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Original Article

Prevalence of STOP BANG questionnaire and association with major cardiovascular events in hospitalized population: is it enough with currently used cardiovascular risk measurements?

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ABSTRACT

Cardiovascular risk (CR) is associated with obstructive sleep apnea hypopnea syndrome (OSAHS). This association enhances the risk of major adverse cardiovascular events (MACE); nevertheless, data from hospitalized populations and interactions among these conditions remain unclear.

Purpose: To evaluate the risk of MACE in the population with risk of OSAHS using the STOP-BANG questionnaire.

Methods: We performed a prospective study in an academic hospital from 2017 to 2018. Data included demography, admissions, STOP-BANG score and CR using AHA scores. The primary outcome was risk of MACE in participants with low risk of OSAHS (STOP-BANG 0–2 points), risk of OSAHS (≥3 points) and risk of moderate/severe OSAHS (≥5 points). Risk of MACE was evaluated using odds ratios (OR), and average CR was evaluated using the t-test.

Results: A total of 441 participants were included. The cumulative prevalence of STOP BANG ≥3 points was 80.9%, and that of ≥5 points was 41.6%. OR of MACE ≥3 points was 3.93 (CI 2.08–7.24) ($p < 0.001$) compared with <3 points, and Average CR was 10.91% (SD ± 2.13) at <3 points versus 24.3% (SD ± 1.24) for ≥3 points for ≥5 points OR of MACE was 1.72 (CI 1.18–2.59) ($p = 0.005$) and average CR was 26.14% (SD ± 1.63). However, after multivariable analysis, gender differences and previous heart failure were independently associated to MACE.

Conclusion: The risk of OSAHS in the hospitalized population is high. This population has a higher risk of MACE and higher CRs than do low-risk participants. Conversely, gender and heart failure are potential cofounders.

1. Introduction

Cardiovascular death is the leading cause of mortality worldwide [1]. However, in developed countries, this cause has decreased because of several interventions in both primary and secondary prevention [2]. Classic cardiovascular risk factors include modifiable and non-modifiable factors, and physicians can estimate risk using predictive tools. The Framingham Heart Study (FHS) was the first predictive rule for cardiovascular events; although this tool increased the risk in populations with low mortality rates [3].

Late in 2013, the American College of Cardiology published the pooled cohort ASCVD risk equation, incorporating similar risk factors included in the FHS but adding race [4]. This tool is useful for populations between 40 and 79 years old and is able to predict both cardiovascular and cerebrovascular risks in 10 years.

Despite these predictive tools, several publications have associated various diseases with increased cardiovascular risk. One of these diseases is obstructive sleep apnea hypopnea syndrome (OSAHS) [5].

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OSAHS is a prevalent condition; the HypnoLaus study reported that the prevalence of moderate-to-severe OSAHS was 23.4% for women and 49.7% for men. Moderate-to-severe disease was also associated with a higher risk of hypertension, diabetes mellitus, metabolic syndrome and higher cardiovascular morbidity and mortality [5–9]. Validated OSAHS diagnostic methods are costly and require technical equipment and specialized personnel, making them poorly accessible for most populations. Therefore, OSAHS has become a severe public health problem because of its high prevalence, lack of access to diagnostic procedures, and its association with social, occupational, and cardiovascular risk problems [10].

Predictive rules are useful for identifying populations at risk and prioritizing available health resources. The STOP BANG questionnaire uses a combination of clinical and anthropometric variables and is an inexpensive tool for screening for OSAHS [11]. This questionnaire, based on four questions, was called STOP (Snore, Tired, Observed Apnea, and Pressure) and BANG because of four parameters (Body mass index (BMI), Age, Neck Circumference, and Gender) and reported 90% sensitivity to detect risk of any OSAHS using a cutoff of ≥3 points and 96% for detecting risk of moderate/severe OSAHS using a cutoff of ≥5 points [12].

The aim of this study was to use the STOP BANG questionnaire to determine the risk of OSAHS in a population admitted to an internal medicine service and to explore the potential associations between STOP BANG scores and cardiovascular events.

2. Methods

This study followed the current recommendations of the STROBE statement for observational studies [13]. We performed a prospective, observational study from August 2017 to August 2018. Patients with medical disease admitted to the internal medicine service at one academic hospital were consecutively included. Exclusion criterial were as follows: (1) any surgical procedure; (2) end-of-life condition; (3) patient transfer to intensive care unit in <24 h and; (4) refusal to sign the consent form. The ethics board of our institution approved the protocol and consent form (resolution number: 25).

2.1. Study protocol

We included demographic data (eg, age, gender, comorbidities, admission cause, smoking history, and pack/year index) as well as STOP BANG scores using previously published parameters [11,14]. Cardiovascular risk was calculated in participants between 40 and 79 years old using the American Heart Association cardiovascular risk calculator [15]. For total cholesterol and high-density lipid (HDL), we included a lipid profile requested in the first 48 h from admission.

2.2. Outcome measure

We defined the primary outcome as a composite outcome known as major cardiovascular events (MACE), including cardiovascular mortality, acute coronary syndrome, and heart failure decompensation (Supplementary material 1). A blinded research assistant evaluated each admission and determined MACE according to the medical record. According to the MACE definition, mortality was measured at 30 days. We also added a one-year follow-up, without including these data as MACE. Long-term follow-up was achieved through the national registry of mortality consultation (http://www.registrocivil.cl/).

2.3. Confounder assessment

We performed univariate analysis in both groups exploring the following variables associated with high risk of MACE: age, gender, tobacco history, hypertension, diabetes mellitus (DM type 1 or 2), dyslipidemia, severe obesity (>35 kg/m^2), heart failure, chronic obstructive pulmonary disease (COPD), atrial fibrillation, chronic kidney disease (CKD) and STOP BANG total score with cutoffs ≥3 points and ≥5 points.

2.4. Statistical analysis

Extracted data were tabulated in an Excel database. Descriptive data were expressed as the mean and standard deviation. Prevalence was defined as the number of positive tests/total population. For comprehensive analysis, we classified STOP BANG results in three groups: (1) Low risk of OSAHS (0–2); (2) risk of OSAHS (≥3 points); and (3) risk of moderate/severe OSAHS (≥5 points).

Quantitative data were measured using Student's t-test or the nonparametric test using the Mann–Whitney test for non-Gaussian distributions. Qualitative data were analyzed using a Chi-squared test or Fisher's exact test. A p-value <0.05 indicated statistical significance. For confounder assessment, we explored significant variables in univariate analysis using a multivariable Cox regression and we explored gender differences using a stratified analysis. We used odds ratios (OR) and the 95% confidence interval (CI) for qualitative data and used mean differences for quantitative data. Data analysis was performed using IBM SPSS statistic software version 22.0 (IBM statistic, Chicago. USA).

3. Results

During this period, 441 participants were available for the study. The average age was 60.72 years old (SD ± 17.14). In addition, (245/441) 55.5% of the sample were males, and 105/441 (23.80%) were smokers, with average pack years of 16.9 packs/year (SD ± 21.25). Data from 331 participants between 40 and 79 years old were available to calculate cardiovascular risk according to an AHA calculator, with an average score of 19.69% (SD ± 17.56%). Regarding comorbidities, 280/441 (63.49%) reported hypertension, 169/441 (38.32%) reported diabetes mellitus, and 169/441 (27.89%) reported dyslipidemia (Table 1).

MACE was reported in 154/441 (34.92%) admissions. The main causes of MACE were acute coronary syndrome in 87/154 (56.5%), heart failure (decompensation) in 64/154 (41.5%), and cardiovascular mortality in 5/154 (3.2%). Others admission causes were infectious in 77/441 (17.46%) and digestive causes in 44/441 (9.98%). During our follow-up, we recorded mortality of 43/441 (9.75%). Early mortality occurred in 12/43 cases (27.91%). The main causes were sepsis (5/12), respiratory failure (3/12), congestive heart failure 2/12, and others (2/12). Late mortality (after one year of follow-up), was achieved in 31/43 cases (72.09%). The main causes were cancer (7/31), respiratory conditions (7/31), infectious disease (6/31), CKD (4/31) and others (3/31).

3.1. STOP BANG

The average STOP BANG score was 4.02 points (SD ± 1.76). The cumulative prevalence of STOP BANG ≥3 points was 80.9%, and that of ≥5 points was 41.6%.

3.1.1. Risk of OSAHS (STOP BANG ≥ 3 points)

Chi-squared tests showed an association between OSAHS and MACE. STOP BANG <3 points was associated with 12/94 (12.7%) of MACE compared to 42/357 (39.7%) of those with ≥3 points. The

Table 1
Characteristic of included population. AHA, calculator was measure between 40 and 79 years-old. SD: Standard deviation, cm = centimeters, IPA = pack-year index, BMI = body mass index, AHA: American Heart association, COPD: chronic obstructive pulmonary disease, CKD: chronic kidney disease, MACE: major adverse cardivascular event.

	n = 441
Age, (SD)	60.72 (17.14)
Male, (%)	245 (55.5%)
Neck circumference (cm), (SD)	39.81(±4.97)
AHA score, (%) (SD)	19.69 (±15.5%)
Smoker (%)	110 (24.9)
Smoker (IPA), (SD)	16.9, (±21.25)
BMI, (SD)	30.2, (±6.54)
Comorbidities	
Blood pressure	280, (±63.49)
Diabetes mellitus	169, (±38.32)
Dyslipidemia	123, (±27.89)
BMI > 35 kg/m2, (%)	143, (±32.43)
Hearth failure	98, (±22.22)
COPD	41, (±9.30)
CKD	72, (±16.33)
Cirrhosis	19, (±4.31)
STOP BANG, mean (SD)	4.02, (1.76)
0 points, (%)	11, (2.5)
1 point, (%)	29, (6.6)
2 points, (%)	44, (10)
3 points (%)	81, (18.4)
4 points, (%)	92, (20.9)
5 points, (%)	97, (22)
6 points, (%)	53, (12)
7 points, (%)	26, (5.9)
8 points, (%)	8, (1.8)
Admission causes	
MACE, (%)	154, (34.92)
Infectious etiology (any)	77, (17.46)
Cancer (any)	26, (5.90)
Pulmonary decompensation	40, (9.0)
Bleeding (any)	22, (5)
Digestive system	44, (10)
Rheumatology causes	14, (3.17)
Renal failure	21, (4.76)
Others	43, (9.75)
Mortality	
<30 days	12, (27.9)
1 year	31, (72)

calculated OR for this association was 3.93 (CI 2.08–7.24) (p < 0.001).

Cardiovascular risk in the 40- to 79-year-old group was increased. The average cardiovascular risk was 10.91% (SD ± 2.13) versus 24.3% (SD ± 1.24) with scores of 3–4 points. The mean difference was 13.39% (SD ± 3.18) (CI 7.13–19.6), p < 0.0001 (Fig. 1).

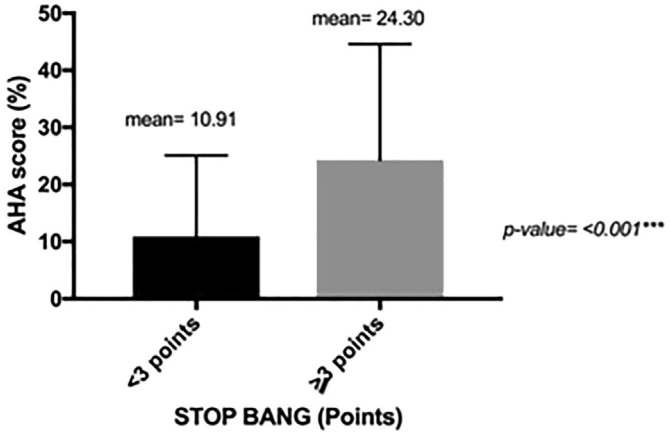

Fig. 1. Cardiovascular risk in population with risk of OSAHS.

3.1.2. Risk of moderate/severe OSAHS (STOP BANG ≥ 5 points)

Patients with risk of moderate/severe OSAHS reported a higher risk of MACE as well. MACE was reported in 76/257 (29.5%) of patients with <5 points and 78/184 (42.4%) of those with ≥5 points. The calculated OR of MACE in this group was 1.72 (CI 1.18–2.59) (p = 0.005).

Cardiovascular risk was also increased in this group: the average AHA risk was 26.14 (SD ± 1.63) compared to 18.91 (SD ± 1.55) with <5 points. The mean difference was 7.23 (SD ± 2.25), p value = 0.001 (Fig. 2).

We found a significant difference between low risk and both intermediate (3–4 points) and moderate/severe risk of OSAHS (p < 0.001), and we found no differences between intermediate and moderate/severe risk (p value = 0.39).

Finally, we found no significant association between atrial fibrillation and either risk of OSAHS (p = 0.164) or risk of moderate/severe OSAHS (0.332).

3.2. Confounder assessment

Using a univariate model, we found a significant association between MACE and the following variables: age (p < 0.001), male gender (p = 0.016), AHA score (p = 0.013), hypertension (p < 0.001), diabetes mellitus (p = 0.013), heart failure (p = <0.001), STOP BANG ≥3 points (p < 0.001) and STOP BANG ≥5 points (p = 0.006). After multivariable analysis, three variables were statistically significant: age (OR = 1.03 (CI 1.01–1.06), p = 0.015), male gender (OR 2.48 (CI 1.45–4.26), p < 0.001) and heart failure (OR = 5.11 (CI 2.74–9.52), p < 0.001). We also found a significant tendency for STOP BANG <5 points with OR (0.59 (0.34–1.027), p = 0.06) (Tables 2–3).

3.3. Gender differences

3.3.1. Female differences

Data from 245 males and 196 females were analyzed. Female patients with low risk of OSAHS had an average cardiovascular risk of 21.21% versus 22.88% in those with risk of OSAHS (p = 0.041). In patients with <5 points, average risk was 19.45% versus 27.21% with risk of moderate/severe OSAHS (p = 0.018) (Fig. 3).

MACE risk was also greater in patients with abnormal STOP BANG. For cutoff ≥3 points, 34.27% had MACE, versus 13.21% in those with <3 points. The calculated OR was 3.426 (CI 1.425–8.605, p = 0.004). We also found an increased risk of MACE in those with ≥5 points (38.89%) versus 24.64% in those with <5 points. The calculated OR was 1.945 (CI 1.006–3.872, p = 0.05).

In this group, univariate analysis revealed a significant association between MACE and age, hypertension, diabetes mellitus, hearth failure, STOP BANG total score, STOP BANG cutoff ≥3 and STOP BANG ≥5 points. However, after multivariable analysis, heart

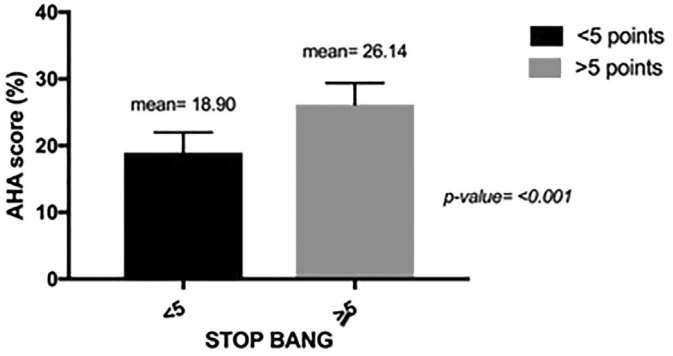

Fig. 2. Cardiovascular risk in population with risk of severe OSAHS.

Table 2
Univariable analysis exploring different cardiovascular risk factors. SD: standard deviation, MACE: major adverse cardiovascular event, AHA: American heart association, DM2: diabetes mellitus type 2, COPD: chronic obstructive pulmonary disease, CKD: chronic kidney disease, BMI: body mass index.

	With MACE (n = 154)	Without MACE (287)	p value
Age, (years)	67.42	57.13	*0.000**
Male, (%)	63.60%	51.20%	*0.016**
AHA score, (mean) (SD)	19.6 ± 1.18	24.86 ± 1.88	*0.013**
Smoker, (%)	31/110 (28.2%)	79/110 (71.8%)	0.106
Hypertension (%)	80.50%	54.40%	*0.000**
DM2 (%)	45.50%	34.50%	*0.031**
Heart failure, (%)	42.20%	11.50%	*0.000**
COPD, (%)	7.80%	10.10%	0.494
CKD, (%)	19.50%	14.60%	0.224
Dyslipidemia, (%)	47.5%	36.7%	0.312
BMI > 35, (%)	37%	47%	*0.999*
STOP BANG ≥ 3 points, (%)	92.20%	75.30%	*0.000**
STOP BANG ≥ 5 points, (%)	50.60%	36.90%	*0.006**

* Statistically significant.

Table 3
Multivariable analysis exploring different cardiovascular risk factors. AHA: American heart association.

Variable	Multivariable model	p value
Age	1.03 (1.01–1.06)	0.015*
Gender (male)	2.48 (1.45–4.26)	*<0.001**
AHA risk	1.00 (0.98–1.01)	0.91
Hypertension	1.64 (0.84–3.20)	0.14
Diabetes mellitus	0.94 (0.55–1.59)	0.81
Heart failure	5.11 (2.74–9.52)	*<0.001**
STOPBANG ≥ 3	1.52 (0.63–3.64)	0.34
STOPBANG ≥ 5	0.59 (0.34–1.027)	0.06

* Statistically significant.

failure was independently associated with this outcome, OR = 6.12 (CI 2.73–13.52), p < 0.001 (Tables S1–S2).

3.3.2. Male differences

Male patients with <3 points reported an average AHA score of 10.46% versus 26.29% for those with ≥3 points (p < 0.001). This difference was also significant for those patients with risk of moderate/severe OSAHS. Patients with <5 points reported an average AHA score of 19.79% versus 26.29% for those with ≥5 points (p < 0.001).

MACE was reported in 93/98 (94.9%) of those with ≥3 points compared to 5/98 (5.1%) of those with <3 points (OR = 3.997 (CI 1.477–9.845, p = 0.003)). Using a cutoff of 5 points, MACE was reported in 41/98 (41.83%) with <5 points and 57/98 (58.16%) with ≥5 points. We found a non-significant OR (1.409 (CI 0.8436–2.391, p = 0.239)).

Univariate analysis revealed a significant association between male gender and age, AHA score, hypertension, hearth failure, STOP

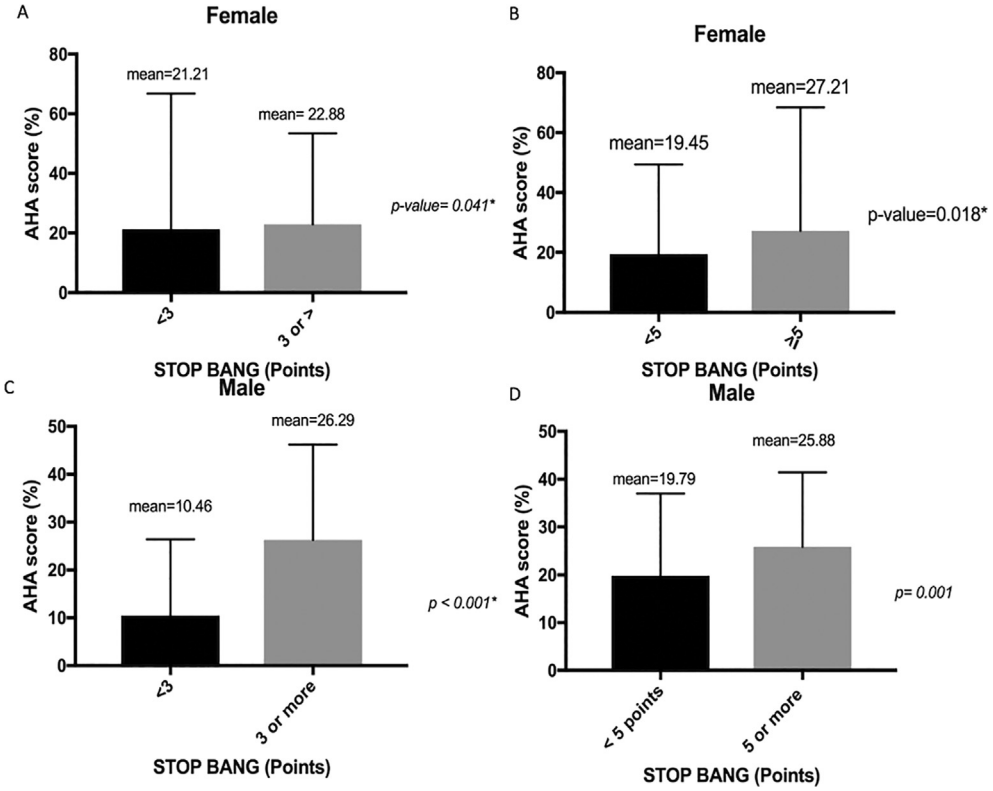

Fig. 3. Cardiovascular risk according to gender. A: Average risk in female with risk of OSAHS. B: Average risk in female with high risk of OSAHS. C: Average risk in male with risk of OSAHS. D: Average risk in male with high risk of OSAHS.

BANG total score, and STOP BANG ≥3 points. However, after multivariable analysis, medical record evidence of heart failure was independently associated with MACE, OR = 2.47 (1.07–5.67), p = 0.032 (Tables S3–S4).

4. Discussion

Our main findings were as follows: (1) a high risk prevalence of either any OSAHS or moderate/severe OSAHS in the hospital setting; (2) a high prevalence of cardiovascular comorbidities; (3) a significant association between MACE and altered STOP BANG; (4) higher AHA scores in patients with risk of OSAHS or moderate/severe OSAHS in patients 40–79 years old; (5) an independent association between MACE and age, gender and medical record evidence of heart failure; as well as (6) an independent association between MACE and medical record evidence of heath failure in both males and females.

Although the STOP BANG questionnaire was originally designed for the perioperative setting, it has been validated in inpatient or outpatient settings [16,17]. Accuracy for populations at risk for OSAHS and risk of moderate/severe OSAHS was >90%. For example, a study performed in a respiratory clinic revealed a sensitivity of 95% for OSAHS using a cutoff ≥3 points [17]. A systematic review and meta-analysis showed an area under the curve with a STOP BANG cutoff point ≥3 of 0.72 with sensitivity and specificity for AHI ≥5 ev/h of 90% (95% CI: 83–91) and 49% (95% CI: 45–54), and for AHI ≥30 ev/h of 96% (95% CI: 95–97) and 25% (95% CI: 23–27) [18].

The plausibility of cardiovascular risks in populations at risk for OSAHS measured by the STOP BANG was previously reported in different settings. First, in a hypertension clinic, the accuracy of STOP BANG reported a sensitivity of 89.5% and a specificity of 45.9% using a cutoff of ≥3 points. Using a cutoff of ≥5 points, the sensitivity was 48.1% and the specificity was 72.4% [19]. Second, in populations with increased cardiovascular risk, including diabetes mellitus, STOP BANG yielded a sensitivity of 81.8% for OSAHS and 67.1% for moderate/severe OSAHS [20]. In another study, the prevalence of OSAHS in the population referred to a tertiary diabetic clinic was 39%, and they reported more comorbidities such as dyslipidemia and obesity [21].

In our study, the prevalence of altered STOP BANG was higher than previously reported. Conversely, most of those studies were developed in outpatient settings such as sleep clinics and hypertension clinics, among others, and included non-Latino populations [20,21]. According to data from the national health survey in Chile, the risk of OSAHS using STOP BANG is 31.2% in the population with 3–4 points and 8.9% for the population with ≥5 points [22].

We used the AHA risk predictive score. In our population, this predictive model gave an AUC of 0.78 (CI 0.68–0.84) compared to the Framingham model (AUC 0.60, CI 0.52–0.74) [23]. We found a significant difference between patients at risk for OSAHS and those at risk for moderate/severe OSAHS as well. To the best of our knowledge, this finding is novel and shows the accumulative cardiovascular risk in populations at risk for OSAHS, especially in hospitalized populations.

Our analysis revealed an increased cardiovascular risk and a dose–response relationship between altered STOP BANG score and MACE. Confounder evaluation also showed that our population reported higher morbidity and altered metabolic parameters such as hypertension, diabetes mellitus, dyslipidemia, obesity, and eventually, OSAHS. The independent variables associated with this outcome were age, male gender, and heart failure. After a stratified analysis exploring the differences between males and females, the main independent risk factor was heart failure. This finding is consistent with the results of a previous publication showing a strong association between heart failure and cardiovascular risk factors [24]. A meta-analysis of six cohort studies reported that the relative risk of all-cause mortality was RR 1.67 (CI 1.23–2.23) and 2.21 (1.61–3.24) for cardiovascular mortality [25].

This study has a few limitations. First, comorbidities and cardiovascular risk were higher than in the general population. Further population-based screenings using both STOP BANG scores and cardiovascular risk tools are needed in order to confirm our findings in the general population, probably modifying our current cardiovascular risk calculator incorporating OSAHS. Second, we limited our OSAHS evaluation to a previously validated questionnaire, without further testing (home sleep apnea test or polysomnography). Nevertheless, the high sensitivity of STOP BANG is a cost-effective initial approach to screen populations at risk for both OSAHS and severe OSAHS. Third, current cardiovascular models estimated the risk of cardiovascular mortality in the next 10 years. We limited our follow-up to one year; therefore, further long-term follow-up studies are needed in order to explore the consistency of our findings. Finally, we found an independent association between MACE and heart failure. We restricted our data to medical records, without further exploration of this comorbidity. Nevertheless, the link between cardiovascular risk, altered STOP BANG scores and heart failure has a strong association with risk of OSAHS and classical cardiovascular risk factors.

5. Conclusion

Cardiovascular risk is increased in hospitalized populations at risk for OSAHS. This population reported more comorbidities. Despite the potential association of risk of OSAHS and increased cardiovascular risk, after confounder analysis, heart failure was an independent risk factor in both men and women. Further analysis and long-term follow-up incorporating risk of OSAHS are needed to improve the accuracy of our current cardiovascular risk predictive models.

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Contribution

Dr. Labarca: Principal investigator, data extraction, data analysis, manuscript redaction and final approval.

Dr. Valdivia, Onate, Navarrete: Data extraction, data synthesis, manuscript redaction and final approval.

Dr. Dreyse, Fernandez-Bussy and Jorquera: Data conception, critical analysis, manuscript redaction and final approval.

All authors approve final version of this manuscript.

Conflict of interest

None declared.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2019.02.019.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.sleep.2019.02.019.

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