

Obstructive sleep apnea in women: Prevalence, risk factors and relation to menopausal status



Apneia obstrutiva do sono nas mulheres: Prevalência, factores de risco e relação com a menopausa

Dear Editor,

Obstructive sleep apnea (OSA) is two to five times more prevalent in men than women but this gender-related difference is attenuated after menopause.¹⁻⁴ Many of the hypotheses that explain the gender differences in OSA are based on the hormonal dissimilarities between men and women and the protective effect of circulating progesterone in women. So, one would expect that postmenopausal women with OSA would benefit from hormonal replacement therapy (HRT) but studies done so far are based on small series and still inconclusive.^{2,5}

The aim of this study was to describe the prevalence, severity and risk factors for OSA in women, particularly menopause and HRT. We performed a retrospective analysis of women submitted to overnight polysomnography (PSG) at the Sleep Laboratory of Coimbra's University Hospital between 2004 and 2013. Clinical reports were reviewed and pertinent clinical data retrieved, including Body Mass Index (BMI), sleep-related complaints, comorbidities and previous medication. Menopausal status, according to the WHO classification, and other gynecological details were attained by a preset telephone questionnaire.

From a total of 745 consecutive female patients, 403 met the inclusion criteria of this study, 55.3% of which had OSA. Univariate analysis showed that OSA women were significantly older ($p=0.000$), had more frequent complaints of nycturia and dry mouth ($p<0.025$), more cardiovascular risk factors ($p=0.000$) and a higher risk for overweight/obesity, diabetes, arterial hypertension and dyslipidemia ($p<0.032$). Women without OSA had more daytime sleepiness ($p=0.012$), which could be explained by the higher risk for depression and/or anxiety ($p=0.001$). After menopause, women with OSA were older ($p=0.000$), had more frequent complaints of nycturia ($p=0.000$) and a two-to-fourfold higher risk for diabetes, dyslipidaemia and arterial hypertension ($p<0.037$) but had no significant polysomnographic differences compared to premenopausal women. AHI was significantly higher for postmenopausal women without HRT compared to those with HRT, but there were no relevant symptomatic differences between the two groups (Table 1). Multiple regression showed that age and BMI independently predicted OSA while depression and/or anxiety was negatively correlated with the risk of OSA. No significant association between OSA and menopause or HRT could be found in multivariate analysis adjusted for age.

In conclusion, age and BMI were the two main independent risk factors for OSA in women. Menopause was associated to OSA as part of the aging process itself, which has been corroborated by previous studies that showed a higher prevalence of OSA after menopause.^{2,3} HRT showed no benefits for decreasing the risk for OSA. Clinical presentation was similar between women with or without OSA.

Table 1 Comparison of descriptive variables among women submitted to overnight polysomnography between January 2004 and December 2013.

Variable	All women			OSA women			OSA postmenopausal women		
	OSA n=223	Non-OSA n=180	p-Value	Premenopausal n=64	Postmenopausal n=159	p-Value	w/HRT n=13	w/o HRT n=146	P-Value
Age, years	57.4 ± 10.1	49.2 ± 11.2	0.000	48.4 ± 10.5	61.0 ± 7.3	0.000	54.9 ± 5.6	61.6 ± 7.2	0.002
AHI, events/h	23.9 ± 25.5	1.1 ± 1.3	0.000	21.5 ± 24.9	24.9 ± 25.8	ns	15.4 ± 13.5	25.7 ± 26.5	0.026
SpO ₂ < 90%, min	27.3 ± 54.7	3.2 ± 17.0	0.000	18.7 ± 37.0	30.7 ± 60.1	ns	5.6 ± 9.5	33.0 ± 62.2	ns
ODI, events/h	17.0 ± 17.1	2.7 ± 8.7	0.000	13.3 ± 14.0	18.5 ± 18.0	0.038	12.5 ± 13.2	19.1 ± 18.3	ns
Lowest SpO ₂ , %	80.2 ± 10.2	91.0 ± 5.4	0.000	81.6 ± 10.9	79.6 ± 9.9	ns	79.7 ± 8.1	79.6 ± 10.0	ns
BMI, kg/m ²	32.9 ± 8.2	28.6 ± 5.6	0.000	33.1 ± 7.8	32.8 ± 8.3	ns	34.5 ± 15.3	32.6 ± 7.5	ns
ESS, score	9.4 ± 6.5	8.8 ± 6.7	ns	9.7 ± 6.2	9.3 ± 6.6	ns	10.3 ± 7.1	9.2 ± 6.6	ns
OSA-related symptoms, n	4.6 ± 2.2	4.6 ± 2.1	ns	4.4 ± 2.2	4.7 ± 2.2	ns	4.3 ± 2.0	4.8 ± 2.2	ns
Cardiovascular risk factors, n	0.9 ± 0.2	0.8 ± 0.4	0.000	0.9 ± 0.3	1.0 ± 0.2	ns	2.1 ± 1.0	2.0 ± 1.1	ns

OSA: obstructive sleep apnea; HRT: hormonal replacement therapy; w/: with; w/o: without; AHI: apnea-hypopnea index; SpO₂: oxygen peripheral saturation; ODI: oxygen desaturation index; BMI: body mass index; ESS: Epworth sleepiness scale; ns: not significant. Values are displayed as mean ± standard deviation.

References

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328:1230–5. <http://dx.doi.org/10.1056/NEJM199304293281704>.
2. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A, et al. Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med*. 2001;163 3 Pt 1:608–13. <http://dx.doi.org/10.1164/ajrccm.163.3.9911064>.
3. Dancey DR, Hanly PJ, Soong C, Lee B, Hoffstein V. Impact of menopause on the prevalence and severity of sleep apnea. *Chest*. 2001;120:151–5. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/11451831> [accessed 13.01.14].
4. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep-disordered breathing in the Wisconsin Sleep Cohort Study. *Am J Respir Crit Care Med*. 2003;167:1181–5. <http://dx.doi.org/10.1164/rccm.200209-1055OC>.
5. Manber R, Kuo TF, Cataldo N, Colrain IM. The effects of hormone replacement therapy on sleep-disordered breathing in postmenopausal women: a pilot study. *Sleep*. 2003;26:163–8. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12683475> [accessed 13.01.14].

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Diagnosis of pulmonary sequestration using imaging methods



Diagnóstico de sequestro pulmonar usando métodos de imagiologia

Dear Editor:

Pulmonary sequestration (PS) is a rare congenital malformation. Several reports describe preoperative diagnostic difficulties with this pathology.^{1–3} The authors report a case of intralobar sequestration (IS) and intend to draw attention to the importance of imaging modalities, including Computed Tomography (CT) with contrast for the diagnosis, in order to reduce the number of patients sent for surgery without an accurate diagnosis.

IS corresponds to 75–90% of all sequestrations consisting of non-functioning lung tissue that falls within the normal lung parenchyma without its own visceral pleura; it is usually unrelated to the tracheobronchial tree. It is irrigated by an abnormal systemic artery derived mainly from the aorta. About 20% of cases are irrigated by more than one artery.^{1–3}

IS presents frequently in adolescents and young adults, but rarely in adults over 40 years, with recurrent infection and hemoptysis; 15% of patients are asymptomatic.^{1,2}

The diagnosis is suggested by the identification of the abnormal blood vasculature in imaging studies. The average rate of reported preoperative misdiagnosis in the literature is high. Angiography used to be considered the method of choice for preoperative diagnosis, has been replaced by non-invasive methods. Conventional CT does not consistently show anomalous arterial supply. Thus, CT with contrast has emerged as the imaging test of choice.^{1–5}

Surgical resection is the treatment of choice.^{1,2}

Case report: Female patient, Caucasian, 64 years old, house cleaner, no smoking or alcohol habits. Referred for pulmonology evaluation because she had asthenia and adynamia with one-year evolution, weight loss (13 kg in six months), a CT showing fibrosis areas, traction bronchiectasis and an encysted and loculated left pleural effusion.

In relation to antecedents, the key was anaemia of unknown cause and an episode of respiratory infection which dragged on (one year ago). She did not report any hemoptysis episodes.

A thoracic CT with contrast was performed and showed a vascular image in the internal basal segment of the left lower lobe, 47 mm × 47 mm, with at least two arteries originating from the lower thoracic aorta; this was suggestive of PS. There were also fibrosis areas and bronchiectasis (Fig. 1).

A left lower lobectomy was performed and the anomalous arteries were joined up. An anatomopathological examination showed a cavitory formation of 5 cm, with apparent communication with the bronchial tree and pulmonary condensation of the surrounding parenchyma. Microscopically, this was a cystic lesion lined with respiratory epithelium; adjacent to this, there were many dilated and mucous retention airspaces, tortuous blood vessels and wall thickening, which are consistent with IS.

A diagnostic hypothesis suggested from the first CT (without contrast) was encysted pleural effusion. The performance of CT with contrast was central to the correct diagnosis of preoperative PS.

In this case, the highlights are the unusual age of onset of clinical presentation, nonspecific and uncharacteristic symptomatology, PS irrigated by two arteries and communication with the tracheobronchial tree.