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Case Report

Obstructive sleep apnea-induced chronic cough: three case reports and a review of the literature

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Abstract: Objective: The aim of this study was to analyze the clinical features of obstructive sleep apnea (OSA)-induced chronic cough and improve the current understanding of the association between chronic cough and OSA. Methods: We used *chronic cough* and *obstructive sleep apnea* as the key words with which to retrieve relevant literature from the PubMed database from January 2002 through November 2014. Eight articles were included: four were case reports of nine patients (three male and six female patients aged 46-73 years); the other four articles were epidemiological studies of patients with concurrent OSA and chronic cough. A literature review was performed by combining the characteristics of the cases reported in the literature and the three cases in the present report. Results: Case 1 involved a 60-year-old woman with a 3-month history of cough. Case 2 involved a 52-year-old man with a 3-year history of cough. Case 3 involved a 63-year-old man with a 5-year history of cough and chest tightness. Common causes of chronic cough were excluded in these three cases. Polysomnography indicated a diagnosis of severe OSA, and the patients' symptoms were resolved after continuous positive airway pressure therapy. Conclusions: OSA is a possible etiology of chronic cough. Chronic cough can be the sole presenting symptom in patients with OSA. The incidence of chronic cough is significantly high in patients with OSA. Continuous positive airway pressure treatment can significantly improve chronic cough.

Keywords: Chronic cough, obstructive sleep apnea, continuous positive airway pressure

Introduction

Chronic cough is defined as a cough that lasts for ≥8 weeks in patients with no obvious anomalies on chest X-ray and who present with cough as the main or sole symptom; it is the most common symptom in medical patients [1]. Epidemiological studies have shown that the main causes of chronic cough include cough variant asthma (CVA), upper airway cough syndrome (UACS), eosinophilic bronchitis, gastroesophageal reflux cough (GERC), and allergic cough, all of which account for 70% to 95% of the etiologies of chronic cough. Other causes account for a small proportion of the etiologies and vary widely [2, 3].

Obstructive sleep apnea (OSA) is a sleep disorder with frequent apnea as the prominent manifestation. OSA is caused by complete or partial repetitive obstruction of the upper airway during sleep. Its prevalence is approximately 4% in adults and can reach 20% to 40% in the older population aged >60 years [4]. Recent studies have found that patients with OSA also frequently have chronic cough in addition to other symptoms such as snoring, nighttime awakenings, and daytime sleepiness; a few case reports on OSA-induced chronic cough have been published [6-8]. We recently treated three patients with OSA with chronic cough as the main etiological factor. We herein report the diagnosis and treatment of these three patients and present a review of the relevant literature.

Clinical data

The clinical data were summarized in **Table 1**.

Case 1

A 60-year-old woman was admitted to our hospital because of a >3-month history of recurrent cough. The patient reported that the cough

Table 1. Clinical features of three patients with obstructive sleep apnea-induced chronic cough

	Case 1	Case 2	Case 3
Age (years)	60	52	63
Sex	Female	Male	Female
Snoring	Yes	Yes	Yes
Daytime sleepiness	Not significant	Yes	Yes
Height (cm)	160	168	157
Weight (kg)	75	75	65
BMI (kg/m^2)	29.3	26.6	26.4
Baseline VAS score	60	55	70
Post-CPAP VAS score	0	5	20
Routine blood tests			
WBC (10^9)	7.77	8.83	6.23
EOS (%)	2.9	1.3	1.5
Total IgE (kU/L)	Not tested	73.9	89.5
FVC (L)	2.53	3.31	2.24
FEV1 (L)	1.94	3.07	1.66
FEV1/FVC (%)	85.9	92.87	74.17
Induced sputum neutrophils (%)	89.5	86.0	94.0
Induced sputum eosinophils (%)	2.0	0.0	0.0
FENO (ppb)	15	30	13
AHI	37.1	50.7	54.6
Post-treatment AHI	7.2	2.8	NA

BMI, body mass index; VAS, visual analogue scale; WBC, white blood cells; EOS, eosinophils; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s; FENO, fractional exhaled nitric oxide; AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure; NA, not available.

occurred with no obvious cause. It manifested as a paroxysmal irritating cough with no circadian rhythm, wheezing, acid reflux, belching, nasal congestion, runny nose, or postnasal drip (PND) sensation. The cough attacks were unrelated to smelling a pungent odor, and the patient had no obvious nasal congestion, runny nose, or dyspnea. Approximately 2 weeks before admission, the patient had developed an upper respiratory tract viral infection (a cold), resulting in a more severe cough. Associated symptoms include fever, sore throat, and a small amount of white sputum. Her body temperature fluctuated from 37.2°C to 38.8°C. She visited the clinic in a local hospital, and a chest X-ray showed no clear lesions in the parenchyma of both lungs. The patient's fever and sore throat resolved after antibiotic treatment. However, during the 10 days before admission, her cough did not improve and she developed an itchy throat and felt tightness while breathing during severe coughing. One week before admission, she developed an itchy throat and felt tightness while breathing during severe coughing. One week before admission, she developed a persistent right-sided headache and was thus hospitalized for further treatment.

The patient had no history of smoking; no medical history of hypertension, coronary heart disease, or diabetes; and no relevant personal or family history. Physical examination showed pharyngeal stenosis, a hypertrophic tongue, soft palate hypertrophy, and a large uvula. The patient's breathing was smooth, and her breath sounds were clear in both lungs. Auxiliary examination by chest X-ray in the clinic of our hospital showed no clear lesions in the parenchyma of both lungs. On admission, we considered that the possible etiologies of the patient's cough were UACS and CVA. Auxiliary echocardiographic examination after admission indicated aortosclerosis and left atrial enlargement. Sinus and chest computed tomography (CT) suggested bilateral maxillary sinus and ethmoid sinus inflammation and slight chronic inflammation/fibrotic foci in the lower lingular segment of the left upper lobe and left lower lung. A lung function test indicated normal ventilatory function, and a bronchial provocation test was negative. After admission, an ear, nose, and throat (ENT) consultation led us to consider chronic rhinitis. The patient was treated with budesonide nasal spray with azithromycin for infection; however, her symptoms did not resolve.

When asked about her medical history, the patient reported nocturnal snoring accompanied by intermittent apnea with no obvious daytime sleepiness; occasionally, she had dizziness and headache. Polysomnography (PSG) was applied, and the results were as follows: total number of central sleep apneas, 0; total number of complex sleep apneas, 0; total number of obstructive apneas, 228 (longest, 106.5 s; associated blood oxygen desaturation [OD] of 212 times); and total number of hypopneas, 34 (longest, 49.5 s; associated blood OD of 27 times). The apnea-hypopnea index (AHI) was 37.1 times/h. The total duration of a <90% oxy-

gen saturation (SaO_2) was 66.5 min, accounting for 15.7% of the total sleep time. The mean SaO_2 while awake was 95%, and the lowest value was 62%. The maximum OD was 35%, and the longest duration of OD was 94 s. Severe OSA with severe nocturnal hypoxemia (NH) was diagnosed and treated by pressure titration using a ventilator. The patient had good compliance. The 90% confidence pressure set range was 4.0 to 9.8 cm H_2O, the mean automatic continuous positive airway pressure (CPAP) was 7.3 cm H_2O, and the AHI under CPAP therapy was 7.2 times/h. Her cough was significantly improved on the second day of pressure titration, and she wore a ventilator for daily treatment after discharge. The patient took no other medications and had almost no cough at follow-up 1 month later.

Case 2

A 52-year-old man was admitted to our hospital because of a >3-year history of a recurrent cough and 6-month history of chest tightness. The cough manifested as repeated paroxysms of coughing, especially during the day and after dinner, with a small amount of white phlegm. Smelling an irritating odor such as soot or gasoline stimulated the cough, but violent activities did not; coughing was independent of postural, seasonal, or weather changes. The patient also experienced snoring at night and nighttime awakenings. Six months prior to presentation, he had developed chest tightness mainly at night with intermittent acid reflux and belching. He had no palpitations, precordial pain, or shortness of breath after activity. He visited a local hospital and was diagnosed with chronic bronchitis. However, the cough did not improve after antispasmodic asthma treatment by aerosol inhalation.

After admission, the patient underwent routine blood testing, biochemistry evaluation, and measurement of myocardial enzyme parameters, all of which showed no abnormalities. Specific allergen screening was negative, and a 24-hour ambulatory electrocardiogram and echocardiogram were normal. Lung function testing showed the following results: ventilatory function was within the normal range; a bronchial provocation test was negative; the total lung capacity (TLC) was reduced, while the residual volume (RV) and respiratory RV/TLC were normal; and airway resistance was within the normal range. PSG indicated a diagnosis of severe OSA with severe NH. The total duration of a <90% SaO_2 was 158.9 min, and the lowest SaO_2 was 69%. The longest duration of OD was 61 s. An artificial airway pressure titration test was performed; the mean pressure during automatic CPAP therapy was 11.5 cm H_2O, and the AHI under CPAP therapy was 2.8 times/h. The patient's cough and chest tightness were significantly improved after 1 week of CPAP therapy.

Case 3

A 63-year-old woman was admitted to our hospital because of a 5-year history of repeated coughing and chest tightness. Smelling a pungent odor or being present in a sealed environment induced symptoms of coughing and chest tightness. She had a paroxysmal cough with no chest pain, palpitations, or expectoration. She visited a local hospital, and the preliminary diagnosis was bronchitis; however, symptomatic treatment was ineffective. Approximately 6 months before admission, she was evaluated at a local hospital. A lung function test was normal, and a bronchial provocation test was positive. CVA was considered, and the patient was treated with formoterol-budesonide dry inhalation powder and theophylline tablets; however, this treatment had no significant effects. Six years previously, she had experienced snoring at night with nighttime awakenings since six years ago and undergone PSG at a local hospital because of snoring. The results indicated moderate OSA, but the patient refused CPAP therapy.

After admission, the patient underwent routine blood testing, biochemistry evaluation, and measurement of myocardial enzyme parameters; no abnormalities were found. Specific allergen screening was negative, and a 24-hour ambulatory electrocardiogram and echocardiogram were normal. Lung function testing showed that ventilatory function was within the normal range, a bronchial provocation test was negative, the TLC was reduced while the RV and respiratory RV/TLC were normal, and airway resistance was within the normal range. The peak expiratory flow variability was <20%. Bronchoscopy revealed no abnormalities, and bronchial mucosa biopsies showed chronic inflammatory changes. PSG indicated severe OSA with severe NH. The total duration of a <90% SaO_2 was 88 min, and the lowest SaO_2

was 69%. The longest duration of OD was 61 s. The cough and chest tightness were significantly improved after 1 month of CPAP therapy.

Literature review

We used *chronic cough* and *obstructive sleep apnea* as keywords with which to search the PubMed database and obtained eight valid articles. Four of them were case reports and the other four were epidemiological investigations of patients with concurrent OSA with chronic cough. The four case reports involved nine patients (three men, six women; age, 46-73 years). Birring et al. [6] first reported four cases of OSA-induced chronic cough in 2007 and found that the cough disappeared or was significantly alleviated in these patients after CPAP therapy. In their study, a causal relationship was established between OSA and chronic cough for the first time. These four cases are summarized below.

Case 1 involved a 52-year-old man with a history of smoking. He visited a general practitioner because of a 3-month history of coughing and expectoration. The coughing had no circadian pattern and was aggravated in the lying position. Multiple courses of antibiotic treatment were ineffective, and the patient was transferred to a university hospital. Chest X-rays, lung function testing, bronchial provocation testing, chest CT, and induced sputum results were normal. GERC and rhinitis were considered, and the patient's symptoms were improved after treatment with nasal corticosteroids and a proton pump inhibitor (PPI); however, the cough still remained, and treatment was stopped. The patient visited a doctor 3 years later for evaluation of the cough with daytime sleepiness. Physical examination showed oropharyngeal stenosis, and PSG indicated OSA (AHI of 36). Complete remission was observed after 6 weeks of CPAP therapy.

Case 2 involved a 73-year-old woman with a history of smoking. She had an 18-month history of a dry cough that mainly occurred during the day. She also reported daytime sleepiness and snoring secondary to the coughing. PSG indicated severe OSA (AHI of 86), but the patient refused to undergo CPAP therapy. She was more concerned about resolving the cough. Chest X-ray examination revealed no anomalies; lung function testing showed restrictive ventilatory dysfunction; and nasal endoscopy, an otolaryngology consultation, bronchoscopy, and echocardiography were normal. Inhaled corticosteroids (ICS), inhaled short-acting β2-agonists, and long-acting β2-agonists (LABA) were ineffective. The patient agreed to undergo noninvasive ventilation by CPAP therapy at her second doctor visit because of the chronic cough. The cough was obviously improved after 1 week of treatment.

Case 3 involved a 46-year-old nonsmoking woman. She had a 1-year history of a cough and expectoration, with coughing symptoms both day and night. She had no rhinitis or GERD. Nasal corticosteroids and ICS were ineffective. Chest X-ray examination and lung function tests were normal. The initial diagnosis was asthma and eosinophilic bronchitis. Lung function, high-resolution CT (HRCT) findings, and induced sputum testing were normal. PSG indicated OSA. The cough resolved after 5 days of CPAP therapy.

Case 4 involved a 63-year-old man with a 4-month history of coughing and expectoration. The coughing was severe both day and night and was more severe in the supine position. No symptoms of rhinitis or GERD were found. Chest X-ray examination, lung function, bronchial provocation testing, and induced sputum testing were normal. The patient complained of snoring and daytime sleepiness. Nocturnal oximetry monitoring indicated OSA (AHI of 10). The cough was improved after 2 days of CPAP therapy and completely resolved 3 months later.

Sundar and Daly [7] reported two cases of OSA-induced chronic cough. Case 1 involved a 61-year-old nonsmoking woman with an 18-year history of chronic cough. The cough was dry and more severe at night and in winter. Associated symptoms included reflux, nausea, nasal congestion, and PND. She had frequent upper respiratory tract infections accompanied by bronchitis. She had used antibiotics and corticosteroids to ease dyspnea, nasal congestion, and chest tightness, but the nighttime cough significantly affected her sleep. Multiple chest X-ray examinations and lung function tests were normal. Bronchial provocation testing, an ENT consultation, endoscopy, and imaging showed no abnormalities. The fractional exhaled nitric oxide level was normal

(13 ppb). During a 5-year follow-up period, she intermittently used second-generation antihistamines, ICS, montelukast, and PPI, and occasionally used LABA/ICS therapy. Antibiotics and LABA/ICS were added when her condition became aggravated. The cough temporarily improved with each treatment and subsequently recurred, affecting sleep and inducing daytime sleepiness. The final diagnosis was OSA (AHI of 47). The cough was significantly improved after CPAP therapy. Application of a PPI was maintained, while antihistamines and leukotriene antagonists (LTRAS) were continuously used to control nasal symptoms. Case 2 involved a 60-year-old nonsmoking woman with a 6-month history of coughing after pneumonia. She was diagnosed with severe OSA (AHI of 50) 3 months before admission, and the cough completely resolved after CPAP therapy.

Yokohori et al. [8] reported the first two cases of OSA-induced chronic cough in Japan. Case 1 involved a 63-year-old nonsmoking woman who visited a community physician because of a dry cough for multiple years. ICS, LABA/ICS, theophylline, LTRAS, and a long-acting M-receptor antagonist were ineffective. The patient was transferred to a university hospital for treatment. She had a cough both day and night, with no wheezing or GERD symptoms. She also had hypertension and diabetes. Chest X-ray examination, lung function testing, a bronchial dilation test, and HRCT findings were normal. An IgE test indicated allergies to house dusts and ticks. CVA was diagnosed. Treatment with budesonide/formoterol, LTRAS, and antihistamines for 3 months was ineffective. A subsequent ENT consultation excluded PND syndrome (PNDS), and chronic rhinitis was diagnosed. The patient was treated with clarithromycin and carbocisteine; however, the PNDS was aggravated a few days later, and the drugs were withdrawn. The patient was obese with a body mass index (BMI) of 33.2 kg/m^2. She had pharyngeal narrowing and snoring. PSG indicated severe OSA (AHI of 90.2), and the cough disappeared after 1 month of CPAP therapy. The LABA/ICS, LTRAS, and antihistamines were withdrawn.

Case 2 involved a 66-year-old nonsmoking woman. She had experienced a dry cough and dyspnea for 5 months and had a history of sick sinus syndrome. GERD was diagnosed by digestive endoscopy. PPI therapy resulted in no improvement, and the patient was transferred to a university hospital 3 months later. She had a pacemaker and hypertension, but did not take any angiotensin-converting enzyme inhibitors or exhibit PNDS or GERD symptoms. Chest X-rays showed no lung abnormalities, and lung function was normal. She had upper airway symptoms was preliminarily diagnosed with UACS. Antihistamine therapy was ineffective during the following 2 months, and subsequent ICS and LABA/ICS therapy only temporarily resolved the cough. The patient had no daytime sleepiness. She was mildly obese (BMI of 26.1 kg/m^2) and had pharyngeal narrowing and snoring. PSG indicated severe OSA (AHI of 47), and the cough was significantly improved after 1 month of CPAP therapy. The LABA/ICS was finally withdrawn.

Faruqi et al. [9] described a 61-year-old nonsmoking man with a chronic cough. He had heartburn, but no dyspnea, wheezing, or PND. Chest X-rays and lung function were normal. Pharyngeal pH measurements suggested the presence of GERD. The cough was slightly improved after tentative lansoprazole treatment, but experimental treatment with nasal corticosteroids and ICS was ineffective. He had a BMI of 31 kg/m^2. His medical history included snoring for several years with daytime sleepiness. OSA was diagnosed by PSG (AHI of 45). The cough disappeared after CPAP therapy. The outcome was satisfactory at the 1-year follow-up, the patient's quality of life was improved, and cough sensitivity was significantly improved. GERC and OSA were finally considered to be the causes of the cough [9].

Chan et al. [10] investigated the prevalence of chronic cough, the severity of cough, and associated symptoms in patients with sleep-disordered breathing (SDB). A total of 108 patients with suspected SDB participated in the survey and filled out a questionnaire form on respiratory and sleep health. The results showed that 33% of the patients with SDB had chronic cough; most were women, and most had nocturnal heartburn and rhinitis. However, there were no statistically significant differences in the Epworth sleepiness scale, respiratory disturbance index, BMI, dyspnea, wheezing, snoring, dry mouth, or asphyxia between the patients with and without cough.

Sundar et al. [11] performed a retrospective analysis of patients with chronic cough from a community pulmonary center in 2010. The analysis included 75 patients with chronic cough, and most of them had GERD, UACS, and CVA; none had common causes of cough such as smoking, chest radiographic abnormalities, pulmonary function abnormalities, and structural lung disease. Thirty-eight patients underwent PSG, and 33 were found to have OSA (mean BMI, 32 kg/m^2; mean age, 57 years; female: male ratio, 1.3:1). However, OSA was considered to be the sole etiology of chronic cough for only one of these patients. The cough improved or even disappeared in 27 patients after intervention for the OSA and GERD, UACS, and CVA.

Sundar et al. [12] also conducted a prospective study of 28 patients with chronic cough. Twenty-two patients with suspected OSA were screened out by a questionnaire survey and nocturnal oximetry. Nineteen patients were confirmed to have OSA by sleep apnea monitoring using PSG (mean age, 58.7 years; female:male ratio, 2.17:1). Therefore, the prevalence of OSA in the patient population with chronic cough was 68%. Patients with both chronic cough and OSA were generally older and their BMI was significantly higher than that of patients with chronic cough without OSA. Cough symptoms were significantly improved after CPAP and symptomatic treatment for UACS, GERD, and asthma.

Wang et al. [13] performed a retrospective study of 131 patients who visited the sleep center of a university hospital during a 2-year period. They found that the prevalence of chronic cough was higher in patients with than without OSA (39.4% vs. 12.5%, respectively). Multiple regression analysis showed that GERD was a risk factor for chronic cough in patients with OSA. Twelve of 18 patients with OSA who underwent CPAP therapy showed remission of their cough; the cough was resolved in only two of 21 patients who did not undergo CPAP.

Discussion

The main symptoms of OSA include snoring, nighttime awakenings, and daytime sleepiness. Chronic cough was recently found to be the main and even sole symptom of OSA in some patients.

Pathogenesis of OSA-induced chronic cough

The pathogenesis of OSA-induced chronic cough is presently unclear, but may be related to the following factors: (1) Patients with OSA exhibit mouth breathing while snoring at night; this readily causes chronic inflammation of the pharynx and induces upper respiratory tract infection [16], which in turn become risk factors for chronic cough [17]. In the present study, two patients had a medical history of upper respiratory tract infection with aggregated cough. (2) Cough, snoring, and OSA can cause abnormalities in the soft palate, uvula, and upper airway mucous membrane structure, as well as neural reflexes [18]. These changes increase the cough reflex sensitivity, which may also be a cause of chronic cough. The patients in this study did not undergo cough sensitivity testing, and their cough sensitivity before and after treatment was unknown. Whether upper airway abnormalities stimulate cough receptors remains unclear. (3) Patients with OSA have airway inflammation and increased sputum neutrophil levels [19], consistent with our observation in the present study (the sputum neutrophil level was >50% in all three patients). Moreover, patients with OSA have increased concentrations of airway inflammatory mediators [20], decreased levels of inflammatory factors in the exhaled concentrate after CPAP therapy, and relieved airway inflammation [21]. (4) OSA may be associated with an increased incidence of rhinitis [22]. In patients with OSA, nasal disease is involved in the development of chronic cough by the same mechanism as UACS. Two patients in the present study had mild sinusitis. In Case 1, symptomatic treatment had no significant effect, while the cough was resolved after CPAP therapy; this suggests that sinusitis was not the main cause of cough in these two patients. (5) A large proportion (60-70%) of patients with OSA also have gastroesophageal reflux [23]. GERC is a common cause of chronic cough. Apnea repeatedly occurs in patients with OSA during nighttime sleep, with recurrent large fluctuations in intrathoracic pressure [24]. CPAP therapy can improve the negative intrathoracic pressure and increase the lower esophageal sphincter tone, thereby alleviating the gastroesophageal reflux and associated cough in patients with OSA.

Clinical features of OSA-induced chronic cough

OSA is often misdiagnosed in female patients because of their atypical symptoms [4]. Considering the cases in the literature and the three cases in the present report, we found that OSA-induced chronic cough is more common in middle-aged women with a relatively high BMI.

Based on previous case reports, epidemiological studies, and the cases in the present study, OSA-induced chronic cough is most common in middle-aged and older women, patients who are obese, and patients who snore. Chronic cough can be regarded as the primary or sole symptom of OSA. It usually manifests as a dry cough that occurs both day and night. Patients often wake up during the night because coughing disturbs their sleep, and the cough may be associated with a burning sensation of the stomach or throat discomfort [10]. The cell counts in induced sputum, lung function, and fractional exhaled nitric oxide concentration are mostly normal, and many patients have been misdiagnosed with common causes of chronic cough such as asthma/CVA, GERD, and UACS. CPAP therapy is effective in relieving cough. OSA-induced chronic cough often develops in middle-aged and older women possibly because the incidence of OSA increases with age [14], and this increasing trend is significant in postmenopausal women [15]. Epidemiological studies show that many patients with OSA (33.0-39.4%) have chronic cough [10, 13], and CPAP therapy can relieve cough symptoms. Meanwhile, other epidemiological studies show that the prevalence of OSA is high (up to 68%) in patients with chronic cough patients; most such patients have UACS, GERD, and CVA.

Diagnosis

OSA-induced chronic cough is relatively rare. First, it is important to raise awareness of the diagnosis. For patients with risk factors for OSA, such as obesity and chin retrusion, it is necessary to obtain a detailed medical history, perform a physical examination, and arrange PSG. When common causes are excluded in the diagnostic procedure recommend by existing guidelines for management of chronic cough, or when the cough is not relieved or does not completely disappear after treatment of common causes, we should consider the possibility of OSA as the independent or common cause of chronic cough and perform the corresponding examination in a timely manner. A definitive diagnosis can be made if PSG indicates the presence of OSA and the cough is relieved or disappears after CPAP or bilevel positive airway pressure ventilation.

Treatment of OSA-induced chronic cough

Either CPAP or bilevel positive airway pressure treatment is the preferred option for OSA-related chronic cough. The existing literature and the present case reports suggest that cough symptoms can be significantly improved after 1 to 2 days of treatment; however, it takes 5 days to 6 weeks for these symptoms to completely disappear, and great individual differences are observed among patients. In Case 1 of the present study, CPAP therapy for 1 day effectively resolved the cough symptoms. In Case 2, CPAP therapy for 1 week also achieved a marked effect in resolving chronic cough. The mechanism of CPAP therapy for OSA-related chronic cough may involve the following: (1) CPAP therapy maintains airway patency and prevents upper airway collapse and wallowing tongue, reducing stimulation of local cough receptors; (2) it increases intrathoracic and esophageal pressure and blocks or alleviates gastroesophageal reflux, improving cough symptoms; and (3) it corrects intermittent hypoxia and reduces mechanical airway damage caused by repeated airway obstruction, inhibiting airway inflammation. The course of treatment refers to the treatment for OSA, and long-term maintenance therapy is recommended.

Conclusion

OSA is a newly recognized cause of chronic cough, and its mechanism has not been elucidated. However, the prevalence of OSA is relatively high, and epidemiological studies show a high incidence of chronic cough in patients with OSA. Therefore, it is necessary to enhance awareness of OSA-related chronic cough. OSA should be considered in patients with unexplained chronic cough. In particular, patients with obesity, a chunky neck, and mandibular retrusion should be asked about symptoms such as snoring, apnea, nighttime awakenings, dizziness, and daytime sleepiness and should

undergo a detailed physical examination. If the above features are observed, OSA-related chronic cough should be highly considered. PSG is useful in the diagnosis of OSA, and non-invasive ventilation therapy can be used after obtaining a definitive diagnosis. Caution should be taken to avoid misdiagnosis.

Disclosure of conflict of interest

None.

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