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“MANAGEMENT OF ANIDRA WITH TAGARA (VALERIANA WALLICHII DC.) CHURNA WITH SPECIAL REFERENCE TO PRIMARY INSOMNIA: A CASE REPORT”

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ABSTRACT:

Background: Nidra (sleep) is enumerated among the Trayopasthambha — the three sub-pillars that sustain the body — and its derangement, Anidra or Nidranasha, is a Vata-Pitta predominant Manasika-Sharirika vyadhi. Contemporary insomnia affects an estimated 10–30% of adults globally, and conventional hypnotics carry well-documented risks of tolerance, dependence and rebound insomnia. Tagara (Valeriana wallichii DC.) is a classically documented Nidrajanana and Medhya dravya whose use in Anidra has preliminary clinical support.

Case presentation: A 38-year-old male information-technology professional presented to the outpatient department with an eight-month history of difficulty in initiating sleep, three to four nocturnal awakenings, non-restorative sleep, morning Shirogaurava, Jrimbha, Angamarda, Tandra and reduced work concentration. He had discontinued zolpidem four weeks earlier because of morning sedation and apprehension about dependence. Secondary causes were excluded by clinical assessment and laboratory investigation. He was diagnosed as a case of Anidra (Vata-Pittaja) corresponding to chronic insomnia disorder.

Intervention and outcome: Tagara Churna 3 g twice daily with 100 mL lukewarm cow's milk as Anupana was administered for 30 days, together with Nidana Parivarjana and structured sleep hygiene; no other Ayurvedic medicine or procedure was given. Sleep-onset latency fell from 105 to 18 minutes, total sleep time rose from 3.8 to 6.9 hours and sleep efficiency from 48% to 86%. The Pittsburgh Sleep Quality Index improved from 15 to 5, the Insomnia Severity Index from 20 to 7 and the Athens Insomnia Scale from 17 to 5. The composite Ayurvedic symptom score improved by 68.18%. Gains were largely maintained at the 30-day drug-free follow-up, with no

rebound insomnia and no adverse event.

Conclusion: Tagara Churna with Godugdha Anupana produced a clinically meaningful, well-tolerated and sustained improvement in chronic Anidra in this patient. The observation is consistent with the GABAergic and adenosinergic pharmacology of valerianic acid and valepotriates, and supports the conduct of adequately powered randomised controlled trials employing objective sleep measurement.

Keywords: Anidra, Nidranasha, Tagara, Valeriana wallichii, primary insomnia, Nidrajanana, case report, Ayurveda, Pittsburgh Sleep Quality Index.

1. INTRODUCTION:

Ayurveda situates sleep within its most fundamental physiological scheme. Acharya Charaka describes Ahara, Nidra and Brahmacharya as the *Trayopasthambha* — the three subordinate pillars upon which the body, its strength, complexion and continued growth rest.¹ The dependence is stated in still stronger terms in the Nidra-specific verse of Sutrasthana: happiness and misery, nourishment and emaciation, strength and debility, potency and sterility, knowledge and ignorance, and life itself are said to be contingent upon sleep.² Sleep is therefore not treated as a passive interval between periods of activity but as an active, restorative physiological process whose disturbance carries systemic consequences.

The physiology of Nidra is explained through the interplay of *Kapha Dosha* and *Tamo Guna*. When the mind (Manas) becomes fatigued and the sensory and motor faculties withdraw from their objects, sleep ensues.² Charaka classifies Nidra into six varieties — Tamobhava, Shleshmasamudbhava, Manah-Sharira-Shrama-sambhava, Agantuki, Vyadhyanuvaritini and Ratriwabhavaprabhava — of which only the last is considered physiological (Bhutadhatri).³ Any process that diminishes Kapha and Tamas, or that aggravates Vata and Rajas in the Manovaha Srotas, therefore obstructs the natural onset of sleep.

Anidra, also termed Nidranasha or Nidra-alpata, is described as a symptom in several disorders and as an independent clinical entity. Charaka enumerates its causative factors as Vata-Pitta Vriddhi, Kapha Kshaya, Manasika Santapa (mental affliction), Dhatu Kshaya, Abhighata (trauma) and Vyadhi-karshana (debility due to disease).⁴ Vagbhata adds Ratri Jagarana, Chinta, Krodha, Bhaya, Shoka and excessive Ruksha-Katu-Tikta Ahara among its precipitants.⁵ The clinical presentation described in the classics — Jrimbha (yawning), Angamarda (body ache), Shiogaurava and Shirashula (heaviness and pain in the head), Tandra and Klama (lassitude), Alasya (lethargy), Bhrama (giddiness), Apakti (indigestion) and Glani — maps closely onto the daytime impairment domain that modern nosology requires for a diagnosis of insomnia disorder.

In contemporary sleep medicine, the International Classification of Sleep Disorders, Third Edition, defines chronic insomnia disorder as difficulty in initiating or maintaining sleep, occurring at least three nights per week for at least three months despite adequate opportunity and circumstances for sleep, accompanied by clinically significant daytime impairment.⁶ The DSM-5-TR criteria are broadly concordant, distinguishing episodic from persistent forms at the three-month boundary.⁷

Globally, 10–30% of adults report chronic insomnia; in India, community and national survey data indicate that insomnia symptoms affect roughly one third of older adults, with treatment-seeking behaviour remaining strikingly low.^{8,9} The World Health Organization has estimated that 3.6 million years of productive, healthy life are lost annually as a consequence of primary insomnia.¹⁰

Standard pharmacotherapy relies on benzodiazepines and non-benzodiazepine benzodiazepine-receptor agonists (the so-called Z-drugs). Both act at the classical benzodiazepine binding site formed at the α/γ_2 interface of the GABA_A receptor. Their efficacy is well established for short-term use, but meta-analytic data show that in older adults the number needed to treat for improved sleep quality is small while the number needed to harm for adverse cognitive and psychomotor events is smaller still.¹¹ Tolerance, dose escalation, dependence, next-day sedation, falls and rebound insomnia on withdrawal are recognised limitations, and current European and American College of Physicians guidelines accordingly position cognitive behavioural therapy for insomnia as first-line, restricting hypnotics to short courses.^{12,13} This therapeutic gap has renewed interest in classical Nidrajanana dravyas.

Tagara (*Valeriana wallichii* DC., syn. *Valeriana jatamansi* Jones; Fam. Valerianaceae/Caprifoliaceae) is one such drug. Bhavaprakasha Nighantu places it in the Karpuradi Varga and attributes to it Tikta, Katu and Kashaya Rasa, Laghu and Snigdha Guna, Ushna Virya and Katu Vipaka, with Tridoshahara action and specific indications in Visha, Unmada, Apasmara, Netraroga and Nidranasha.¹⁴ Sushruta includes it within the Vachadi and Eladi Ganas. The Ayurvedic Pharmacopoeia of India recognises the dried rhizome and root as the official part and prescribes a Churna dose of 3–6 g per day.¹⁵ A comparative clinical trial has reported that Tagara Churna produced a 76.00% improvement in initiation of sleep and a 55.17% improvement in duration of sleep in primary insomnia, outperforming Jatamansi on all four cardinal domains.¹⁰ Its principal constituents — valerenic acid, valepotriates such as valtrate and isovaltrate, sesquiterpenoids and lignans — have documented central nervous system activity.

Notwithstanding this evidence, single-drug case documentation of Tagara in Anidra using validated sleep instruments remains sparse in the indexed literature. The present case report describes the clinical course of a patient with chronic Anidra treated with Tagara Churna as the sole pharmacological intervention, assessed simultaneously through classical Ayurvedic symptom grading and three internationally validated insomnia scales. The report follows the CARE (Case Report) guidelines.¹⁶

2. MATERIALS AND METHODS (CASE PRESENTATION):

2.1 Patient Information:

A 38-year-old married Hindu male, employed as a software engineer in an information-technology firm, belonging to the middle socio-economic class and residing in an urban locality, reported to the outpatient department of Hospital with complaints of disturbed sleep of eight months'

duration.

2.2 Chief Complaints:

Table 1: Chief complaints with duration

Complaint (Ayurvedic term)	Clinical description	Duration
Nidra-anarambha	Difficulty in initiating sleep despite adequate opportunity	8 months
Vichhinna Nidra	Three to four awakenings per night with difficulty resuming sleep	8 months
Nidra-alpata	Reduced total sleep duration; non-restorative sleep	8 months
Shirogaurava / Shirashula	Heaviness and dull ache in the head on waking	6 months
Jrimbha, Angamarda	Excessive yawning and generalised body ache through the day	6 months
Tandra, Klama, Alasya	Daytime drowsiness, fatigue without exertion, lethargy	6 months
Manasika Klama	Irritability and impaired concentration at work	4 months

2.3 History of Present Illness:

The patient reported having slept satisfactorily until approximately nine months prior to presentation, with a habitual sleep-onset latency of about 15 minutes and a total sleep time of seven to eight hours. His difficulty began after he was assigned to a rotational-shift project that required him to remain awake and engaged with illuminated screens until 01:00–02:00 hours on most working days (Ratri Jagarana). Over the same period his tea consumption rose to five or six cups per day, the last being taken after 21:00 hours; meals became irregular in quantity and timing (Vishamashana), and a family financial difficulty introduced sustained Chinta and Shoka.

Sleep-onset latency progressively lengthened to 90–120 minutes. Total sleep time fell to three and a half to four hours, interrupted by three to four awakenings, after each of which he required a further 15–20 minutes to fall asleep. He typically woke finally at about 05:00 hours without feeling refreshed. Daytime symptoms of headache, yawning, body ache and impaired concentration followed within two months of the sleep disturbance and had begun to affect his occupational performance.

He initially self-medicated with over-the-counter sedating antihistamines intermittently for about six weeks with partial and inconsistent benefit. He was subsequently prescribed zolpidem 5 mg at bedtime elsewhere, which he took for three weeks. Although sleep latency shortened, he experienced persistent morning grogginess and became apprehensive about dependence, and discontinued the drug of his own accord. He remained free of all hypnotic and sedative medication for four weeks before

presenting to the Ayurvedic outpatient department, and this interval served as a wash-out period.

2.4 Past, Family, Personal and Treatment History –

Table 2: Systematic history

Domain	Findings
Past history	No history of diabetes mellitus, hypertension, thyroid disorder, bronchial asthma, epilepsy, chronic pain syndrome, head injury or any diagnosed psychiatric illness. No previous hospitalisation or surgery.
Family history	No family history of sleep disorder, psychiatric illness or substance dependence.
Ahara	Mixed diet; Vishamashana (irregular in quantity and time); predominance of Katu, Tikta and Ruksha Ahara; frequent late dinner after 22:30 hours; fast food two to three times per week.
Vihara	Sedentary occupation with 9–11 hours of screen exposure daily; no regular Vyayama; occasional Diwaswapna on holidays; Ratri Jagarana on most working days.
Vyasana	Tea 5–6 cups per day. No alcohol, tobacco, smoking or other substance use.
Koshtha / Mala	Krura Koshtha; Mala Pravritti once in two days with straining, hard consistency.
Mutra	5–6 times per day, 1–2 times per night; no burning micturition.
Nidra	Alpa and Vishama (Anidra) — the presenting complaint.
Manasika Bhava	Chinta and Shoka predominant; occasional Krodha; Rajas–Tamas imbalance with Rajo Vriddhi.
Treatment history	OTC sedating antihistamines (intermittent, ~6 weeks); zolpidem 5 mg HS for 3 weeks, self-discontinued 4 weeks before enrolment. No Ayurvedic treatment taken previously.

2.5 Ashtavidha and Dashavidha Pariksha:

Table 3: Ashtavidha Pariksha (eight-fold examination)

Parameter	Finding	Parameter	Finding
Nadi	Vata-Pittaja, 78/min	Shabda	Spashta
Mala	Krura, once in 2 days	Sparsha	Ruksha, Anushna-sheeta
Mutra	Prakruta, 5–6/day	Druk	Prakruta with periorbital darkening
Jihva	Ishat Sama (mildly coated)	Akruti	Madhyama

Table 4: Dashavidha Pariksha (ten-fold examination)

Parameter	Finding	Remarks
Prakriti	Vata-Pittaja	Predisposes to Nidranasha through inherent Rajas dominance
Vikriti	Madhyama	Dosha, Dushya, Kala and Bala moderately involved
Sara	Madhyama (Rasa-Mamsa Sara)	—
Samhanana	Madhyama	—
Pramana	Height 172 cm, Weight 68 kg, BMI 22.9 kg/m ²	Within normal range
Satmya	Vyamishra	—
Satva	Madhyama	Reduced tolerance to psychological stressors
Ahara Shakti	Abhyavarana — Madhyama; Jarana — Madhyama	Vishamagni
Vyayama Shakti	Avara	Sedentary lifestyle
Vaya	Madhyama (38 years)	Pitta-predominant age group

2.6 Clinical Findings:

On general examination the patient was conscious, cooperative and well oriented to time, place and person. He was afebrile. Pulse rate was 78 beats per minute, regular and of good volume; blood pressure 124/78 mmHg in the right arm, sitting; respiratory rate 16 per minute; oxygen saturation 98% on room air. Pallor, icterus, cyanosis, clubbing, lymphadenopathy and pedal oedema were absent. Mild periorbital hyperpigmentation was noted.

Systemic examination was unremarkable. Cardiovascular examination revealed normal first and second heart sounds with no murmur. The respiratory system was clear on auscultation with vesicular breath sounds bilaterally. The abdomen was soft and non-tender with no organomegaly. Central nervous system examination showed intact higher functions, normal cranial nerves, normal power and tone in all four limbs, and no focal neurological deficit.

Mental status examination revealed a mildly anxious affect with preserved insight and judgement. There was no evidence of delusion, hallucination, formal thought disorder, suicidal ideation or psychomotor retardation. Screening instruments recorded a Generalised Anxiety Disorder-7 score of 8 (mild anxiety) and a Patient Health Questionnaire-9 score of 4 (minimal depressive symptoms), excluding a major depressive episode as the primary driver of the sleep disturbance. The STOP-BANG questionnaire score was 1, and the bed partner denied habitual snoring, witnessed

apnoea or restless leg symptoms.

2.7 Investigations:

Laboratory investigations were performed at baseline to exclude secondary causes of insomnia and to establish a safety baseline.

Table 5: Baseline laboratory investigations

Investigation	Value	Reference range / Inference
Haemoglobin	13.8 g/dL	13.0–17.0 g/dL — normal
Total leucocyte count	7,400 /cmm	4,000–11,000 /cmm — normal
ESR	12 mm/1st hour	Normal
Fasting blood sugar	92 mg/dL	70–100 mg/dL — normal
Post-prandial blood sugar	118 mg/dL	<140 mg/dL — normal
Serum TSH	2.4 µIU/mL	0.4–4.0 µIU/mL — euthyroid
Blood urea / Serum creatinine	24 mg/dL / 0.9 mg/dL	Normal renal function
SGOT / SGPT	26 / 30 IU/L	Normal hepatic function
Serum calcium	9.4 mg/dL	Normal
Serum vitamin B12	340 pg/mL	Normal
Serum 25(OH) vitamin D	28 ng/mL	Insufficient but not deficient; not supplemented during study
Urine routine and microscopy	Normal	No abnormality
Electrocardiogram	Normal sinus rhythm	No abnormality

All investigations were within normal limits, thereby excluding anaemia, thyroid dysfunction, dysglycaemia, renal and hepatic disease as secondary contributors. Polysomnography was not performed as clinical assessment did not suggest sleep-disordered breathing or a movement disorder of sleep.

2.8 Diagnostic Assessment:

Ayurvedic diagnosis:

Anidra (Nidranasha) with Vata-Pitta predominance and Kapha-Tamo Kshaya, arising from Manovaha Srotodushti — Kevala Anidra, that is, occurring as an independent disorder rather than as a lakshana of another vyadhi.

Modern diagnosis:

Chronic insomnia disorder (primary insomnia), satisfying ICSD-3 and DSM-5-TR criteria — sleep initiation and maintenance difficulty occurring nightly for more than three months, with adequate sleep opportunity and demonstrable daytime impairment, not attributable to another sleep disorder, medical condition, mental disorder or substance.

Table 6: Samprapti Ghataka (components of pathogenesis) in this case

Component	Determination in this patient
Nidana	Ratri Jagarana, Ativichintana (Chinta–Shoka), Vishamashana, Ruksha-Katu Ahara Sevana, excessive tea, Ativyavasaya (occupational overstrain), absence of Vyayama
Dosha	Vata (Prana and Vyana) and Pitta (Sadhaka and Alochaka) Vriddhi; Kapha (Tarpaka) Kshaya
Manasika Dosha	Rajo Vriddhi with Tamo Kshaya
Dushya	Rasa Dhatu; Manas
Agni	Jatharagni — Vishamagni
Srotas	Manovaha and Rasavaha Srotas
Srotodushti Prakara	Sanga and Vimarga Gamana
Udbhava Sthana	Amashaya (Pakwashaya for Vata)
Sanchara Sthana	Rasayani (Sarva Sharira)
Adhishthana	Hridaya and Shiras (seats of Manas)
Vyakta Sthana	Manas and Sarva Sharira
Roga Marga	Madhyama
Sadhya-Asadhyata	Krichchhra Sadhya (chronic, eight-month duration, with prior hypnotic exposure)

2.9 Timeline:**Table 7: Chronological timeline of the case**

Time point	Event
– 9 months	Assignment to rotational-shift project; onset of Ratri Jagarana, increased tea intake, irregular meals and psychosocial stress
– 8 months	Onset of sleep-initiation difficulty and nocturnal awakenings
– 6 months	Appearance of daytime symptoms — Shirogaurava, Jrimbha, Angamarda, Tandra
– 4 months	Intermittent self-medication with over-the-counter sedating antihistamines
– 3 to – 2 months	Zolpidem 5 mg at bedtime for three weeks; discontinued because of morning sedation
– 1 month to Day 0	Drug-free wash-out period
Day 0	Presentation to OPD; history, examination, investigations, baseline assessment, written informed consent
Day 1–30	Tagara Churna 3 g twice daily with lukewarm cow's milk; Nidana Parivarjana and sleep hygiene
Day 10, 20, 30	Scheduled interim and end-of-treatment assessments
Day 60	Drug-free follow-up assessment (30 days after stopping treatment)

2.10 Therapeutic Intervention:**Table 8: Details of the intervention**

Attribute	Specification
Drug	Tagara Churna — fine powder of the dried rhizome and root of <i>Valeriana wallichii</i> DC. (Fam. Valerianaceae)
Source and identity	Procured from a GMP-certified manufacturer; batch no. []. Authenticated in the Department of Dravyaguna against the monograph and organoleptic, macroscopic, microscopic and physico-chemical standards of the Ayurvedic Pharmacopoeia of India, Part I. Herbarium specimen retained.
Dose	3 g twice daily (total 6 g/day, within the API range of 3–6 g/day)
Anupana	100 mL lukewarm cow's milk (Koshna Godugdha)
Kala (time)	Morning at 08:00 hours after breakfast, and at night one hour before the intended sleep time
Route	Oral
Duration	30 days
Follow-up	Further 30 days without any medication
Co-interventions	Nidana Parivarjana and structured sleep-hygiene counselling only. No Shirodhara, Abhyanga, Nasya, Shirobasti, Panchakarma procedure, other Ayurvedic formulation, hypnotic, anxiolytic or antidepressant was administered at any point during the study period.

2.11 Pathya–Apathya and Sleep Hygiene Advice:**Table 9: Pathya and Apathya prescribed**

Pathya (to be observed)	Apathya (to be avoided)
Godugdha, Ghrita, Shali and Godhuma preparations; Madhura Rasa predominant, warm, well-cooked, Snigdha Ahara	Ruksha, Katu, Tikta and Kashaya Rasa predominant diet in excess; dry and stale food
Fixed sleeping and waking hours; retiring by 22:30 hours	Ratri Jagarana; screen exposure in the hour preceding sleep
Draksha, Kharjura and other Madhura Snigdha dravyas	Tea and coffee beyond two cups per day, none after 16:00 hours
Regular moderate Vyayama to half of one's capacity, in the morning	Ativayayama, particularly in the evening; complete physical inactivity
Dhyana, Bhramari and Anuloma-Viloma Pranayama, Shavasana before sleep	Ativichintana, Chinta, Shoka, Krodha and Bhaya, particularly at night
Quiet, dark and well-ventilated bedroom; bed used only for sleep	Diwaswapna; daytime napping
Warm-water bath in the evening; loose comfortable clothing	Vishamashana; heavy meal within two hours of sleep

2.12 Assessment Criteria:

Outcomes were assessed at Day 0, Day 10, Day 20, Day 30 and at the Day 60 drug-free follow-up, using four complementary sets of measures.

2.12.1 Subjective sleep parameters

The patient maintained a seven-day sleep diary preceding each assessment point, from which sleep-onset latency (SOL), total sleep time (TST), number of nocturnal awakenings, wake after sleep onset (WASO) and sleep efficiency were derived. Sleep efficiency was calculated as total sleep time divided by time in bed, expressed as a percentage; time in bed was held constant at eight hours (480 minutes) throughout.

2.12.2 Classical symptom grading

Cardinal and associated symptoms of Anidra were graded on a four-point ordinal scale (0–3) constructed for this case on the basis of previously published Anidra grading schemes.

Table 10: Grading scale for cardinal and associated symptoms of Anidra

Symptom	Grade 0	Grade 1	Grade 2	Grade 3
Nidra-anarambha (sleep-onset latency)	< 15 min	15–30 min	31–60 min	> 60 min
Nidra-alpata (total sleep duration)	7–8 hours	6–7 hours	4–6 hours	< 4 hours
Vichhinna Nidra (awakenings/night)	Nil	Once	2–3 times	> 3 times
Shirogaurava / Shirashula	Absent	Mild, no interference	Moderate, tolerable interference	Severe, requires analgesic
Jrimbha	Absent	Occasional (< 5/day)	Frequent (5–10/day)	Very frequent (> 10/day)
Angamarda	Absent	Mild, only after exertion	Moderate, present through the day	Severe, impairs routine work
Tandra / Klama	Absent	Occasional daytime drowsiness	Persistent, affects routine	Severe, compels daytime rest
Alasya	Absent	Mild, initiates work with effort	Moderate, delays routine tasks	Severe, avoids routine tasks
Manasika Klama (irritability, poor concentration)	Absent	Mild, noticed only by self	Moderate, noticed by others	Severe, affects relationships/work

Maximum obtainable composite score: 27 (nine parameters × grade 3).

2.12.3 Validated instruments

Three validated psychometric instruments were administered: the Pittsburgh Sleep Quality Index (PSQI; range 0–21, global score > 5 indicating poor sleep quality),¹⁷ the Insomnia Severity Index (ISI; range 0–28, with bands of 0–7 no clinically significant insomnia, 8–14 subthreshold, 15–21 moderate and 22–28 severe),¹⁸ and the Athens Insomnia Scale (AIS; range 0–24, cut-off ≥ 6 for a diagnosis of insomnia).¹⁹ Daytime sleepiness was assessed with the Epworth Sleepiness Score (ESS; range 0–24, > 10 indicating excessive daytime sleepiness)²⁰ and anxiety with the GAD-7.²¹

2.12.4 Safety assessment

Vital parameters were recorded at every visit. Complete blood count, liver function tests and renal function tests were repeated at Day 30. The patient was instructed to report any untoward symptom immediately, and a structured enquiry for adverse events — morning sedation, headache, giddiness, gastric irritation, nausea, palpitation and skin reaction — was conducted at each visit.

2.13 Ethical Considerations:

Written informed consent was obtained from the patient for treatment, for the use of his clinical data for academic and scientific publication, and for the publication of this report. Anonymity has been maintained throughout; no identifying photograph or personal identifier is included. The report has been prepared in accordance with the CARE guidelines for case reports. Institutional Ethics Committee approval / exemption reference: [].

3. RESULTS (OBSERVATIONS):

The patient completed the full 30-day course of treatment and attended all scheduled assessments, including the Day 60 drug-free follow-up. Compliance, verified by pill-diary and returned-container count, exceeded 95%.

3.1 Effect on Subjective Sleep Parameters:

Table 11: Serial change in subjective sleep parameters

Parameter	Day 0	Day 10	Day 20	Day 30	Day 60 (F/U)
Sleep-onset latency (min)	105	62	35	18	20
Total sleep time (hours)	3.8	4.9	5.8	6.9	6.8
Nocturnal awakenings (n)	4	3	1	1	1
Wake after sleep onset (min)	65	40	20	10	12
Sleep efficiency (%)	48	61	73	86	85

Sleep efficiency computed against a fixed time in bed of 480 minutes.

Sleep-onset latency showed the earliest and most substantial change, falling by 41% within the

first ten days and by 82.86% by the end of treatment, from 105 minutes to 18 minutes — a value within the normal physiological range. Total sleep time rose progressively from 3.8 hours to 6.9 hours, an increase of 81.58%. The number of nocturnal awakenings fell from four to one by Day 20 and remained at one thereafter, while wake after sleep onset reduced from 65 minutes to 10 minutes. Sleep efficiency improved from 48% to 86%, crossing the conventional threshold of 85% that separates disturbed from consolidated sleep. At the Day 60 assessment, conducted 30 days after the last dose, all five parameters remained close to their end-of-treatment values, with only a marginal drift in sleep-onset latency (18 to 20 minutes) and total sleep time (6.9 to 6.8 hours).

3.2 Effect on Cardinal and Associated Symptoms:

Table 12: Serial change in graded symptoms of Anidra

Symptom	Day 0	Day 10	Day 20	Day 30	Day 60	% relief (Day 30)
Nidra-anarambha	3	2	2	1	1	66.67
Nidra-alpata	3	2	2	1	1	66.67
Vichhinna Nidra	3	2	1	1	1	66.67
Shirogaurava / Shirashula	2	2	1	0	0	100.00
Jrimbha	2	1	1	0	0	100.00
Angamarda	2	1	1	1	0	50.00
Tandra / Klama	3	2	1	1	1	66.67
Alasya	2	2	1	1	0	50.00
Manasika Klama	2	2	1	1	1	50.00
Composite score	22	16	11	7	5	68.18

The composite symptom score reduced from 22 at baseline to 7 at the end of treatment, representing 68.18% relief, and reduced further to 5 at follow-up (77.27% relief from baseline). Complete relief was obtained in Shirogaurava/Shirashula and Jrimbha by Day 30, and additionally in Angamarda and Alasya by Day 60. The three cardinal sleep parameters — Nidra-anarambha, Nidra-alpata and Vichhinna Nidra — each improved from grade 3 to grade 1, a 66.67% reduction, but did not reach grade 0 within the observation period. Manasika Klama showed the slowest response, improving from grade 2 to grade 1 by Day 20 and remaining there, which is consistent with the persistence of the underlying occupational and financial stressors, which were not themselves modifiable.

3.3 Effect on Validated Instruments:**Table 13: Change in validated psychometric scores**

Instrument	Day 0	Day 30	Day 60	% change (Day 30)	Change in severity band
PSQI (0–21)	15	5	5	66.67	Poor sleeper → Good sleeper (≤ 5)
ISI (0–28)	20	7	8	65.00	Moderate insomnia → Not clinically significant
AIS (0–24)	17	5	5	70.59	Above cut-off → Below cut-off (< 6)
ESS (0–24)	12	6	5	50.00	Excessive daytime sleepiness → Normal
GAD-7 (0–21)	8	3	3	62.50	Mild anxiety → Minimal

All five instruments moved in a concordant direction, and in each case the magnitude of change exceeded the minimal clinically important difference reported for that instrument. The PSQI global score of 5 at Day 30 placed the patient within the “good sleeper” category for the first time in eight months. The ISI reduction of 13 points is well above the six-point threshold generally accepted as a clinically meaningful response and the eight-point threshold for remission. The parallel reduction in GAD-7 suggests that improvement in sleep and reduction in anxiety proceeded together, although the direction of causality cannot be determined from a single case.

3.4 Overall Response and Follow-up:

Taking the composite symptom score, the overall response was categorised as marked improvement (50–75% relief) at Day 30 and as marked-to-complete improvement (77.27%) at Day 60. Critically, no deterioration was observed in the 30 days following withdrawal of the drug. Sleep-onset latency, total sleep time and awakenings remained stable, and there was no rebound insomnia — the phenomenon of transient worsening beyond baseline that characteristically follows discontinuation of benzodiazepine-receptor agonists, and which this patient had himself experienced after stopping zolpidem. The patient reported that he no longer anticipated bedtime with apprehension, and his self-rated occupational concentration returned to what he described as his pre-illness level.

3.5 Safety and Adverse Events:

No adverse event was reported or observed at any point. Specifically, the patient did not experience morning sedation, hangover effect, headache, giddiness, gastric irritation, nausea,

palpitation, altered taste or any cutaneous reaction. Vital parameters remained stable throughout (blood pressure range 118/76 to 126/80 mmHg; pulse 72–80 per minute). Repeat complete blood count, liver function tests and renal function tests at Day 30 were within normal limits and showed no clinically relevant deviation from baseline. The drug was found palatable with milk and no compliance difficulty was reported.

4. DISCUSSION:

This case documents a clinically meaningful and durable improvement in chronic Anidra following 30 days of Tagara Churna administered as the sole pharmacological agent, with benefit sustained through a 30-day drug-free follow-up and no adverse effect. Three aspects merit detailed consideration: the Ayurvedic understanding of the pathogenesis in this particular patient, the probable mode of action of the intervention viewed both classically and pharmacologically, and the position of this observation within the existing evidence base.

4.1 Nidana and Samprapti in this Patient:

The nidana profile in this case corresponds almost point for point with the classical enumeration. Ratri Jagarana is described by Vagbhata as inherently Ruksha in its effect and is the single most direct Vata-prakopaka nidana for Nidranasha.⁵ Sustained Chinta and Shoka act as Manasika nidana, producing Rajo Vriddhi in the Manas; since Nidra is fundamentally Tamobhava, an ascendant Rajas necessarily obstructs its onset. Vishamashana and predominance of Ruksha-Katu-Tikta Ahara compounded Vata Vriddhi while simultaneously depleting Kapha, and repeated tea intake — Katu, Ushna and Rukshana in its effect — acted as an additional Vata-vardhaka and, in modern terms, as a direct adenosine-receptor antagonist with a half-life sufficient to interfere with sleep initiation when taken after 21:00 hours.

The resulting Samprapti may be stated succinctly: the aggregate of these nidanas produced Vata and Pitta Vriddhi with concurrent Kapha and Tamo Kshaya; the vitiated Doshas localised in the Manovaha Srotas, whose Adhishthana are Hridaya and Shiras, producing Sanga; the physiological withdrawal of the Indriyas from their Vishayas that constitutes normal sleep was thereby obstructed, manifesting as Nidranasha with its Vataja anubandha lakshanas of Shirashula, Angamarda, Jrimbha and Klama. The identification of Krura Koshta and Vishamagni in this patient further supports Vata dominance and has therapeutic relevance, since Vata-shamana was the primary requirement.

4.2 Probable Mode of Action of Tagara — Ayurvedic Perspective:

Table 14: Rasapanchaka of Tagara and its therapeutic correlation in Anidra

Attribute	Property of Tagara	Correlation with the Samprapti of Anidra
Rasa	Tikta, Katu, Kashaya	Tikta and Kashaya act as Deepana-Pachana correcting Vishamagni; Katu Rasa performs Srotoshodhana of the obstructed Manovaha Srotas
Guna	Laghu, Snigdha	Snigdha Guna is directly Vata-shamaka and counteracts the Rukshata generated by Ratri Jagarana; Laghu Guna prevents Srotorodha
Virya	Ushna	Ushna Virya breaks Vata-Kapha Sanga and, with Snigdha Guna, produces Vata-anulomana — the essential prerequisite for Nidra
Vipaka	Katu	Sustains Agni and prevents Ama formation during therapy
Prabhava	Nidrajanana	The specific and non-derivable action by which Tagara induces sleep; classically enumerated among Nidrajanana dravyas
Karma	Vatakaphahara, Medhya, Vedanasthapana, Vishaghna, Krimighna, Sangyasthapana	Medhya action addresses Manasika Klama and impaired concentration; Vedanasthapana explains the complete relief of Shirashula by Day 30

It is worth pausing on an apparent paradox that postgraduate scholars frequently raise. Tagara is Ushna, Tikshna and Katu-Vipaka, yet Nidra is a Kapha-Tamas dependent phenomenon. How does a heating, penetrating drug induce sleep? The resolution lies in three considerations. First, the primary obstacle to sleep in this patient was not deficient Kapha in isolation but Vata-mediated Sanga in the Manovaha Srotas; Ushna-Tikshna-Snigdha properties relieve that Sanga, after which the physiological Kapha-Tamas process of sleep can proceed unimpeded. Second, the Snigdha Guna of Tagara, uncommon among Tikta-Ushna drugs, provides direct Vata-shamana that offsets its Rukshata. Third, and most importantly, the sleep-inducing action of Tagara is attributed to Prabhava — an action not deducible from Rasa, Guna, Virya or Vipaka — and this is precisely why the classical texts list it as a Nidrajanana dravya despite its Ushna nature.

The choice of Anupana deserves equal emphasis, and is often treated carelessly in student practice. Godugdha is Madhura in Rasa and Vipaka, Snigdha and Guru in Guna and Sheeta in Virya; it is Jeevaniya, Balya, Rasayana and — significantly for this indication — is itself enumerated among the Nidrakara dravyas by Charaka.⁴ Milk therefore performs three functions here simultaneously: it

moderates the Ushna Virya and Rukshata of Tagara, it supplies the Snigdha-Guru-Madhura qualities that augment Tarpaka Kapha and physiological Tamas, and it contributes an independent Nidrajanana effect. The combination is thus synergistic rather than merely vehicular. Modern nutritional pharmacology adds that milk supplies tryptophan, the precursor of serotonin and thence of melatonin, providing a plausible convergent mechanism.

4.3 Probable Mode of Action — Modern Pharmacological Perspective:

The rhizome and root of *Valeriana wallichii* contain sesquiterpenoids (chiefly valerenic acid, acetoxy-valerenic acid and valeranone), iridoid esters known as valepotriates (valtrate, isovaltrate, didrovaltrate), lignans, flavonoids and a volatile oil containing maaliol and patchouli alcohol. Several distinct and complementary mechanisms have been demonstrated.

GABA_A receptor modulation. Valerenic acid is a positive allosteric modulator of the GABA_A receptor, but — and this is the pharmacologically decisive point — it acts at a site distinct from that of the benzodiazepines. Two-microelectrode voltage-clamp studies on receptors expressed in *Xenopus* oocytes have shown that stimulation of chloride current by valerenic acid occurs only in channels incorporating β_2 or β_3 subunits; substitution with β_1 drastically reduces sensitivity. Valerenic acid shifts the GABA concentration–effect curve towards lower GABA concentrations and, at concentrations of 30 μ M and above, gates substantial current directly. The effect is independent of the γ_2 subunit and is insensitive to flumazenil, and the binding site is believed to be the loreclezole pocket on the β subunit.²² In vivo work in mice carrying a point mutation rendering the β_3 subunit insensitive has confirmed that β_3 -containing GABA_A receptors mediate the anxiolytic action of valerenic acid.²³ This subunit selectivity offers a coherent explanation for the clinical profile observed in the present case — sedation and anxiolysis without the next-morning impairment and without the rebound insomnia that follows γ_2 /benzodiazepine-site agonists such as zolpidem, which the same patient had previously experienced.

Inhibition of GABA catabolism and reuptake. Valepotriates and valerenic acid have been reported to inhibit GABA transaminase, the enzyme responsible for the degradation of GABA, and to reduce neuronal GABA reuptake. The net effect is increased availability and prolonged residence of GABA in the synaptic cleft, reinforcing inhibitory tone in sleep-promoting circuits.²⁴

Adenosinergic modulation. Adenosine accumulating during waking is the principal humoral substrate of sleep pressure. Valerenic acid and the lignan hydroxypinoresinol act as allosteric modulators at the adenosine A₁ receptor, amplifying the effect of endogenous adenosine; in vitro evidence indicates that A₁ activation and GABA_A modulation contribute independently to the sleep-inducing capacity of *Valeriana*.²⁵ This mechanism is of particular relevance in the present patient, whose heavy tea consumption represented chronic competitive antagonism at the same receptor.

Effects on sleep architecture and monoamines. In Sprague-Dawley rats, aqueous root extract of *V. wallichii* at 200 and 300 mg/kg significantly increased total sleep time and the duration of non-rapid

eye movement sleep and decreased sleep latency and wakefulness in a dose-dependent manner, with a significant increase in electroencephalographic delta activity during NREM sleep — an objective index of sleep depth. The same study documented reductions in noradrenaline and serotonin in the frontal cortex and brainstem.²⁶ Suppression of ascending arousal-system monoamines is exactly the physiological correlate of the classical concept of the Indriyas withdrawing from their Vishayas. A partial agonist action at 5-HT_{5A} receptors within the suprachiasmatic nucleus has additionally been proposed as a mechanism of circadian re-entrainment, which is pertinent in a patient whose disorder began with shift work.

4.4 Comparison with Existing Literature:

The magnitude and pattern of improvement observed here align closely with the only substantial published clinical trial of this drug in this indication. Toolika et al. administered Tagara Churna 4 g three times daily with milk for one month to fifteen patients with primary insomnia and reported improvements of 76.00% in initiation of sleep, 55.17% in duration of sleep, 69.58% in disturbed sleep and 73.95% in disturbance of routine work, all at $P < 0.001$, with Tagara outperforming Jatamansi across all four domains.¹⁰ In the present case, improvement of 66.67% in each of the three cardinal sleep domains and of 68.18% in the composite score falls within the same range, obtained at a lower daily dose of 6 g. The same group's experimental study, using pentobarbitone-induced sleeping time and actophotometer scores, demonstrated significant hypnotic and locomotor-depressant activity for Tagara relative to control, supporting a pharmacological rather than purely placebo basis for the clinical findings.²⁷ A systematic review and meta-analysis of Valeriana preparations for sleep concluded that the evidence, while methodologically heterogeneous, is consistent with improvement in subjective sleep quality and with an adverse-event profile no different from placebo.²⁸

What the present report adds is the concurrent application of three validated instruments alongside classical symptom grading, a documented four-week wash-out from a benzodiazepine-receptor agonist, deliberate exclusion of all confounding Ayurvedic procedures, and a drug-free follow-up demonstrating absence of rebound. These are the features that distinguish a publishable case report from an anecdote, and they are the features that postgraduate scholars should build into their own documentation from the outset.

4.5 Role of Nidana Parivarjana:

Charaka places Nidana Parivarjana ahead of Aushadha in the management of every vyadhi, and Anidra is a disorder in which this sequence is not merely conventional but mechanistically necessary. Continued Ratri Jagarana, evening caffeine and unmodified psychological stress would have regenerated the Samprapti as rapidly as the drug dismantled it. The sleep-hygiene component of this case — fixed sleep and wake times, screen abstinence for one hour before bed, caffeine restriction to two cups before 16:00 hours, the bed reserved for sleep, and Bhramari Pranayama with Shavasana

before retiring — is the operational equivalent of Nidana Parivarjana and overlaps substantially with the stimulus-control and sleep-restriction elements of cognitive behavioural therapy for insomnia. It must be acknowledged, and is acknowledged below, that this same overlap constitutes a methodological confounder.

4.6 Strengths and Limitations:

The principal strengths of this report are the clean single-drug design free of Panchakarma or polyherbal confounding, the documented wash-out period, the use of validated instruments in parallel with classical grading, the inclusion of laboratory safety data, and the drug-free follow-up period.

The limitations are substantial and must be stated plainly. First, this is a single uncontrolled observation; it can establish plausibility, tolerability and a hypothesis, but it can never establish efficacy. Second, all primary outcomes were subjective; polysomnography and actigraphy, which would have provided objective corroboration of sleep-onset latency and sleep architecture, were not employed. Third, placebo response, expectancy and the Hawthorne effect of frequent clinical contact are unquantified and, in a disorder as expectancy-sensitive as insomnia, potentially large. Fourth, sleep-hygiene counselling was delivered concurrently, so the observed benefit cannot be attributed to Tagara alone — although it is relevant that the patient had already been attempting several of these measures without success before enrolment. Fifth, the follow-up of 30 days is short and permits no comment on durability beyond that window. Sixth, no phytochemical assay such as HPTLC quantification of valerenic acid was performed on the specific batch used, so batch potency is undocumented.

5. CONCLUSION:

In this 38-year-old male with eight months of chronic Anidra corresponding to chronic insomnia disorder, Tagara (*Valeriana wallichii* DC.) Churna 3 g twice daily with lukewarm cow's milk for 30 days, combined with Nidana Parivarjana, produced a marked and clinically meaningful improvement across every domain assessed. Sleep-onset latency fell by 82.86%, total sleep time increased by 81.58%, sleep efficiency rose from 48% to 86%, and the composite Ayurvedic symptom score improved by 68.18%. All three validated instruments crossed from the pathological into the normal or subthreshold band. The improvement was sustained at 30 days after cessation of the drug without rebound insomnia, and no adverse event, clinical or biochemical, was recorded.

The observation is coherent both with the classical attribution of Nidrajanana Prabhava to Tagara and with the demonstrated β_2/β_3 -selective GABAergic and adenosinergic pharmacology of its constituents. Tagara therefore merits consideration as a well-tolerated option in chronic Anidra, particularly for patients who wish to avoid or discontinue benzodiazepine-receptor agonists. A single case, however, cannot substitute for controlled evidence; the appropriate next step is an adequately powered, randomised, placebo-controlled trial employing polysomnographic or actigraphic endpoints,

standardised and assayed drug material, and follow-up sufficient to establish durability.

LEARNING POINTS FOR POSTGRADUATE SCHOLARS:

1. Anidra is a Manasika-Sharirika vyadhi. Nidana Parivarjana precedes Aushadha in Charaka's sequence, and in insomnia this is not a formality — an unmodified nidana will regenerate the Samprapti faster than any drug can dismantle it.
2. Always exclude secondary insomnia before labelling a case as Kevala Anidra. Anaemia, thyroid dysfunction, dysglycaemia, depression, obstructive sleep apnoea, restless legs, chronic pain and drug-induced insomnia must be ruled out clinically and, where indicated, biochemically. A case report without this exclusion will not survive peer review.
3. Document a wash-out period whenever the patient has been on hypnotics. Rebound insomnia following withdrawal of a benzodiazepine-receptor agonist can be mistaken either for baseline severity or for treatment failure.
4. Use validated instruments alongside classical grading, never instead of it. PSQI, ISI and AIS make the work comparable with global literature; the Ayurvedic grading preserves the classical clinical logic. Reporting both is what makes an Ayurvedic case report indexable.
5. Explain the mode of action at both levels. Stopping at “Tagara is Vatashamaka” is insufficient at postgraduate level; state the Rasapanchaka correlation and the receptor pharmacology side by side, and be able to reconcile them.
6. Treat Anupana as part of the prescription, not as a convenience. Godugdha is itself Nidrajanana and modifies the Ushna-Ruksha character of Tagara; changing the Anupana changes the therapy.
7. Keep the intervention clean. Adding Shirodhara, Abhyanga or a second formulation may help the patient but makes attribution impossible in a single-case design. Decide at the outset whether the objective is maximal clinical benefit or interpretable documentation.
8. Report negatives explicitly — absence of adverse events, stable laboratory values, absence of rebound. Negative safety data are among the most useful contributions a case report can make.
9. State limitations without hedging. A case report establishes plausibility and a safety signal; it never establishes efficacy. Reviewers penalise overclaiming far more heavily than they penalise a modest, well-bounded conclusion.
10. Follow the CARE guidelines, obtain written informed consent for publication, and secure Institutional Ethics Committee clearance or documented exemption before submission.

DECLARATIONS:

Patient consent: Written informed consent was obtained from the patient for treatment and for publication of this case report and any accompanying data. The patient's identity has been fully anonymised.

Ethical approval: Institutional Ethics Committee approval / exemption reference [].

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Conflict of interest: None declared.

Authors' contributions: [Author 1] managed the case and drafted the manuscript; [Author 2] conceptualised and supervised the work and critically revised the manuscript; [Author 3] contributed to the literature review and pharmacological discussion. All authors read and approved the final manuscript.

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