

Central sleep apnea in a patient with clinically isolated syndrome

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Abstract. Central sleep apnea (CSA) is characterized by the cessation of air flow without respiratory effort during sleep. Various neurological diseases have been shown to be associated with CSA.

In this case, we report a 37-year-old male with who admitted to us for sleep respiratory disturbance. CSA was diagnosed after a full-night polysomnography. Although the neurological examination of the patient was normal, we detected hyperintense brain lesions in magnetic resonance imaging (MRI), past history for optic neuritis and delayed visual evoked potential responses on the left eye and diagnosed as clinically isolated syndrome (CIS) accompanying to CSA.

These findings show and emphasize the importance of MRI in patients with CSA even their neurological examination is normal. Up to our knowledge, this is the first case of CSA reported in a patient with CIS.

Key words: Central sleep apnea, clinically isolated syndrome, multiple sclerosis

1. Introduction

Central sleep apnea (CSA) is characterized by the cessation of air flow for at least 10 seconds without respiratory effort during sleep (1,2). It is relatively rare comparing to obstructive sleep apnea syndrome (OSAS) and usually both syndromes are overlapped (1). Various neurological diseases including Arnold-Chiari malformation, stroke and multiple sclerosis (MS) have been shown to be associated with CSA (2-4). Clinically isolated syndrome (CIS) is a condition which is the first episode of a demyelination disease, mostly MS (5). In this case, we report a patient with sleep respiratory disturbance, who was diagnosed as both CIS and CSA after our investigation. Up to our knowledge, this is the first case of CSA reported in a patient with CIS.

2. Case report

A 37-year-old male with body mass index (BMI) 29.4kg/m² was referred to our department for severe daytime sleepiness and witnessed apnea attacks. He had no history for any neurological disease and was not taking any medication. He was diagnosed CSA after full-night polysomnographic investigation with apnea-hypopnea index (AHI)=85.5/h [index of the apnea patterns; obstructive apnea=19.7/h, central apnea=48.3/h, mixed apnea=4.1/h, all hypopnea=13.5/h] (Figure 1). Sleep disordered breathing events were scored manually according to American Academy of Sleep Medicine criteria (6). His neurological examination was normal. He was consulted to cardiology, internal medicine and chest disease departments and no abnormality was found. A detailed questioning of his medical history revealed an episode of blurry vision on left eye for fifteen days which healed spontaneously 2 years ago. Contrast-enhanced magnetic resonance imaging (MRI) demonstrated a few non-active small demyelinated plaques perpendicular to the lateral ventricles (Figure 2). Spinal MRI showed no abnormality. We performed visual evoked potentials and detected delayed P 100 potential on the left side (135 msec). We also performed brainstem auditory evoked potentials (BAEP), and the result was

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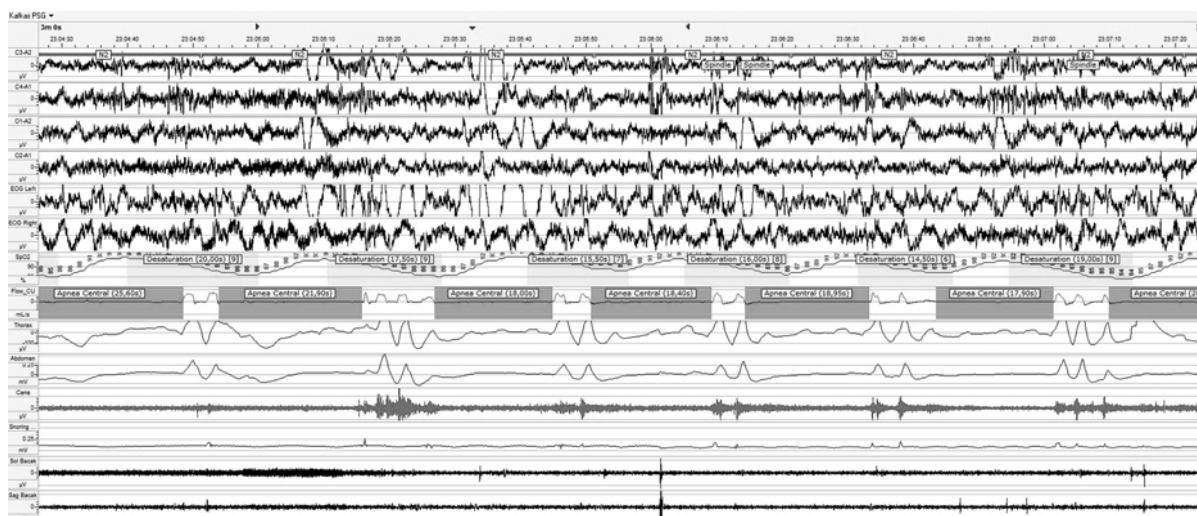


Fig. 1. Sample from the patient's polysomnographic recording.

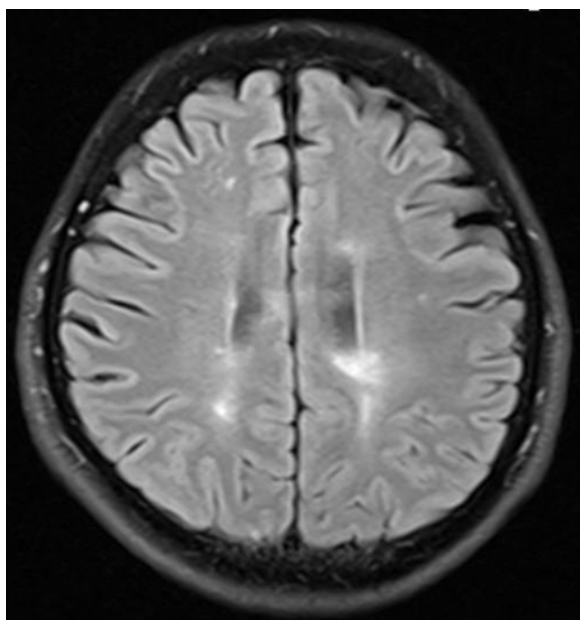


Fig. 2. T2 weighted MRI, Hyperintense lesions perpendicular to the lateral ventricles.

within normal limits. Routine blood tests including thyroid function tests, vitamin B12 and folic acid levels and vasculitis markers were normal. He had no prior history of oral and genital aphthae, uveitis, skin lesions or arthritis. Ophthalmological examination including bilateral visual field examination was normal. The patient refused lumbar puncture for cerebrospinal fluid investigation. Due to a unilateral optic neuritis attack history and delayed Visual Evoked Potential (VEP) response on the same eye, the patient was considered as CIS. He was given continuous positive airway pressure (CPAP) therapy and there was a clinical improvement in the symptoms of the patient. After more than one

year follow-up the patient remains without any new complain.

3. Discussion

Brainstem is the most important center of the respiratory control. Any pathology affecting the brainstem may cause sleep-disordered breathing disorders including sleep apnea disturbances (2-4).

In a recent study, OSAS frequency was found as 58% in patients with MS, but there was no significant difference between the age and sex matched control group (7).

In another study, CSA was reported in 2 of 25 (8%) patients with MS coexisting with another sleep related disorder, such as periodic limb movement disorder during sleep (8). There was a report of a patient with MS who developed severe fatigue and CSA and was treated successfully (5). Also a severe form of CSA called as Ondine's curse has been reported in two patients with MS and both patients died in their sleep (4).

CIS is the first and solo episode of a demyelinating disease of the central nervous system. It may suggest the possibility of developing MS. Commonly it is presented with signs of involvement of optic nerve, spinal cord and brainstem (5).

Our patient had a history of optic neuritis but the first symptoms for a doctor visit were witnessed apnea and daytime sleepiness. Although the neurological examination of the patient was normal and no brainstem pathology was detected in MRI or BAEP, we believe that these symptoms were associated with an undetectable brainstem lesion with 1.5 tesla MRI.

These findings show and emphasize the importance of MRI in patients with CSA even

their neurological examination is normal. This is the first reported case of CIS and CSA in the same patient. Although it might be just co-occurrence of both diseases, in our opinion due to absence of other causes of CSA, CSA might be associated with CIS in this case. This report adds variety to clinical presentations of CIS.

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