

Reduced Cataplexy Symptoms While on Suboxone: A Case Report

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Abstract

Background: Narcolepsy type 1 is a sleep disorder characterized by excessive sleepiness along with cataplexy and/or hypocretin deficiency. We present a case of narcolepsy type 1 in which the cataplexy symptoms were reduced while on suboxone. **Aim:** This case report aims to note the reduction of cataplexy symptoms with suboxone. **Case Presentation:** A 39-year-old female patient with narcolepsy type 1 was referred to our pain clinic for management with suboxone. The patient had tried various other medications for the management of narcolepsy. With opioid management, the patient reported reduced cataplexy symptoms. **Conclusion:** The case illustrates the scientific thought that opioids could increase hypocretin and reduce symptoms in narcolepsy type 1.

Keywords

Narcolepsy, Cataplexy, Suboxone, Sleep, Muscle, Tone

1. Introduction

Narcolepsy type 1 is a sleep disorder characterized by excessive sleepiness along with cataplexy and/or hypocretin deficiency [1]. Narcolepsy type 1 is a sleep disorder that affects 0.02% of adults globally [2]. Patients exhibit Excessive Daytime Sleep (EDS), sudden loss of muscle tone triggered by emotions (cataplexy), hallucinations, and sleep paralysis [3].

The integration of opioid medication such as tramadol in the management of cataplexy to help reduce and ease the impact of cataplexy symptoms has been noted with positive results [4].

We present a case of a patient with narcolepsy with cataplexy symptoms who presented to our pain clinic for the management of suboxone with the reported

reduction in cataplexy symptoms.

2. Case Presentation

The patient is a 39-year-old female who presented to our pain clinic with a diagnosis of narcolepsy with cataplexy referred by her pulmonologist for management with suboxone. The patient was diagnosed with narcolepsy with cataplexy at the age of 28. Before the diagnosis, she experienced chronic fatigue and sleepiness without a clear cause. She adjusted her lifestyle to be able to manage her activities. To get through college, she opted for night classes which suited her since her narcolepsy inclined her to sleep until late in the day. Along with the narcolepsy she also had symptoms of cataplexy whereby she would lose all muscle control with any intense emotions such as heavy laughter. Deep laughter would cause her knees to give out whereby she would have to sit from a standing position. The cataplexy symptoms also happened in other states of intense emotion such as when she was happy, nervous, scared, and surprised. The cataplexy symptoms at times only abated upon cessation of the emotion. If she was laughing, she would regain muscle control only when she stopped laughing. Other episodes of cataplexy lasted a few seconds. The patient states she lost work opportunities due to her symptoms of cataplexy at one point being dismissed from work due to the symptoms.

Upon diagnosis, her pulmonologist placed the patient on medications that included armodafinil, fluoxetine, sodium oxybate, methylphenidate with ongoing symptoms.

The patient was then started on a trial with low dose opioid (oxycodone 5 mg bid) for her condition. With this treatment, the patient endorsed reduced cataplexy symptoms. She continued on the low dose opioid for a year and was then referred to pain management as the patient was experiencing withdrawal symptoms on the oxycodone and management with suboxone was recommended. The patient subsequently presented to the pain clinic where management with suboxone (1 - 4 mg sublingual bid) was initiated after a thorough evaluation. While on suboxone, the patient endorsed ongoing reduced symptoms of cataplexy noting fewer episodes of cataplexy. She endorsed rare events of muscle loss even with instances of increased emotional levels. She stated she could laugh heartily without falling to the ground due to a loss of muscle tone.

3. Discussion

Narcolepsy type 1 manifests with Excessive Daytime Sleep (EDS) along with cataplexy. It is an autoimmune disease that results from the destruction of orexin-producing neurons within the hypothalamus, resulting in reduced orexin levels [5]. Orexin is responsible for the regulation of the sleep-wake cycle [6].

Buprenorphine/naloxone is an opioid agonist-antagonist medication. It is a compound consisting of buprenorphine, the opioid agonist, and naloxone, the opioid antagonist. Buprenorphine is a partial μ -opioid receptor agonist [7]. The

partial agonist characteristic of buprenorphine gives buprenorphine/naloxone a low risk of overdose [7]. Buprenorphine can be taken intravenously or intranasally [7]. To limit its abuse, naloxone, which lacks effect on the sublingual use of buprenorphine, acts as a deterrent to limit the abuse of buprenorphine through intravenous or intranasal administration.

Opioids primarily stimulate neurons within the hypothalamus to produce orexin [8]. Thus, opioid agonists like buprenorphine are postulated to aid the treatment of narcolepsy with cataplexy by stimulating the hypothalamic neurons to produce more orexin, alleviating symptoms of narcolepsy with cataplexy in patients. Treatment of narcolepsy with other opioid agonists such as codeine [9] and tramadol [4] has been described and supports the observation that opioid treatment reduces cataplexy symptoms.

4. Conclusions

In this patient, management with buprenorphine/naloxone reduced symptoms of cataplexy. This case report supports the observations and hypothesis opiates can increase hypocretin/orexin production.

This is one case report, there are no randomized studies trials to support the observation and more studies need to be done on the management of narcolepsy type 1 symptoms with low-dose opioid medication to give more insight into efficacy, risks, side effects profile, and dosing.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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