

Undiagnosed sleep apnoea syndrome in patients with acute myocardial infarction: Potential importance of the STOP-BANG screening tool for clinical practice

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To the Editor:

Sleep apnoea hypopnoea syndrome (SAHS) has been identified as an independent risk factor for adverse cardiovascular events in patients admitted with an acute coronary syndrome (ACS) [1]. Most people with SAHS, including those with ACS, are undiagnosed. Konecny and colleagues [2] reported a prevalence of previously diagnosed or suspected SAHS (specifically obstructive sleep apnoea) of 14% among 74 patients with acute MI. However, the frequency of SAHS defined by overnight polysomnography (PSG) was 69%, more than half of whom had PSG findings indicative of moderate–severe SAHS.

Whilst PSG is the gold standard for the diagnosis of SAHS, it is time consuming and costly to perform, and its routine application to patients with ACS is impractical. By contrast, a simple screening tool capable of stratifying patients into high and low risk groups for SAHS could potentially be applied to this patient group. The STOP-BANG screen for SAHS comprises four binary questions and four items, paraphrased here as: Snore?, Tiredness during daytime?, Observed apnoea?, High Blood Pressure?, Body mass index, Age, Neck circumference and Gender [3].

We applied the STOP-BANG screen to all patients admitted with acute MI within a one month period to a tertiary referral centre, an urban general hospital and a rural general hospital. A total of 135 patients were diagnosed with MI during the study period. 101 (75%) were male, the mean age was 66 ± 14.6 years, and the mean body mass index (BMI) was 28 ± 5.7 kg/m². There was no significant difference in these variables between the three centres. A STOP-BANG score suggestive of SAHS (≥ 3) was present in 100 (74%) patients. The two patients who had a pre-existing diagnosis of SAHS both had a STOP-BANG score suggesting high risk of SAHS (6,7). The proportion of significant scores was similar across centres ($P=0.88$). A significant score was more common in males compared with females (83% v 47%; $\chi^2 = 17.3$, $P < 0.001$) and this was not explained by BMI.

In this study of patients presenting with ACS to UK hospitals, 74% of those with confirmed MI had a STOP-BANG score suggestive of pre-existing SAHS. While this proportion appears surprisingly high, our data are consistent with those from two recent PSG-based studies of patients with acute MI. In these studies, the prevalence of SAHS defined by an apnoea hypopnoea index (AHI) > 5 , was 68.9% and 74.9%, respectively (Fig. 1) [2,4].

For patients with an AHI > 5 the sensitivity of the STOP-BANG for SAHS is 83.6%, rising to 92.9% for those with an AHI > 15 [3]. In the present study population, the STOP BANG is likely to have identified the majority of patients across the spectrum of SAHS severity but with greatest sensitivity implied for those with moderate–severe SAHS. That only two of our patients who had significant STOP-BANG scores had a prior diagnosis of SAHS reinforces the finding of Konecny and colleagues [2] that this potentially important underlying condition is not well recognised. The high sensitivity of the STOP-BANG makes it a potentially useful screening tool for SAHS and its simplicity means that it is ideal for use in patients presenting with acute MI. Indeed, we were able to apply the STOP-BANG to all patients who presented with MI whereas studies utilising PSG have generally excluded patients with co-morbidities such as diabetes [4], COPD, stroke and valvular heart disease [5], and concomitant treatment with sedatives and narcotics [5].

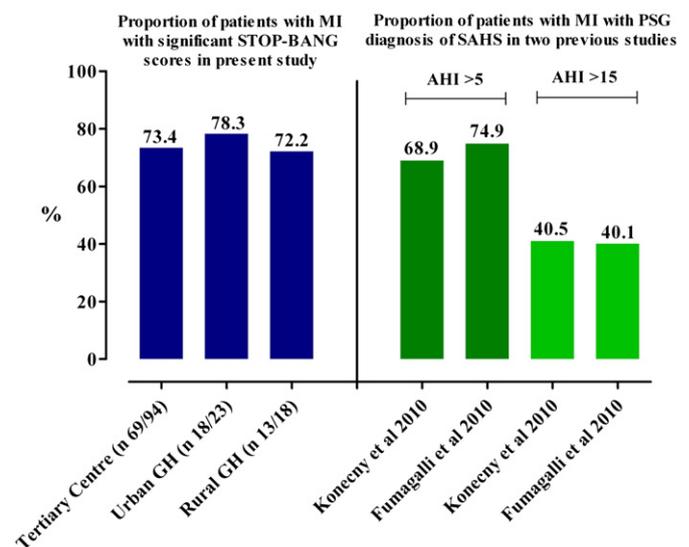


Fig. 1. Proportion of patients presenting with confirmed MI who had STOP-BANG scores (≥ 3) suggestive of SAHS. Also provided for comparison are data published in two studies (2, 4), indicating the proportion of patients presenting with MI who had PSG confirmed mild (AHI ≥ 5) and moderate–severe (AHI ≥ 15) SAHS (specifically obstructive sleep apnoea). MI: myocardial infarction. PSG: polysomnography. AHI: apnoea hypopnoea index.

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The predecessor to the STOP-BANG screen was the Berlin Sleep Questionnaire (BSQ). When applied to 200 patients with ACS (25% of whom had unstable angina), 47% had scores indicative of SAHS (obstructive sleep apnoea) [1]. The lower prevalence of SAHS in this study compared with our own was likely accounted for by differences in sensitivity of the BSQ [3] and by the broader inclusion criteria of ACS in the BSQ-based study. The AHI is reportedly comparable between patients with a diagnosis of MI compared to unstable angina, although the central apnoea index might be higher in patients with MI [5].

We have found a high prevalence of SAHS in our study of MI patients despite the relatively modest mean BMI (28 kg/m²). In Fumagalli et al.'s study [4], the prevalence of SAHS was similar at 74.9% despite a mean BMI of 26.6 kg/m². They report that body weight but not BMI correlated significantly with AHI [4]. Obesity is associated with greater neck circumference and upper airway obstruction ('obstructive apnoea'), whereas the mechanism underlying 'central apnoea' is less dependent on obesity. Both apnoea patterns are found in patients with ACS [5] and there is evidence to suggest that the frequency of central apnoeas is reduced six months after the acute event while the frequency of obstructive apnoeas remains unchanged. It has been postulated therefore that obstructive apnoeas are a risk factor for MI whereas central apnoeas are a consequence of MI [5].

Several pathophysiological mechanisms could explain a causal link between SAHS and ACS, but hypoxia is the most straightforward and provides a potential target for therapeutic intervention. We suggest that the STOP-BANG screen is used to bridge the gap between the failure to recognise patients with SAHS and the practical constraints of PSG. Its ready application at the bedside provides for a standardised approach to the identification of a presently undiagnosed group of patients whose prognosis might be improved by treatment for SAHS.

References

- [1] Jesus EV, Dias-Filho EB, Mota M, B, et al. Suspicion of obstructive sleep apnea by Berlin Questionnaire predicts events in patients with acute coronary syndrome. *Arq Bras Cardiol* 2010;95:313–20.
- [2] Konecny T, Kuniyoshi FHS, Orban M, et al. Under-diagnosis of sleep apnea in patients after acute myocardial infarction. *J Am Coll Cardiol* 2010;56:742–3.
- [3] Chung F, Elsaid H. Screening for obstructive sleep apnea before surgery: why is it important? *Curr Opin Anaesthesiol* 2009;22:405–11.
- [4] Fumagalli S, Tarantini F, Cipriani C, et al. Obstructive sleep apnea after myocardial infarction. *Int J Cardiol* 2010;145:550–2.
- [5] Bahammam A, Al-Mobeireek A, Al-Nozha M, Al-Tahan A, Binsaeed A. Behavior and time-course of sleep disordered breathing in patients with acute coronary syndromes. *Int J Clin Pract* 2005;59:874–80.

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Modified criteria for determining cardiometabolic syndrome in Asian Indians living in the USA: Report from the diabetes among Indian Americans national study

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The disproportionate burden of cardiovascular disease (CVD) in the Asian Indian (AI) diaspora is well documented but not well understood [1–4]. Foucan et al. found AIs with type-2 diabetes mellitus (DM) had a 5-fold higher risk of cardiometabolic syndrome (CMetS) than the

general US population [1]. Typical risk factors (RFs) like elevated low-density lipoprotein cholesterol (LDL-C) levels do not account for the high CMetS risk. Decreased levels of high-density lipoprotein subfraction 2b (HDL_{2b}) are associated with higher rates of coronary artery disease [5]. Higher levels of abdominal adiposity, visceral fat, dyslipidemia, insulin resistance, and high-sensitivity C-reactive protein (hsCRP) have also been reported as contributory factors [6–8]. In this study we aimed to propose an explanatory model of CMetS in AIs, controlling for confounders such as demographic variables, traditional risk factors, and novel biomarkers.

As the first randomized large-scale investigation of the prevalence and risk factors for DM and CVD in AIs in the US, the Diabetes among Indian Americans study included conventional and emerging CMetS RFs [2]. The primary outcome measure was prevalence of CMetS, which the International Diabetes Federation (IDF) defines as the presence of abdominal obesity (i.e., waist circumference [WC] ≥ 35.4 in for male and ≥ 31.5 in for female South Asians) and ≥ 2 other RFs [9]. Because AIs are at high risk for CMetS, progression in CMetS RFs was differentially examined based on a proposed set of AI-specific CMetS criteria (elevated WC and ≥ 1 other RFs).

Using computer randomization, 5000 participants were selected from a database of 43,000 AIs compiled from area telephone, ethnic association, faith-based organization, and professional association

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