GMR
The Original

INTEGRATIVE AYURVEDIC MANAGEMENT OF IDIOPATHIC INTRACRANIAL HYPERTENSION WITH CLASS III OBESITY: A CASE REPORT

Dr. Mohamed Mirdaz K[1], Dr. Sonu Jamewal[2], Dr. Nisha Sharma[3], Dr. Pratibha[4], Dr. Anjnee Bijlwan[5], Dr. Saurabh Garg[6]

[1]Professor and HOD, Department of Rog Nidan Evam Vikriti Vigyan, Sri Sai Institute of Ayurveda PG Medical College, Aligarh, Uttar Pradesh, India. drmirdas@gmail.com
[2]Assistant Professor, Department of Rog Nidan Evam Vikriti Vigyan, Motherhood Ayurveda Medical College, Roorkee, Uttarakhand, India. sonuharidwar84@gmail.com, 0009-0006-7801-0059
[3]Associate Professor, Department of Swasthyritta evum Yoga, Dr. Sarvesh Kumar Shukla Ayurvedic Medical College and Hospital, Bahraich, Uttar Pradesh, India. drnisha4341@gmail.com
[4]Associate Professor, Department of Dravyaguna Vigyana, Sanskriti Ayurvedic Medical College and Hospital, Sanskriti University, Mathura, Uttar Pradesh, India. pratibhaj004@gmail.com
[5]Assistant Professor, Department of Kriya Sharir, Motherhood Ayurveda Medical College, Roorkee, Uttarakhand, India. bijlwananjnee@gmail.com
[6]Assistant Professor, Department of Rog Nidan Evam Vikriti Vigyan, Mangalayatan Ayurvedic Medical College and Research Centre, Mangalayatan University, Aligarh, Uttar Pradesh., India. gargsaurabh133@gmail.com, 0000-0003-1534-9865

ABSTRACT

Idiopathic intracranial hypertension (IIH) mainly affects women with obesity, and papilledema threatens vision. Weight loss and carbonic anhydrase inhibitors are standard treatment, but drug intolerance is common. We report a 33-year-old woman with class III obesity (body mass index 44.3 kg/m²) and IIH diagnosed in 2018. After 4 years of acetazolamide (up to 1,500 mg/day), topiramate, and furosemide, she had disabling adverse effects, morning epistaxis, transient visual obscurations, falls, and moderate obstructive sleep apnea. These drugs were cross-tapered over 10 days while oral Ayurvedic medicines were introduced, and cerebrospinal fluid opening pressure at admission, after the taper, was 38.5 cm H_2O. She then received a 26-day, three-phase inpatient Panchakarma protocol with oral herbal and herbo-mineral medicines, followed by 30 days of oral medicines, a calorie-restricted diet, walking, and head-elevated sleep. At follow-up 86 days after admission, headache had fallen from 8/10 to 0/10 and epistaxis had stopped. Papilledema resolved from Frisén grades 3 (right) and 4 (left) to grade 0, retinal nerve fiber layer thickness fell from 168 and 214 µm to 94 and 91 µm, and visual field mean deviation improved from −4.85 and −8.40 dB to −1.05 and −1.60 dB. Opening pressure was 18.0 cm H_2O, weight fell by 10.5 kg (9.4%), and kidney and liver tests remained normal. Weight loss and natural fluctuation may have contributed to the pressure and papilledema changes, and drug withdrawal to the relief of adverse effects, so causality cannot be established. The case supports controlled studies with systematic safety monitoring.

KEYWORDS: Idiopathic intracranial hypertension; Pseudotumor cerebri; Papilledema; Obesity, morbid; Medicine, Ayurvedic; Panchakarma

INTRODUCTION

Idiopathic intracranial hypertension (IIH), also called pseudotumor cerebri, is characterized by raised intracranial pressure without a space-occupying lesion, venous sinus thrombosis, or abnormal cerebrospinal fluid (CSF) composition (Friedman et al., 2013). It predominantly affects women of reproductive age with obesity, and its burden has grown with rising population body mass index (Mollan et al., 2019). Papilledema is the central threat to vision. Visual field loss is found on perimetry in most patients and is often reversible, but a minority deteriorate and some are left with permanent loss (Wall and George, 1991). Neuroimaging often shows a partially empty sella, distension of the perioptic subarachnoid space, and flattening of the posterior globe (Agid et al., 2006), and transverse sinus stenosis is its most sensitive MR imaging correlate (Morris et al., 2017).

Consensus guidelines place weight loss at the center of management and use acetazolamide as first-line drug therapy, with topiramate or loop diuretics as adjuncts. CSF diversion, optic nerve sheath fenestration, or venous sinus stenting are considered when vision is threatened or medical therapy fails (Mollan et al., 2018). Weight loss is associated with lower intracranial pressure and resolution of papilledema (Johnson et al., 1998; Kupersmith et al., 1998; Sinclair et al., 2010), and in a randomized trial of women with a body mass index of 35 kg/m² or more, bariatric surgery lowered pressure more than community weight management (Mollan et al., 2021). Acetazolamide with a low-sodium diet gave a modest gain in perimetric mean deviation in mild visual loss, but adverse effects, including paresthesias, were more frequent than with placebo (NORDIC Idiopathic Intracranial Hypertension Study Group Writing Committee, 2014), and metabolic acidosis may complicate long-term use.

Ayurveda, the traditional medical system of India, interprets obesity with raised cranial pressure through Medo-dhatu dushti (disturbed adipose tissue metabolism) and Kapha-Medo avarana (obstruction of Vata by Kapha and fat). It treats this pattern with Shodhana (purificatory) and Shamana (palliative) measures; the purificatory

procedures are collectively known as Panchakarma. Objective ophthalmic and CSF outcomes after such treatment in IIH have, to our knowledge, not been reported in detail.
We describe a woman with drug-intolerant IIH and class III obesity whose conventional drugs were withdrawn and replaced by an inpatient Ayurvedic protocol, with serial documentation of symptoms, papilledema, optical coherence tomography, perimetry, CSF pressure, imaging, and safety laboratory tests. Our aim was to report the clinical course and to ask whether the improvement can be separated from concurrent weight loss. It cannot be fully separated, as discussed below.

MATERIAL AND METHODS

Patient and reporting standards

This report follows the CARE guidelines for case reports (Gagnier et al., 2014; Riley et al., 2017). The patient was admitted to an Ayurvedic teaching hospital in India for 26 days in 2022 and reviewed 86 days after admission (61 days after discharge). Inpatient day 1 was the day of admission and day 26 the day of discharge. Key events are summarized in Table 1. Written informed consent for publication of anonymized clinical details was obtained from the patient. Ethics committee approval was waived for this single case report.

Case presentation

The patient, a 33-year-old woman, received 18 months of ovulation induction with clomiphene citrate, human menopausal gonadotropins, and exogenous progestins for primary infertility between 2016 and 2018, with hyperphagia, fluid retention, and a 26 kg weight gain over 24 months. In mid-2018 she developed subacute visual deterioration and bilateral headache. Emergency neuro-ophthalmic evaluation found Frisén grade 4 papilledema, and lumbar puncture at diagnosis showed an opening pressure of 41 cm H_2O with normal cytology and protein 14 mg/dL; these findings satisfy the revised diagnostic criteria for IIH (Friedman et al., 2013). Over the next 4 years, escalating acetazolamide, topiramate, and furosemide failed to control her symptoms and caused persistent digital and perioral paresthesias, anorexia, brain fog, fatigue, and recurrent metabolic acidosis (bicarbonate 17-19 mmol/L).
By admission, headache had become daily and severe (visual analogue scale 8/10), centered over the vertex, occiput, and temples. It worsened with coughing, straining, and lying down, and sometimes woke her from sleep. She also reported transient visual obscurations lasting 2 to 5 seconds on coughing, straining, or standing, and projectile vomiting without nausea during pressure flares. Morning epistaxis had occurred 3 to 4 times weekly for 14 months. Giddiness with head movement, coughing, or bending had caused 2 falls in the 3 months before admission. She had fragmented sleep, loud snoring, morning bifrontal heaviness, and neck stiffness radiating to the trapezius.
She had moderate COVID-19 in 2020 and reinfection in 2021, both managed at home without systemic corticosteroids, a recognized secondary cause of intracranial hypertension. She also had seasonal allergic rhinitis, and there was no surgical history. Her family history was noncontributory apart from obesity, and her menstrual cycles were irregular. She was a homemaker, and her symptoms disrupted her daily life. The ovulation-induction drugs had been completed before symptom onset, and pressure remained raised 4 years later. She reported no exposure to tetracyclines, vitamin A derivatives, or other drugs associated with intracranial hypertension.
The Ayurvedic diagnosis was *Kaphaja*/*Tridoshaja Shiroroga* (head disorder arising from *Kapha* or all three *Doshas*) with *Kapha-Medo Avarana*, *Shirogata Shopha* (cranial edema), secondary *Urdhwaga Raktapitta* (bleeding from the upper orifices, here epistaxis), *Bhrama* (giddiness), and *Atisthaulya* (morbid obesity), with *Kapha-Vata* predominance and secondary *Pitta* and *Rakta* involvement.

Table 1. Timeline of key events (day 1 = day of admission).

Timing	Event
2016-2018	18 months of ovulation induction (clomiphene citrate, human menopausal gonadotropins, exogenous progestins) for primary infertility; 26 kg weight gain over 24 months
2018	Subacute visual deterioration and bilateral headache; Frisén grade 4 papilledema
2018 (diagnosis)	Lumbar puncture opening pressure 41 cm H_2O; MRI brain; MR venography 2 days later
2018-2022	Daily headache; escalating acetazolamide, topiramate, and furosemide with adverse effects; morning epistaxis for 14 months and 2 falls in the 3 months before admission
2020 and 2021	Moderate COVID-19 and reinfection, managed at home without systemic corticosteroids
2022	Repeat MR venography
Days −10 to −1	10-day cross-taper of conventional drugs
Day 1	Admission; opening pressure 38.5 cm H_2O after the taper; weight 112.0 kg
Days 1-14	Phase 1: external and cranial pouring therapies; oral medicines on days 1-10, then 11-14

Timing	Event
Days 15-19	Phase 2: internal oleation with massage and steam, rest day, therapeutic purgation on day 19; no oral medicines
Days 20-26	Phase 3: nasal instillation, cranial pouring, dry powder massage; final oral regimen started on day 20
Day 26	Discharge; weight 106.8 kg; oral regimen continued for 30 days
86 days after admission	Follow-up; opening pressure 18.0 cm H_2O; weight 101.5 kg

Assessments

Assessments were made at admission (day 1), discharge (day 26), and follow-up (86 days after admission). Height, weight, and body mass index were recorded, and headache severity was scored on a 0-10 visual analogue scale. Distance and near visual acuity were measured with Snellen charts, and pupils were examined with the swinging flashlight test using a neutral density filter to grade any relative afferent pupillary defect. Optic disc swelling was graded by dilated funduscopy with the Frisén scale (Frisén, 1982). Average retinal nerve fiber layer (RNFL) thickness was measured by spectral-domain optical coherence tomography, and visual fields by Humphrey 24-2 SITA-Fast perimetry.

CSF opening pressure was measured by lumbar puncture at admission and follow-up, with cell count, protein, glucose, and cytology; the 2018 value comes from the diagnostic record. Both punctures were performed in the lateral decubitus position with the legs extended, using the same technique. Pressures are given in cm H_2O (1 cm H_2O = 0.098 kPa). Baseline MRI and MR venography findings, including cerebellar tonsil position and optic nerve sheath diameter, were taken from previous treatment records, and MRI of the brain and craniovertebral junction was repeated at follow-up.

Morning epistaxis was investigated with anterior rhinoscopy, beta-2 transferrin testing of nasal secretions, and bone-window CT of the skull base to exclude a CSF leak. Sleep-disordered breathing was assessed with the STOP-Bang questionnaire and overnight portable polysomnography. Multiple occipital nodules found on examination were evaluated by soft-tissue ultrasound and MR venography to exclude a venous anomaly communicating with the dural sinuses. Kidney function, electrolytes, bicarbonate, liver enzymes, bilirubin, and urinalysis were tracked at all three time points because the protocol included a mercury-containing herbo-mineral preparation, described below.

Therapeutic intervention

Decision to withdraw conventional drugs

The decision to withdraw conventional drugs was made with a neuro-ophthalmologist. Surgical options (CSF diversion, optic nerve sheath fenestration, and venous sinus stenting) were offered, and the patient declined them. Return to conventional or surgical treatment was planned if visual fields worsened.

Pre-admission cross-taper

Before the taper she took acetazolamide 1,500 mg/day (500 mg three times daily), topiramate 50 mg twice daily, and furosemide 20 mg on alternate days. To guard against possible rebound intracranial hypertension after carbonic anhydrase inhibitor withdrawal, the drugs were withdrawn over the 10 days before admission (days −10 to −1): furosemide was stopped at the start, and acetazolamide and topiramate were reduced stepwise (Supplementary Table S1). Oral *Punarnavadi Kashayam* was introduced for gentle diuresis, and *Chandraprabha Vati* was added from day −7. Topiramate was stopped on day −7 and acetazolamide on the day of admission. The taper was supervised on an outpatient basis by her previous treating physician.

Inpatient procedures

The 26-day inpatient protocol ran in three phases (Table 2). Phase 1 (days 1-14) combined external and cranial pouring therapies. Phase 2 (days 15-19) gave internal oleation alongside massage and steam, then a rest day and therapeutic purgation. Phase 3 (days 20-26) combined nasal instillation, cranial pouring, and dry powder massage. Oral medicines were given on days 1-14 and 20-26, but not during oleation, rest, and purgation (days 15-19). Because whole-body heat and head-down posture can in theory raise venous and intracranial pressure, procedures were adapted with hemodynamic precautions, and vital signs were checked afterward. During admission she received a vegetarian diet high in protein and dietary fiber, providing roughly 1,200-1,500 kcal per day.

Table 2. Inpatient Ayurvedic procedures.

Phase	Procedure	Medicine or medium	Inpatient days	Notes
1	*Utsadana* (powder massage) and *Nadi Swedana* (localized steam)	*Kola Kulathadi Choornam*	1-14	*Lekhana* (fat-reducing) and *Kaphahara* (*Kapha*-reducing) intent; localized mild heat only, to avoid systemic vasodilation; 30-45 min per session
1	*Mukha Lepa* (facial paste)	*Mukkadi Gutika* with *Ksheera* (milk)	1-14	Applied to the face and periorbital region for local *Shophahara* (anti-edema) action; up to 60 min per session

Phase	Procedure	Medicine or medium	Inpatient days	Notes
1	*Kashaya Shirodhara*	Decoction of *Dashamoola Choornam* and *Triphala Choornam*	1-14	Continuous warm oscillating flow, cranial pouring therapy; 30-45 min per session
1	Oral medicines	See *Internal medicines during admission*	1-14 (days 1-10, then 11-14)	
2	*Vicharana Snehapana* (internal oleation)	*Guggulutiktaka Ghritam* 30 mL in the early morning with warm water	15-17	Given on the same days as massage and steam
2	*Sarvanga Abhyanga* (whole-body oil massage) and *Bashpa Swedana* (whole-body steam)	*Shatavahadi Tailam*, then steam	15-17	Cabin below 40 °C, maximum 12 minutes, head outside with a continuous cold forehead compress; vital signs checked after each session
2	*Visrama Kala* (rest)	None	18	Rest day before purgation
2	*Virechana* (therapeutic purgation)	*Avipathi Choornam* 25 g in fresh honey	19	14 watery bouts (*Madhyama Shuddhi*, moderate purification); no hypotension or clinical signs of dehydration
3	*Nasya* (nasal instillation)	*Anu Tailam*, 10 drops per nostril (*Uttama Matra*, maximum dose)	20-26	Modified supine position, 15° neck extension for 90 seconds, gentle inhalation afterward, no breath-holding or straining; secretions expectorated
3	*Takradhara*	Medicated buttermilk (*Takram* processed with *Musta* and *Amalaki*)	20-26	Cranial pouring therapy; 15-30 min per session
3	*Udwarthana* (dry powder massage) and *Nadi Swedana*	*Kola Kulathadi Choornam*	20-26	Fat-reducing intent; localized mild heat only; 30-45 min per session
3	Oral medicines	See *Internal medicines during admission*	20-26	

Internal medicines during admission

Oral medicines (*Abhyantara Aushadha*) were given in three schedules. None were given on days 15-19, during internal oleation, the rest day, and purgation. These day ranges differ from the procedural phases in Table 2. All formulations follow Sahasrayogam (Nishteswar and Vidyanath, 2007), as listed in Supplementary Table S2.

Days 1-10: Deepana-Pachana (digestive stimulation) and Shophahara (anti-edema)

- *Punarnavadi Kashayam* 60 mL, warm, in the early morning on an empty stomach, for intended anti-edema (*Shophahara*) and diuretic action.
- *Pathyashadangam Kashayam* 60 mL in the evening, 1 hour before dinner, a classical formulation for head disorders (*Shiroroga*) and eye pain (*Netra-shoola*).
- *Chandraprabha Vati* 1 tablet (500 mg) with each morning and evening decoction, for intended fat-reducing (*Medohara*) and mild diuretic (*Mootrala*) action.
- *Brihat Kasturi Bhairava Rasa* 1 tablet (125 mg) twice daily after meals with fresh ginger juice (*Ardraka Swarasa*), given as a *Rasayana* (rejuvenative). It contains a purified mercury-sulfur preparation (*Kajjali*), so kidney and liver function were monitored.
- *Drakshadi Kashayam* 40 mL at mid-day, to pacify *Pitta* and control epistaxis (*Nasagata Raktapitta*).

Days 11-14: Koshtha Shodhana (bowel preparation)

- *Vaishwanara Choornam* 5 g twice daily after meals with fresh spiced buttermilk (*Takram*), for *Agni Deepana* (digestive stimulation) and *Apana Vatanulomana* (restoring the downward movement of *Vata*).

Days 20-26: regimen later continued at discharge

- *Punarnavadi Kashayam* 60 mL in 60 mL warm boiled water twice daily, 30 minutes before breakfast and dinner, for anti-edema and tissue-clearing action.
- *Pathyashadangam Kashayam* 60 mL in 60 mL warm water twice daily before meals, for headache prevention and neuro-ophthalmic protection.
- *Hingutriguna Tailam* 5 mL at bedtime with 50 mL warm water, for *Vatanulomana* and relief of *Avarana*.
- *Dashamoola Haritaki Lehyam* 10 g (1 rounded teaspoon) at bedtime with warm water, for *Srotoshodhana* (channel clearing) and *Kaphahara* action and as support against sleep-disordered breathing.

Discharge regimen (30 days)

Punarnavadi Kashayam, *Pathyashadangam Kashayam*, *Hingutriguna Tailam*, and *Dashamoola Haritaki Lehyam* were continued as discharge medicines for 30 days, in the doses and timings given above for days 20-26. Diet and activity advice (*Pathya-Apathya*) was also given:

- Diet: low-glycemic, calorie-restricted (1,400 kcal/day) *Medohara* diet, without refined sugars, heavy curd, fermented batter, or excess sodium.
- Activity and sleep: 45 minutes of brisk walking daily and head-elevated sleep on a 30° head-of-bed wedge, intended to reduce nocturnal venous engorgement and sleep apnea.

After the 30-day discharge regimen, she was advised to continue only *Dashamoola Haritaki Lehyam*, at a reduced dose of 5 g at bedtime with warm water.

RESULTS

Baseline examination and investigations

She was alert and in moderate distress from headache. Height was 159 cm and weight 112.0 kg (body mass index 44.3 kg/m²). Blood pressure was 128/82 mmHg, heart rate 76/min, respiratory rate 16/min, and oxygen saturation 98% on room air. Neurological examination was normal apart from mild unsteadiness on tandem gait with rapid head turning and grade 1 tenderness at the temporal and suboccipital insertions.

Distance acuity was 6/6 in both eyes and near acuity N6. The left pupil reacted sluggishly with a relative afferent pupillary defect of +0.4 log units. Papilledema was Frisén grade 3 in the right eye and grade 4 in the left. Average RNFL thickness was 168 µm (right) and 214 µm (left; reference 85-105 µm). Perimetry showed enlarged blind spots and an inferonasal step in both eyes, with an early superior arcuate defect on the left (mean deviation −4.85 dB right, −8.40 dB left).

Admission CSF opening pressure, measured after the cross-taper, was 38.5 cm H_2O, with clear fluid, protein 16 mg/dL, glucose 62 mg/dL, and a normal cell count. MRI at diagnosis in 2018 had shown fluid-distended perioptic subarachnoid spaces with posterior scleral flattening and a partial empty sella. Previous treatment records documented cerebellar tonsils 3.5 mm below the foramen magnum and an optic nerve sheath diameter of 6.4 mm. MR venography (2018, repeated in 2022) showed bilateral narrowing of the mid-to-distal transverse sinuses, consistent with extrinsic compression.

Rhinoscopy showed engorged Kiesselbach's plexus without mucosal tears. Nasal secretions were negative for beta-2 transferrin, and bone-window CT showed an intact skull base, excluding a CSF leak. The STOP-Bang score was 5/8, and polysomnography showed moderate obstructive sleep apnea (22 events/hour, oxygen saturation nadir 86%). The epistaxis was considered most consistent with raised cranial venous pressure acting on a fragile venous plexus (see Discussion).

Examination also found multiple soft, mobile, non-pulsatile occipital nodules (1.0-1.8 cm). They were hyperechoic and encapsulated on ultrasound, and MR venography showed no communication with the dural sinuses, excluding a venous anomaly; they were diagnosed as benign nuchal fibrolipomatosis.

Clinical course and outcomes

No rebound headache or visual deterioration occurred during the cross-taper or the admission. Distance acuity stayed 6/6 and near acuity N6 throughout. The afferent pupillary defect was trace at discharge and absent at follow-up. Papilledema regressed to Frisén grade 1 (right) and 2 (left) at discharge and resolved completely (grade 0) in both eyes by follow-up. RNFL thickness fell to 122 and 138 µm at discharge and to 94 and 91 µm at follow-up, within the reference range. Because average RNFL can appear normal as swelling resolves even after some axonal loss, these values do not exclude early atrophy. On direct funduscopy, the temporal margin of the left optic disc appeared slightly paler than normal. Perimetric mean deviation improved to −1.05 dB (right) and −1.60 dB (left), with near-complete resolution of the nasal step and isolated mild blind-spot enlargement on the left (Table 3).

Headache fell from 8/10 to 2/10 at discharge and 0/10 at follow-up. There was no epistaxis from the second week of admission through follow-up. Giddiness had eased to mild postural lightheadedness at discharge and resolved by follow-up, with normal tandem gait. Weight fell by 5.2 kg during the 26-day admission and by 10.5 kg (9.4%) by follow-up. CSF opening pressure at follow-up was 18.0 cm H_2O, a 53.2% fall from 38.5 cm H_2O, with unremarkable cytology and biochemistry. It was not measured at discharge.

The formal follow-up MRI report stated no significant intracranial or craniovertebral junction abnormality. The cerebellar tonsils had retracted above the foramen magnum, optic nerve sheath diameter had fallen from 6.4 to 4.8 mm, and posterior scleral flattening had resolved.

Table 3. Clinical, neuro-ophthalmic, CSF, and imaging outcomes.

Parameter	Admission (day 1)	Discharge (day 26)	Follow-up (86 days)
Neuro-ophthalmic (right / left)			
Distance acuity (Snellen)	6/6 / 6/6	6/6 / 6/6	6/6 / 6/6

Parameter	Admission (day 1)	Discharge (day 26)	Follow-up (86 days)
Near acuity	N6 / N6	N6 / N6	N6 / N6
Relative afferent pupillary defect	Left, +0.4 log units	Trace (<0.2 log units)	Absent
Frisén grade	3 / 4	1 / 2	0 / 0
Average RNFL thickness (µm)	168 / 214	122 / 138	94 / 91
Perimetry mean deviation (dB)	−4.85 / −8.40	−2.60 / −4.10	−1.05 / −1.60
Symptoms and anthropometry			
Headache, visual analogue scale (0-10)	8	2	0
Morning epistaxis	3-4 per week	None from week 2	None through follow-up
Giddiness and imbalance	Daily; 2 falls in 3 months	Mild postural lightheadedness	Resolved; normal tandem gait
Weight (kg) / body mass index (kg/m²)	112.0 / 44.3	106.8 / 42.2	101.5 / 40.1
CSF and imaging			
CSF opening pressure (cm H_2O)	38.5 (after taper)	Not measured	18.0
Cerebellar tonsils	3.5 mm below foramen magnum[a]	Not imaged	Above foramen magnum
Optic nerve sheath diameter (mm)	6.4[a]	Not imaged	4.8

[a] From previous treatment records, not from imaging at admission. RNFL, retinal nerve fiber layer; reference range for average RNFL thickness 85-105 µm.

Safety and laboratory monitoring

Kidney and liver tests were within reference ranges at admission, at discharge (16 days after the 10-day course of *Brihat Kasturi Bhairava Rasa* ended), and at follow-up, and urinalysis was negative for protein and blood (Table 4). Bicarbonate rose from 20.2 to 24.6 mmol/L by discharge, after acetazolamide withdrawal. Purgation on day 19 produced 14 watery bouts without hypotension or clinical signs of dehydration, and vital signs were stable after steam therapy.

Table 4. Safety laboratory monitoring.

Parameter	Admission (day 1)	Discharge (day 26)	Follow-up (86 days)	Reference range
Blood urea nitrogen (mg/dL)	14.2	13.0	12.4	7.0-20.0
Creatinine (mg/dL)	0.82	0.78	0.74	0.50-1.10
Estimated glomerular filtration rate (mL/min/1.73 m²)	98	102	105	>90
Sodium / potassium / chloride (mmol/L)	139 / 4.2 / 101	141 / 4.4 / 102	140 / 4.3 / 102	135-145 / 3.5-5.0 / 96-106
Bicarbonate (mmol/L)	20.2	24.6	25.1	22.0-29.0
AST (U/L)	28	24	21	10-40
ALT (U/L)	32	29	24	7-56
Alkaline phosphatase (U/L)	78	74	70	44-147
Total bilirubin (mg/dL)	0.65	0.58	0.52	0.2-1.2

Electrolytes and bicarbonate are in mmol/L, numerically equal to the mEq/L in the source record. AST, aspartate aminotransferase; ALT, alanine aminotransferase.

DISCUSSION

Principal findings

In this woman with drug-intolerant IIH and class III obesity, a 26-day inpatient Ayurvedic protocol begun during withdrawal of acetazolamide, topiramate, and furosemide was followed by resolution of headache, epistaxis, and imbalance. Papilledema grade, RNFL thickness, the afferent pupillary defect, and CSF opening pressure normalized, and perimetry returned to normal or near-normal, within 86 days of admission. Visual acuity was 6/6 throughout, whereas disc grading, RNFL thickness, perimetry, and pupillary testing documented both the optic nerve injury and its recovery, in keeping with the frequent perimetric loss in IIH despite preserved acuity (Wall and George, 1991). The residual findings were slight pallor of the temporal margin of the left optic disc and a mildly enlarged left blind spot, both in the eye with the most severe baseline swelling and field loss.

Weight loss and other concurrent measures

The main alternative explanation is weight loss. She lost 10.5 kg (9.4%) over 86 days, more than the roughly 6% loss associated with resolution of marked papilledema in one series (Johnson et al., 1998). In other series, a loss of at least 2.5 kg over 3 months was associated with faster improvement in papilledema and visual fields (Kupersmith et al., 1998), and CSF pressure fell during a three-month low-energy diet (Sinclair et al., 2010). The

protocol also included a calorie-restricted diet, daily walking, head-elevated sleep, and withdrawal of three drugs, and none of these can be separated here from the herbal and procedural components.

The early change needs separate comment. Between admission and discharge on day 26, left RNFL thickness fell from 214 to 138 µm and Frisén grades fell by two while she lost 5.2 kg (4.6% of body weight). Purgation and the diuretic action attributed to the herbal formulations may have mobilized fluid, so weight alone does not cleanly separate the contributions, and no pressure was measured at discharge to show whether early decompression occurred.

Two other explanations need comment. Withdrawal of acetazolamide, topiramate, and furosemide plausibly explains the resolution of paresthesias, cognitive slowing, fatigue, and acidosis, but not the fall in pressure, which occurred after three pressure-lowering drugs were stopped; topiramate withdrawal would, if anything, have favored weight gain. Spontaneous improvement occurs in IIH, but papilledema had persisted for 4 years before admission, making coincidental remission within 86 days less likely, though not excluded.

Proposed Ayurvedic rationale

Ayurvedic reasoning links this picture to *Kapha-Medo Avarana* of *Vata* with obstruction of cranial channels, and the protocol aimed to clear it through *Virechana* and *Nasya* and to reduce adiposity (*Lekhana*) through *Utsadana* and *Udwarthana*. These constructs have not been mapped to venous outflow or CSF absorption, and no mechanistic measurement was made, so they remain hypotheses.

Safety and procedural precautions

Procedures were adapted to avoid raising venous and intracranial pressure (Table 2), and no hypotension, clinical dehydration, or hemodynamic instability was documented. Withdrawal of acetazolamide was accompanied by normalization of bicarbonate and no rebound symptoms.

Brihat Kasturi Bhairava Rasa contains a mercury preparation, and heavy metal content is a recognized concern in Ayurvedic products (Saper et al., 2004, 2008). Kidney and liver tests stayed normal after the 10-day course, but these tests do not measure mercury exposure, and blood or urine mercury is not reported. Any future study should include it.

Imaging and associated findings

Regression of tonsillar descent, optic nerve sheath distension, and posterior scleral flattening at follow-up is consistent with these signs reflecting raised pressure rather than fixed anatomy, although the baseline measurements come from 2018 records rather than imaging at admission. Cerebellar tonsillar descent occurs in IIH, and a descent of 2-4 mm was classed as cerebellar ectopia in one series (Banik et al., 2006); her 3.5 mm falls in that range.

Morning epistaxis and moderate obstructive sleep apnea plausibly share a venous mechanism: raised cranial venous pressure, with nocturnal hypercapnia and possibly morning blood pressure surges, acting on a fragile venous plexus. Seasonal allergic rhinitis may also have contributed. The epistaxis stopped, but no repeat polysomnography is reported, so any change in sleep apnea after weight loss is unknown.

LIMITATIONS

This is a single uncontrolled case with follow-up to 86 days after admission (61 days after discharge), and several interventions were applied at once. Relapse after weight regain is common in IIH (Ko et al., 2011), so durability is unknown. Persistent papilledema after years of maximum tolerated drug therapy argues against simple natural fluctuation, but does not exclude it. Outcome assessors were not blinded, formulations were not chemically standardized, and no pressure was measured at discharge. Controlled studies with standardized formulations, heavy metal monitoring, and prespecified ophthalmic endpoints are needed before any conclusion about efficacy, and this report does not support withdrawing conventional therapy outside a closely monitored setting.

CONCLUSION

In a woman with drug-intolerant IIH and class III obesity, a structured 26-day Ayurvedic inpatient protocol begun during withdrawal of acetazolamide, topiramate, and furosemide was followed by normal CSF pressure, resolved papilledema, normal RNFL thickness, and near-normal visual fields, with normal kidney and liver tests and a 10.5 kg weight loss. Weight loss and the other concurrent measures cannot be excluded as causes, so the report shows an association, not efficacy. It supports controlled studies with standardized formulations, heavy metal monitoring, and objective ophthalmic outcomes.resolved.

Table 5. Symptoms, anthropometry, CSF pressure, and imaging.

Parameter	Baseline (26 Jul 2022)	Discharge (8 Aug 2022)	Follow-up (20 Oct 2022)
Headache, visual analogue scale (0-10)	8	2	0
Morning epistaxis	3-4 per week	None in week 2	None through follow-up
Giddiness and imbalance	Daily; 2 falls	Mild postural lightheadedness	Resolved; normal tandem gait
Weight (kg) / body mass index (kg/m²)	112.0 / 44.3	106.8 / 42.2	101.5 / 40.1

Parameter	Baseline (26 Jul 2022)	Discharge (8 Aug 2022)	Follow-up (20 Oct 2022)
CSF opening pressure (cm H_2O)	38.5	Not measured	18.0
Cerebellar tonsils	3.5 mm below foramen magnum (previous records)	Not imaged	Above foramen magnum
Optic nerve sheath diameter (mm)	6.4 (previous records)	Not imaged	4.8

Baseline cerebellar tonsil position and optic nerve sheath diameter are from previous treatment records, not from imaging at admission.

Safety and laboratory monitoring

Kidney and liver tests stayed within reference ranges across the 11-day course of Brihat Kasturi Bhairava Rasa, and urinalysis was negative for protein and blood (Table 6). Bicarbonate rose from 20.2 to 24.6 mmol/L by discharge, after acetazolamide withdrawal. Purgation produced 14 watery bouts without hypotension or electrolyte imbalance, and vital signs were stable after steam therapy.

Table 6. Safety laboratory monitoring.

Parameter	Admission	Discharge	Follow-up	Reference range
Blood urea nitrogen (mg/dL)	14.2	13.0	12.4	7.0-20.0
Creatinine (mg/dL)	0.82	0.78	0.74	0.50-1.10
Estimated glomerular filtration rate (mL/min/1.73 m^2)	98	102	105	>90
Sodium / potassium / chloride (mmol/L)	139 / 4.2 / 101	141 / 4.4 / 102	140 / 4.3 / 102	135-145 / 3.5-5.0 / 96-106
Bicarbonate (mmol/L)	20.2	24.6	25.1	22.0-29.0
AST (U/L)	28	24	21	10-40
ALT (U/L)	32	29	24	7-56
Alkaline phosphatase (U/L)	78	74	70	44-147
Total bilirubin (mg/dL)	0.65	0.58	0.52	0.2-1.2

Electrolytes and bicarbonate are in mmol/L, numerically equal to the mEq/L in the source record. AST, aspartate aminotransferase; ALT, alanine aminotransferase.

DISCUSSION

Principal findings

In this woman with drug-intolerant IIH and class III obesity, an inpatient Ayurvedic protocol begun during withdrawal of acetazolamide, topiramate, and furosemide was followed by resolution of headache, epistaxis, and imbalance. Papilledema grade, RNFL thickness, the afferent pupillary defect, and CSF opening pressure normalized, and perimetry returned to normal or near-normal, within 86 days of admission. Visual acuity was 6/6 throughout, whereas disc grading, RNFL thickness, perimetry, and pupillary testing documented both the optic nerve injury and its recovery, in keeping with the frequent perimetric loss in IIH despite preserved acuity (Wall and George, 1991). The residual findings were slight pallor of the temporal margin of the left optic disc and a mildly enlarged left blind spot, both in the eye with the most severe baseline swelling and field loss.

Weight loss and other concurrent measures

The main alternative explanation is weight loss. She lost 10.5 kg (9.4%) over 86 days, more than the roughly 6% loss associated with resolution of marked papilledema in one series (Johnson et al., 1998). In other series, a loss of at least 2.5 kg over 3 months was associated with faster improvement in papilledema and visual fields (Kupersmith et al., 1998), and CSF pressure fell during a three-month low-energy diet (Sinclair et al., 2010). The protocol also included a calorie-restricted diet, daily walking, head-elevated sleep, and withdrawal of three drugs, and none of these can be separated here from the herbal and procedural components.

The early change needs separate comment. Between admission and discharge, left RNFL thickness fell from 214 to 138 µm and Frisén grades fell by two while she lost 5.2 kg (4.6% of body weight). Purgation and the diuretic action attributed to the herbal formulations may have mobilized fluid, so weight alone does not cleanly separate the contributions, and no pressure was measured at discharge to show whether early decompression occurred.

Ayurvedic reasoning links this picture to Kapha-Medo avarana of Vata with obstruction of cranial channels, and the protocol aimed to clear it through Virechana and Nasya and to reduce adiposity (Lekhana). These constructs

have not been mapped to venous outflow or CSF absorption, and no mechanistic measurement was made, so they remain hypotheses.

Safety and procedural precautions

Procedures were adapted to avoid raising venous and intracranial pressure (Table 3), and no hypotension, electrolyte disturbance, or hemodynamic instability was documented. Withdrawal of acetazolamide was accompanied by normalization of bicarbonate and no rebound symptoms.

Brihat Kasturi Bhairava Rasa contains a mercury preparation, and heavy metal content is a recognized concern in Ayurvedic products (Saper et al., 2004, 2008). Kidney and liver tests stayed normal after the 11-day course, but these tests do not measure mercury exposure, and blood or urine mercury is not reported. Any future study should include it.

Imaging and associated findings

Follow-up MRI was reported as showing no significant intracranial or craniovertebral junction abnormality. Compared with previous records, the cerebellar tonsils had retracted above the foramen magnum, the optic nerve sheath diameter had fallen from 6.4 to 4.8 mm, and posterior scleral flattening had resolved, as would be expected if these signs reflected raised pressure. Cerebellar tonsillar descent occurs in IIH, and a descent of 2-4 mm was classed as cerebellar ectopia in one series (Banik et al., 2006); her 3.5 mm falls in that range.

Morning epistaxis and moderate obstructive sleep apnea plausibly share a venous mechanism, raised cranial venous pressure with nocturnal hypercapnia, and the epistaxis stopped. No repeat polysomnography is reported, so any change in sleep apnea after weight loss is unknown.

Limitations

This is a single uncontrolled case with follow-up to 86 days after admission (73 days after discharge), and several interventions were applied at once. Relapse after weight regain is common in IIH (Ko et al., 2011), so durability is unknown. Persistent papilledema and high pressure through years of maximum tolerated drug therapy argue against simple natural fluctuation, but they do not exclude benefit from stopping drugs that were causing harm. Outcome assessors were not blinded and formulations were not chemically standardized, and no pressure was measured at discharge. Controlled studies with standardized formulations, heavy metal monitoring, and prespecified ophthalmic endpoints are needed before any conclusion about efficacy.

CONCLUSION

In a woman with drug-intolerant IIH and class III obesity, a structured Ayurvedic protocol begun during withdrawal of acetazolamide, topiramate, and furosemide was followed by normal CSF pressure, resolved papilledema, normal RNFL thickness, and near-normal visual fields, with normal kidney and liver tests and a 10.5 kg weight loss. Weight loss and the other concurrent measures cannot be excluded as causes, so the report shows an association, not efficacy. It supports controlled studies with standardized formulations, heavy metal monitoring, and objective ophthalmic outcomes.

Conflict of Interest

The authors declare that they have no conflict of interest.

Author Contributions

Dr. Mohamed Mirdaz K managed the case. Dr. Sonu Jamewal and Dr. Anjnee Bijlwan drafted the manuscript. Dr. Nisha Sharma and Dr. Pratibha contributed to Panchakarma procedure planning and manuscript revision. Dr. Mohamed Mirdaz K, Dr. Nisha Sharma and Dr. Pratibha supervised the case management, verified the clinical and classical references. Dr. Sonu Jamewal, Dr. saurabh Garg and Dr. Anjnee Bijlwan critically revised the manuscript. All authors read and approved the final manuscript.

ACKNOWLEDGEMENT

The authors acknowledge the cooperation of the patient in permitting the publication of her clinical course.

REFERENCES

1. Agid R, Farb RI, Willinsky RA, Mikulis DJ, et al. (2006). Idiopathic intracranial hypertension: the validity of cross-sectional neuroimaging signs. Neuroradiology 48: 521-527.
2. Banik R, Lin D and Miller NR (2006). Prevalence of Chiari I malformation and cerebellar ectopia in patients with pseudotumor cerebri. J. Neurol. Sci. 247: 71-75.
3. Charaka Samhita (2014). Sharma PV (Ed. and Trans.). Sutrasthana, Chapter 21 (Ashtau Ninditiya Adhyaya), verses 3-9; Chikitsasthana, Chapter 28 (Vatavyadhi Chikitsitam), verses 59-64 and 235-240. Chaukhambha Orientalia, Varanasi.
4. Friedman DI, Liu GT and Digre KB (2013). Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 81: 1159-1165.
5. Frisén L (1982). Swelling of the optic nerve head: a staging scheme. J. Neurol. Neurosurg. Psychiatry 45: 13-18.
6. Gagnier JJ, Kienle G, Altman DG, Moher D, et al. (2014). The CARE guidelines: consensus-based clinical case report guideline development. J. Clin. Epidemiol. 67: 46-51.
7. Johnson LN, Krohel GB, Madsen RW and March GA Jr (1998). The role of weight loss and acetazolamide in the treatment of idiopathic intracranial hypertension (pseudotumor cerebri). Ophthalmology 105: 2313-2317.

8. Ko MW, Chang SC, Ridha MA, Ney JJ, et al. (2011). Weight gain and recurrence in idiopathic intracranial hypertension: a case-control study. Neurology 76: 1564-1567.
9. Kupersmith MJ, Gamell L, Turbin R, Peck V, et al. (1998). Effects of weight loss on the course of idiopathic intracranial hypertension in women. Neurology 50: 1094-1098.
10. Mollan SP, Davies B, Silver NC, Shaw S, et al. (2018). Idiopathic intracranial hypertension: consensus guidelines on management. J. Neurol. Neurosurg. Psychiatry 89: 1088-1100.
11. Mollan SP, Aguiar M, Evison F, Frew E, et al. (2019). The expanding burden of idiopathic intracranial hypertension. Eye 33: 478-485.
12. Mollan SP, Mitchell JL, Ottridge RS, Aguiar M, et al. (2021). Effectiveness of bariatric surgery vs community weight management intervention for the treatment of idiopathic intracranial hypertension: a randomized clinical trial. JAMA Neurol. 78: 678-686.
13. Morris PP, Black DF, Port J and Campeau N (2017). Transverse sinus stenosis is the most sensitive MR imaging correlate of idiopathic intracranial hypertension. AJNR Am. J. Neuroradiol. 38: 471-477.
14. Nishteswar K and Vidyanath R (2007). Sahasrayogam (text with English translation). Chaukhambha Sanskrit Series Office, Varanasi.
15. NORDIC Idiopathic Intracranial Hypertension Study Group Writing Committee, Wall M, McDermott MP, Kieburtz KD, et al. (2014). Effect of acetazolamide on visual function in patients with idiopathic intracranial hypertension and mild visual loss: the idiopathic intracranial hypertension treatment trial. JAMA 311: 1641-1651.
16. Riley DS, Barber MS, Kienle GS, Aronson JK, et al. (2017). CARE guidelines for case reports: explanation and elaboration document. J. Clin. Epidemiol. 89: 218-235.
17. Saper RB, Kales SN, Paquin J, Burns MJ, et al. (2004). Heavy metal content of Ayurvedic herbal medicine products. JAMA 292: 2868-2873.
18. Saper RB, Phillips RS, Sehgal A, Khouri N, et al. (2008). Lead, mercury, and arsenic in US- and Indian-manufactured Ayurvedic medicines sold via the Internet. JAMA 300: 915-923.
19. Sinclair AJ, Burdon MA, Nightingale PG, Ball AK, et al. (2010). Low energy diet and intracranial pressure in women with idiopathic intracranial hypertension: prospective cohort study. BMJ 341: c2701.
20. Khan A, Naquvi KJ, Haider MF and Khan M A (2024). Quality by design—newer technique for pharmaceutical product development. Intelligent Pharmacy 2: 122–129.
21. Gupta J, Sharma B, Sorout R, Singh RG, Ittishree and Sharma MC (2025). Ginger (Zingiber officinale) in traditional Chinese medicine: a comprehensive review of its anti-inflammatory properties and clinical applications. Pharmacol. Res. Mod. Chin. Med. 14: 100561.
22. Sushruta Samhita (2017). Srikantha Murthy KR (Ed. and Trans.). Uttaratantra, Chapters 25-26. Chaukhambha Orientalia, Varanasi.
23. Wall M and George D (1991). Idiopathic intracranial hypertension. A prospective study of 50 patients. Brain 114: 155-180.

SUPPLEMENTARY MATERIAL

Supplementary Table S1. Medication cross-taper before admission (day 1 = day of admission).

Period	Acetazolamide	Topiramate	Furosemide	Ayurvedic oral drugs
Days −10 to −8	1,000 mg/day (500 mg twice daily)	50 mg at night	Stopped	*Punarnavadi Kashayam* 30 mL twice daily
Days −7 to −4	500 mg at night	Stopped	Stopped	*Punarnavadi Kashayam* 60 mL twice daily; *Chandraprabha Vati* 1 tablet twice daily
Days −3 to −1	250 mg at night	Stopped	Stopped	Continued
Day 1 (admission)	Stopped	Stopped	Stopped	Inpatient protocol began

Supplementary Table S2. Formulations used and their classical sources (Nishteswar and Vidyanath, 2007).

Formulation	Dosage form	Classical source
Punarnavadi Kashayam	Decoction (oral)	**Sahasrayogam**
Pathyashadangam Kashayam	Decoction (oral)	**Sahasrayogam**
Drakshadi Kashayam	Decoction (oral)	**Sahasrayogam**
Chandraprabha Vati	Tablet (oral)	**Sahasrayogam**
Brihat Kasturi Bhairava Rasa	Herbo-mineral tablet (oral)	**Sahasrayogam**
Vaishwanara Choornam	Powder (oral)	**Sahasrayogam**
Hingutriguna Tailam	Medicated oil (oral)	**Sahasrayogam**
Dashamoola Haritaki Lehyam	Confection (oral)	**Sahasrayogam**
Guggulutiktaka Ghritam	Medicated ghee (oral oleation)	**Sahasrayogam**
Avipathi Choornam	Powder (purgation)	**Sahasrayogam**
Anu Tailam	Medicated oil (nasal)	**Sahasrayogam**
Shatavahadi Tailam	Medicated oil (massage)	**Sahasrayogam**
Kola Kulathadi Choornam	Powder (external)	**Sahasrayogam**

Formulation	Dosage form	Classical source
Mukkadi Gutika	Pill, applied as paste with milk (external)	**Sahasrayogam**
Dashamoola Choornam and *Triphala Choornam*	Powders for decoction (*Shirodhara*)	**Sahasrayogam**
Takram processed with *Musta* and *Amalaki*	Medicated buttermilk (*Takradhara*)	-