



EDITORIAL

Type I diabetes: a new risk factor for obstructive sleep apnea



Obstructive sleep apnea (OSA) is common in the general population but remains largely under recognized.¹ Risk factors for OSA include male gender, obesity and increasing age.^{1,2} There is growing evidence that OSA may cause several consequences including not only poor sleep quality and excessive daytime sleepiness but also contribute to detrimental cardiometabolic consequences.^{3,4} Therefore, recognizing populations that are at increased risk should help to detect OSA. In the present number of the Portuguese Journal of Pulmonology, Vale and colleagues provide novel evidence suggesting that type I diabetes is strongly associated with OSA.⁵ The authors used portable monitoring to evaluate the presence of sleep disordered breathing among 46 adults with diabetes, divided in 23 with type I and 23 type II diabetes. Because OSA and type II diabetes share several common risk factors such as obesity, it is not surprising that OSA was extremely frequent in this population. This finding is therefore in line with several studies and point out to the low levels of OSA recognition among patients with type 2 diabetes.³ Vale and colleagues took a step forward and showed a similar high frequency of OSA among adult patients with type I diabetes as compared with those with type II diabetes. Despite the fact that the patients with type I were not particularly obese (mean body mass index of 28 kg/m²) and were in fact much younger than those with type II diabetes (mean age of 39 vs 62 years old, respectively) the frequency of OSA was similar among the two groups and ranged from 26 to 70% depending of the apnea hypopnea index criterion for OSA definition. The study is therefore in line with recent evidence pointing out that not only type II diabetes, but also type I diabetes is associated with a high prevalence of OSA.^{6,7} Another important message of the study of Vale and colleagues was that there was no association between OSA and complaints of excessive daytime sleepiness among patients with diabetes. In contrast to patients referred to sleep laboratories, among consecutive patients

with cardiovascular diseases such as hypertension as well among those with metabolic syndrome and diabetes, the association between OSA and excessive daytime sleepiness is very poor or non-existent.^{4,8} This raises one important question: What is the real significance of an altered sleep study among patients with no overt symptoms? The current working hypothesis is that OSA is more than a common condition but when present contributes to poor cardiovascular and metabolic outcome.³ The primary mechanisms that are deleterious to the cardiovascular and metabolic system includes increased negative intrathoracic pressure during the efforts for breath against an occluded airway, arousals from sleep and intermittent hypoxia. There is growing evidence that these primary events that occur during sleep trigger a cascade of detrimental effects, including increased sympathetic activity, oxidative stress, metabolic deregulation such as insulin resistance and lipid dysfunction, endothelial dysfunction and accelerated atherosclerosis.³ Consistent with the hypothesis that OSA may contribute to poor glycaemic control, Vale and colleagues found that the AHI was significantly lower and in the normal range among patients with type II diabetes and normal glycosylated hemoglobin (HbA1c) as compared to patients with type II diabetes and elevated HbA1c. The serum level of HbA1c is known to reflect average glycemia over the preceding 2–3 months and therefore one possible conclusion is that OSA contributed to poor glycaemic control. The interpretation of such cross sectional data is, however, limited and the authors acknowledged that due to the relatively small sample they were under powered to correct for confounding variables. There is some evidence that OSA treatment promotes better glucose control in type 2 diabetes.⁹ Future randomized studies are necessary in order to evaluate whether treatment of OSA contributes to a better control of type I diabetes. Another important aspect that the study of Vale and colleagues raises is the relationship between type I diabetes and sleep. It must be stressed that type I diabetes and glycaemic control may affect circadian rhythms, sleep duration and quality of sleep.¹⁰ Therefore the interaction of such factors may go far beyond OSA. One attractive hypothesis

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is that diabetes may cause subtle alterations in the upper airway function. Consistent with this hypothesis, diabetes was recently associated with decreased nasal mucociliary function.¹¹ Although not directly related to OSA pathogenesis, it raises the hypothesis that other functions such as upper airway neuro reflexes that are important to stabilize the upper airway during sleep are impaired among patients with long lasting type I diabetes. Recent studies showed that OSA was independently associated with macrovascular complications, retinopathy as well as cardiovascular autonomic neuropathy among patients with type I diabetes.^{7,12} Therefore, OSA could also be seen as a complication of diabetes. Independent of the exact mechanism linking type I diabetes to OSA, the study of Vale and colleagues contribute to the hypothesis that OSA should be actively searched not only among patients with type II diabetes but also among those with type I diabetes.

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