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CLINICAL INVESTIGATIONS

Is obstructive sleep apnea associated with ventricular tachycardia? A retrospective study from the National Inpatient Sample and a literature review on the pathogenesis of Obstructive Sleep Apnea

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Background: Obstructive sleep apnea (OSA) is a known independent risk factor for a multiple cardiovascular morbidities and mortality. The association of OSA and ventricular arrhythmias is less well understood. The aim of this analysis is to study the relationship between OSA and ventricular tachyarrhythmias.

Hypothesis: OSA is associated with increased ventricular arrhythmias.

Methods: Data from the national inpatient sample (NIS) 2012 to 2014, were reviewed. Discharges associated with OSA were identified as the target population using the relevant ICD-9-CM codes. The primary outcome was a diagnosis of ventricular tachycardia (VT) in the OSA population. Secondary outcomes include the rate of ventricular fibrillation (VF) and cardiac arrest. Multivariable analyses were performed to examine the association of VT with multiple potential confounding clinical variables.

Results: Of 18 013 878 health encounters, 943 978 subjects (5.24%) had a diagnosis of OSA. VT and VF were more prevalent among patients with OSA compared to those without a diagnosis of OSA (2.24% vs 1.16%; $P < 0.001$ and 0.3% vs 0.2%; $P < 0.001$, respectively). Odds ratio for cardiac arrest in OSA group was not statistically significant (1, 95% confidence interval 0.97-1.02, $P < 0.76$). In unadjusted analyses, all examined comorbidities were significantly more common in those with OSA, including diabetes mellitus, hypertension, chronic kidney disease, acute coronary syndrome, and heart failure.

Conclusion: OSA is associated with increased rates of ventricular tachyarrhythmia.

KEYWORDS

obstructive sleep apnea (OSA), ventricular arrhythmias, ventricular fibrillation (VF), ventricular tachycardia (VT)

1 | INTRODUCTION

Obstructive sleep apnea (OSA) is characterized by recurrent partial or complete pharyngeal airway collapse with subsequent temporary cessation of breathing during sleep. It is a common condition that affects at least 10% of the general population, primarily obese men.[1] OSA results in multiple physiological consequences that affect the cardiovascular system. The cardiovascular complications of OSA have been an important research topic in the past decade. The role of OSA in the development of atrial fibrillation, hypertension and heart failure is well explored in the literature.[2,3] OSA has been linked to sudden cardiac death.[4] However, the association of OSA and ventricular arrhythmias, particularly ventricular tachycardia (VT) is less well understood.

Ventricular arrhythmia is classified electrocardiographically into premature ventricular contractions (PVCs), VT and ventricular fibrillation (VF).[5] PVCs are generally benign, but if the burden exceeds

10%/24 hours., they can result in cardiomyopathy.[6] Nonsustained VT is defined as a series of at least three repetitive ventricular beats with duration of less than 30 seconds and not requiring emergency therapy while sustained VT lasts for more than 30 seconds or requires termination earlier because of hemodynamic compromise.[7] We hypothesize that OSA could be a significant independent predictor for development of ventricular tachyarrhythmia. We focused on VT and VF because of the associated mortality.

2 | METHODS

2.1 | Data source

We utilized the national inpatient sample (NIS), the largest national inpatient database in the United States. The NIS approximates a 20% stratified sample of all discharges from US community hospitals participating in the Healthcare Cost and Utilization Project (HCUP) and sponsored by the Agency for Healthcare Research and Quality (AHRQ).[8]

The data excludes rehabilitation and long-term acute care hospitals. When the data expands to estimate nation-wide discharges, it provides estimates corresponding to about 38 million annual hospitalizations.[9] Each hospitalization is treated as an individual database entry.

2.2 | Subjects and variables

The study population was derived from hospital discharges (18013878) available in the NIS. Patients with VT and OSA were identified using International Classification of Diseases-9th Edition (ICD-9) codes 427.1 and 327.23, respectively. The primary outcome was a diagnosis of VT in OSA population. Secondary outcome was the rate of VF and cardiac arrest in an OSA population. VF and cardiac arrest were also identified using the ICD 9 codes (427.41 and 427.5, respectively). Analyses of clinical variables were performed to examine the association of multiple confounding factors and VT. Confounding variables were identified using ICD-9 codes including both patient and hospital level variables, in addition to data year. Patient characteristics included age, gender, obesity, diabetes mellitus, heart failure, hypertension, acute coronary syndrome, prior stroke, chronic kidney disease (CKD), and Charlson-Deyo Index (CDI). The CDI is a validated measure to predict mortality in studies based on administrative databases. It is based on 17 indicators for comorbidities that affect the in-hospital mortality, such as heart failure, diabetes mellitus, chronic kidney disease, myocardial infarction, cerebrovascular disease, cancer, COPD, and AIDS. Higher score indicates more comorbidities and correlates with an increased risk of death.[10]

2.3 | Statistical analysis

Normally distributed continuous variables were expressed as means ± SD and compared using *t* tests. Continuous variables that were not normally distributed were expressed as medians with interquartile ranges and compared using Mann-Whitney test. Categorical variables were compared using the χ^2 test. In order to adjust for potential confounders and perform interaction testing, multivariable logistic regression models were employed.

All analyses were performed using STATA v25.0 (Stata Corp LLC, College Station, Texas). Two-tailed *P*-values <0.05 were considered statistically significant.

3 | RESULTS

We identified 18 013 878 discharges of patients ≥8 years from the NIS from 2012 to 2014. Of these, 943 978 subjects (5.2%) had a diagnosis of OSA, and 17 069 900 (94.8%) did not have OSA among their discharge diagnoses. The baseline characteristics of patients with and without OSA diagnosis are shown in Table 1.

In unadjusted analyses, patients with OSA were middle aged (mean 62.1 years) and 55.9% were males. Many comorbidities were significantly more common in those with OSA including diabetes mellitus (40.5% vs 20.2%, $P < 0.001$), hypertension (76.9% vs 50.4%, $P < 0.001$), CKD (15.8% vs 8.4%, $P < 0.001$), acute coronary syndrome (ACS) (5.4% vs 4.3%, $P < 0.001$) and heart failure (32.9% vs 14.1%, $P < 0.001$). Median Charlson-Deyo index was 2 in OSA group.

TABLE 1 The baseline characteristics of patients with and without obstructive sleep apnea diagnosis

Variables	OSA (n = 943 978)	No OSA (n = 17 069 900)	*P*-value
Age in years, means (SD)	62.1 (±13.5)	57.0 (±20.8)	< 0.001
Female, % (n)	44.1% (416223)	59.9% (10225940)	< 0.001
Obesity, % (n)	50.35% (475317)	10.11% (1726613)	< 0.001
Diabetes mellitus, % (n)	40.52% (382458)	20.22% (3450746)	< 0.001
Hypertension, % (n)	76.86% (725520)	50.39% (8601813)	< 0.001
Chronic kidney disease, % (n)	15.77% (148846)	8.40% (1433161)	< 0.001
Prior cerebrovascular accident, % (n)	1.61% (15202)	1.82% (311287)	< 0.001
Acute coronary syndrome, % (n)	5.39% (50875)	4. 26% (727730)	< 0.001
Heart failure, % (n)	32.87% (310315)	14.05% (2398101)	< 0.001
Charlson-Deyo index median(IQR)	2 (1-4)	1 (0-3)	< 0.001
Ventricular tachycardia, % (n)	2.24% (21.132)	1.16% (198257)	< 0.001
Ventricular fibrillation, % (n)	0.3% (2811)	0.2% (35, 319)	< 0.001

TABLE 2 Independent predictors of ventricular tachycardia in subjects with obstructive sleep apnea

Variables	Odds ratio	95% confidence interval	*P*-value
Age	1.012	1.011–1.012	< 0.001
Male gender	1.98	1.96–1.99	< 0.001
Obstructive sleep apnea	1.22	1.20–1.24	< 0.001
Diabetes mellitus	0.80	0.80–0.81	< 0.001
Hypertension	1.04	1.03–1.05	< 0.001
Acute kidney injury	1.42	1.41–1.46	< 0.001
Obesity	1.10	1.08–1.11	< 0.001
Acute coronary syndrome	3.12	3.08–3.16	< 0.001
Coronary artery disease	1.71	1.70–1.73	< 0.001
Heart failure	2.71	2.68–2.75	< 0.001
Diastolic heart failure	2.10	2.07–2.14	< 0.001
Systolic heart failure	6.30	6.22–6.38	< 0.001
Hyperlipidemia	1.14	1.13–1.16	< 0.001
Tobacco smoking	1.07	1.06–1.08	< 0.001
Charlson-Deyo index	1.019	1.017–1.022	< 0.001

TABLE 3 Independent predictors of ventricular fibrillation in subjects with obstructive sleep apnea

Variables	Odds ratio	95% confidence interval	*P*-value
Age	0.996	0.996–0.997	< 0.001
Male gender	2.02	1.99–2.08	< 0.001
Obstructive sleep apnea	1.02	0.98–1.07	0.27
Diabetes mellitus	0.79	0.77–0.81	< 0.001
Hypertension	0.91	0.89–0.93	< 0.001
Acute kidney injury	2.16	2.11–2.22	< 0.001
Obesity	1.12	1.08–1.16	< 0.001
Acute coronary syndrome	12.35	12.05–12.65	< 0.001
Coronary artery disease	1.86	1.81–1.91	< 0.001
Heart failure	2.47	2.39–2.55	< 0.001
Diastolic heart failure	0.99	0.94–1.05	0.78
Systolic heart failure	3.27	3.17–3.37	< 0.001
Hyperlipidemia	0.97	0.95–1.00	0.03
Tobacco smoking	0.99	0.97–1.01	0.24
Charlson-Deyo index	0.98	0.97–0.98	< 0.001

A total of 2.24% (21132) VT were observed in the OSA group vs 1.16% (198257) in the group with no OSA ($P < 0.001$). The adjusted odds rate of the VT in OSA group was 1.22 (1.20-1.24, $P < 0.001$). Table 2 shows independent predictors of VT in a multivariable analysis model. In addition to OSA, other independent predictors of the detected presence of VT were: male gender (1.98; 95% CI 1.96-1.99, $P < 0.001$), heart failure (2.71; 95% CI 2.68-2.75; $P < 0.001$) and ACS (3.12; 95% CI 3.08-3.16, $P < 0.001$).

Also, VT was higher in the acute kidney injury (AKI) (1.42, 95% CI 1.41-1.46, $P < 0.001$), hyperlipidemia (1.14, 95% CI 1.13-1.16, $P < 0.001$) and obesity group (1.10, 95% CI 1.08-1.11, $P < 0.001$). Further analysis shows systolic heart failure carries higher odds to demonstrate VT (6.30, 95% CI 6.22-6.38, $P < 0.001$) as compared to diastolic heart failure (2.10, 95% CI 2.07-2.14, $P < 0.001$).

Diabetes mellitus (0.80, 95% CI 0.80-0.81, $P < 0.001$), HTN (1.04, 95% CI 1.03-1.05, $P < 0.001$), tobacco smoking (1.07, 95% CI 1.06-1.08, $P < 0.001$) and Charlson-Deyo index (1.019, 95% CI 1.017-1.022, $P < 0.001$) were not associated with significant increased odds for VT detection.

Ventricular fibrillation was more common in the OSA 0.3% vs 0.2% than in the group with no OSA group, $P < 0.001$. Interestingly after multivariable regression analysis, the odds ratio of VF in OSA group was not significantly high (1.02, 95% CI 0.98-1.07, P 0.27) (Table 3). OSA population with ACS (12.35, 95% CI 12.05-12.65, $P < 0.001$), systolic heart failure (3.27, 95% CI 3.17-3.37, $P < 0.001$) and AKI had (2.16, 95% CI 2.11-2.22, $P < 0.001$) higher odds for development of VF. Similarly, odds ratio for cardiac arrest in OSA group was not statistically significant (1, 95% CI 0.97-1.02, $P < 0.76$).

4 | DISCUSSION

Despite the strong evidence regarding the role of OSA in development of hypertension, myocardial infarction, and heart failure, the evidence of a role in the development of cardiac arrhythmias, particularly VT, remains scarce, and conflicting. In 1970, MacGregor et al. first described an increased relative risk of sudden cardiac death in OSA patients of (23%).[11] Subsequent reports revealed higher prevalence of nocturnal tachyarrhythmia (14%) in OSA.[12] Also, individuals with sleep disorder breathing (SDB) were found to have a 3-fold risk of NSVT, and almost twice the odds of complex ventricular ectopy as compared to a population without SDB.[13]

In contrast to above mentioned, Flemons et al., found no differences in the prevalence of ventricular arrhythmias, atrioventricular block, and sinus arrest in 76 subjects diagnosed with SDB.[14]

This study analyzed a large database and found a positive relationship between OSA and VT, indicating an increased prevalence of VT in patients with OSA compared to those without (2.24% vs 1.16% $P < 0.001$). Even after adjustment for possible confounders, the odds for having VT in OSA patients was significantly higher than those

TABLE 4 Independent predictors of cardiac death in subjects with obstructive sleep apnea

Variables	Odds ratio	95% confidence interval	*P*-value
Age	1.010	1.009-1.011	<0.001
Male gender	1.43	1.41-1.44	<0.001
Obstructive sleep apnea	1.00	0.97-1.02	0.76
Diabetes mellitus	0.95	0.93-0.96	<0.001
Hypertension	0.84	0.83-0.85	<0.001
Acute kidney injury	3.66	3.61-3.71	<0.001
Obesity	1.02	1.00-1.04	0.06
Acute coronary syndrome	4.95	4.87–5.03	<0.001
Coronary artery disease	1.17	1.15-1.19	<0.001
Heart failure	1.76	1.72-1.79	<0.001
Diastolic heart failure	1.01	0.99-1.04	0.42
Systolic heart failure	1.54	1.51-1.58	<0.001
Hyperlipidemia	0.71	0.70-0.72	<0.001
Tobacco smoking	0.85	0.84-0.86	<0.001
Charlson-Deyo index	1.08	1.08-1.09	<0.001

without OSA (1.22; 95% CI 1.20-1.24, $P < 0.001$). Past medical illnesses, as expected, were more common in the OSA group for example, diabetes mellitus, hypertension, CKD, ACS and heart failure. This was also reflected in the higher CDI in the OSA group.

Interestingly, VF was slightly higher in the OSA group than the group with no OSA (0.3% vs 0.2%, $P < 0.001$). However, after multivariable regression analysis, the odds ratio for having VF in OSA was no longer statistical significant (Table 3). This suggests, either VT does not predict the occurrence of VF or the improvement in early management of patients with VT and the expansion of evidenced-based medical and device therapies may have lowered the burden for VF development. Of note, the odds ratio of atrial fibrillation in our subjects with OSA (vs not) was 1.64 (95% CI 1.63-1.66; $P < 0.001$), which is consistent with the current literature that emphasizes increased odds for AF of up to 4-fold in SDB.[15]

Because of the type of data in this administrative database, the possibility of a causative relationship between OSA and VT was not directly studied. However, there are theoretical potential mechanisms to explain the development of ventricular arrhythmias in OSA patients. Apnea-induced hypoxia and associated hypercapnia causing autonomic nervous system imbalance in addition to myocardial ischemia. Sympathetic nervous system stimulation then results in tachycardia and a surge in left ventricular afterload, with subsequent relative myocardial ischemia as the oxygen saturation is at its lowest. Not only is there increased sympathetic activity during the post-apneic periods, but also the daytime sympathetic nervous activity is increased.[16] Moreover, OSA causes acidosis, vascular endothelial dysfunction, systemic inflammation, and elevated oxidative stress, all of which can be potent stimuli for myocardial ischemia.[17] Individuals with OSA also have a paradoxical increase in coagulability during the night. Platelet activation and aggregation are increased while the fibrinolytic activity is decreased during sleep.[4] The latter might contribute to higher ischemic events with subsequent cardiac arrhythmias.

Hypoxia and respiratory acidosis trigger abnormal automaticity, which results in spontaneous cardiac impulse formation in the ventricles, and the enhanced sympathetic tone present in SDB can exacerbate this.[13] During the apneic spells, the inspiratory efforts against the occluded pharynx cause abrupt reduction in the intrathoracic pressure, with enhancement of the venous return, distension of the right ventricle, and leftward shift of the interventricular septum causing a reduced filling of the left ventricle.[18] This significant variation in the intrathoracic pressure produces transmural forces that contributes to atrial and ventricular free wall cardiac stretch and hence induces arrhythmias via mechanical electrical feedback mechanisms.[19] Parasympathetic nervous system activation results in QTc prolongation and increased heterogeneity of ventricular repolarization that is manifested as increased QTc dispersion. Distention of the right ventricle by an acute increase in pulmonary artery pressure, fluctuating intrathoracic pressure, cardiac distortion, and changes in venous return contribute to the myocardial electrical instability and hence the ventricular arrhythmias and sudden cardiac death.[20,21]

Finally, reentry mechanisms may occur through the vagal stimulation that results from respiration against a partially occluded airway, which may lead to bradycardia-dependent increased dispersion of atrial repolarization predisposing to intra-atrial entry.[13] Some studies have shown substantial decrease in VT after successful treatment of OSA, providing compelling evidence that at least in some patients, the OSA is the likely culprit.[7]

Continuous positive airway pressure (CPAP) is known to maintain upper airway patency by increasing transmural pressure of upper airways and consequently does not lead to sympathetic stimulation or cardiovascular consequences. It also decreases the cardiovascular events as shown in a large, nonrandomized trial with a mean follow-up of approximately 10 years.[22] CPAP treatment was shown to improve cardiac parameters, such as heart rate, QT-interval dispersion, systemic and pulmonary arterial pressures, and stroke volume in patients with OSA. CPAP results in 58% reduction of the frequency of ventricular premature complexes in patients with SDB and CHF during sleep and a parallel reduction in nocturnal urinary norepinephrine concentrations.[23] On the other hand, the SAVE study, a multicenter randomized open-label trial, showed that the CPAP did not prevent cardiovascular events in patients with moderate to severe OSA but it reduced snoring, daytime sleepiness, and improved health-related quality of life.[24]

4.1 | Study power

To the best of our knowledge, this is the first epidemiological study that incorporates such a large number of patients to assess the association of OSA and ventricular arrhythmias. Our primary analysis confirms the increased prevalence of VT in OSA, even after adjustment for possible biological confounders. The findings have strength, in that the HCUP is a well-validated database and has been previously utilized in similar research studies.

4.2 | Study limitation

ICD-9 codes were used to identify patient comorbidities. It was not possible to validate the accuracy of these ICD-9 codes. However, prior studies have demonstrated that ICD-9 codes exhibit high (>90%) sensitivity, specificity, and positive predictive value for cardiovascular disorders, such as HF, myocardial infarction, and arrhythmias, when compared to a full medical chart review.[25] Also, because of the nature of the NIS database analysis, functional outcomes and long-term data were not available.

Moreover, we did not have specific information regarding medications, electrophysiological studies, and echocardiographic parameters, which could have implications for VT outcome. Finally, as with most observational studies, residual confounding is always a concern and causality cannot be demonstrated.

5 | CONCLUSIONS

OSA results in a multitude of cardiovascular pathophysiological changes that result in cardiovascular morbidity and mortality. Our analysis finds that OSA is associated with VT and might be a predictor for development of ventricular arrhythmias. VT. Physician counseling should be provided regarding the importance of OSA treatment.

Despite the large number of subjects analyzed to examine the relationship between OSA and VT, the nature of the database prohibits the establishment of causality. Prospective studies are needed to confirm the theory that OSA causes VT.

CONFLICTS OF INTEREST

The authors declare no potential conflict of interests.

Author contribution

All authors had access to the manuscript and contributed to the manuscript.

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