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Late-Onset Narcolepsy Type 1 With Inconclusive Sleep Studies: A Case Report

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Patient: Male, 60-year-old
Final Diagnosis: Narcolepsy type 1
Symptoms: Cataplexy-like episodes • excessive daytime sleepiness • sleep paralysis-like episodes
Clinical Procedure: —
Specialty: Psychiatry

Objective: Unusual clinical course

Background: Narcolepsy type 1 (NT1) is characterized by excessive daytime sleepiness, cataplexy, and cerebrospinal fluid (CSF) hypocretin-1 deficiency. Although typically occurring in younger individuals, late-onset NT1 can present with diagnostic challenges, particularly when symptoms overlap with psychiatric, neurological, or sleep-related disorders.

Case Report: We report a case of a 60-year-old man with recurrent emotion-triggered muscle weakness and progressive excessive daytime sleepiness. The patient had been treated for 6 years under a diagnosis of dissociative (conversion) disorder. The weakness episodes were clinically compatible with cataplexy, as they were emotion-triggered and occurred with preserved awareness. Two polysomnography and multiple sleep latency testing evaluations demonstrated pathological sleepiness but did not meet the conventional sleep-onset rapid eye movement period (SOREMP) criterion for narcolepsy. Interpretation was complicated by moderate-to-severe obstructive sleep apnea, fragmented sleep, previous psychotropic medication exposure, and cerebral ischemic changes. CSF hypocretin-1 concentration was markedly reduced (12.08 pg/mL), confirming the diagnosis of NT1. Treatment with clomipramine and pitolisant improved daytime sleepiness and cataplexy-like episodes, with the Epworth Sleepiness Scale score decreasing from 24 at admission to 9 after 4 months of follow-up.

Conclusions: Late-onset NT1 should be considered in older adults presenting with progressive excessive daytime sleepiness and emotion-triggered weakness, even when conventional sleep studies are inconclusive. CSF hypocretin-1 measurement can provide important diagnostic evidence, while atypical neurological manifestations should be evaluated separately for alternative or coexisting disorders.

Keywords: Cataplexy • Narcolepsy

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/955007

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Introduction

Narcolepsy is a chronic neurological sleep disorder characterized by impaired sleep–wake regulation, excessive daytime sleepiness, sleep attacks, and disturbed nocturnal sleep. Narcolepsy type 1 is further characterized by cataplexy and reduced cerebrospinal fluid hypocretin-1 concentrations, reflecting loss or dysfunction of hypocretin-producing neurons in the hypothalamus [1]. The disorder most commonly begins in childhood, adolescence, or early adulthood, whereas onset in later life is uncommon [2,3]. Diagnosis may be delayed when excessive daytime sleepiness is accompanied by atypical neurological, psychiatric, or sleep-related manifestations [4,5].

Polysomnography and multiple sleep latency testing are important in the diagnosis of narcolepsy, but their findings can be affected by sleep comorbidities, medication use, and poor sleep quality [6]. We report a case of a 60-year-old man with recurrent emotion-triggered loss of muscle tone who presented with progressively worsening excessive daytime sleepiness in later life. Although 2 rounds of sleep testing did not meet the typical diagnostic criteria for narcolepsy, cerebrospinal fluid hypocretin-1 was markedly reduced to 12.08 pg/mL, establishing the diagnosis of narcolepsy type 1 [7]. Unlike previous reports focusing mainly on older age at onset, this case highlights the diagnostic challenges of late-onset narcolepsy type 1 in the presence of psychiatric misclassification, inconclusive sleep studies, and atypical neurological manifestations.

Case Report

The patient was a 60-year-old Chinese man with a history of hypertension and coronary artery disease. There was no family history of narcolepsy or related symptoms, and HLA-DQB1*06:02 testing was not performed. No clear preceding infection was identified. He received COVID-19 vaccination after symptom onset and reported no other recent vaccinations. In October 2025, he was admitted to the sleep medicine department because of progressively worsening excessive daytime sleepiness and recurrent emotion-triggered episodes of muscle weakness. The weakness episodes had begun 6 years earlier and had become more frequent during the 5 days preceding admission.

History and Symptomatology

The patient first developed episodic limb weakness in February 2019, at approximately 54 years of age. The episodes initially occurred while he was talking with other people and were characterized by sudden weakness of the left leg, difficulty walking, and occasional slurred speech. They occurred 3 to 4 times per day, lasted several minutes, and became more frequent during emotional arousal. Awareness was generally preserved, and he was able to recall the events afterwards (**Figure 1**).

Over time, the episodes became more frequent and varied in presentation. He developed generalized weakness, tremor,

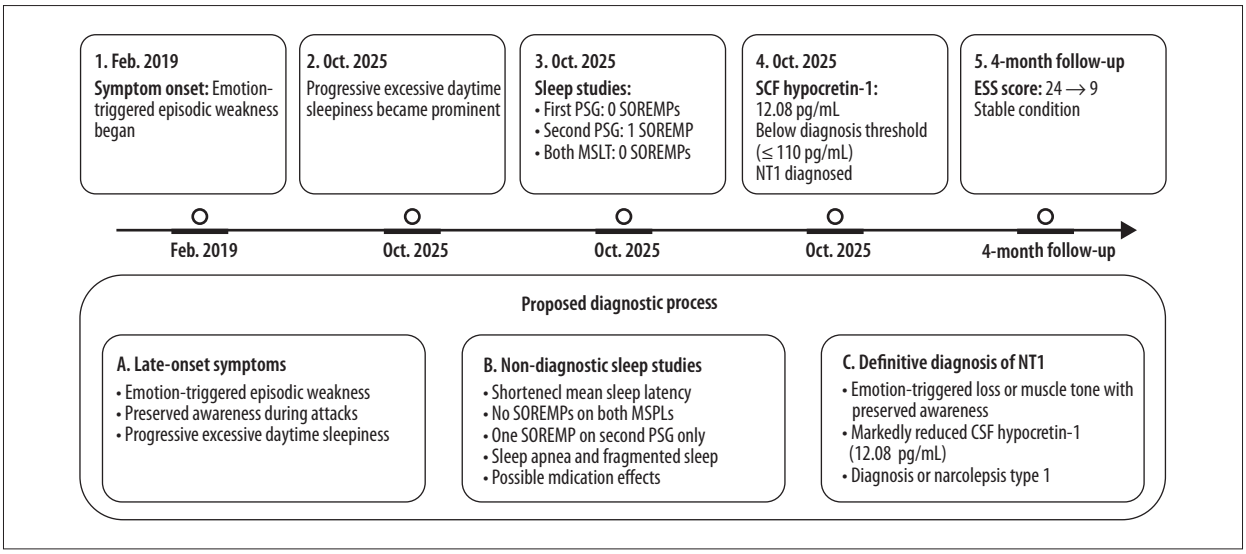


Figure 1. Evidence-informed case timeline of symptom development, examinations, and results in narcolepsy type 1 (2019-2025). The figure illustrates the chronological clinical course, diagnostic examinations, and the proposed diagnostic process. The upper panel shows symptom development and key examinations over time. The lower panel summarizes the proposed diagnostic pathway: late-onset symptoms, non-diagnostic sleep studies, and definitive diagnosis of NT1. Arrows and timeline markers indicate the sequence of clinical events and diagnostic evaluations. This figure is based on a single case and does not represent a validated diagnostic model. CSF, cerebrospinal fluid; ESS, Epworth Sleepiness Scale; MSLT, Multiple Sleep Latency Test; NT1, narcolepsy type 1; PSG, polysomnography; SOREMP, sleep-onset rapid eye movement period.

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Table 1. PSG and MSLT results.

PSG findings		
Variable	**First PSG**	**Second PSG**
Sleep latency (min)	11.0	7.0
REM latency (min)	220.0	1.5
N1 (% TST)	12.5	57.9
N2 (% TST)	70.0	17.7
N3 (% TST)	0.0	0.2
REM (% of TST)	17.6	24.2
Total sleep time (TST)(min)	216.5	279.0
Sleep efficiency (%)	45.4	54.8
Apnea–hypopnea index total (/h)	17.7	38.5
PLMS index (/h)	24.9	0.0
Arousal index (/h)	15.0	17.6
Number of SOREMPs	0.0	1.0

MSLT findings			
MSLT session	**Mean sleep latency (min)**	**Mean REM latency (min)**	**Number of SOREMPs**
First MSLT	6.8	-	0
Second MSLT	1.5	-	0
EEG/ECG	No abnormal findings		

difficulty holding objects, and occasional falls. Social interaction and emotional distress often triggered the episodes. He also reported vivid dreams, nightmares, and sleep talking.

During the following 6 years, he was repeatedly evaluated in psychiatric and neurological settings. He was initially suspected of having a somatic symptom disorder and was later diagnosed with dissociative (conversion) disorder. He received treatment with several antidepressants and antipsychotics under this diagnosis, which produced partial or temporary improvement. However, the episodic weakness recurred after treatment discontinuation. In October 2025, the weakness episodes became more frequent, and progressively worsening excessive daytime sleepiness became prominent, leading to his admission to the sleep medicine department for further evaluation (**Figure 1**).

Sleep-Related Symptoms and Clinical Evaluation

On admission to the sleep medicine department, he reported severe excessive daytime sleepiness and slept for more than 10 hours per day. He frequently fell asleep while sitting or talking and had difficulty resisting sleep during the daytime. His Epworth Sleepiness Scale score was 24.

He described having recurrent episodes of sudden loss of muscle strength, most commonly triggered by emotional distress or social interaction. The episodes usually involved the legs, arms, or neck. In some instances, he developed slurred speech or fell to the ground. Consciousness was usually preserved, and he was generally able to recall the events afterwards. The emotion-triggered loss of muscle tone with preserved awareness showed clinical features compatible with cataplexy. Some episodes lasted several minutes and varied in severity.

He also reported having recurrent sleep paralysis-like episodes during sleep onset, characterized by preserved awareness of surrounding conversations despite an inability to speak or move. He could recall the events after awakening. The duration and frequency were unclear, and eye movements were not documented. Spontaneous breathing was preserved in similar episodes observed during clinical interviews and ward rounds. No definite hypnagogic or hypnopompic hallucinations were reported.

His nocturnal sleep was characterized by difficulty initiating sleep, frequent awakenings, light sleep, vivid dreams, nightmares, and sleep talking. During the clinical assessment, he

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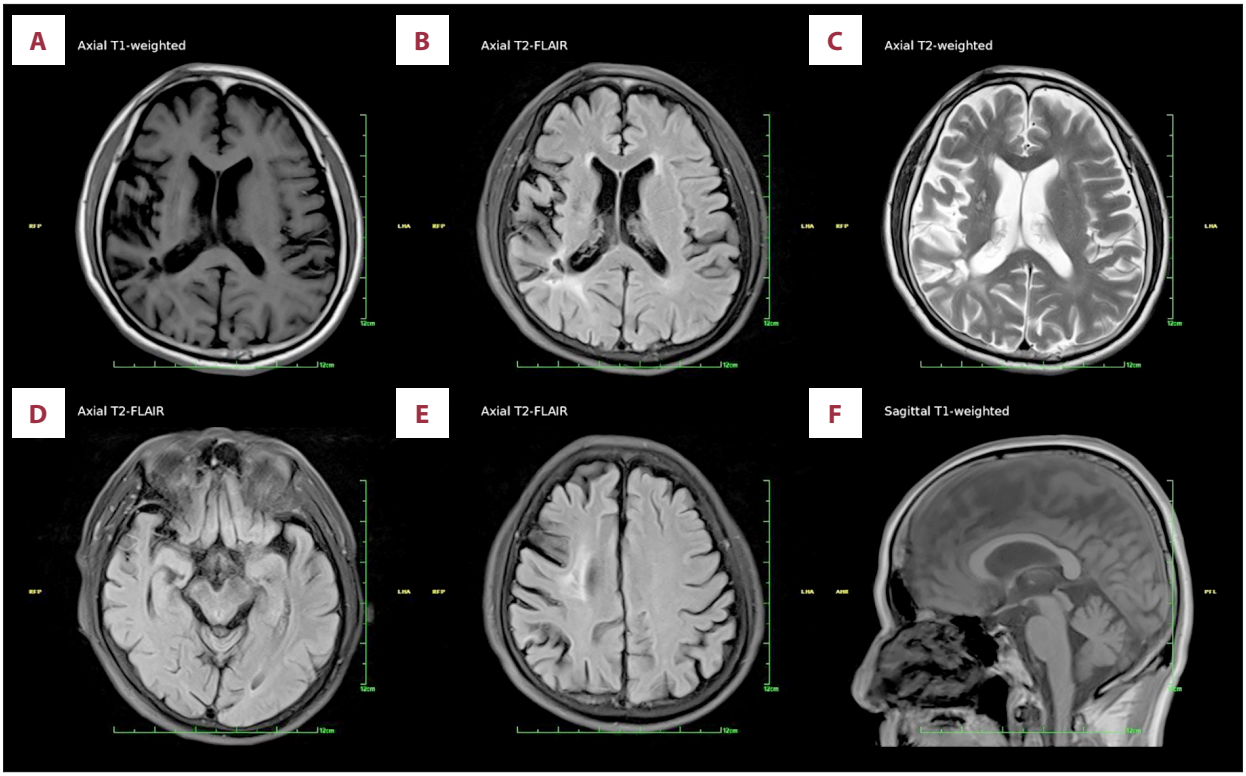


Figure 2. Representative brain magnetic resonance imaging (MRI) findings in a patient with late-onset narcolepsy type 1. The figure presents 6 representative MRI images obtained using different imaging sequences and anatomical planes. (**A**) Axial T1-weighted image; (**B**) axial T2-weighted fluid-attenuated inversion recovery (FLAIR) image; (**C**) axial T2-weighted image; (**D, E**) axial T2-FLAIR images at different anatomical levels; and (**F**) sagittal T1-weighted image. Brain MRI revealed areas of encephalomalacia and gliosis adjacent to the right lateral ventricle, multifocal ischemic changes involving the cerebral hemispheres and periventricular white matter, and cerebral atrophy. Additional ischemic lesions involving the brainstem and basal ganglia were identified during the overall MRI assessment. No discrete hypothalamic lesion was identified. These findings demonstrate coexisting cerebrovascular abnormalities but do not establish a secondary structural cause of narcolepsy. FLAIR, fluid-attenuated inversion recovery; MRI, magnetic resonance imaging.

repeatedly fell asleep while sitting and experienced several episodes of weakness involving the upper limbs, head, and neck. Between episodes, he was alert, cooperative, and fully oriented. Neurological examination showed no focal abnormalities, and no hallucinations, delusions, or other psychotic symptoms were identified.

Results

The patient underwent polysomnography (PSG) and multiple sleep latency testing (MSLT) on 2 occasions. The first PSG showed poor sleep efficiency, no sleep-onset rapid eye movement period (SOREMP), and moderate sleep apnea with an apnea–hypopnea index (AHI) of 17.7 events/h. The subsequent MSLT showed a mean sleep latency of 6.8 minutes without SOREMPs (**Table 1**).

The second PSG again showed disturbed sleep and revealed a shortened REM latency with 1 SOREMP. Severe obstructive sleep apnea was also present, with an AHI of 38.5 events/h. The second MSLT showed a markedly shortened mean sleep latency of 1.5 min but no SOREMPs. Thus, both MSLTs confirmed pathological sleepiness but did not meet the conventional SOREMP criterion for narcolepsy [1] (**Table 1**).

Brain magnetic resonance imaging (MRI) showed multiple areas of encephalomalacia with gliosis adjacent to the right lateral ventricle. Additional multifocal ischemic and infarct lesions were present in the brainstem, bilateral frontal and parietal lobes, basal ganglia, and periventricular white matter. Cerebral atrophy was also noted. No discrete hypothalamic lesion was identified (**Figure 2**). Routine cerebrospinal fluid findings were unremarkable. Cerebrospinal fluid hypocretin-1, measured using radioimmunoassay (RIA), was markedly reduced to 12.08 pg/mL (**Table 2**), fulfilling the criterion for CSF hypocretin deficiency according to the International Classification of Sleep Disorders criteria (≤ 110 pg/mL) [1]. Together with persistent excessive daytime sleepiness and emotion-triggered loss of muscle tone, this finding established the diagnosis of narcolepsy type 1.

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Table 2. CSF hypocretin-1 testing.

Parameter	Method	Result	Unit
Hypocretin-1 (Orexin A)	Radioimmunoassay (RIA)	12.08	pg/mL

Treatment and Follow-Up

Before the first PSG/MSLT, the patient had been taking paroxetine and olanzapine, both of which were discontinued 2 days before the first PSG/MSLT. The first PSG/MSLT was performed before these medications were restarted and was non-diagnostic for narcolepsy. Because he had previously experienced partial symptomatic improvement with paroxetine and olanzapine, paroxetine 20 mg daily and olanzapine 1.25 mg nightly were restarted after the first PSG/MSLT. After approximately 1 week of treatment, his excessive daytime sleepiness and cataplexy remained inadequately controlled. Given the persistent symptoms and unresolved diagnostic uncertainty, a second PSG/MSLT was performed while the patient was receiving paroxetine and olanzapine. CSF hypocretin-1 was subsequently found to be markedly reduced at 12.08 pg/mL, confirming the diagnosis of narcolepsy type 1. Treatment was then adjusted to clomipramine 25 mg nightly and pitolisant 9 mg daily, with pitolisant increased to 18 mg daily after 1 week. Continuous positive airway pressure (CPAP) therapy was recommended after obstructive sleep apnea was identified on PSG. However, CPAP was discontinued because of poor tolerance.

On the day of discharge, the Epworth Sleepiness Scale score had decreased from 24 at admission to 12. At the 2-month follow-up, he was taking clomipramine 12.5 mg twice daily and pitolisant 18 mg daily, with marked reductions in excessive daytime sleepiness and cataplexy.

At the 4-month follow-up, his condition remained stable, and the Epworth Sleepiness Scale score had further decreased to 9. He was able to maintain his daily activities, with stable mood and satisfactory nocturnal sleep, and continued the same treatment regimen. No adverse effects related to clomipramine or pitolisant were reported during follow-up.

Discussion

Narcolepsy type 1 is characterized by excessive daytime sleepiness, cataplexy, and cerebrospinal fluid hypocretin deficiency [8]. Although it typically begins in childhood or early adulthood, late-onset cases are uncommon [3]. In this patient, emotion-triggered episodes of transient muscle weakness with preserved awareness first occurred at approximately 54 years of age and were considered compatible with cataplexy. We therefore regarded the onset of these cataplexy-compatible episodes at approximately 54 years of age as the earliest clinically suggestive manifestation of NT1, supporting the late-onset designation in this case. Progressive excessive daytime sleepiness became prominent later, around the age of 60, and the diagnosis was ultimately confirmed by markedly reduced cerebrospinal fluid hypocretin-1 levels. The late presentation, prolonged psychiatric treatment, sleep comorbidities, and atypical neurological manifestations contributed to diagnostic delay.

The inconclusive PSG and MSLT findings may have reflected several confounding factors. The patient had a history of treatment with REM-suppressing antidepressants, including duloxetine, sertraline, and paroxetine, as well as sedating antipsychotics. Immediately before the first PSG/MSLT, he had been taking paroxetine and olanzapine. Both medications were discontinued 2 days before the first PSG/MSLT. Given the short washout interval, residual medication effects on REM sleep and SOREMP occurrence during the first sleep evaluation could not be excluded. After the first PSG/MSLT, paroxetine and olanzapine were restarted because of previous partial symptomatic improvement. As the symptoms remained inadequately controlled after approximately 1 week of treatment, a second PSG/MSLT was performed while the patient was receiving both medications. Therefore, the potential effects of ongoing psychotropic medication exposure should also be considered when interpreting the second sleep study [6]. In addition, poor sleep efficiency, fragmented sleep, and clinically relevant sleep apnea observed on both PSG examinations may also have affected the interpretation of the MSLT findings.

Secondary narcolepsy was considered because of the late onset and cerebrovascular abnormalities. However, brain MRI showed no hypothalamic lesion, although ischemic changes were present in the brainstem, which is involved in sleep–wake regulation. These findings may partly explain the atypical neurological manifestations but were insufficient to establish a secondary structural cause of narcolepsy.

Not all historical neurological manifestations were attributable to narcolepsy. Only the emotion-triggered episodes with preserved awareness were considered compatible with cataplexy, whereas tremor, slurred speech, impaired coordination, prolonged weakness, and dream-related behaviors were regarded as atypical and may have involved other contributing factors. REM sleep behavior disorder was considered but was not supported by PSG findings [9].

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This case highlights the diagnostic complexity of late-onset narcolepsy type 1, particularly in the presence of cataplexy-compatible symptoms, atypical neurological manifestations, and sleep-related comorbidities. In older adults with progressive excessive daytime sleepiness and emotion-triggered weakness, narcolepsy type 1 should remain a diagnostic consideration despite inconclusive PSG–MSLT findings. Cerebrospinal fluid hypocretin-1 measurement can provide critical diagnostic evidence, while atypical neurological and dream-related manifestations require separate evaluation for other or co-existing disorders.

Conclusions

This case illustrates that narcolepsy type 1 can present in later life and may be overlooked when comorbidities and non-diagnostic sleep studies obscure the clinical picture. In patients with persistent excessive daytime sleepiness and emotion-triggered loss of muscle tone, cerebrospinal fluid hypocretin-1 measurement may provide decisive diagnostic evidence, while atypical manifestations should be evaluated independently.

Institution Where Work Was Done

Shandong Daizhuang Hospital, Jining, Shandong, PR China.

Consent

Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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