Patients with obstructive sleep apnoea (OSA) have a higher incidence of cardiovascular morbidity and mortality\textsuperscript{1,2}. Recent data suggest that OSA may be associated with a number of cardiovascular risk factors, such as hypertension, insulin resistance, impaired glucose tolerance, and dyslipidaemia, which together comprise the metabolic syndrome (MS)\textsuperscript{1,3}. A growing recognition of the presence of various metabolic abnormalities in subjects with OSA has been observed during the past two decades, and the association of OSA and MS was highlighted as “syndrome Z” in the late 1990s\textsuperscript{4}. There are multiple potential mechanistic pathways potentially involved in the interaction between OSA and MS. Chronic intermittent hypoxia and sleep fragmentation with sleep loss present in OSA can lead to generation of reactive oxygen species and neurohumoral changes, respectively. These key triggers likely initiate or contribute to a low grade inflammation, a prominent phenomenon of OSA and a shared feature with MS\textsuperscript{1,3,5,6}. Furthermore, intermittent hypoxia and oxidative stress have been implicated in the upregulation of the transcription factors related with the sterol regulatory element binding protein (SREBPs), contributing to the development of hyperlipidaemia\textsuperscript{7,8}, an abnormality seen in MS. Despite the rather prolific data that suggest a contributing role of OSA towards the various components of MS and the entity itself, the exact relationship between OSA and MS remains controversial, since obesity constitutes a powerful confounding factor. Some studies have already showed that OSA is associated with metabolic abnormalities even in non obese patients\textsuperscript{9}, however, others have shown that obese OSA patients may have an increased rate of MS and more pronounced metabolic dysfunction\textsuperscript{10,11}. Prevalence of MS in OSA patients is high, varying approximately between 60 and 90 per cent\textsuperscript{12-15}. However, this prevalence differs according to MS diagnostic criteria applied and type of the populations studied concerning demographics (age, gender, ethnicity, tertiary and primary care health services) and OSA features, such as severity, determined by apnoea-hypopnoea index (AHI), respiratory disturbance index (RDI) and desaturation index (DI).

In this issue Agrawal et al\textsuperscript{16} contribute to our understanding of the burden of MS in OSA patients in a hospital-based population of a tertiary health care centre in New Delhi, India. In this prospective cross-sectional study, the authors have analyzed 227 consecutive patients who underwent an overnight 16-channel polysomnography for evaluation of excessive daytime somnolence and snoring. From the total of patients enrolled 187 (82\%) had OSA. Anthropometry, body composition analysis, blood pressure (BP), fasting blood glucose, insulin resistance by homeostasis model assessment (HOMA-IR) and fasting blood lipid profile measures were performed. They found that diastolic BP, fasting plasma insulin, HOMA-IR, waist circumference and waist-hip ratio were higher in patients with OSA, with a trend towards higher systolic BP, fasting blood glucose, triglycerides and LDL cholesterol. Body composition analysis showed higher fat mass, per cent body fat and skin fold thicknesses in patients with OSA. These findings are in accordance with previous studies showing OSA to be associated with higher BP, insulin resistance and deranged lipid profile and body composition\textsuperscript{13,17-19}.

MS was defined using the National Cholesterol Education Program Adult treatment panel III criteria\textsuperscript{20}. Prevalence of MS in OSA group was 4-fold higher as compared to non-OSA group (79 versus 48\%).
Despite within the aforementioned range, Agrawal et al\textsuperscript{16} pointed out some methodological issues which could explain the different prevalences found in other studies. The lower prevalences described by Sharma et al\textsuperscript{13} (77 versus 40\%) and Lam et al\textsuperscript{13} (58 versus 21\%) can be explained by the fact that these were community-based studies, emphasizing the referral bias, and participants had a lower BMI compared to this study\textsuperscript{16}.

On the other hand, a higher prevalence was reported by Coughlin et al\textsuperscript{12} in a previous hospital-based study reporting prevalence of MS in OSA, probably due to ethnic differences of the populations (Europeans versus Asians) and a much higher BMI of participants in the study by Coughlin et al. In another study, Mota et al\textsuperscript{14} found 63.5 per cent prevalence of MS in an OSA tertiary hospital population. Once again methodological heterogeneity, concerning main objectives, sample size and existence of a control group, can be used to explain the differences found.

Agrawal et al\textsuperscript{16} also showed that prevalence of MS increased with severity of OSA (mild: 66\%; moderate: 72\%; severe: 86\%), suggesting the positive association between the severity of OSA and the presence of MS, already described in previous studies\textsuperscript{14,21}. However, as Agrawal et al\textsuperscript{16} fairly signaled, a causative relationship between OSA and MS cannot be established since obesity, a major risk factor for both conditions, that acts as a significant confounder, was not matched in both groups in the present study.

In conclusion, the scientific evidence provided by Agrawal & colleagues\textsuperscript{16}, as the first prospective hospital-based study performed in Asia which enrolled a large sample size, is of an additional value for supporting the higher prevalence of MS and its components in OSA patients, and the importance of ruling out presence of MS in OSA and vice versa, in order to an early detection and adequate treatment of both conditions. Matching for obesity in future studies will be important to better clarify the interaction between sleep disturbance and metabolic abnormalities in Asian populations.

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