shabda-Prakrit	Sparsha - Samyak
Druk-Prakrit	Aakriti-Madhyam
Kshuda-Kshudamandya	Nidra-Samyak

MATERIALS AND METHODS

Table 1: The treatment plan.

Sl. No.	Type of treatment	Treatment periods	Intervention
1	Panchakarma Procedures (shodhana): Virechan	Deepan-panchana: 5days Sneha Pana: 5days Abhyanga swedana: 2 days	Deepana pachana with Avipattikar choorna 1 tsf BD before meal and Chitrakadi vati 2 vati BD after meal

			Sneha pana , 1st day - 30 ml 2nd day - 60 ml 3rd day - 90 ml 4th day - 120 ml 5th day - 150 ml Then 2 day Abhyanga swedan with mahanarayana taila Then 1 day gap and next day virechana with Trivrit Avaleham - 30 gm,Eranda taila -20 ml,Triphala kwath-20 ml
2	Systemic treatment	3 month, continue upto follow up	Tab Brihatvat chintamoni Ras 1 od/Yogendra ras 1 od Yograj gugglu 1pilL+khanjankari ras 1 pill + maharasnadi Kwath 20 ml, 1 dose twice daily after meal. Ashwaganda aveleha 1tsf twice daily (when patient had fever above medicine stopped) Avipattikar choorna 1tsf BD before food. Bala arista 2 tsf BD after meal for 20 days.
3	Panchakarma procedure: Snehan, Swedan; Sarvang (mainly lower limb) swastic shali Pind-sweda; followed by kalabasti [Niruha and Anuvasan (Matra basti) A/M)] per rectally	Swastic shali pinda swedan 7days Kalabasti 16 days Snehan swedan 16 days	Snehan-Ksheerabala taila/Narayan tail Followed by Pindsweda Rice Udad dal Black sesame seeds cooked in kuath made up Dashamool, Niruh: Dashmool+ Erandmool + Saidhav salt 3 gm+ Madhu (Honey) 5 ml Narayan tail 20 ml -Total quantity 400 ml. Anuvasan/Matra Basti ksheerabala tail 40ml and Narayan taila 40 mlA/D
4 5	Nasya Majja basti	Nasya 7 day (Except patient had fever) Majja basti 7 day	Nasya with ksheerabala taila A cycle of 3 days starting Firast day Niruh,second day Majjabasti & Third day Matra basti (Dashmool tail/) 40 ml in this order for 7 cycles.
6	Kati basti /	7days	With sahacharadi +

	merudanda basti		mahanarayan taila
7	Physiotherapy		Active Range of Motion for upper limb; Hip and knee flexion exercise; Ankle-toes movements Hip abduction, hip extension Walker support; Thoracic expansion exercises; UE strengthening exercises.

Pathya and Apathya

The patient was advised to take easily digestible food and refrain from non-vegetarian food products, milk products like *Abhishyandh*i with *viruddha aahar*.

Pathya	**Apathya**
Sunthi, ahraka, ajwain, souf, marich, hingu, Saindhava, jiraka, lahsun, yava, takra, eranda taila, ushnadaka etc.	Dadhi. Fish, guda, milk, anupa mamsa, asatmya bhojana, vegadharana, ratri jagarana, alasya, cinta shoka, etc.

RESULTS

Table 2: Symptom-wise outcomes.

Symptoms Before Treatments	**After 1 month**	**After 45 days**	**After 60 days**
Loss of reflexes of lower limbs	++	+	-
Weakness of muscles of extremities (Specially lower)	+++	++	+
Tinglings and Numbness of extremities	+++	+	-
Pain paresthesia	+++	++	+
Unable to walk	Unable	Able with support	Able to walk

1st Day Visit in KC OPD, GACH(Can't Walk) Support)

After 45 Days of Treatment in GACH (Walking with Support)

After 60 Days of Treatment in GACH(Walking without

DISCUSSION

Describing chronic inflammatory demyelinating polyneuropathy (CIDP) and its connection to autoimmune disorders. C.I.D.P. Auto- immune diseases like Multiple sclerosis, Guillain Barre Syndrome, Myasthenia gravis, C.I.D.P. etc are known in Ayurved as Aam -Vat. Free CIDP indeed involves damage to the myelin sheath, leading to nerve dysfunction. Ayurveda often associates such conditions with the concept of "Aam-Vat," which relates to toxins and imbalances in the body. Free radicals and Reactive oxygen Species (ROS) play a role in cellular damage, potentially exacerbating conditions like CIDP. Managing oxidative stress and inflammation is crucial disorders holistical. In the patient case, triggering factor which vitiated the *Kapha* and *Vata* due to its *sheeta* and *Drava* guna.

Vayu is called as Tantra-Yantra dhar. It has complete control over all physiological activities & it takes part in to all kriyas in the body. Any derangement in function of any organ is due to vitiated Vayu/Vat (Cha. Su. 12/8). This disorder occurs due to *Kaphavrutta Vata*. Vat vriddhi in Kapha sthan occurs due to Ati-sevan of Rukshadi aahar, Adhyashan, Mal-Mutradi Vegavrodh, Ati- shram, Anil sevan, living in sheet/ humid climate leads to vitiated vat which combines with Kapha, causing the pathogenesis of this disorder.

Samprapti of Strotorodh pradhan vatvyadhi

Hetusevan

↓

Kaphaprakop/ *Aamdoshotpatti* (Sheet guna sevan)

↓

Strotorodh

↓

Vimarg gaman of vata

↓

Sthansanshray of Samvayu at Strotovaigunya

↓

Vatavyadhi-strotorodh pradhan

Strotorodh Balbhransh Gaurav Anilmudhata I

Alasya apakti nisthiv Malsangh Aruchi Klam: II

Vag. Su 13/23-24

Strotorodh, or obstruction of the channels (Strotas), can indeed disrupt various physiological processes in the body. When there's obstruction in the Mal mutradi and other micro and macro channels, it affects circulation, function, and nutrient transport along the digestive, urinary, circulatory, lymphatic, and nervous systems, as well as others. This disruption can have adverse effects on the formation of poshya dhatu (Nutrient tissues).

The mode of action of the prescribed drugs and panchakarma therapies can be understood on the basis of inherent properties of drugs and therapies.

- This disorder is due to Aam related pathogenesis, therefore it is crucial to treat Aam at Jathragni level and at Dhatvagni/ tissue level, following which, we have to put forward disease specific therapy and Apunarvabhav chikitsa like deepa pachan and virechana therapy.
- Granthakaras have remarked that Basti chikitsa is the half chikitsa in Vata disorders. Further in Marmaghat also Basti Chikitsa is very effective. In this case Prakop of Vata occurred due to strotorodh, therefore we decided to remove strotorodh first and then administered Anuloman chikitsa. Treament aimed Vat chikitsa, to treat vitiated Vata by Snehan-Swedan, Merudand basti/kati basti followed by Yog basti (Niruh and Anuvasan-Matra basti). As the disease pathology is related to nervous system and Charakacharya remarked that *Sneho anilam hanti mrudu karoti deham, Malanam vinihanti Sangam.*[9]
- Ashwagandha (Withania somniphera), Bala (Cida cordifolia), Dashamulkwath and tab Brihatvat chintamani and yogendra ras ,maharasnadi kwath, yograj guggulu were aimed as disease specific and Balyaa-Rasayan-Apunarbhav chikitsa of Vata (Majjavahstrotas) that helped to improve the cellular metabolism. Thus Ayurvedic treatment restored the imbalance of Vata and improved microcirculation and carried out repair of myelin sheath, too.
- Vagbhat mentioned – ‘Nasa hi shirso dwaram' and Nasya nourishes Uttamang (Vital organs), increases Indriya bala and Manobala and reduces Sanchit doshas; we performed Nasya karma as it digests. Kapha and directing Aam directing the Vatanuloman in proper way.

CONCLUSION

The case study highlights the efficacy of Ayurveda in treating autoimmune disorders like C.I.D.P., showing not only a halt in disease progression but also a reversal of its pathology. This suggests that Ayurvedic therapy could be considered as an alternative or complementary

approach to modern treatments relying heavily on steroids, immunosuppressants, and plasmapheresis.

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