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Ayurvedic management of narcolepsy secondary to post-viral autoimmune encephalitis - a case report

Abstract

Narcolepsy is a chronic neurological condition characterized by disrupted sleep-wake cycle, excessive daytime sleepiness, sudden sleep attacks, fatigue, and sudden loss of muscle tone leads to collapse/falling. Narcolepsy is classified into two types, Narcolepsy type 1 (Na-1) corresponds to narcolepsy with cataplexy and Narcolepsy type 2 (Na-2) corresponds to narcolepsy without cataplexy. There is no current cure or disease modulating therapies for treating narcolepsy. The present case report deals with the *Ayurvedic* management of a 12-years old male patient, who came with the diagnosis of narcolepsy secondary to post-viral autoimmune encephalitis. *Ayurvedic* diagnosis of *Tandra* has been made for the present case and treated accordingly. *Ayurvedic* medicines including *Smritisagara Ras, Vachadi Gutika, Rasaraja Ras,* and *Saraswatarishta* were prescribed and the Ullanlinna Narcolepsy Scale (UNS) has been used for assessment. Total two assessments have been carried out, one at baseline/before starting *Ayurvedic* treatment and the second assessment after completion of 6 months of *Ayurvedic* treatment. *Ayurvedic* intervention has provided clinically meaningful improvement in excessive daytime sleepiness, fatigue, cataplexy-like falling episodes and in memory impairment. Clinical trials with large sample sizes and extended follow-up periods are required to substantiate the current case report findings.

Keywords: autoimmune, *ayurveda*, cataplexy, encephalitis, sleep, *tandra*

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Introduction

Narcolepsy is a chronic neurological disorder that disrupts the normal sleep-wake cycle. It primarily affects the regulation of REM (rapid eye movement) and NREM (non-rapid eye movement) sleep. Narcolepsy is characterized by episodic excessive daytime sleepiness (EDS) often with sudden 'sleep attacks,' night time wakefulness, sleeping difficulties, abnormally rapid eye movements, mild muscular weakness, cataplexy (sudden loss of muscle control triggered by strong emotions, ranging from mild weakness in the face to complete collapse), all of which lead to sleep paralysis or body collapse. Narcolepsy is a form of hypersomnia, but its exact cause is unknown though it can be linked to traumatic brain injury (TBI), hypothalamic damage, loss of hypocretin receptors, and other neurological and metabolic disorders. Narcolepsy is categorized into three types, with cataplexy, without cataplexy and secondary.[1] Secondary narcolepsy can be caused by brain TBI, infections (streptococcal infections, dengue virus infection, and H1N1 epidemics), stroke, demyelination, often by an autoimmune attack on the wakefulness-promoting neurons in the lateral hypothalamus.[2]

The management of secondary or symptomatic narcolepsy differs from other types of narcolepsy. According to the International classification of sleep disorders, 3rd edition (ICSD-3), narcolepsy is classified into two types, Narcolepsy type 1 (Na-1) corresponds to narcolepsy with cataplexy and Narcolepsy type 2 (Na-2) corresponds to narcolepsy without cataplexy. Narcolepsy might be secondary to an underlying structural brain injury, which can manifest as either Na-1 or Na-2 depending on the presence of cataplexy and CSF (cerebrospinal fluid) hypocretin levels.[3] Sleep disorders (insomnia, hypersomnia, EDS, narcolepsy, and REM sleep behaviour disorder) are common in about two-thirds of patients with autoimmune encephalitis.[4]

An infectious-like prodrome can be found (sore throat, rhinorrhoea, and low grade fevers) in patients with autoimmune encephalitis.[5,6] Only symptomatic treatments are currently available for narcolepsy, and no disease-modifying cure is available to date. Chronic pharmacological treatments are frequently associated with side effects and may not sufficiently impact the overall disease burden. These issues often lead to significant challenges in patient management and outcomes.[7] The present case report described a male patient diagnosed with narcolepsy with cataplexy (Na-1?) secondary to post-viral autoimmune encephalitis who opted for *Ayurvedic* treatment for better and sustained relief after the failure of conventional treatment.

Case description

A 12-year-old male patient came to our care on 30th Mar 2025 accompanied by his parents with the diagnosis of hypersomnolence with bilateral frontal epileptiform discharges? Autoimmune encephalitis? He has been suffering with excessive sleepiness throughout the day, lack of interest in daily activities due to severe fatigue, decreased clarity of speech, imbalance in walking, dizziness, frequent sudden falls and abnormal movements, objects tend to fall from hands due to lack of grip, feeling lethargic, and decline in academic functioning. The symptoms have manifested in the first week of Mar 2025 insidiously, followed by a viral flu-like illness. As per the advice of the neurologist in Mar 2025, the patient has undergone several investigations, including an MRI (magnetic resonance imaging) brain, sleep video EEG (electroencephalogram), and NCS (nerve conduction study) (Table 1). The patient was prescribed with Fluoxetine and Aripiprazole. The condition was provisionally diagnosed as autoimmune encephalitis. The patient was admitted for six days (13th to 18th Mar 2025) and received management for altered behavior, fall of things from hands, excessive sleepiness and giddiness. The condition has been gradually progressive, and the patient has not received satisfactory relief with conventional treatment. The parents of the patient opted for *Ayurvedic* treatment and arrived to our healthcare facility on 30th Mar 2025.

The patient is a 7th standard student, has been suffering with EDS, and sudden uncontrollable sleep attacks while sitting in the classroom, reading, watching television and throughout the day with no history of head injury. The sleep attacks are associated with the dropping of

the head or sudden falls or dropping objects from the hands and loss of consciousness. Past history has revealed a viral or flu-like illness with high grade fever, sore throat, and severe body pain a week before the manifestation of EDS. The patient was immunized up to age with an uneventful birth history, normal development history and negative family history for any psychiatric or neurological illness. Patient was not allergic to any drug or a food item. General examination has revealed normal heart rate, blood pressure, respiratory rate, body temperature, $Sp0_2$, and other vital signs. Mucous membranes were moist and pink. No conjunctival pallor, cyanosis found, edema and enlargement of lymph nodes seen. Heart sounds (S_1 & S_2) were normal and without murmurs. Normal bilateral vesicular breath sounds heard. Abdomen was soft, non-tender, and without any organ enlargement on palpation. Body weight was 60.6 kg and height 162 cm (comes under overweight category with BMI 23.1 kg/m^2). EEG, brain MRI, NCS and polysomnography done on 30th Mar & 10th Apr 2025 were normal (Table 1). Various hematological and biochemical investigation reports (15th & 17th Apr 2025) were within normal limits. CSF analysis for glucose, cell count, culture and sensitivity was normal. A negative autoimmune encephalitis panel using CSF cells has been found (which doesn't rule out the diagnosis of antibody-negative autoimmune encephalitis). Signs of generalized epilepsy, a few generalized epileptiform discharges with a frontal dominance were seen in sleep and awake EEG. Terminal zones of myelination were found in MRI brain.

Table 1 Investigation reports

Date	Name of the investigation	Findings
11th Mar 2025	MRI (magnetic resonance imaging) brain	Periventricular FLAIR (fluid-attenuated inversion recovery) hyperintensity
13th Mar 2025	Video EEG (electroencephalogram)	Normal awake and sleep EEG record with positive Hyperventilation and photic stimulation
30th Mar 2025	NCS (nerve conduction study), Polysomnography, EEG and MRI brain	Normal
10th Apr 2025	Polysomnography, MRI brain and EEG	Normal
15th Apr 2025	Complete blood count (CBC), renal and liver function tests, serum electrolytes, Vitamin B12, ESR (erythrocyte sedimentation rate), random blood sugar	WNL (with in normal limits)
	CSF (cerebrospinal fluid) analysis	CSF Glucose – 53 mg/dl Culture report – No growth after 48 hours of incubation
	Awake and sleep EEG	Signs of generalized epilepsy, a few generalized epileptiform discharges with a frontal dominance – 'likely incidental finding.'
	MRI brain	Terminal zones of myelination
17th Apr 2025	Autoimmune encephalitis panel by using CSF cell based assay and immunofluorescence assay (IFA)	NMDA (Anti-glutamate receptor against NR1 subunit) –Negative AMPA (Anti-glutamate) – Glu R1 – Negative AMPA (Anti-glutamate) – Glu R2 – Negative GABA-B receptor antibody – Negative LGI-1 antibody (VGKC type) – Negative CASPR2 antibody (VGKC type) - Negative

The patient was alert, fully conscious, and oriented at the time of the first interview (30th Mar 2025). A Glasgow Coma Scale (GCS) score was E4V5M6 (indicates spontaneous eye-opening, oriented verbal responses, and an ability to follow motor commands). Examination of cranial nerves has revealed normal findings with no meningeal signs and nystagmus. Sensory examination has revealed normal perception of pain, touch, temperature, and vibration. Equal movements of all four limbs with bilateral 2+ deep tendon reflexes were found on motor system examination. A bilateral plantar flexor response was seen. Normal findings were seen on a neurological examination of the cerebellar system, extrapyramidal system, gait, skull, and spine. Fine tremors were noticed in both upper limbs. On Mental status examination (MSE), the patient looks euthymic and maintained the eye contact. Speech was coherent and but reduced. Thought form, content and course were normal. No psychotic symptoms were found. Orientation to time, place and person was normal. Immediate, recent and remote memory was normal. MSE findings have revealed lack of interest in activities and episodic irritability/aggression. Patient had sleep attacks with sudden onset at the time of interview. Parents used to assist the patient while walking as the patient had a tendency to fall suddenly and frequently. Systemic, neurological and MSE examination have not found any significant signs pointing to a specific disease (except narcolepsy and cataplexy-like episodes).

Diagnosis, assessment and intervention

According to the ICSD-3 diagnostic criteria, Na-1 is defined by EDS and either (a) cataplexy plus a positive MSLT (Multiple Sleep Latency Test), or (b) hypocretin deficiency (CSF hypocretin-1 level < 1/3 of normal or ≤ 110 pg/mL). Na-2 is defined by EDS and a positive MSLT, but without cataplexy or hypocretin deficiency. A positive Multiple Sleep Latency Test (MSLT) requires a mean sleep latency of ≤ 8 minutes and 2 or more SOREMPs (Sleep onset REM periods), with a SOREMP on the preceding nocturnal polysomnogram counting towards this total. The diagnosis of Na-2 remains challenging

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because it relies heavily on clinical findings and the MSLT, which has limitations, including low test-retest reliability and the false-positive results. There is no definitive biomarker for Na-2, unlike Na-1, which is often associated with a clear hypocretin deficiency.[3] The hypothalamic injury (caused by stroke, tumor, TBI, sarcoidosis, demyelination and inflammatory conditions) often leads to low CSF hypocretin levels. These same pathological processes can cause EDS alone, hence a positive MSLT is required to diagnose secondary Na-2.[3]

In present case, the patient has not undergone a MSLT and CSF hypocretin level testing, hence there is a diagnostic dilemma for establishing a definitive diagnosis of narcolepsy and distinguishing between its two types (Na-1 & Na-2). In present case, the patient's presentation of "loss of consciousness" during sudden falling episodes that appear "cataplexy-like," combined with "generalized epileptiform discharges with frontal dominance" on EEG (Table 1), makes the diagnosis of pure cataplexy highly ambiguous, as preserved consciousness is its defining characteristic. Ambiguous episodes of sudden loss of muscle tone and collapse (cataplexy-like episodes) that present with positive EEG generalized epileptiform discharges as in present case, may also indicate absence seizures or atonic seizures rather than pure cataplexy. Given the history of viral infection, the ambiguous clinical symptoms (cataplexy/absence or atonic seizures), and no evidence of CNS inflammation, the diagnosis of narcolepsy secondary to post-viral autoimmune encephalitis can't be ruled out even with a negative standard autoimmune encephalitis panel. The accurate diagnosis of complex conditions like primary vs. secondary narcolepsy, cataplexy vs. absence or atonic seizures, and positive vs. negative autoimmune encephalitis panels relies on a multifaceted approach (a combination of clinical history, physical examination, neurological tests, laboratory and radiological findings) due to significant clinical overlap and the lack of a single definitive test as seen in the present case. The absence of specific antibodies does not exclude the diagnosis of autoimmune encephalitis.[8] *Ayurvedic* diagnosis of *Tandra* (drowsiness/hypersomnia associated with fatigue) has been made based on the clinical features[9] and the patient treated accordingly.

Ullanlinna Narcolepsy Scale (UNS) has been used as assessment criteria.[10] The UNS is an 11-item scale designed to evaluate a wide variety of narcolepsy-related symptoms, including EDS and cataplexy. The score of each item ranges from '0' to '4,' to indicate how frequently the patient experiences narcolepsy symptoms. Total scores can range from 0 to 44 with higher scores denoting greater narcoleptic tendencies.[10] A total of two assessments have been taken, baseline (30.03.2025) and after completion of six months of treatment (30.09.2025), either telephonically or in-person. Fluoxetine and Aripiprazole were stopped before starting *Ayurvedic* medicines. *Smriti Sagara Ras* and *Vachadi Gutika* were prescribed initially (from 30.03.2025 to 27.04.2025), followed by *Rasaraja Ras* and *Saraswatarishta* (from 28.04.2025 to 30.09.2025).

Discussion

Autoimmune encephalitis can be caused by Herepes simplex virus, Japanese encephalitis virus, West Nile virus, SARS-CoV-2, and Mycoplasma pneumoniae. Seropositivity for specific autoantibodies are found more frequently in patients with Na-1 shortly after disease onset and it is rarely seen in Na-2 or in those with more chronic disease.[11] Autoimmune encephalitis symptoms can include seizures, cognitive impairment, neuropsychiatric and autonomic dysfunction, headache, visual disturbances, and/or movement disorders. Since a seronegative autoimmune syndrome is possible, relying on a constellation of clinical, imaging, and laboratory findings and importantly after excluding the other potential causes.[5,6] The symptoms of autoimmune encephalitis are diverse often mimicking primary psychiatric illnesses or neurodegenerative diseases. A much clearer understanding of the diverse clinical presentations of autoimmune encephalitis has been growing. Sleep tests including MSLT and polysomnography could be used for diagnosis.[4] The onset of autoimmune encephalitis is usually subacute with rapid progression.[5,6]

The combination of vitiated *Vata* and *Kapha Doshas* with the *Tamas* (quality of inertia) can cause excessive drowsiness (*Tandra*). This condition can be described as a psycho-somatic disorder with symptoms including mental sluggishness, heavy senses, and a desire for sleep. According to Acharya *Sushruta*, *Tandra* is characterized by a feeling of heaviness in the senses, excessive yawning, fatigue, inability to wake up, and activities resembles a seeping person. Management of *Tandra* includes, *Kapha*-pacifying drugs, physical exercise, bloodletting (*Rakta Mokshana*), massage with herbal powders (*Udwartana*), purificatory procedures (*Shodhana*), and usage of bitter (*Tikta*) and pungent (*Katu*) food items.[9]

Smritisagara Ras is a herbo-mineral formulation, commonly used in disorders of consciousness, memory impairment and in epilepsy. *Smritisagara Ras* is prepared with purified and/or incinerated *Parada* (mercury), *Gandhaka* (sulphur), *Haratala* (orpiment), *Manashila* (realgar), *Tamra Bhasma* (copper), and *Swarnamakshika Bhasma* (incinerated copper pyrite), triturated in the herbal decoctions of *Vacha* (Acorus calamus), *Brahmi Swarasa* (juice of Bacopa monnieri) and *Jyotishmati Beeja Taila* (seed oil of Celastrus paniculatus) and then formed into pills/tablets.[12] *Smritisagara Ras* is given with the dose of 125mg, twice a day, after meals.

Vachadi Gutika is mentioned in *Visha Vaidhya Jyotsnika* in the chapter *Sarvavisha Chikitsa* (treatment of poisoning). It is an herbal compound having the drugs like *Vacha*, *Hingu* (Ferula northax), *Lashuna* (Allium sativum), *Maricha* (Piper nigrum), *Pippali* (Piper longum), *Aardraka* (Zingiber officinale) and *Kaanjika* (fermented rice and grain-based liquid) is the *Bhavana Dravya* (liquid substances used for wet grinding or to triturate). The ingredients of *Vachadi Gutika* have *Vata* and *Kapha* pacifying properties and *Sanjna Prabodhana* (drugs used to restore consciousness) action (especially *Vacha* and *Hingu*),[13] hence prescribed in the present case with the dose of 250mg, twice a day, after meals.

Rasaraja Ras is composed of purified, incinerated metals/minerals, including mercury (*Parada*), mica (*Abhraka*), gold (*Swarna*), silver (*Raupya*), iron (*Loha*), and tin (*Vanga*); herbs such as *Ashwagandha*, *Lavanga*, *Jatiphala*, and *Ksheea Kakoli* triturated in the decoctions/juices of *Kumari* and *Kakamachi*; and then formed into pills/tablets. *Rasaraja Ras* is indicated in *Daruna Vata Vyadhi* (severe neurological illness) and its ingredients have proven antidepressants, anxiolytic, and rejuvenating properties.[14] *Saraswatarishta* is a multi-ingredient herbo-mineral formulation consists of 18 herbs including *Medhya Rasayanas* (brain tonics) which are known to improve memory and cognitive functions. Ingredients of *Saraswatarishta* are *Brahmi, Shatavari* (Asparagus racemosus), *Vidarikanda* (Pueraria tuberosa), *Haritaki* (Terminalia chebula), *Aardraka*, *Mishreya* (Anethum sowa), *Trivrit* (Operculina ipomoea), *Pippali, Lavanga* (Syzygium aromaticum), *Vacha, Kushta* (Saussurea lappa), *Ashwagandha, Vibhitaki* (Terminalia belirica), *Guduchi* (Tinospora cordifolia), *Ela* (Elettaria cardamomum), *Vidanga* (Embelia ribes), *Dalchini* (Cinnamomum zeylanicum), and *Swarna* (pure gold). *Saraswatarishta* is useful in the management of anxiety, fatigue, insomnia, memory impairment, low grasping power, slurred speech etc.[15] *Rasaraja Ras* is given with

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the dose of 100mg, twice a day, after meals along with 20 ml of *Saraswatarishta*.

The patient experienced no negative reactions or side effects from the medication he was prescribed. Baseline score/first assessment score (dated on 30th Mar 2025) on UNS was 23 out of 44 and the second assessment score (dated on 30th Sep 2025, after 6 months' completion of treatment) was 9 out of 44 which indicates 60.87% of relief on the total score of UNS (Figure 1). Good improvement is seen in various items of UNS such as 'falling down suddenly (item-4),' 'falling asleep quickly (item-5),' 'naps during the day (item-6),' 'fall asleep while reading (item-7),' and 'falling asleep during other unusual times (item-11) with 66.7%, 75%, 75%, 66.7% and 50% of relief respectively on UNS. Patient's mother has informed wonderful improvement in EDS, naps during daytime (both in frequency and duration), cataplexy-like episodes, and fatigue. Improvement in daily activities, active participation in sports and games, academic performance and memory were reported by the patient and his parents. *Ayurvedic* treatment found beneficial in the management of narcolepsy in present case. The patient had missed further appointments, has not been seen after 30th Sep 2025 and 'lost to follow-up' due to unknown reasons. The lack of CSF hypocretin levels and a MSLT are major limitations in the present case report.

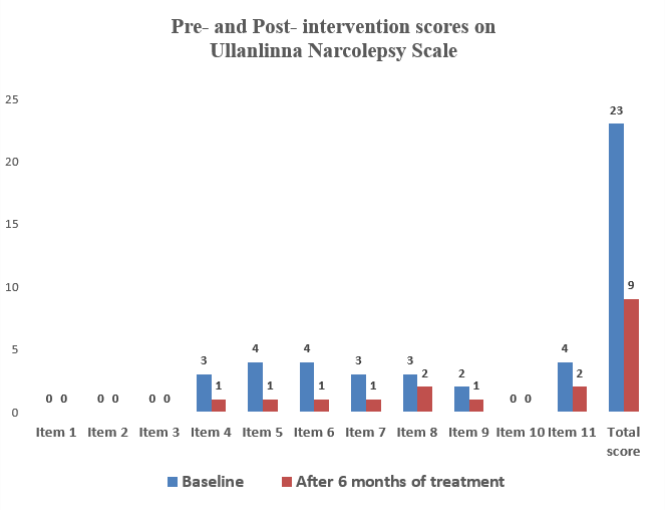

Figure 1 Assessment on Ullanlinna narcolepsy scale.

Conclusion

Narcolepsy is a chronic neurological disorder that disrupts the sleep-wake cycle and causes excessive daytime sleepiness and sleep attacks. There is no definitive, disease-modifying cure currently available for narcolepsy. An *Ayurvedic* diagnosis of '*Tandra'* has been made and treated accordingly in the present case having the contemporary diagnosis of 'Narcolepsy secondary to post-viral autoimmune encephalitis.' *Ayurvedic* medicines such as *Smritisagara Ras, Vachadi Gutika, Rasaraja Ras,* and *Saraswatarishta* have provided satisfactory relief on the Ullanlinna Narcolepsy Scale (UNS) in managing excessive daytime sleepiness, cataplexy-like falling episodes, memory impairment, and fatigue in present case. Clinically meaningful improvement (60.87% of relief) was found in the total score of UNS after receiving 6 months of *Ayurvedic* intervention. As the findings of current case report can't be generalized, studies with large sample sizes and extended follow-up periods are required to substantiate.

Limitations of the study

As the patient has not undergone an MSLT and CSF hypocretin level testing, there is a diagnostic dilemma for establishing a definitive diagnosis of narcolepsy and distinguishing between its two types (Na-1 & Na-2) in the present case. No questionnaire or scale related to *Tandra* has been used for the evaluation of the efficacy of the *Ayurvedic* treatment protocol. Long-term follow-up observation was not possible as the patient was 'lost to follow-up.' The findings of the present case study couldn't be generalized.

Declaration of patient consent

The patient's mother has given consent for reporting the clinical information in any journal. Though anonymity cannot be guaranteed due efforts will be made to conceal the patient's identity. The patient and his parent understands that his name and initials will not be published.

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Conflicts of interest

The author declares there is no conflict of interest.

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