Predictors of obstructive sleep apnoea in patients admitted for acute coronary syndrome

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ABSTRACT   Identifying undiagnosed obstructive sleep apnoea (OSA) patients in cardiovascular clinics could improve their management. Aiming to build an OSA predictive model, a broad analysis of clinical variables was performed in a cohort of acute coronary syndrome (ACS) patients.

Sociodemographic, anthropometric, life-style and pharmacological variables were recorded. Clinical measures included blood pressure, electrocardiography, echocardiography, blood count, troponin levels and a metabolic panel. OSA was diagnosed using respiratory polygraphy. Logistic regression models and classification and regression trees were used to create predictive models.

A total of 978 patients were included (298 subjects with apnoea–hypopnoea index (AHI) <15 events·h^{-1} and 680 with AHI ⩾15 events·h^{-1}). Age, BMI, Epworth sleepiness scale, peak troponin levels and use of calcium antagonists were the main determinants of AHI ⩾15 events·h^{-1} (C statistic 0.71; sensitivity 94%; specificity 24%). Age, BMI, blood triglycerides, peak troponin levels and Killip class ⩾II were determinants of AHI ⩾30 events·h^{-1} (C statistic of 0.67; sensitivity 31%; specificity 86%).

Although a set of variables associated with OSA was identified, no model could successfully predict OSA in patients admitted for ACS. Given the high prevalence of OSA, the authors propose respiratory polygraphy as a to-be-explored strategy to identify OSA in ACS patients.

Introduction

Cardiovascular diseases represent the main cause of death worldwide, with 17.5 million victims in 2012 [1]. Coronary artery disease (CAD) is responsible for more than half of all cardiovascular deaths, and acute coronary syndrome (ACS), ranging from unstable angina to myocardial infarction, is often the first manifestation of underlying CAD [1, 2]. Obstructive sleep apnoea (OSA), defined as the presence of repetitive episodes of upper airway collapse during sleep, is a common chronic condition, affecting 10% of middle-aged men and 3% of middle-aged women [3]. Increased sympathetic activity and oxidative stress induced by recurrent airway obstruction, intermittent hypoxaemia and arousals from sleep, cause endothelial dysfunction and predispose patients to atherosclerosis [4]. OSA is widely recognised as a risk factor for cardiovascular diseases [5, 6] and increasing evidence suggests a key role and a prognostic value of OSA in ACS.

The prevalence of OSA is very high amongst patients with CAD, affecting from 57 to 79% of patients hospitalised for ACS [7–9]. Furthermore, several authors have observed worse cardiovascular outcomes when OSA coexists with ACS [10]. In patients with myocardial infarction undergoing a percutaneous coronary intervention, for example, OSA seems to promote atheroma progression, increasing the recurrence of cardiac and cerebrovascular events such as re-infarction, stroke, sudden death and repeated revascularisations [10–13]. Among such complex patients, the early identification and treatment of those with OSA may help to reduce these complications. Unfortunately, OSA remains greatly underdiagnosed in cardiology settings [14, 15].

Few studies to date have tried to predict the risk of suffering from OSA among patients with acute coronary syndrome using clinical variables. Furthermore, all of them have focused on a limited number of clinical variables [16, 17]. Using data from the Impact of sleep apnoea syndrome in the evolution of acute coronary syndrome cohort (ISAACC), we performed a broad analysis of multiple clinical variables in a large cohort of ACS patients, with the aim of identifying the main determinants of OSA in such a group of patients.

Methods

Study population

This is an ancillary study of the ISAACC study, which is a multicentre, open-label, parallel, prospective, randomised, controlled trial (registered 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 [18]. Starting in June 2011, patients admitted for ACS to coronary care units or cardiology hospitalisation wards at 14 teaching hospitals in Spain (male and females aged ⩾18 years) were evaluated regards their suitability for the trial [18]. All patients underwent respiratory polygraphy during the first 48–72 h after admission. Patients with an apnoea–hypopnoea index (AHI) >15 events·h^{-1} and ⩽50% of central apnoeas were randomised to conservative or CPAP treatment. Those patients with an AHI ⩽15 events·h^{-1} were considered controls. For the current study, we used information about the first 1000 patients recruited consecutively in the ISAACC study, excluding patients with more than 50% of missing variables. We assessed individual predictors for OSA and developed predictive models to determine the pre-test probability of OSA based on a broad range of baseline variables in non-sleepy ACS patients, thus using the results of the respiratory polygraphy as the outcome variable.

Acute coronary syndrome was defined as the acute presentation of coronary disease with or without ST elevation infarction, unstable angina, or type 1 MI [19]. The exclusion criteria for the current study included the following: previous treatment with CPAP; psychophysical inability to complete questionnaires; the presence of any previously diagnosed sleep disorder; patients with >50% central apnoeas or the presence of Cheyne–Stokes respiration, daytime sleepiness (Epworth Sleepiness Scale (ESS) >10), patients with chronic diseases (e.g. neoplasms, renal insufficiency (glomerular filtration rate <15 mL·min^{-1}·1.73 m^{-2}), severe chronic obstructive pulmonary disorder (a forced expiratory volume in 1 s <50%), chronic depression and other limiting chronic diseases), a medical history that could interfere with the study objectives, any processes, whether cardiovascular or otherwise, that reduce life expectancy to <1 year, and patients in cardiogenic shock.

The ethics committee of each participating centre approved the study (approval number in the coordinator centre: 2010-852), and all patients provided written informed consent.

Data collection
Outcomes
OSA diagnosis was based on the results of overnight cardio-respiratory polygraphy, which is in accordance with the guidelines of the Spanish national consensus on apnoea–hypopnoea syndrome [20]. All participating centres used the same model of polygraph (Embletta; ResMed, Bella Vista, Australia). Nasal pressure airflow, thoracoabdominal movements, electrocardiography and pulsioxymetry were recorded. Obstructive apnoea was defined as an absence of airflow lasting $\geqslant 10$ s in presence of abdominal and thoracic movements. Central apnoeas were defined in absence of both thoracic and abdominal wall movement and airflow lasting $\geqslant 10$ s. Obstructive hypopnoea was defined as a reduction in airflow lasting $\geqslant 10$ s associated with oxygen desaturation in presence of both thoracic and abdominal movements. Oxygen desaturation was defined as a decrease in arterial oxygen saturation $\geqslant 4\%$. Respiratory polygraphy studies were performed without supplemental oxygen. The AHI was defined as the number of episodes of apnoea and hypopnoea per hour of recording. OSA was defined as an AHI >15 events·h^{-1} with $\leqslant 50\%$ of central apnoeas; and severe OSA as an AHI >30 events·h^{-1} with $\leqslant 50\%$ of central apnoeas. A minimum of 3 h of satisfactory signal recording were required to consider the test as valid.

Covariates
Sociodemographic variables as well as information regarding lifestyle habits, clinical background and usual pharmacological treatment were recorded using questionnaires. The degree of self-reported sleepiness/drowsiness was analysed by the Spanish version of the Epworth Sleepiness Scale (ESS) test [21]. Quality of life was assessed with the EuroQol EQ-5D questionnaire. Anthropometric measures included weight, height, body mass index (BMI), neck, waist and hip circumferences, and the waist-to-hip ratio. Blood pressure measures, electrocardiography and echocardiography were routinely performed during patient admission. Similarly, variables related to ACS severity (ejection fraction, Killip classification, number of affected vessels, number of stents implanted and peak troponin) and short-term prognosis (length of stay in the coronary unit, length of hospitalisation, complications and mortality) were measured during patient admission. Killip classification is a clinical scale that uses physical examination to define the severity of ventricular dysfunction after a cardiac ischaemic event, predicting the risk of death, and ranging from class I (absence of heart failure signs) to class 4 (cardiogenic shock) [22]. Fasting blood samples were obtained and analysed including a complete blood count and a basic metabolic panel.

Statistical analyses
Data for each participant were uploaded to a database. Only the coordinating centre (Hospital Arnau de Vilanova and Santa Maria, IRBLleida, Lleida, Spain) had full access to the database. Given the heterogeneity of troponin measurement methods among centres, method-specific deciles of peak troponin were computed and an overall troponin variable grading from 1 to 10 was created for each subject. The mean±SD, median (interquartile range) or frequency (%) were computed to evaluate the differences between OSA and control patients, assessing the significance of such differences with the Chi-squared test, t-test or Mann–Whitney test, as appropriate.

Multiple imputation techniques were used to estimate values for those patients with missing variables. Multiple imputation was implemented under the assumption that the missing data were missing at random. For each missing value, ten imputed values were generated on the basis of AHI, sex, age and reported hypertension, dyslipidaemia and diabetes. This was generated using multiple imputation by chained equations, specifying the univariate imputation model appropriate for each variable, using "mi impute chained" command in Stata (StataCorp, College Station, TX, USA), version 12.1. Crude logistic regression models were used to identify potential determinants of OSA. R^2 using Fisher's z over imputed data and C statistic over imputed data were used to identify variables with predictive value for OSA. Top 20 variables according to R^2 were considered for multivariate logistic regression models. After a first multivariate model including all selected variables, variables with p>0.100 were removed from the model. In a subsequent final model, only significant variables were kept. Potential interactions among such variables were tested. Predictive value of the final model was assessed in terms of R^2 using Fisher's z over imputed data and C statistic over imputed data. The same methodology was applied to assess predictors for severe OSA (AHI >30 events·h^{-1}). Additionally, sensitivity analyses excluding subjects with more than 20% of missing variables were performed.

Classification and regression trees (CART) [23] were also used as alternatives to the previously mentioned logistic regression approach to assess OSA and severe OSA. Briefly, CART provide top-down set of hierarchical variables with specified cutoff points which classify all subjects according to their probability of having OSA. Therefore, an inverted tree structure is generated, with each node corresponding to a variable and a given cutoff point, and with each dead-end branch providing the probability of such subjects having OSA. Sensitivity, specificity and predictive value of CART were assessed. CART methods were used before any multiple imputations. CART analyses were performed using the R statistical package.

Data analysis was conducted using Stata 12.1 (StataCorp, College Station, TX, USA). The threshold for significance was set at 0.05.

Results

A total of 978 patients were included in the analysis, after the exclusion of 22 patients with more than 50% of missing data. Up to 298 patients had AHI <15 events·h^{-1} and 680 had AHI ⩾15 events·h^{-1} (379 with AHI ⩾30 events·h^{-1}). The main cardio-respiratory polygraphy variables of the cohort's subjects are shown in table 1.

Tables 2, 3 and online supplementary table S1 show all the characteristics of the ISAACC patients that were considered as potential determinants of OSA, according to AHI. The tables include information about anthropometric variables, biological determinants, usual pharmacological treatment, clinical background and lifestyle habits, cardiovascular variables and hospitalisation-related variables. OSA patients were significantly older than controls (p<0.001). Similarly, OSA patients were significantly more obese than controls according to weight (p<0.001), body mass index (p<0.001), neck circumference (p<0.001) and waist and hip circumferences (p<0.001). Other very significant differences were found for peak troponin (p<0.001), blood glucose (p=0.005), use of calcium antagonist (p=0.001), hypertension (p=0.001) and the Epworth Sleepiness Scale (p=0.001), for which OSA patients showed increased values.

After computing R^2 using Fisher's z over imputed data and C statistic over imputed data to identify variables with the highest predictive value for OSA, the top 10 variables were BMI (R^2=0.054), weight (R^2=0.037), waist (R^2=0.035), hip (R^2=0.030) and neck (R^2=0.016) circumferences, age (R^2=0.011), peak troponin (R^2=0.010), calcium antagonists (R^2=0.010), Epworth sleepiness scale (R^2=0.009) and hypertension (R^2=0.009). The final logistic regression models attempting to predict subjects with an AHI ⩾15 events·h^{-1}, as well as their prediction capacity according to R^2 and the C statistic are shown in table 4. The adjusted model had a C statistic of 0.71, with a sensitivity of 94.1% and a specificity of 24.3%. Similarly, table 5 shows the final logistic regression models attempting to predict severe OSA. The adjusted model had a C statistic of 0.67, with a sensitivity of 30.5% and a specificity of 85.5%. Age, BMI and the ESS were common variables to both OSA and severe OSA models. Given the poor predictive capacity of the developed models, no validation studies were performed. Sensitivity analyses excluding subjects with more than 20% of missing values reported very similar results and did not increase the predictive capacity of the models.

The use of CART did not provide better prediction capacity. Briefly, the first nodes of the classification tree for AHI ⩾15 events·h^{-1} involved BMI, age and ESS, while for AHI ⩾30 events·h^{-1} included BMI, prothrombin time and peak troponin levels. The former model had a sensitivity of 95% and a specificity of 43% while the later had a sensitivity of 53% and a specificity of 87%. Overall, none of the resulting classification trees had a good enough performance to justify a validation study.

Discussion

In this ancillary study including 978 non-sleepy subjects from the ISAACC trial with an episode of ACS, we measured a broad range of clinical variables and assessed their association to OSA. At the same time, we tried to develop a predictive model, which could be used to identify ACS patients who should be referred for a sleep study. Age, BMI, the ESS, peak troponin levels and usual intake of calcium antagonists were the main determinants of having AHI ⩾15 events·h^{-1}. Similarly, we identified age, BMI, blood triglycerides, peak troponin levels and having a Killip class II or higher as the main determinants of AHI ⩾30 events·h^{-1}.

In accordance to previous literature, we found age, BMI and the ESS to be strongly associated with OSA [16, 17, 24]. However, no associations for sex were found although being previously reported [25]. Intake of one of the most widely used antihypertensive drugs, calcium antagonists, was also related to OSA, thus indicating an association between OSA and hypertension in our cohort. Additionally, we found higher

TABLE 1 Cardio-respiratory polygraphy results of the ISAACC patients according to apnoea–hypopnoea index (AHI)

	AHI		p-value[#]
	<15 events·h^{-1}	⩾15 events·h^{-1}	
Patients n	298	680	
AHI events·h^{-1}	6 (3–10)	32 (22–46)	<0.001
Oxygen desaturation index >4% h^{-1}	5 (2–10)	26 (16–42)	<0.001
Mean S_{aO_2} %	92.7±4	92.0±4	0.018
Minimum S_{aO_2} %	84.4±11	81.0±10	<0.001
Time with S_{aO_2} <90% %	0.6 (0–6.1)	4.3 (0.9–19)	<0.001

Data are presented as mean±SD or median (interquartile range), unless otherwise stated. S_{aO_2}: arterial oxygen saturation; AHI: apnoea–hypopnoea index. [#]: t-test or Kruskal–Wallis test as appropriate.

TABLE 2 Baseline characteristics of the ISAACC patients according to apnoea–hypopnoea index (AHI)

	AHI		p-value[#]
	<15 events·h^{-1}	≥15 events·h^{-1}	
Patients n	298	680	
General characteristics			
Age years	58±12	60±11	<0.001
Males n (%)	246 (83)	564 (83%)	0.881
Body mass index kg·m^{-2}	27.0±4	29.5±5	<0.001
Epworth Sleepiness Scale	4.9±2.5	5.4±2.5	0.001
Biological determinations			
Hematocrit %	42.5±5	41.8±5	0.046
Glucose mg·dL^{-1}	102 (87–134)	109 (93–142)	0.005
Triglycerids mg·dL^{-1}	137.5±83	146.8±77	0.132
Total cholesterol mg·dL^{-1}	181±46	177±43	0.286
Uric acid mg·dL^{-1}	5.5 (4.5–7.2)	6.1 (5–7.2)	0.007
Creatinine mg·dL^{-1}	0.89±0.2	0.92±0.3	0.093
Peak troponin (deciles)[¶]	4 (1–7)	5 (3–8)	<0.001
Creatine phosphokinase UI·mL^{-1}	236 (103–828)	254 (118–844)	0.328
Usual pharmacological treatment			
Diuretics	45 (15)	128 (19)	0.183
β-blockers	73 (25)	149 (22)	0.305
ACE inhibitors	38 (22)	90 (22)	0.943
Angiotensin II receptor antagonists	20 (12)	63 (16)	0.228
Calcium antagonists	19 (7)	92 (14)	0.001
Hypolipidaemics	91 (31)	262 (39)	0.022
Oral antidiabetics	46 (16)	132 (20)	0.158
Insulin	21 (7)	47 (7%)	0.908

Data are presented as mean±SD, n (%) or median (interquartile range), unless otherwise stated. [#]: Chi-squared test, one-way ANOVA or Mann–Whitney test as appropriate; [¶]: test-specific deciles of peak troponin (computed separately for each testing method to account for differences in sensitivity among centres).

peak troponin levels in OSA patients compared to controls. Some studies have also observed increased levels of plasma troponin, a sign of subclinical myocardial injury, in patients with OSA [26–28]. Moreover, a relation between troponin levels and OSA severity has been prudently suggested regardless of concerns involving a potential clustering of cardiovascular risk factors in subjects with OSA [26–28]. Finally, it is noteworthy that although not making it into the final predictive models, blood glucose levels but not diabetes was related to the risk of suffering OSA, probably due to diabetes under-diagnosis [29, 30].

Regarding severe OSA patients, we identified blood triglycerides and the Killip class as the main predictors together with age, BMI and peak troponin levels. The presence of dyslipidaemia in patients suffering OSA has been shown in several studies [31, 32], and the levels of blood triglycerides represent a risk factor for cardiovascular diseases and have a prognostic role among ACS patients [33, 34]. However, Lavie et al. [35] did not find differences in triglycerides in patients with and without OSA and CAD. Several trials have shown a worse prognosis in patients with ACS and OSA [10–13, 36]. Nonetheless, no differences in troponin levels were found in a study comparing a small group of patients with CAD and untreated OSA to controls [37]. Similarly, Valo et al. [38] studied 21 patients with CAD and OSA without observing any differences with the control group. Finally, a broader study with 136 myocardial infarction patients with and without OSA showed lower levels of troponin among OSA patients, and even a cardio-protective role of OSA suggesting that OSA (or episodic hypoxia) might act as a "preconditioning factor" [39]. However, as acknowledged by the authors of previous studies, small study sample sizes could be the underlying factor explaining many of the contrasting results.

The current study has several strengths including a large sample size, the novelty of the setting in non-sleepy patients with an episode of ACS, and the measurement of a broad range of sociodemographic, anthropometric, lifestyle, biological, clinical, pharmacological and cardiovascular variables, while using respiratory polygraphy to determine OSA status. On the other hand, several limitations should be acknowledged. 1) The study is lacking the dimension of genetics; however, genetic tests are not usually available in standard clinical settings and would hinder the usefulness of the predictive model. 2) No information regarding snoring was collected as such information is not investigated in coronary units.

TABLE 3 Baseline clinical characteristics and variables related to acute coronary syndrome severity of the ISAACC patients according to apnoea–hypopnoea index (AHI)

	AHI		p-value[#]
	<15 events·h^{-1}	≥15 events·h^{-1}	
Patients n	298	680	
Clinical background			
Hypertension	125 (42)	362 (53)	0.001
Dyslipidaemia	144 (48)	356 (52)	0.246
Diabetes mellitus	64 (22)	168 (25)	0.274
Cardiomyopathy	69 (23)	145 (22)	0.569
Stroke	7 (2)	25 (4)	0.274
Cardiovascular variables			
First ACS episode	238 (85)	520 (82)	0.199
Anomalies in ECG	233 (90)	518 (91)	0.621
ACS category			
Unstable angina	33 (13)	67 (12)	
Non-STEMI	125 (50)	268 (48)	
STEMI	94 (37)	224 (40)	0.736
Ejection fraction	56.9±10	54.9±11	0.017
Killip Class			0.060
I	221 (95)	497 (90)	
II	11 (5)	47 (8)	
III	0 (0)	6 (1)	
IV	0 (0)	3 (1)	

Data are presented as n (%) or mean±SD, unless otherwise stated. ACS: acute coronary syndrome; ECG: electrocardiogram; STEMI: ST-elevation myocardial infarction. [#]: Chi-squared test, one-way ANOVA or Mann-Whitney test as appropriate.

3) The exclusion of part of the controls modified the proportion of cases and controls, which ultimately affects the sensitivity and specificity of the predictive models; however, this fact does not diminish the predictive capacity of the models but rather shifts the model from a specificity-driven model toward a sensitivity-driven one. 4) No additional confirmatory polygraphy was available to ensure that fluid accumulation, as a symptom of acute cardiac dysfunction, was not distorting OSA diagnosis; however, polygraphies made in a subset of 57 participants after 1 year showed minor differences in median AHI: 32.0 events·h^{-1} at baseline and 28.7 events·h^{-1} after 1 year, thus suggesting that acute fluid accumulation was unlikely to distort OSA diagnosis. 5) The inclusion in the imputation models of subjects with up to 49% of missing variables could also be seen as a limitation; however, it is well known that analyses including only complete cases are likely to be biased due to substantial loss of precision and power [40]. Moreover, sensitivity analyses including only subjects with up to 19% of missing variables reported very similar results, thus confirming that the current results were not driven by missing values and/or

TABLE 4 Logistic regression models of potential determinants of obstructive sleep apnoea defined as apnoea–hypopnoea index ≥15 events·h^{-1}

	Crude models				Adjusted model[#]			
	OR	95% CI	R^{2}[¶]	C statistic[+]	OR	95% CI	R^{2}[¶]	C statistic[+]
Age years	1.02	1.01–1.04	0.011	0.57	1.03	1.01–1.04		
Body mass index kg·m^{-2}	1.15	1.11–1.20	0.054	0.67	1.15	1.11–1.20		
Epworth Sleepiness Scale	1.10	1.04–1.16	0.009	0.57	1.08	1.02–1.15		
Calcium antagonists	2.28	1.37–3.81	0.010	0.54	1.91	1.11–3.28		
Peak troponin deciles[§]	1.08	1.03–1.13	0.010	0.57	1.08	1.03–1.13		
Full model							0.090	0.71

Positive predictive value over imputed data for the adjusted model=73.94%. Negative predictive value over imputed data for the adjusted model=64.50%. [#]: all covariates are together in a single model. [¶]: R^2 using Fisher's z over imputed data; [+]: C statistic over imputed data; [§]: test-specific deciles of peak troponin (computed separately for each testing method to account for differences in sensitivity among centres).

TABLE 5 Logistic regression models of potential determinants of severe OSA defined as Apnoea–hypopnoea index $\geqslant$30 events·h^{-1}

	Crude models				Adjusted model[#]			
	OR	95% CI	R^{2}[¶]	C statistic[+]	OR	95% CI	R^{2}[¶]	C statistic[+]
Age years	1.02	1.006–1.03	0.007	0.55	1.03	1.01–1.04		
Body mass index kg·m^{-2}	1.11	1.08–1.15	0.040	0.63	1.11	1.08–1.15		
Triglycerides cg·dL^{-1}	1.03	1.007–1.04	0.007	0.55	1.03	1.01–1.05		
Peak troponin deciles[§]	1.07	1.02–1.11	0.007	0.56	1.07	1.02–1.12		
Killip class >I	2.31	1.39–3.88	0.010	0.53	2.06	1.21–3.52		
Full model							0.072	0.67

Positive predictive value over imputed data for the adjusted model=57.15%. Negative predictive value over imputed data for the adjusted model=66.04%. [#]: all covariates are together in a single model; [¶]: R^2 using Fisher's z over imputed data; [+]: C statistic over imputed data; [§]: test-specific deciles of peak troponin (computed separately for each testing method to account for differences in sensitivity among centres).

imputation technique. 6) The abovementioned strength of using non-sleepy subjects (ESS $\leqslant$10) could also be considered a weakness, as this exclusion handicaps the feasibility of an effective predictive model; however, sleepy subjects tend to be managed in sleep units and thus are not the main target of a hypothetical predictive model. 7) Not having data on the performance of screening questionnaires, such the Berlin questionnaire or the obstructive sleep apnoea in acute coronary syndrome score for the patients with ACS, precluded a comparison between them and the developed models.

This study tried to define the clinical variables that characterise patients hospitalised for ACS at higher risk of undiagnosed OSA, especially when such patients do not show significant daytime sleepiness. As expected, OSA could be suspected in older patients with high BMI and more reported sleepiness even if it would be considered not relevant enough to classify the patient as sleepy. Moreover, regular use of calcium antagonists and higher peak troponin levels, were also associated with a higher risk of OSA. However, grouping these variables into a single predictive model was insufficient to create an effective predictor model for OSA. It could be argued that, as none of the non-sleepy ACS patients are undergoing sleep tests (0% sensitivity and 100% specificity scenario), a predictive model calibrated in order to maximise specificity could be of some use while having a small impact on medical costs. However, such a model would have a poor sensitivity and many OSA patients would never undergo a sleep test. Therefore, options beyond predictive modelling should be explored. In this sense, the broadening application of respiratory polygraphy devices and the reduction in costs associated with domiciliary sleep tests, as well as the potential to-be-demonstrated beneficial effects of CPAP treatment, sleep testing of patients admitted for ACS could be a sound option in the near future.

In conclusion, our study failed to construct a model capable of successfully predicting OSA in patients admitted for ACS, although a set of variables associated with OSA in such patients including age, BMI and ESS but also peak troponin levels and regular use of calcium antagonists was identified. While the clinical value of correctly identifying and managing non-sleepy OSA patients who are admitted to hospital with ACS awaits the results of large randomised controlled trials of OSA treatment such as ISAACC, the authors propose the exploration of a broad use of respiratory polygraphy, rather than clinical variables, to identify OSA in ACS populations.

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