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Case series and reports

Treatment-emergent central sleep apnoea after surgery for obstructive sleep apnoea

Apnee notturne centrali post chirurgia disostruttiva delle prime vie aeree nei pazienti affetti da OSAS

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SUMMARY

Central sleep apnoea (CSA) is a lack of drive to breathe during sleep, which can occur in physiologic as well as in pathologic conditions. A particular type of CSA, defined treatment-emergent CSA (TECSA), may occur after the treatment of obstructive sleep apnoea syndrome (OSAS), either with CPAP or surgery. TECSA is transitory and seems to be related to the severity of OSAS. We describe a 51-year-old man affected by severe OSAS who developed severe, transient CSA immediately after upper airways surgery. We believe that CSA was triggered by the sudden variation in nocturnal arterial PCO_2, which decreased from 52.3 mmHg before surgery to 42.0 mmHg after surgery. It is conceivable that, due to long-lasting severe OSAS, our patient lowered his chemosensitivity to PCO_2. Consequently, the resolution of obstructive apnoeas and the restoration of normal nocturnal values of PCO_2 may have reduced the nocturnal PCO_2 to the point of being inadequate to stimulate ventilation.

KEY WORDS: Treatment Emergent Central Sleep Apnoea • OSA • CSA • Complex Sleep Apnoea • UPPP • PCO_2

RIASSUNTO

L'Apnea Centrale nel Sonno (CSA) è caratterizzata da un mancato input a compiere l'atto respiratorio durante il sonno e può verificarsi sia in condizioni fisiologiche che in condizioni patologiche. Un particolare tipo di CSA, definito come "Apnea Centrale nel Sonno Emergente dal Trattamento" (TECSA), può verificarsi dopo il trattamento della Sindrome delle Apnee Ostruttive durante il Sonno (OSAS), sia dopo ventiloterapia con CPAP che dopo intervento chirurgico. La TECSA è di solito transitoria e sembra correlata alla gravità dell'OSAS. Descriviamo un uomo di 51 anni affetto da OSAS grave, che ha sviluppato gravi e transitorie apnee centrali nel sonno, immediatamente dopo l'intervento chirurgico delle vie aeree superiori. Ipotizziamo che la CSA sia stata innescata dalla brusca variazione dei valori notturni di PCO2 arteriosa, passata da 52,3 mmHg prima dell'intervento chirurgico a 42,0 mmHg dopo l'intervento chirurgico. È ipotizzabile che, a causa della lunga durata e della severità dell'OSAS, il paziente abbia sviluppato una bassa chemiosensibilità alla PCO2 arteriosa. Di conseguenza, la risoluzione delle apnee ostruttive e il ripristino dei valori normali notturni di PCO2, potrebbero aver ridotto i valori di PCO2 notturna fino al punto di essere inadeguati per stimolare la ventilazione.

PAROLE CHIAVE: *Apnea Centrale nel Sonno Emergenti dal Trattamento • OSA • CSA • UPPP • PCO_2*

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Introduction

Central sleep apnoea (CSA) is characterised by a lack of drive to breathe during sleep. CSA can occur in a variety of physiologic and pathologic conditions, which include sleep at high altitude, drug intake, neuromuscular disorders, congestive heart failure, idiopathic CSA; moreover, CSA can be associated with obstructive sleep apnoea syndrome (OSAS) [1].

The present report concerns the polysomnographic findings in a 51-year-old man affected by severe OSAS, who developed severe, transient CSA immediately after upper airway surgery for OSAS.

Case report

A 49-year-old man referred to our sleep centre for symp-

toms of OSAS. No remarkable medical history was reported; in particular, neurologic, heart and respiratory diseases were ruled out, and he took no drugs. Body mass index (BMI) was 27.8 kg/m^2, neck circumference was 42.5 cm, Mallampati class IV. Spirometry and pulmonary function tests were normal. He underwent full-night, laboratory based nocturnal video-polysomnography (PSG). PSG montage included three EEG leads, two EOGs, submental PNG, chest and abdominal respiratory effort, airflow (nasal cannula), EKG, SpO_2, body position, snoring, audio and video recording. PSG showed apnoea-hypopnoea index (AHI) = 40 events/hour, and oxygen desaturation index (ODI) = 41.8 events/hour. The index of central events was 4.6. Treatment with positive airways pressure was started (CPAP = 12 cmH_20). CPAP was titrated to suppress all sleep-related pathologic respiratory events. The patient came for follow-up evaluation 24 months later, complaining of poor compliance with CPAP and persistence of symptoms, despite the use of a humidifier and the adoption of alternatively a nasal and a facial mask. A trial with a BiPAP was also attempted, without satisfactory results. He underwent a pre-surgical evaluation protocol. BMI and neck circumference were unmodified. Arterial blood gas analysis was performed in the morning, within minutes from awakening, while breathing room air. Gas analysis showed PCO_2 = 52.3 mmHg, PO_2 = 90.2 mmHg, pH = 7.4. Sleep endoscopy showed three sites of obstruction: nasal (hypertrophy of inferior turbinates); retro palatal-oropharyngeal (total antero-posterior collapse); and laryngeal (collapse of the epiglottis on the lumen of larynx). Therefore, he underwent a surgical procedure which included mucotomy with radiofrequency, uvulopalatopharyngoplasty (UPPP) and partial epiglottectomy. No surgical complications occurred. The patient presented no clinically relevant post-operative pain, and no narcotic therapy was administered. PSG performed three days after surgery (to rule out possible residual effect of anaesthetics); PSG showed a clear reduction of the obstructive events, but a striking increase of the central apnoea index (Fig. 1). At this time, the patient reported no symptoms and other subjective complaints. Central apnoeas progressively decreased in follow-up PSGs, whereas the index of obstructive+mixed events remained stable. PSG results, apnoea-hypopnoea and oxygen desaturation indexes and values of arterial blood gas analysis are reported in Table I.

Discussion

A particular type of CSA, defined treatment-emergent central sleep apnoea (TECSA) and previously known as complex sleep apnoea, has been reported during the titration CPAP in patients with OSAS [2]. In a large retrospective study of patients with OSAS, 6.5% had CPAP emer-

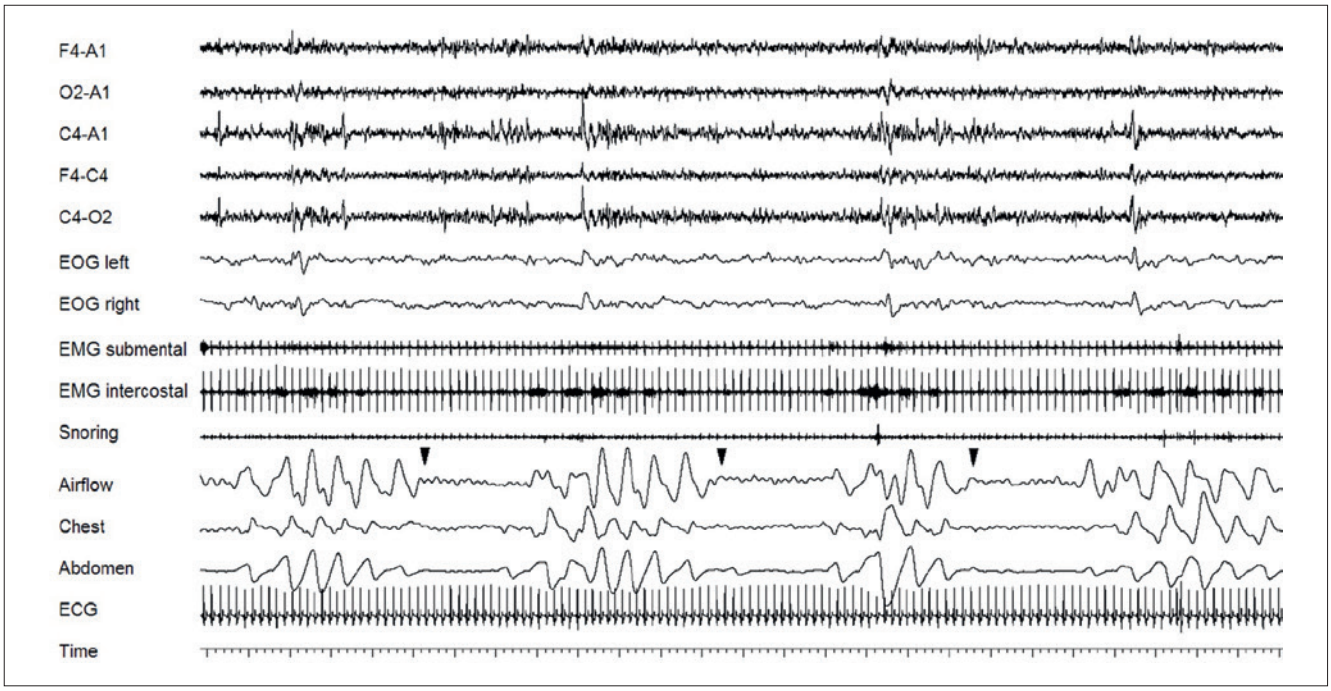

Fig. 1. PSG recording (about 2 minutes) obtained during night #3 postsurgery. Three central apnoeas are shown (arrows).

Table I. Polysomnography and arterial blood gas analysis before and after surgery.

	Before surgery	After surgery			
Polysomnography		**Day #3**	**Day #15**	**Day #30**	
Total Sleep Time	435	331	505	399	Minutes
Sleep Efficiency Index	96,6	95,8	88,9	98,2	%
Total Apnea-Hypopnea Index	48,2	71,2	21	14,6	Events/hour
Central events	4,6	64,1	10,4	4,7	Events/hour
Obstructive + Mixed events	43,6	7,1	10,6	9,9	Events/hour
AHI supine	45,5	69,1	18,6	27,1	Events/hour
AHI lateral	6,6	68,1	5,4	3,1	Events/hour
Oxygen Desaturations Index	42	72	21	26	Events/hour
Basal SpO_2	95	96	96	97	%
Lowest SpO_2	76	75	76	74	%
Arterial blood gas analysis					
PCO_2	52	42		42	mmHg
PO_2	91	80		88	mmHg
pH	7,41	7,46		7,42	
Bicarbonates	25,8	29,9		27,2	mol/l
Base excess (blood)	2	5,4		2,4	mol/l
Base excess (extra-cellular fluid)	1,9	6,1		2,7	mol/l

gent complex sleep apnoea; however, it was generally transitory and was eliminated within eight weeks of CPAP therapy [2]. The occurrence of CSA during CPAP treatment is related to the severity of OSAS [2]. TECSA has also been occasionally described after the surgical treatment of OSAS, in particular with tracheostomy [3] and maxillomandibular advancement [4]. Central sleep apnoea has not been reported following palatal or pharyngeal surgery [5-7].

The pathogenesis of CSA after the resolution of OSAS is unclear. CSA during sleep is due to abnormalities in ventilation control mechanisms, and ventilation during sleep, in particular during NREM sleep, is entirely dependent on the metabolic control systems. This means that the ventilatory drive during NREM sleep is dependent on the fluctuations of the levels of arterial PCO_2.

We believe that, in our patient, transient, self-limiting CSA was triggered by the sudden variation of nocturnal PCO_2 that occurred immediately after surgery in a patient with reduced chemosensitivity due to long-term exposure to nocturnal hypercapnia. In fact, values of arterial PCO_2 on awakening decreased from 52.3 (before surgery) to 42.0 mmHg (after surgery). Severe OSAS may be associated with high levels of nocturnal arterial PCO_2, due to sleep disordered breathing itself, even in the absence of other respiratory impairment [8]. This, in turn, can result in reduced sensitivity of brainstem chemoreceptors to PCO_2. The resolution of sleep-related obstruction and restoration of normal nocturnal values of PCO_2 might have decreased the response of brainstem chemoceptors to PCO_2 to the point of apnoea [1]. In other words, the levels of arterial PCO_2 became inadequate to stimulate ventilation during sleep.

The loop-gain model provides a good explanation for breathing instability in our patient [9]. Loop gain is an engineering term used to quantify the gain of a system controlled by feedback loops. Respiration is in fact such a system. According to the model, a strong interaction occurs between airway caliber and ventilatory stability. It can be hypothesised that, before surgery, our patient presented a low critical pressure for upper airways closure and, consequently, obstructive apnoeas occurred. After surgery, which reduced upper airways resistance, our patient presented a high loop gain (due to reduced sensitivity to CO_2) and, consequently, respiratory instability. Some weeks later, after a progressive adaptation to the new levels of nocturnal PCO_2, CSA progressively decreased.

In conclusion, our observation suggests that PSG monitoring may be useful, in the days immediately after surgical intervention, in order to recognise CSA. It seems conceivable that routine assessment of complex sleep apnoea should be performed after surgery at least in patients with preoperative evidence of nocturnal hypercapnia. Moreover, monitoring the levels of arterial PCO_2 may help to predict, and eventually prevent, the occurrence of CSA. In our patient, no symptoms of central apnoeas were re-

ported, the condition was self-limiting and no specific treatment was needed.

Conflict of interest statement

None declared.

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