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Case Study

OSMOTIC DEMYELINATION SYNDROME AFTER CORRECTION OF HYPONATREMIA

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ABSTRACT

Osmotic demyelination syndrome is a rare neurological disease resulting in cerebral apoptosis and loss of myelin due to osmotic stress. Based on anatomical localization and pathological attributes, ODS can be categorized as Central Pontine Myelinolysis (CPM) and Extrapontine Myelinolysis (EPM). It has a biphasic course, with first phase reflecting electrolyte imbalance and the second with pontine dysfunction, impaired vigilance and movement disorders. In an autopsy-based study, a prevalence rate of 0.25–0.5% was seen in the general population and 10% in patients undergoing liver transplantation. Being a rare disease with variable, but preventable outcome, the present case was managed with Ayurvedic oral medication and *Panchakarma* therapy with an aim to improve the quality of life. In Ayurvedic perspective, *Vata kshaya lakshanas* occur during the phase of hyponatremia and further correction of this leads to disruption of osmolality (*Kapha pitta* accumulation) resulting in *Avarana* of *Vata*. This may be the reason for demyelination and localisation of *Vata* in *Sira* and *Snayu* leading to *Sira snayu shoshana* on both halves of the body resulting in *Sarvangaroga* (full body afflicted with *Vata*). Thus *Avaranahara*, *Srotosodhana* and *Vatanulomana* principles were adopted. An improvement in Barthel index and modified rankin scale were observed after a course of treatment for 30 days.

INTRODUCTION

Osmotic demyelination syndrome (ODS) which encompasses Central Pontine Myelinolysis (CPM) and Extrapontine Myelinolysis (EPM), is a severe neurologic disorder characterised by demyelinative lesions in the brain[1]. Occurrence of these lesions in pons results in central pontine myelinolysis and that outside the pons in extrapontine myelinolysis[2]. It is an osmotic brain injury in which rapid correction of hyponatremia triggers apoptosis in astrocytes followed by loss of communication between astrocytes and oligodendrocytes that causes secondary inflammation and finally leads to demyelination[2]. Central Pontine myelinolysis, mostly affecting alcoholics and malnourished typically presents with severe electrolyte disturbances that improves within 48-72 hours as normonatremia is restored[3].

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Following this, deterioration during secondary phase include dysarthria, dysphagia and flaccid quadriparesis that later becomes spastic[3]. Extrapontine myelinolysis shares the same pathology and time course as that of central pontine myelinolysis but differs in clinical manifestation such that behavioural changes and movement disorders are also involved[3]. Though, the exact incidence of ODS is not known, an autopsy-based study documented a prevalence rate of 0.25–0.5% in the general population and 10% in patients undergoing liver transplantation[4]. It affects men more often than women and is most common in middle aged patients[5]. A suspected case of ODS is confirmed by MRI demonstration of demyelination sites, typically localised in pons, cerebellum, lateral geniculate body, thalamus and external capsule[6]. An individual prognosis is difficult, as neither clinical features nor extend of radiological changes are predictive[6]. Thus, the outcome may be death, disability, or recovery to a virtually normal level of function[6]. Extensive and prolonged neuro-rehabilitation is the only treatment possible in those who survive the disease[7].

Patient Information

A 44-year-old hypertensive male patient with the complaints of weakness of bilateral upper limb and lower limb associated with difficulty in speech for the past 10 months visited Kayachikitsa OPD of Government Ayurveda College, Thiruvananthapuram, on 2/6/2022. He was diagnosed with Osmotic Demyelination Syndrome from NS Memorial Hospital on 20/8/2021. In addition, his USG Abdomen was suggestive of chronic liver disease with portal hypertension. Then he was hospitalized for 3 and 1/2 months and was on conservative management and physiotherapy. Patient being aware of irreversible nature of the disease took admission at our hospital aiming improvement in quality of life.

Timeline

The patient, a teacher by profession and an alcoholic for the past 6 years was apparently normal till 1 year back. Then he had an acute episode of epistaxis and was diagnosed with systemic hypertension. He was advised antihypertensive (cilapress 10mg) since then. 2 months later he developed muscle cramps over b/l calves associated with pedal edema. Within a week he developed excessive fatigue followed by recurrent episodes of vomiting. On consultation, blood investigation revealed hyponatremia (serum sodium- 103mEq/L) and IV correction was done. Course in the hospital was uneventful. 2 days after discharge, he developed an episode of seizure followed by mild weakness of b/l lower limb and slurred speech. On reaching hospital, weakness progressed, and he had a fall with loss of consciousness. He was completely paralysed and admitted in ICU for 3 days. Further investigation revealed Osmotic Demyelination Syndrome and Chronic Liver Disease with Portal Hypertension and was managed conservatively. Physiotherapy was initiated after 3 days, and liquid diet was advised. After 1 month started solid food but had nasal regurgitation often. Within next 3 months, he started to walk with support, but speech was spastic. At the time of admission here, he presented with weakness of b/l upper limb and lower limb but was able to walk without support. Generalised muscle spasm associated with rigidity and difficulty in speech was also noticed. The patient also had sleep disturbance and anxiety.

Clinical Findings

General Examination: The patient was conscious, well oriented, with no pallor, icterus, cyanosis, clubbing, lymphadenoapathy and edema. Gait was spastic with right knee hyper extended, left knee semiflexed, b/l upper limb abducted, semiflexed and clenched at fist.

Vital signs

BP: 140/100mmhg, left arm sitting

PR: 74/min, regular, full volume

HR: 74/min, no added sounds

RR: 22/min

Central nervous system examination

- Higher mental function– Speech (spastic dysarthria), emotional state (desperate).
- Cranial nerve examination- Table 1 shows cranial nerve examination findings.

Table 1: Cranial Nerve Examination

Cranial nerves involved	Findings
Cranial nerve III, IV, VI	Increased palpebral fissure, dilated pupil, slow saccades and pursuits, diminished pupillary light reflex
Cranial nerve V	Side to side movement of jaw difficult
Cranial nerve VII	Buccinator, orbicularis oris and platysma weak bilaterally
Cranial nerve XI	Trapezius and sternocleidomastoid weak bilaterally
Cranial nerve XII	Lateral movements of tongue impaired

Motor system- Table 2 shows motor system examination findings

Table 2: Motor System Examination

Bulk	b/l UL and LL symmetrical
Tone	B/l UL and LL spastic -grade 2
Power	4- over b/l shoulder, elbow, hip, knee 2 over b/l wrist, fingers, ankle and toes
Reflex	Biceps, triceps, supinator and ankle jerk- 2+ Knee jerk- 3+
coordination	could not be elicited due to weakness
Involuntary movements	No tremor/ dystonia/ chorea

Sensory system- intact

Investigations

Haematological: All blood parameters were within normal limit

Imaging Techniques

- MRI Brain (20/8/2021)- Bilateral symmetrical FLAIR and T_2W_1 hyperintensities with minimal diffusion restriction in bilateral precentral gyrus, caudate nucleus and lentiform nucleus suggestive of Osmotic Demyelination Syndrome
- USG Abdomen and Pelvis (20/8/2021)- Liver normal in size with multiple fine nodularity of parenchyma suggestive of CLD with PHT

Ayurvedic Clinical Assessment

Ayurvedic clinical assessment (*Dashavidha pareeksha*) is tabulated in table 3.

Table 3: *Dashavidha Pareeksha*

Prakriti	*Kapha vata*
Vikriti	*Dosha- Vata kapha pradhana tridoshaja* *Dushya- Dhatu: Rasa rakta mamsa meda majja* *Upadhatu: Sira snayu* *Srotas- Rasa rakta mamsa meda majja* *Sroto dushti- Sangam, vimargagamanam*
Saram	*Madhyamam*
Samhananam	*Madhyamam*
Pramanam	*Madhyamam*
Satmyam	*Madhyamam*
Satwam	*Madhyamam*
Aharashakthi	*Abhyavaharana shakthi- Madhyamam* *Jaranashakthi- Madhyamam*
Vyayama shakthi	*Avaram*
Vaya	*Madhyamam*

Diagnostic Focus

The disease having a biphasic course, of which rapid correction of hyponatremia resulting in disruption of blood brain barrier due to altered intracellular and extracellular osmolality constituted the initial phase. This could be understood as increased permeability as a result of *Lavana atyupayoga* due to its *Stambha sanghata bandha Vidmapana* action (removing rigidity and clearing the obstruction of channels and pores). This further initiated demyelination (*Vatavahi sira vishoshana*) and damage to oligodendrocytes due to cytokines and inflammatory markers that entered CNS and got localised at pons. Clinically, this initial phase showed mixed features of *Pittavritha* and *Kaphavritha vata* (*Kapha pitta vridhi* due to *Lavana atiyoga* causing *Avarana* of *Vata,* disrupting normal *Vata gati*) corresponding to symptoms such as loss of consciousness (*Murcha*), loss of strength (*Bala pranasha*), difficulty in speech (*Vak graha*) and gait abnormality (*Skhalitha gati*). However, in the secondary stage, where spastic quadriplegia (*Sarvangaroga*) was evident *vata kapha* features were predominant. Prognostically, being an *Upadhatu pradoshaja* and *Mahamarmasraya vyadhi* it is *Krichra sadhya.*

Therapeutic Intervention

The management was focused on improvement in quality of life, giving emphasize to *Upadhathu pradhoshaja chikitsa* with *Vatha kapha* predominance.

Table 4 and table 5 shows internal medicines and procedures advised in the case respectively.

Table 4: Internal medicines

Name of drug	Dose	Time	Number of Days	Rationale
Ashtavargam kashayam	90 ml	Twice daily	2/6/22-15/6/22	*Vatakapha shamanam, Avaranaharam, Srotosodhanam*
Yogaraja guggulu gulika	1	Twice daily	2/6/22-15/6/22	*Vatakapha shamana, Vatanulomanam*
Chandanadi kashayam	90 ml	Twice daily	16/6/22-2/7/22	Indicated in *Mastulungahrasam*
Sarvamayantaka ghritam	5gm	Twice daily	16/6/22-22/6/22	Indicated in *Hanustambham*, *Karastambham*

Table 5: Procedures done

Procedures	Medicines used	Days of treatment
Jihwa lepam	*Kalyana avaleha choornam* with *Kalyanaka ghritam*	2/6/22-7/7/22
Talam	*Bala choornam* + *Vatashini tailam*	2/6/22-8/6/22
Abhyangam	*Shatahwadi tailam* + *Masha saindhava tailam*	2/6/22-8/6/22
Nasa pichu	*Anu tailam*	9/6/22-15/6/22
Patra potala sweda	*Tailam: Shatahwadi thailam* + *Masha saindhava tailam* *Talam* with *Bala choornam* + *Vatashini tailam*	9/6/22-15/6/22
Kayasekam	*Dhanwantaram tailam* + *Sahacharadi tailam*	16/6/22-22/6/22
Yoga vasti	*Kashaya vasti* *Saindhawam* - 15g *Madhu* - 120ml *Dhanwantaram mezhukupaka tailam* - 240ml *Sathapushpa kalkam* - 30g *Erandamooladi Kashayam* - 480 ml *Sneha vasthi* *Dhanwantaram mezhukupaka tailam* - 90 ml	23/6/22-130/6/22
Talapothichil	*Musta, Amalaka, Panchagandha choornam* with *Ksheerabala tailam*	1/7/22-7/7/22

Outcome Measures

The patient was assessed using Barthel index[8] and modified rankin scale[9] (mRS). At the time of admission, i.e., on 2/6/22, the Barthel index score was 50 (severe dependency) and mRS was 3 which was improved to 90 (moderate dependency) and 2 respectively at the time of discharge i.e., on 7/7/22.

DISCUSSION

In ODS, increased sodium administration during correction of initial phase (*Lavana atyupayoga*) leads to increased permeability due to *Stambha sanghata bandha vidmapana* action of *Lavana* (removing rigidity and clearing the obstruction of channels and pores) resulting in blood brain barrier disruption. Thus, immune mediators enter brain (*Vimargagamana*) and localises at pons (*Ashrayasthana*). This further results in demyelination (*Vatavahi sira shoshana*) and manifests as quadriplegia (*Sarvanga roga*).

In the present case, initially *Vata kshaya lakshanas* were presented by the patient, evident from features such as fatigue (*Sadam*), vomiting (*Chardi*) and calf muscle cramps (*Pindikodveshtana*) due to hyponatremia. Rapid correction of this lead to *Kapha pitta vridhi* due to *Lavana atiyoga*. This increased *Kapha pitta* caused *Avarana* to normal *Vata gati* in *Ashrayasthana* (*Shiras*) resulting in *Sammisra lakshana* of *Pittavritha vata* and *Kaphavritha vata* (corresponding to demyelination- *Vatavahi sira vishoshana*). *Pittavritha prana* resulted in dizziness (*Bhrama*) and fainting (*Murcha*) and symptoms such as exhaustion (*Klama*) and hindrance to movement of body parts (*Anga cheshta sangam*) were produced by *Pittavritha vyana*[10]. Further symptoms such as difficulty in speech (*Vak svara graha*) and loss of strength (*Bala pranasha*) were contributed by *Kaphavritha udana* and that of impaired gait (*Skhalitha gati*) by *Kaphavritha vyana*[10]. But the later sequalae phase was mostly *Vata kapha* predominant with symptoms such as weakness (*Akarmanyathwam*), muscle spasm (*Sankocham*), rigidity (*Deha stambham*) and spastic speech (*Vak stambham*) corresponding to *Sarvangaroga*[11]. Hence treatment line adopted was that of *Srotosodhana*, *Avaranahara* and *Vatanulomana*.

Initially *Ashtavargam kashaya* and *Yogarajaguggulu* were given internally aiming *Srotosodhana*, *Avaranavatahara* and *Vathanulomana*. In the later stage, *Vyadhiprathyanika chikitsa* was adopted. *Chandanadi kashaya* indicated in *Mastulungahrasa* and *Sarvamayantaka ghrita* indicated in *Hanustambha*, *Karastambha* and *Siroroga* were hence given.

Table 6 represents the rationale for the external treatment adopted.

Table 6: Rationale of treatment

Treatment given	Rationale
Abhyangam	*Abhyanga* was done initially with *Shatahwadi taila* and M*ashasaindhava taila*, which are indicated in *Kapha vata roga* and *Sankuchitha vatha* respectively
Talam	*Talam* with *Bala choornam* in *Vatashini taila* was done considering *Vata vridhi* in the *Udbhava sthana* i.e., *Shiras*
Patra potala swedam	As *Snigdha sweda* done with *Vatahara patra* relieves *Stambha*
Kayasekam	As it is indicated in *Vata rogas* and results in *Dhatu dridathwam* and *Indriya prasadam*
Yoga vasti	• *Erandamooladi vasti* removes *Srotorodha* and is indicated in *Kapha vatika* conditions • *Dhanwanthara mezhupaka thaila* was opted for *Anuvasana* as it is *Sarvavata vikarajith*
Talapothichil	Done with *Musta*, *Amalaka*, *Panchagandha choorna* and *Ksheerabala tailam*, as the patient had sleep disturbances and anxiety

Nasyam was not opted here, as the patient had nasal regurgitation often and hence *Nasa pichu* was done with *Anutailam*. Throughout the course *Jihwa lepa* was also done with *Kalyanavaleha choornam* for correction of *Vakstabdhatha.*

At the time of discharge, the patient had improvement in weakness and spasticity. The patient was better in performing day to day activities than before.

CONCLUSION

This case is a rare incidence of iatrogenic occurrence (*Asudha chikitsa* – mentioned in *Ashtanga hridaya suthrasthana* 13th chapter). The equilibrium of *Tridoshas* serves the key to a healthy constitution. Whenever this balance is disrupted, it leads to localisation of one or more *Doshas* leading to pathology. Here, the disturbance in osmolality results in localisation of *Pitha* and *Kapha* in *Shiras* (pons) leading to *Avarana* of *Vatha* and manifests as quadriparesis (*Sarvangaroga*). Ayurvedic medical intervention and procedures will be helpful in improving quality of life in such cases on a rehabilitative perspective.

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