

## Narcolepsy and Psychotic States – A Case Report

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**Abstract:** Narcolepsy is characterized by symptoms that include excessive sleepiness during the daytime, cataplexy, hypnagogic hallucinations and sleep paralysis. We present a single unusual case report of a patient who was admitted to a psychiatric facility. A 20-year-old single woman was hospitalized in a general hospital due to involuntary movements in her limbs. Following complaints of continual daily sleepiness and instances of a loss of muscle tone she was referred for sleep assessment. The patient was diagnosed with narcolepsy and cataplexy. Later, due to auditory hallucinations she was referred to a psychiatric hospital. Her reality judgement was poor, with partial insight regarding her illness.

The patient was treated with methylphenidate 20 mg/day and antipsychotic medication; risperidone of up to 4 mg/day, and paroxetine 20 mg/day to prevent cataplexy. Following further exacerbation, pharmacotherapy was changed to risperidone 6 mg/day and modafinil 200 mg/day. This treatment led to a significant improvement in her condition. The report presented here suggests that combined treatment with a psycho-stimulant drug, an SSRI in combination with antipsychotic treatment, may be indicated in narcolepsy with cataplexy and vivid psychotic production. Multidisciplinary cooperation of neurologists and psychiatrists enabled this therapy to be administered for the patient's benefit.

### Introduction

Narcolepsy is characterized by symptoms that include excessive sleepiness during the daytime, cataplexy, hypnagogic hallucinations and sleep paralysis. Hallucinations and delusions sometimes appear in patients with narcolepsy, and are often considered to be secondary to the hypnagogic hallucinations, either as a side effect of stimulant medications or as a complication of schizophrenia. The differential diagnosis between the disorders is not always simple. The following are examples which illustrate this overlap.

Douglass et al. (1) described a case of narcolepsy in which the hallucinatory component was the primary factor in the illness, and led to a course of illness which could not be differentiated from schizophrenia. These findings support the theory of REM intrusion among some patients suffering

from schizophrenia. The authors posit that some narcoleptic patients are so hallucinatory that they become delusional and thus receive a diagnosis of schizophrenia, and that the cataplexy may erroneously be diagnosed as a catatonic symptom of schizophrenia.

Mlynczak (2) found that 17% of psychotic patients suffered from a variant of narcolepsy. Saucerman (3) suggested that some patients diagnosed with schizophrenia or other psychotic disorders may actually be suffering from a variant of narcolepsy with a genetic base, involving dominant hypnagogic hallucinations.

In 1993, Douglass et al. (4) showed that an antigen tied to narcolepsy, HLA DR15 DQ6, was nearly four times more prevalent among schizophrenic patients as compared with a control group.

A case study by Bhat and Galang (5) reported on a 48-year-old male patient who was admitted

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to hospital due to the sudden appearance of bizarre behavior and delusions of persecution. His behavior and thought content did not change in response to antipsychotic treatment. The patient suffered from hypersomnia; therefore he was referred to a sleep laboratory and was diagnosed with narcolepsy. The hypersomnia was then successfully treated with psychostimulants. We present a single unusual case report of a patient who was admitted to a psychiatric facility.

## Case Report

A 20-year-old single woman was hospitalized in a general hospital due to involuntary movements in her limbs. Later, due to auditory hallucinations she was referred to a psychiatric hospital. The patient was the younger of two daughters. From an early age she experienced developmental difficulties, completing 12 years of school in a special education framework. She later entered a rehabilitational treatment center for adolescents. There was no history of mental illness in her family. Her aunt suffered from epilepsy.

From the age of four she suffered from atonic spells diagnosed as epilepsy. These attacks occurred approximately once a year. During the two years preceding her first hospitalization, she experienced these attacks as often as one to three times a day. The patient received valproate which was ineffective.

A few months prior to her hospitalization she underwent an MRI brain scan and video EEG that showed no abnormalities or epileptic activity.

Due to her complaints of continual daily sleepiness and instances of a loss of muscle tone she was referred for sleep assessment. The results showed sleep latency of 20 minutes and a sleep-onset REM period. The total sleep time was six hours and REM sleep comprised about 17% of total sleep time. Her sleep was characterized by the presence of many awakenings. The Multiple Sleep Latency Test the following day showed a mean sleep latency of six minutes and four sleep-onset REM periods. Between the tests she had at least two prolonged episodes of cataplexy with waxing and waning muscle tone during the episodes accompanied by rapid eye movements. The patient was diagnosed with narcolepsy with cataplexy.

She began treatment with methylphenidate to heighten alertness and paroxetine for control of cataplexy.

The patient then reported experiencing command auditory hallucinations. Pharmacotherapy was stopped, but the psychotic state continued. Under the influence of the voices she heard, the patient inflicted self-harm, and was then hospitalized in a psychiatric hospital.

Upon admission the patient reported hearing a woman's commanding voice. However, she repeatedly stated that: "this woman is actually me." No suicidal ideation appeared to be present. Her reality judgement was poor, with partial insight regarding her illness.

The patient was treated with 20 mg methylphenidate and antipsychotic medication; risperidone of up to 4 mg, and paroxetine 20 mg to prevent cataplexy. This treatment led to a significant improvement in her condition. She became alert and the hallucinations ceased. She was then discharged from the hospital. Treatment with risperidone was gradually reduced from 4 mg to 0.5 mg daily.

The patient was rehospitalized after another psychotic relapse with vivid auditory hallucinations and severe regression manifested by childish behavior, crying spells and inability for self-care. A new battery of tests was conducted, including a CT scan, which showed normal results. Risperidone was increased to 6 mg/daily, with no improvement.

Methylphenidate treatment was gradually reduced and switched to modafinil (Modiodal) titrated to 200 mg/daily. Three weeks later the psychotic symptoms ceased completely. Blood tests were conducted to determine genetic markers tied to narcolepsy, which were found to be negative (HLA DQ, DR). The patient refused to undergo LP sampling of CSF to confirm hypocretin levels. The hallucinations have not recurred and the patient has been receiving this stable drug regimen for more than two years.

## Discussion

Great effort has been made to categorize various subtypes of schizophrenia and its genetic base. The case presented here delineates a specific disorder, narcolepsy, which is expressed through psychotic

symptomatology and may be mistakenly diagnosed as schizophrenia. Narcolepsy in which the hallucinatory component is unusually prominent is rare, but it has been previously described (6) and may lead to the development of an illness indistinguishable from the schizophrenic syndrome.

Takehuchi et al. (7) described a case of a narcoleptic female patient whose delusions and hallucinations appeared before a diagnosis of narcolepsy had been made, and prior to the use of pemolin or clomipramine. Her psychotic state improved with anti-psychotic medication. The authors found that the patient's auditory hallucinations matched the sleep-related symptoms of rapid eye movements during the course of her illness, and they concluded that her hallucinations and delusions were caused by the narcoleptic symptoms.

Royant-Parola (6) found that nearly half of 11 narcolepsy patients manifested a psychiatric disorder in which the hallucinatory component was central, leading to a misdiagnosis of schizophrenia.

Stores (8) described isolated sleep paralysis with vivid, terrifying visual, auditory and somatic hallucinatory episodes at sleep onset, associated with a sense of evil influence and presence.

These findings also led to studies of HLA narcolepsy-associated antigens (NAA) in schizophrenic patients, which found a frequency of the NAA that was 3.89 times higher than in controls. Two of these patients were found to have narcolepsy. Interestingly, the HLA subgroup of HLA-DR15, DQ6 antigens discovered in 1990 as also associated with narcolepsy, mark a group of severe schizophrenia patients with a history of more refractory illness and more hospitalizations, even in the absence of narcolepsy (1).

In most cases with mixed symptoms of narcolepsy and schizophrenia, the psychiatric symptomatology improves with stimulant treatment, or with a combination of antipsychotic agents and stimulants.

In our case we present a patient whose psychotic symptoms worsened upon treatment with methylphenidate. The central dilemma was whether the psychotic symptoms were part of narcolepsy or a result of the treatment with methylphenidate

(Ritalin) or comorbid schizophrenia. Following a joint discussion with the patient, her family and the treating neurologists and psychiatrists, and based upon her sleep study, it was decided that the patient's psychotic symptoms were basically part of her narcoleptic illness. Thus, we decided to renew treatment with methylphenidate, which was augmented with antipsychotic treatment but this option did not have the expected effect. The patient's psychosis improved only with modafinil treatment.

Modafinil is indicated to improve wakefulness in patients with excessive daytime sleepiness. It has an essentially unknown mechanism of action as a sleep inhibitor and perhaps a subtle behavioral activating agent; it appears not to be directly monoaminergic, unlike traditional stimulants. It has been hypothesized that modafinil might promote wakefulness indirectly by inhibiting the release of aminobutyric acid (GABA) in the forebrain, perhaps through a serotonin-mediated process. We chose this compound for our patient and even in a daily dose of 100 mg it seemed to be effective for her sleepiness and alleviation of psychosis. However, Narendran (9) describes a schizophrenic patient treated with modafinil for excessive sedation due to clozapine treatment who developed a florid psychosis.

The report presented here suggests that combined treatment with a psycho-stimulant drug (amphetamine or a substitute), an SSRI in combination with antipsychotic treatment, may be indicated in narcolepsy with cataplexy and vivid psychotic production as a manifestation of hypnagogic hallucinations, thus providing hope for improvement, as we have demonstrated here. Multidisciplinary cooperation of neurologists and psychiatrists enabled this therapy to be administered for the patient's benefit.

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