Sleep and Breathing (2019) 23:1301–1302
https://doi.org/10.1007/s11325-019-01800-y

Finding a needle in the haystack—narcolepsy and obstructive sleep apnea

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Received: 22 September 2018 / Revised: 3 February 2019 / Accepted: 6 February 2019 / Published online: 18 February 2019

Dear Editor,

Narcolepsy is a rare disorder characterized by excessive daytime sleepiness (EDS), sleep paralysis, cataplexy, and hypnagogic hallucinations, which diagnosis depends on careful clinical consideration and confirmation for polysomnography (PSG) and multiple sleep latency test (MSLT) [1]. EDS is an essential symptom that may be the primary concern for narcolepsy patients, leading to distress and impairment regarding The International Classification of Sleep Disorders (ICSD-3) [1]. However, it can also be found in other conditions as insufficient sleep syndrome and sleep disturbances such as obstructive sleep apnea (OSA) with a vast impact on general quality of life too.

OSA syndrome and narcolepsy are conditions that lead to daytime sleepiness. They can coexist in the same person, but the accurate diagnosis can be delayed by overlap of symptoms, risk factors, and comorbidities. OSA exists in narcolepsy patients due to the higher prevalence of obesity, and narcolepsy is a differential diagnosis of OSA patients with excessive daytime sleepiness. In fact, the differential diagnosis in this overlap is a challenge.

We report a 28-year-old male with EDS last year. His wife reported snoring and many breathing pauses at night. He also reported weight gain, anxiety, and cocaine abuse. On physical examination, his blood pressure was 110/70 mmHg with a body mass index (BMI) of 22.46. He had hypertrophy of turbinates with web palate and brachyfacial typology and was affected with a skeletal class II malocclusion, due to a mandibular retrusion (Fig. 1). The overnight PSG showed a REM sleep latency of 282 min and AHI of 25.9 events per hour. A pressure of 6 cm H_2O controlled OSA during a CPAP titration PSG with a nasal mask. He did not initiate CPAP therapy. One year later, new overnight PSG showed apnea-hypopnea index (AHI) of 42.7 events per hour. CPAP therapy was initiated with a pressure of 8 cm H_2O, however without improvement of EDS. He visited our EDS clinic for a second opinion. During the interview, he related a few episodes of cataplexy during the life, sleep attacks, sleep fragmentation, and sleep paralysis. A new PSG using CPAP followed by MSLT was performed. MSLT revealed low sleep latency (2 min) and three sleep onset REM periods (SOREM) in five naps. The HLA-DQB1*0602 was present, and the CSF hypocretin-1 level was zero. Methylphenidate 10 mg twice a day was initiated with the improvement of EDS.

EDS is the inability to stay wakeful and alert during the day when one would be expected to be wakeful. Patients with EDS frequently have a lower quality of life, low workplace motivation, and increased risk of work-related accidents. Medications; alcohol; substance abuse; and certain medical, neurologic, and psychiatric disorders can cause EDS [1]. EDS can be the main complaint of OSA patients that is related to sleep fragmentation.

Many symptoms are common between narcolepsy and OSA patients; in real life, the differential diagnosis is not simple. EDS, hypnagogic hallucinations, and sleep paralysis are narcolepsy symptoms; however, they can be seen in sleepy patients due to other diseases such as OSA. Sleep fragmentation is a frequent finding in OSA and narcolepsy too. In fact, the differential diagnosis between OSA and narcolepsy type 2 can be tough since the cataplexy is an exclusive characteristic of narcolepsy type 1.

EDS, obesity, and OSA are prevalent conditions in many parts of the world. In obese individuals, defined by a BMI > 35 kg/m^2, the prevalence of EDS is about 30% [2]. The role of weight gain in narcolepsy types 1 and 2 is well documented to

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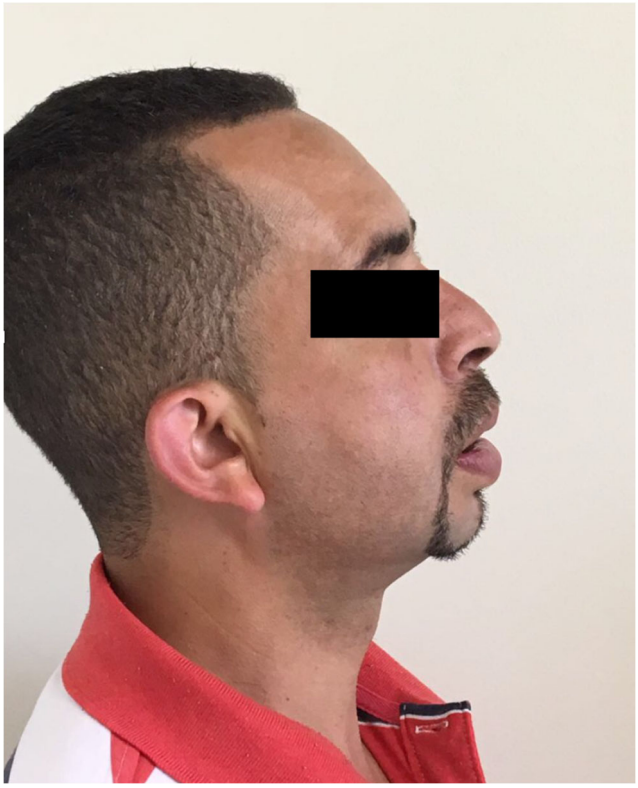

Fig. 1 Brachyfacial typology, due to a mandibular retrusion

increase the risk for OSA [3]. The etiology of obesity in narcolepsy is multifactorial: hypocretin deficiency, sedentary lifestyle, decreased energy expenditure due to sleepiness, and comorbid depression [4].

Our patient was not obese. However, he had a severe mandibular retrognathia. Craniofacial abnormalities can drive OSA [5]. An increased upper airway collapsibility due to maxillary deficiency, mandibular retrusion, inferior displacement of the hyoid bone, and cranial base dimorphisms are common findings [2]. The overlap between craniofacial morphology and obesity probably determines the severity of OSA in part of these patients.

Narcolepsy and OSA can coexist. The differential diagnosis of narcolepsy in sleepy patients without cataplexy and OSA may be difficult. In these cases, the residual EDS after satisfactory continuous positive airway pressure is considered a red flag, especially in patients with other complains such as hypnagogic hallucinations, sleep paralysis, or cataplexy. A careful medical history and follow-up with serial sleep studies including MSLT may allow an early recognition of overlap between narcolepsy and OSA.

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