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Case Study

PANCHAKARMA INTERVENTION IN HERIDITARY SPASTIC PARAPLEGIA

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ABSTRACT

Hereditary Spastic Paraplegia (HSP) is a large group of inherited neurological disorders, in which the prominent feature is a progressive spastic paraparesis. it can be Multiple genetic mutations with various inheritance patterns (autosomal dominant, autosomal recessive, X-linked recessive). Direct correlation of Hereditary Spastic Paraplegia (HSP) is not found in Ayurvedic classics. It can be studied within the broad spectrum of *Vatavyadhi*. In the present cases, based on the symptoms observed in patients, it can be considered under the category of *Pangu*. **Objectives:** By considering *Pangu* is manifested from vitiated *Vata dosha* in combination with other *Dosha* and presents with symptoms of reduced strength in bilateral lower limb and abnormal gait. An attempt has been made to evaluate the efficacy of *Panchakarma* procedures in the conservative management of the disease. **Method:** Two cases of HSP presented with symptoms of difficulty in walking, weakness in bilateral lower limb along with stiffness managed with *Dashamoola Kashaya seka, Shastikashalipinda sweda, Balavarnakara basti* in *Kalabasti* pattern. **Results:** The patient was assessed based on sign and symptoms of HSP. **Conclusion:** *Panchakarma* treatment having encouraging results in improving quality of life of patients.

INTRODUCTION

In today's competitive world, independence and self-reliance are highly valued, both personally and professionally. HSP primarily affects the lower limbs, leading to difficulties with walking, balance, and coordination. Over time, this can result in increased dependence on mobility aids or assistance from others for daily activities. Hereditary Spastic Paraplegia is a genetic condition marked by increasing stiffness and weakness in leg muscles, typically inherited within families, with some cases appearing in adulthood. HSP is usually transmitted as an autosomal trait; most adult-onset cases are dominantly inherited. There are more than 80 genetic types of HSP in which causative mutations in more than 60 genes have been identified. Symptoms usually begin in the third or fourth decade of life, presenting as progressive spastic weakness beginning in the lower extremities. In advanced stages of the condition there may be urinary urgency and incontinence[1].

In pure forms of HSP, the spastic leg weakness is often accompanied by posterior column (vibration and position) abnormalities and disturbance of bowel and bladder function. Some family members may have spasticity without clinical symptoms. Neuro-pathologically, in HSP there is degeneration of the cortico-spinal tracts, which appear nearly normal in the brainstem but show Increasing atrophy at more caudal levels in the spinal cord; in effect, this pathologic picture is of a dying-back or distal axonopathy of long neuronal fibers within the CNS. Defects as numerous loci underlie both dominantly and recessively inherited forms of HSP.

The gene most commonly affected is Spastin (SPAST Gene) and Atlastin (ATL1 Gene). Spastin gene helps in microtubule severing and remodeling, Disrupted microtubule dynamics and axonal transport lead to neuronal degeneration, particularly affecting motor neurons in the corticospinal tract. Atlastin gene helps in Endoplasmic Reticulum membrane fusion and maintenance and Disrupted ER function leads to cellular stress, protein misfolding, and neuronal

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dysfunction. The most common childhood-onset dominant arises from mutations in the atlastin gene.

1. X-Linked Recessive HSP caused by PLP1 gene (Proteolipid Protein 1) and Myelin Proteolipid Protein (PLP) is a major component of the myelin sheath in the central nervous system. PLP1 is crucial for the formation and maintenance of myelin, the insulating layer around nerve fibers that facilitates efficient signal transmission. Defects in the PLP1 gene lead to impaired myelin formation resulting in a variety of neurological symptoms. This form of HSP typically presents in infancy or early childhood.
2. Recessive HSP Caused by Paraplegin Defects SPG7 Gene and Paraplegin protein with homology to metalloproteases. Paraplegin helps in the proper functioning of mitochondria, which are critical for energy production and cellular health. Defects in SPG7 lead to mitochondrial dysfunction, which impairs neuronal health and function.
3. Adrenomyeloneuropathy caused by ABCD1 gene and Adrenoleukodystrophy protein (ALDP) which facilitates the transport of VLCFAs into peroxisomes, where they are degraded. Mutations in the ABCD1 gene lead to a failure in VLCFA transport, resulting in their accumulation. This accumulation causes damage to the myelin sheath and affects both the central and peripheral nervous systems. Typically adult-onset, but the disease can also present in childhood.[1]

Direct correlation of Hereditary Spastic Paraplegia (HSP) is not found in Ayurvedic classics. It can be studied within the broad spectrum of *Vatavyadhi*. In the present cases, based on the symptoms observed in patients, it can be considered under the category of *Pangu*[2] the symptoms includes reduced strength in bilateral lower limb and abnormal gait.

CASE REPORT

Case 1

A 40 years old female patient, presented with complaint of difficulty in walking and weakness in bilateral lower limb since 4 years, associated with stiffness in bilateral lower limb.

History of present illness

A female patient n/k/c/o Diabetes Mellitus and Hypertension, k/c/o Hypothyroidism was asymptomatic 4 years ago. Gradually she developed weakness in bilateral lower limb associated with stiffness and imbalance while walking. But she neglected the condition and continued her daily activities. Later she developed difficulty in walking. The condition progressed and since 1 year and she developed dependency for her routine works. She completely depended on cane for her walking. She consulted many physicians for these complaints but did not get any relief. Seeking better management, she visited OPD of Panchakarma, SJGAUH, Bengaluru, where she was admitted for further care.

History of past illness:

Patient is a known case of Hypothyroidism since 20 years and on medication (Tab. Thyronorm 100mcg 1-0-0 B/F)

Family history: Nothing significant

CASE 2

A male patient aged 18 years, presented with complaints of progressive weakness and stiffness in bilateral lower limb since childhood.

History of present illness

A male patient, not known case of diabetes mellitus and hypertension, noticed gradual weakness and stiffness in his bilateral lower limbs over the past 6 years. He was delivered by normal vaginal delivery with no antenatal or post-natal complications. His developmental milestones were normal until 18 months of age. Gradually, he began experiencing frequent falls while walking. Initially, his parents ignored these issues. Later, they noticed a change in his gait, and the child started complaining of weakness and stiffness in his lower limbs. He was unable to walk for more than 2 minutes and developed pain in back of knee on prolonged walking. Despite visiting many physicians and receiving treatment, his symptoms did not improve. Seeking better management, he was brought to the OPD of Panchakarma at SJGAUH, Bengaluru, where he was admitted for further care.

Family history

Patient's mother has similar complaints.

Table 1: Showing Personal History of both patients

Case 1	Case 2
Name- XYZ	Name- XYZ
Age- 40 years	Age- 18 years
Marital status- Married	Marital status- Unmarried
Occupation- Housewife	Occupation- Student
Diet- Mixed	Diet- Mixed
Appetite - Good	Appetite -Good

Bowel - Regular	Bowel - Regular
Micturation - 4-5/day	Micturation – 5-6/day
Sleep - Disturbed	Sleep - Sound

Table 2: Showing General examination

Case 1		Case 2	
Pallor	Absent	Pallor	Absent
Icterus	Absent	Icterus	Absent
Clubbing	Absent	Clubbing	Absent
Lymphadenopathy	Absent	Lymphadenopathy	Absent
Cyanosis	Absent	Cyanosis	Absent

Table 3: Showing *Asthasthana pareeksha*

	Case 1	**Case 2**
Nadi	*Vatapittaja*	*Vtapittaja*
Mala	*Abaddha*	*Abaddha*
Mutra	*Prakruta*	*Prakruta*
Jihwa	*Alipta*	*Alipta*
Shabda	*Prakruta*	*Prakruta*
Sparsha	*Anushna sheeta*	*Anushna sheeta*
Drik	*Prakruta*	*Prakruta*
Akruti	*Vikruta*	*Vikruta*

Table 4: Showing *Dashavidha pareeksha*

	Case 1	**Case 2**
Prakriti	*Vatakapha*	*Kaphavata*
Vikriti	*Kaphavata*	*Kaphavata*
Sara	*Madhyama*	*Madhyama*
Samhanana	*Madhyama*	*Madhyama*
Satmya	*Katupradhana sarvarasa*	*Katupradhana sarvarasa*
Satva	*Madhyama*	*Madhyama*
Aharashakti	*Madhyama*	*Madhyama*
Vyayamashakti	*Avara*	*Madhyama*
Vaya	*Madhyama*	*Baala*
Pramana	*Madhyama*	*Madhyama*

Table 5: Showing Samprapti ghataka

	Case 1	**Case 2**
Dosha	*Tridosha* with *Vata pradhana*	*Tridosha* with *Vata pradhana*
Dushya	*Rasa, Rakta, Mamsa, Asthi, Snayu*	*Rasa, Rakta, Mamsa, Asthi, Snayu*
Srotas	*Rasavaha, Raktavaha, Mamsavaha, Asthivaha Majjavaha*	*Rasavaha, Raktavaha Mamsavaha, Asthivaha Majjavaha*
Srotodusti	*Sanga*	*Sanga*
Udbhava sthana	*Pakwashaya*	*Pakwashaya*
Sanchara sthana	*Sarvashareera*	*Sarvashareera*
Vyaktha sthana	*Adhoshakha*	*Adhoshakha*
Rogamarga	*Madhyama*	*Madhyama*
Sadhyasadhyata	*Yapya*	*Yapya*
Swabhava	*Chirakari*	*Chirakari*

Table 6: Showing Central nervous system examination

		Case 1			Case 2	
Higher mental function		Consciousness -Fully conscious, Orientation - To time, place, person intact Memory - Immediate, recent, remote intact Hallucination and delusion -Absent Speech disturbance -Absent Handedness- Right			Consciousness -Fully conscious, Orientation - To time, place, person intact Memory - Immediate, recent, remote intact Hallucination and delusion -Absent Speech disturbance - Present (slurred speech) Handedness- Right	
Cranial nerves		Within normal limits			Within normal limits	
Sensory system		No abnormality detected			No abnormality detected	
Gait		Spastic Gait			Spastic Gait	
Coordination		Romberg test - Positive Finger nose test- Negative Tandom gait- Positive			Romberg test - Negative Finger nose test- Negative Tandom gait- Positive	
Involuntary movement		Absent			Absent	
Walking time		6 steps in 10 seconds (with support)			9 steps in 10 seconds (without support)	
Motor system	**Tone**	1.	**Right**	**Left**	**Right**	**Left**
		U/L	Normal	Normal	Normal	Normal
		L/L	Spastic	Spastic	Spastic	Spastic
	Bulk	2.	**Right**	**Left**	**Right**	**Left**
		Biceps	20 cm	20 cm	22 cm	22 cm
		Forearm	17 cm	17 cm	19 cm	19 cm
		Mid-thigh	47cm	49 cm	48 cm	46 cm
		Calf	24.5cm	26 cm	26.5 cm	25 cm
	Power	3.	**Right**	**Left**	**Right**	**Left**
		U/L	5/5	5/5	5/5	5/5
		L/L	4/5	4/5	4/5	4/5
	Reflex	4.	**Right**	**Left**	**Right**	**Left**
		Biceps	++	++	++	++
		Triceps	++	++	++	++
		Knee	+++	+++	+++	+++
		Ankle	+++	+++	+++	+++
		Babinski	Extensor	Extensor	Extensor	Extensor
		Clonus	Present	Present	Absent	Absent
	Deformity		-	-	-	Pes planus

Table 7: Showing *Nidana panchaka*

	Case 1	Case 2
Nidana	*Aharaja- Ruksha, Tikshna, Amla, Katu rasa pradhana sevana.* *Manasika- Atichinta, Krodha*	*Beeja dusti* *Aharaja- Ruksha, Tikshna, Amla, Katu rasa pradhana sevana.*
Purvaroopa	*Avyakta*	*Avyakta*
Roopa	Weakness in bilateral lowerlimb associated with stiffness, difficulty in walking	Weakness in bilateral lower limb associated with stiffness, pain in back of knee on prolonged walking.
Upashaya-Anupashaya	Nothing specific	Nothing specific

Table 8: Showing investigations

	Case 1	Case 2
Thyroid profile	T 3-1.4 ng/ml T4- 11.6 (µg/dL) TSH- 2.9 u IU/ml	-

Table 9: Showing treatment protocol adopted in Case 1

Treatment		Duration	Observation
Sarvanga abhyanga with *Narayana taila* f/b *Dashamoola kashaya seka*		7days	Reduction in stiffness by 20%
Shastikashalipinda sweda		7 days	Stiffness in lower limb reduced by 40%
Vestana to B/L lower limbs with *Mahamasha taila*		14 days	Reduction in stiffness by 60% and improvement in strength of lower limb by 50%
Balavarnakara basti in *Kala basti* pattern		6 days	Gait improved, patient was able to walk without support.
Anuvasana basti with *Guggulutiktaka ghrita* 60 ml		10 days	
Balavarnakara basti *Madhu*- 60ml *Saindavalavana*- 12gms *Guggulutiktakaghrita*- 80ml *Shatapuspha churna*- 12gms *Baladi Kashaya*- 300ml	*Anuvasana basti* with *Guggulutiktaka ghrita*- 60ml *Shatapuspha churna*- 6gms *Saindava lavana*- 6gms		

Table 10: Showing treatment protocol adopted in Case 2

Treatment	Duration	Observation
Sarvanga abhyanga with *Maha naarayana taila* f/b *Dashamoola kashaya seka*	7days	Stiffness in lower limb reduced by 20%
Shastikashalipindasweda	14 days	Stiffness in lower limb reduces by 50%
Matrabasti with *Guggulutiktaka ghrita* 60 ml	10 days	Gait improved, patient could walk to longer distance

Assessment of symptoms

Case 1

		Before treatment	After treatment
1	**Walking distance without pause**	6 steps in 10 seconds (with support)	14 steps in 10 seconds (without support)

			Before treatment		After treatment	
			Right	**Left**	**Right**	**Left**
2.	**Bulk**	**Biceps**	20 cm	20 cm	22 cm	22 cm
		Forearm	17 cm	17 cm	18 cm	18 cm
		Mid-thigh	47cm	49 cm	48 cm	50 cm
		Calf	24.5cm	26 cm	25.5 cm	27 cm

Case 2

		Before treatment	After treatment
1	**Walking distance without pause**	9 steps in 10 seconds (without support)	18 steps in 10 seconds (without support)

			Before treatment		After treatment	
			Right	Left	Right	Left
2.	Bulk	Biceps	22 cm	22 cm	22 cm	22 cm
		Forearm	19 cm	19 cm	19 cm	19 cm
		Midthigh	48 cm	46 cm	49 cm	47 cm
		Calf	26.5 cm	25 cm	27 cm	26 cm

DISCUSSION

Discussion on Intervention

Here we are describing these reports, the mother and son presented with similar clinical feature. Both of them presented with progressive gait disturbance, spasticity of lower limbs, hyper-reflexia and extensor planter responses. Due to cost perspective, genetic study and needed investigation could not be performed in this case. Generally, diagnosis of HSP is confirmed through genetic testing to identify specific gene mutations (e.g., *spastin* or *alastin*). There is no specific treatment to prevent, slow or reverse HSP, treatment of HSP mainly consists regular physical therapy to maintain or improve mobility, strengthen muscles, and enhance coordination and balance, occupational therapy which focus on activities of daily living (ADLs) and adaptive techniques to improve functional independence and quality of life. Direct correlation of HSP cannot be found in Ayurvedic classics; however, it can be considered under the broad spectrum of *Vata vyadhi*. Based on the symptoms, it can be correlated with *Pangu*, primarily involvement of *Beejadosha* along with an imbalance of *Vata, Kapha* and *Pitta*. Treatment was planned accordingly.

Sarvanga Abhyanga

Acharya Dalhana has explained in detail about the absorption of *Sneha* used in *Abhyanga* procedure[3]. The oil used in *Abhyanga* reaches up to the different *Dhatu* if it is performed for the sufficient time. Hence, the drug used in *Abhyanga* gets absorbed by the skin. As *Mahanarayana taila* contains *Bilwa, Ashwagandha, Bruhati, Shyonak, Swadamstra* which posses *Vatakaphahara* property.

Dashamoola kashaya seka

Kayaseka with *Dashamoola Kashaya* was done to relieve the *Stambha* caused by *Kapha-Vata Dosha*. It helps in *Deepana Pachana* of *Ama dosha* present in different *Dhatu*. *Kayaseka* is a *Snigdha ruksha sweda* in which the *Dashamoola kashaya* is poured all over the body. Acharya Sushruta explains that out of the four *Tiryak Dhamanis*, each divides gradually into hundred and thousand times and thus become innumerable. These cover the body like network and their openings are attached to the *Romakupa*. Through these *Veerya* of *Abhyanga* and *Kayaseka* enter into the body after undergoing *Paka* with *Bhrajaka Pitta* in skin. Acharya Vagbhata explains the *Bhrajaka Pitta* is responsible for the *Pachana* of drugs used in *Abhyanga* and *Kayaseka*. The vasodilation due to *Swedana* improves the circulation to the *Twak, Peshi, Snayu, Kandara*. This procedure removes the *Srotavarodha* at *Sthansamshraya* i.e., at the affected *Sira snayu kandaras* there by it improves the movement of *Vyana vata* and improves the motor activity of limbs.

Shastikashalipindasweda

It comes under *Sankara sweda*[4] it has *Brihmana* and has *Snigdha, Guru, Sheeta, Sthiraguna* and is *Tridoshaghna*. It is performed by bolus of boiled *Shashtika Shali* with *Balamoolasidda ksheera* which makes the skin more permeable by opening the skin appendage through sweating, and dilates the blood vessel, all these things helps in absorption of medicine. Most of the medicines are not permeable through the skin but amphipathic nature of the *Ksheera* helps in absorption of other medicine. *Shastika shali* has higher protein content about 16.5%. Protein is made up of amino acids, which act like building blocks for the body. It gives muscles the amino acids necessary to repair and rebuild. A it is also rich in Vitamin B12, it may also help in myelin sheath regeneration So, *Shastikashali Pinda Sweda* is one of the most preferred method of *Swedana*.

Taila Vestana

Vestana is one among *Chikitsa* mentioned in *Vatasya upakrama*[5]. The disease is characterised with *Mamsa shosha*, which is produced predominantly due to *Ruksha guna* of *Vata*[6]. *Mahamasha taila* is indicated for *Vatavyadhi*[7] and have *Brimhana* property. *Vestana* with *Mahamasha taila* imparts *Snehana*, *Swedana* and *Brimhana karma*. Thus it combats with *Rooksha guna* of *vata* and nourishes *Mamsa*.

Balavarnakara basti[8]

In *Vatavyadhichikitsa*, *Basti* has significant importance as it possesses various actions and hence considered as *Ardhachikitsa*[9]. When administered, *Basti* reaches the *Pakwashaya*, the main seat of *Vatadosha*. And from its *Veerya* spreads throughout the entire body. The gastrointestinal tract contains a network of fibers known as the enteric nervous system (ENS) which are in connection with CNS through Gut-Brain Axis. *Balavarnakara basti* consists of *Baladi Kashaya* which has *Rasayana* and *Brimhana* property

and is act as nervine tonic. *Matra basti* with *Guggulutiktaka ghrita* is done in this present case, as it contains drugs like *Nimba, Amruta, Patola, Manjista, Ativisha, Guggulu*, and other *Tikta rasa dravyas,* it is *Prabala samiraanahara*. It is effective in *Dhatukshaya* and acts by reaching the *Asthi* and *Majja dhatus*[10]. Since the dosage of *Sneha* used in *Matrabasti* is low, it stays in the colon for maximum period and will induce all beneficial results.

Discussion on Observation

A moderate improvement in overall symptoms, such as difficulty in walking, stiffness in the bilateral lower limbs, was observed in these cases treated with *Sarvanga Abhyanga* and *Dashamoola Kashaya Seka* helped to reduce stiffness and pain in the bilateral lower limbs. *Balavarnakara Basti* acts as a *Brumhana* and *Rasayana* therapy. *Anuvasana Basti* with *Guggulutiktaka Ghrita* and *Shastikashalipinda Sweda* also function as *Brumhana* by assisting in the repair and rebuilding of muscles. The combined effects of these treatments contribute to reducing the symptoms, as detailed in Tables 9 and 10.

CONCLUSION

Hereditary Spastic Paraplegia (HSP) is a group of genetic disorders that can be caused by mutations in various genes, including spastin and alastin. It is correlated with *Vatavyadhi* in Ayurveda and is challenging to manage. Treatment varies from patient to patient and is tailored based on the individual's presentation. It is planned according to the involvement of *Doshas, Vyadhi avastha, Roga,* and *Rogi bala.* In the first case, the patient experienced significant improvement in her symptoms, enabling her to walk and perform routine activities without support. In the second case, the patient also showed notable improvement in gait, with a moderate reduction in complaints of weakness and stiffness in the lower limbs. Planning a treatment strategy for HSP typically involves a multidisciplinary approach, focusing on managing symptoms, improving quality of life, and addressing specific needs based on the underlying genetic mutation.

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