



Obstructive sleep apnoea in acute coronary syndrome: the invisible threat?

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In patients with acute coronary syndrome polygraphy monitoring is required to detect sleep-disordered breathing <http://ow.ly/JjZi309czta>

Cite this article as: Arzt M, Hetzenecker A, Lévy P. Obstructive sleep apnoea in acute coronary syndrome: the invisible threat? *Eur Respir J* 2017; 49: 1602539 [<https://doi.org/10.1183/13993003.02539-2016>].

Worldwide, coronary artery disease (CAD) is the single most common cause of death [1]. Over seven million people every year die from CAD, accounting for 13.2% of all deaths [1, 2]. The presentation of acute coronary syndrome (ACS) can be unstable angina, non-ST elevation myocardial infarction (NSTEMI) or ST elevation myocardial infarction (STEMI) [1, 3].

Obstructive sleep apnoea (OSA) is a common comorbidity in patients with ACS. There is increasing evidence that sleep disordered breathing is a risk factor for CAD [4, 5]. In the early phase after STEMI and NSTEMI, the heart may be in a vulnerable state, sensitive to the negative consequences of OSA, including increased cardiac workload [6] and endothelial dysfunction [7], which may ultimately lead to a mismatch between oxygen demand and supply. Despite successful percutaneous coronary intervention, patients with STEMI/NSTEMI and OSA, compared with those without OSA, have prolonged myocardial ischaemia [8], less salvaged myocardium [9], and impaired left and right ventricular remodelling [10, 11], all of which predispose to the development of heart failure [5, 12]. In addition, in patients undergoing percutaneous coronary intervention (the majority due to ACS), OSA is a significant risk factor for major adverse cardiac and cerebrovascular events (MACCEs) [13].

In this context, DE BATILLE *et al.* [14] aimed to build a predictive model for OSA in a sample of patients with ACS (n=978; 12% unstable angina, 48% NSTEMI and 40% STEMI). This analysis was an ancillary study of the randomised controlled ISAACC trial (NCT01335087), evaluating the effect of continuous positive airway pressure (CPAP) treatment on the incidence of new cardiovascular events in patients with an episode of ACS and OSA [15]. The present analysis included patients with OSA, assessed with polygraphy 24–72 h after admission [14] and defined as $\leq 50\%$ of central apnoeas and an apnoea/hypopnoea index (AHI) ≥ 15 per h recording time; patients with AHI < 15 per h served as the control group. An unknown number of patients with ACS and predominant central sleep apnoea were excluded [14].

The final adjusted logistic regression models attempting to predict OSA (AHI ≥ 15 per h and AHI ≥ 30 per h, respectively) could not establish a prediction model that would justify a validation study (AHI ≥ 15 per h: $R^2=0.09$; sensitivity 94% and specificity 24%; AHI ≥ 30 ·h⁻¹: $R^2=0.072$; sensitivity 31% and specificity 86%). Significant independent modulators of OSA (AHI ≥ 15 per h) were age, body mass index (BMI),

Received: Dec 24 2016 | Accepted: Jan 03 2017

Support statement: M. Arzt received research grants and lecture fees from ResMed (Martinsried, Germany) and Philips Respironics (Murrysville, PA, USA).

Conflict of interest: Disclosures can be found alongside this article at erj.ersjournals.com

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Epworth Sleepiness Scale, intake of calcium antagonists and peak troponin levels, while for OSA (AHI ≥ 30 per h) they were age, BMI, triglycerides, peak troponin and Killip class >1 [14].

The majority of the reported patient population had NSTEMI or STEMI [14]. During the first 24–72 h after the event, the likelihood is high that subclinical pulmonary congestion was present. During this early phase after NSTEMI or STEMI, the predominant type of sleep-disordered breathing is approximately 50% OSA and 50% central sleep apnoea [16]. In a significant proportion of patients with ACS, left ventricular function recovers after guideline-conforming treatment of ACS. Patients whose left ventricular function improves after NSTEMI or STEMI often experience a significant alleviation of their sleep-disordered breathing and OSA [16, 17]. Thus, when interpreting the predictive models reported by DE BATLLE *et al.* [14], one should take into account that polygraphy between 24 and 72 h after an ACS may be a snapshot of the severity and type of the sleep-disordered breathing and that this may vary considerably afterwards.

In accordance with previous literature, patients presenting with both cardiovascular/cerebrovascular disease and OSA have often no typical clinical presentation helping to clinically suspect OSA, *e.g.* patients with stroke [18], heart failure [19] or NSTEMI/STEMI ACS [13] usually do not report daytime sleepiness. The association of daytime sleepiness and BMI with the occurrence of OSA appears to be weaker compared with observations in community samples without known cardiac disease [14, 20].

The data reported by DE BATLLE *et al.* [14] emphasise that in patients with cardiac disease, modulators reflecting the severity of cardiac impairment, *e.g.* New York Heart Association functional class in patients with heart failure [21], or peak troponin and Killip class >1 in patients with ACS [14], are associated with the presence of sleep-disordered breathing. Ultimately, in patient populations with cardiovascular disease, predictive models for sleep-disordered breathing are too weak to be implemented in clinical practice and failed to show proof of potential contributing to clinical and cost-efficient identification patients with sleep-disordered breathing [14, 21, 22].

In a large sample of patients with ACS and OSA or no sleep-disordered breathing, while excluding patients with central sleep apnoea, DE BATLLE *et al.* [14] could not establish a prediction model with sufficient sensitivity and specificity that would justify a validation study before clinical use. Thus, in such patient populations, monitoring of sleep-disordered breathing has to be performed in order to identify patients with ACS and comorbid OSA. Further evidence from randomised controlled trials (ISAAC, NCT01335087; TEAM-ASV I, NCT02093377) may help elucidate whether OSA should be identified early after ACS or whether routine diagnosis should be performed after the optimal treatment for ACS and ACS-related heart failure is established.

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