

Sleep Apnea Testing and Outcomes in a Large Cohort of Medicare Beneficiaries with Newly Diagnosed Heart Failure

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Rationale: Previous studies have demonstrated a high prevalence of sleep apnea (SA) in patients with chronic heart failure (HF), which is associated with higher rates of morbidity, mortality, and health care use.

Objectives: To investigate the reported incidence, treatment, outcomes, and economic cost of SA in new-onset HF in a large U.S. database.

Methods: This retrospective cohort study used the 2003 to 2005 Medicare Standard Analytical Files and included subjects with newly diagnosed HF from the first quarter of 2004, without prior diagnosis of SA, stratified by testing, diagnosis, and treatment status.

Measurements and Main Results: Among a study population of 30,719 incident subjects with HF, only 1,263 (4%) were clinically suspected to have SA. Of these, 553 (2% of the total cohort) received SA testing, and 545 received treatment. After adjustment for age, sex, and comorbidities, subjects with HF who were tested, diagnosed, and treated for SA had a better 2-year survival rate compared with subjects with HF who were not tested (hazard ratio, 0.33 [95% confidence interval, 0.21–0.51], $P < 0.0001$). Similarly, among subjects who were tested and diagnosed, those who were treated had a better 2-year survival rate than those who were not treated (hazard ratio, 0.49 [95% confidence interval, 0.29–0.84], $P = 0.009$).

Conclusions: In Medicare beneficiaries with HF, comorbid SA is most often not tested and consequently subjects are underdiagnosed and not treated. Meanwhile, in the few subjects in whom a diagnosis of SA is established and treatment is executed, survival improves significantly. These results support the importance of SA testing and treatment for patients newly diagnosed with HF.

Keywords: heart failure; mortality; sleep apnea

Studies of consecutive patients with heart failure (HF) have consistently described that sleep apnea (SA) is a highly prevalent comorbid condition present in approximately 40 to 60% of subjects, a rate far higher than in the general population (1–11). Because SA induces myocardial hypoxia, increases sympathetic stimulation and oxygen demand, and is associated with inflammatory and oxidative stress, it is a likely contributor to HF disease progression, morbidity, and mortality (12–15). A recent American Heart Association/American College of Cardiology Foundation Scientific Statement on SA and cardiovascular disease (15) emphasized the importance of SA testing and diagnosis in the management of HF. Management of SA in HF through timely recognition and effective treatment has been shown to improve left ventricular ejection fraction, sympathetic activity, quality of life, and mortality endpoints (16–23).

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AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Studies of consecutive patients with heart failure (HF) have consistently described that sleep apnea (SA) is a highly prevalent comorbid condition present in approximately 40 to 60% of subjects, a rate far higher than in the general population, but little is known about the actual incidence in the community at large.

What This Study Adds to the Field

Our study demonstrates that among the Medicare population with newly diagnosed HF, those who were tested, diagnosed, and treated for SA had better survival compared with those who were not tested, not diagnosed, and consequently not treated. Meanwhile, only 2% of subjects with new-onset HF were tested for SA over a 2-year period, yet the prevalence of SA in HF has been reported to be about 50%.

Currently, SA is suspected through physician observation and examination and use of accepted questionnaires and diagnosed by in-laboratory polysomnography (PSG), overnight oximetry, or home-based cardiorespiratory testing systems. Today, despite various diagnostic tools that are available, the majority of patients with SA in the general population remain undiagnosed (24), and the rate of SA testing and treatment in patients with HF has not been adequately studied. Furthermore, although several studies have assessed the resource use or economic impact of SA diagnosis and treatment in the general population (24–28), to our knowledge, none has looked specifically at these issues in the HF population.

HF is the most common Medicare diagnosis-related group, and given the high prevalence of comorbid HF and SA and the vicious interactions leading to poor outcomes, the objectives of this study were to perform a population-based retrospective analysis of Medicare claims data to (1) identify the rate of SA testing and treatment in subjects with newly diagnosed HF, and (2) test the hypothesis that SA diagnosis and treatment lead to better economic and mortality outcomes among patients with HF with SA. Some of the results of these studies have been previously reported in the form of an abstract (29).

METHODS

Data Source

This retrospective analysis used the Medicare Standard Analytical Files (SAFs) provided by the Centers for Medicare and Medicaid Services, Baltimore, Maryland (http://www.cms.hhs.gov/IdentifiableDataFiles/02_StandardAnalyticalFiles.asp). The SAFs contain a 5% sample (1.7 million beneficiaries) of randomly selected beneficiaries (i.e., U.S.

citizens aged 65 years or older, those eligible for disability, and/or subjects with end-stage renal disease), except for a small number of beneficiaries who were also enrolled in managed care organizations. The SAF offers a near-census of use and expenditure data for Medicare beneficiaries and provides Medicare and others a near-census snapshot of health care use and expenditure data among the elderly. The SAFs are constructed from weekly data submissions to the National Claims History 100% Nearline File. Final action claims data are obtained by processing National Claims History claims through a series of algorithms designed to match original claims with adjustment claims and resolve all adjustments. There is an 18-month window in which approximately 98% of the claims for services provided in a given year are captured (30, 31). The data allow for longitudinal analyses of health care resource use, clinical epidemiology, and patient outcomes. Each year, the SAF database includes medical claims submitted for laboratory tests, inpatient hospital stays, outpatient care, physician care, skilled nursing facility care, home health care, durable medical equipment use, hospice care, and a denominator file that includes beneficiaries' demographic characteristics (age, sex, race, etc.) and vital status recorded with date of death. In this study, quarterly beneficiaries' claims data from different claims settings were linked via encrypted beneficiary identification numbers at each beneficiary level and were followed longitudinally from the first quarter of 2004 to the fourth quarter of 2005.

Study Population

According to the aim of the study, the population enrolled included all patients with HF newly diagnosed in the first quarter of 2004 who were not coded with diagnosis of HF or SA in 2003. Testing for SA was determined by marked codes for PSG or home-based cardiorespiratory testing in the SAF. The vast majority of testing was documented with codes for PSG. All possible treatments, including continuous positive airway pressure (CPAP), uvulopalatopharyngoplasty, jaw alignment and soft tissue reconstruction, oxygen use, and other surgical treatment, and their representative codes were searched. As a proxy for costs of medical resource use, Medicare payment was one of the key outcome variables in this study, so subjects without valid payment data were also excluded from the analysis.

The identified HF subjects were stratified into groups based on SA testing, diagnosis, and treatment status.

Baseline Assessment

Demographic characteristics (e.g., age, sex, and race) across different study groups were analyzed. To evaluate the role of comorbidities, we analyzed the data sets for subjects' comorbid conditions presented in the year before HF development. The Charlson Comorbidity Index (CCI) (32) was used, a technique validated in longitudinal studies of patients' administrative data, to measure the risk of 1-year mortality attributable to various comorbidities. CCI was based on the diagnosis codes presented in the year before HF development, and the CCI aggregate comorbidity measure contained 19 categories of comorbidity.

SA Outcomes Analyzed

Incidence. Reported incidence of SA among subjects with HF, use of SA testing, and SA treatment.

Treatment. All possible SA treatment codes were searched. Given the limited clinical information available in the medical claims, patient outcomes by SA severity via apnea-hypopnea index or other classification system could not be analyzed.

Hospitalization. The percentage of subjects hospitalized during the new-onset HF episode as well as during the follow-up period after the HF onset was compared. The total number of hospital admissions that occurred during the follow-up period was also calculated for each subject and compared across study groups, adjusted for survival.

Mortality. Using the denominator file, annual mortality in 2004 and 2005 and overall mortality in those 2 years were calculated and compared between subjects based on SA testing, diagnosis, and treatment status. The analysis included deaths that occurred in the inpatient setting (based on the patient status discharge indicator on the inpatient claim form) and also outpatient setting (based on notification of beneficiary death by the Social Security Administration). As the cause of death is not provided in the SAFs, only all-cause mortality was analyzed.

Medicare payment. Health care use costs were assessed from Medicare's perspective. Patient mortality resulted in a variable length of follow-up for each patient when evaluating costs. As the dates of claims in the SAFs were truncated to the nearest quarter, Medicare payment data were censored in the quarter in which a subject expired. The overall 2-year mean Medicare payments per patient and mean Medicare payments per follow-up quarter across all settings of care were calculated.

Statistical Analyses

All analyses were conducted using SAS software version 9.1 (SAS Institute, Inc., Cary, NC). Descriptive statistical analyses were performed for demographics, comorbidities, baseline Medicare payment, treatment rate, mortality, and Medicare payment for medical services across all settings of care.

When using procedure codes to identify a resource used, the indication for which a particular service was rendered could not always be established. For example, in the case in which both HF and chronic obstructive pulmonary disease conditions existed and supplemental O₂ was provided, it was not possible to determine whether the treatment was for HF, SA in HF, or for chronic obstructive pulmonary disease. Therefore, to increase the specificity of identifying treatments indicated for SA, we used a restrictive definition for treatment in which only a claim containing both prespecified SA treatment-related procedure codes and a coded SA diagnosis was deemed an SA treatment claim.

Analysis of variance was used to test for differences between all groups, Tukey-Kramer (correction for multiple comparisons) procedures for all continuous pairwise variables, and Chi-square tests were used for all categorical variables, where appropriate. Kaplan-Meier survival estimation was used to describe the 2-year survival for subjects in each study cohort. The hazard ratio for death occurring during the 2-year follow-up period was estimated using the proportional hazards regression model. Based on the descriptive analysis results, the models were built with an adjustment for confounding variables (i.e., CCI, age, and sex). Linear, nonlinear, and dichotomous association of age and mortality were examined in the model. In the nonlinear analysis, which was based on the observed death rates of the study patients, the age effect was modeled as a constant up to age 60 years, and as the square of age after that. The version of the age term that provided a better fit to the model was used in the analysis. In addition, patients were stratified by age (< 70 yr vs. ≥ 70 yr) and by sex (male vs. female), and the effect on SA testing on survival were explored in these subgroups. As sex was correlated with age, CCI, and mortality in the data set, interactions between sex and these covariates with respect to all-cause mortality were also examined.

The overall level of α significance for each statistical test was set at 0.05.

RESULTS

Study Cohort and Baseline Characteristics

A total of 119,302 subjects were diagnosed with HF in the first quarter of 2004, and after exclusion of subjects with preexisting HF ($n = 86,592$) and SA ($n = 1,027$), there were 31,683 subjects with newly diagnosed HF. Among them, 964 (3%) did not have completed Medicare payment data for 2004 and 2005 and were excluded. Thus, the study cohort consisted of 30,719 subjects. Of this cohort with newly diagnosed HF, 1,263 (4.1%) also had a claim with an SA diagnosis in 2004, and 572 (1.9%) were tested, of which 553 (97.0%) were diagnosed with SA (Figure 1). There were only 19 subjects who were tested and did not receive an SA diagnosis.

Subjects were stratified into groups for further analysis. As will be seen, there were a number of subjects with sleep-disordered symptoms who did not undergo laboratory testing yet were treated for SA. In contrast, there were subjects in whom SA was diagnosed but who were not treated. Therefore, the groups were analyzed based on SA testing, diagnosis, and treatment status in 2004 as follows:

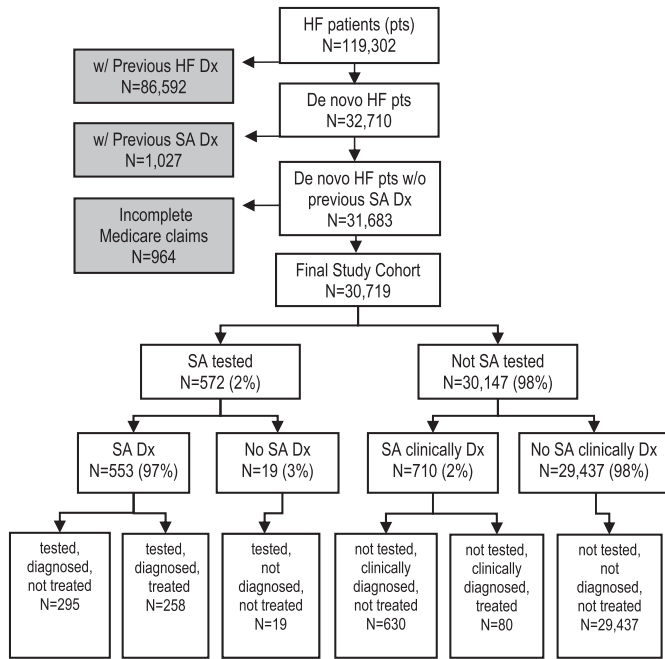


Figure 1. Study design showing the method and the sample sizes by which subjects with new-onset heart failure (HF) without previous sleep apnea (SA) coding was obtained for the study from Medicare Standard Analytic Files (SAFs) provided by the Centers for Medicare and Medicaid Services, Baltimore, Maryland. The SAFs contain a 5% sample (1.7 million beneficiaries). Dx = diagnosis.

- tested, diagnosed, not treated (n = 295)
- tested, diagnosed, treated (n = 258)
- tested, not diagnosed, not treated (n = 19)
- not tested, clinically diagnosed, and not treated (n = 630)
- not tested, clinically diagnosed, treated (n = 80)
- not tested, not diagnosed, not treated (n = 29,437)

Demographics, comorbidity, and treatment characteristics are shown in Table 1. SA-diagnosed (lab tested and clinically diagnosed) subjects tended to be younger and more likely to be male than SA-not diagnosed subjects. The groups did not differ significantly in comorbidity status.

Treatment

Among all of the various possible SA treatments searched, CPAP and oxygen were the only treatments used, with CPAP being more frequently used during both years (n = 512 out of 545 subjects). Some subjects were treated with CPAP and also

received supplemental oxygen. Overall, 545 subjects (1.8%) received SA-related treatment in both years, with 400 (1.3% of total and 73.3% of the subjects treated) having an SA diagnosis. There were 145 subjects who did not have an SA diagnosis in 2004 but were being treated with SA therapy in 2005.

Mortality

In each year, the tested, diagnosed, and treated group had significantly lower mortality than all other groups, whereas the not tested, not diagnosed, and not treated group had the highest mortality (30.0% during the 2 yr; Table 2). Survival analysis was performed between the tested, diagnosed, and treated group and the not tested and not treated groups, adjusted for age, sex, and CCI. Regardless of how age was used in the model (linear term, nonlinear term, or dichotomous), the results remained the same, although the nonlinear analysis produced the highest predictive power (Figure 2). Patients who were tested, diagnosed, and treated for SA had a much better survival than those who were not tested and not treated (hazard ratio [HR], 0.33; 95% confidence interval [CI], 0.21–0.51; *P* < 0.0001). Furthermore, when survival was compared by treatment status among subjects who were tested and diagnosed, the hazard ratio associated with treated subjects was 0.49 (95% CI, 0.29–0.84; *P* = 0.009) after adjustment for age, sex, and CCI (Figure 3).

An age-stratified analysis suggested that SA testing was an independent predictor of survival in both age groups: HR = 0.58 (95% CI, 0.41–0.83) for patients younger than 70 years of age; HR = 0.40 (95% CI, 0.29–0.56) for patients 70 years or older. Similarly, SA testing remained a significant predictor of survival in the sex-stratified analysis. None of the interaction terms between sex and other covariates was significant in predicting death.

Hospitalization

Approximately 51.5% of the study cohort was hospitalized during their new-onset HF episode in the first quarter in 2004. Table 2 (bottom row) shows that of the 23,320 (76%) subjects who were hospitalized one or more times during the 2-year period, the rates for the groups varied from 74.4% (tested, diagnosed, and treated) to 90.5% (not tested, clinically diagnosed, and not treated), with *P* < 0.0001 between all groups.

Medicare Payments

Average Medicare payments per patient over the 2 years studied ranged from \$36,357 to \$63,747 across the groups analyzed. After adjusting for the length of follow-up, subjects who were tested for SA had lower quarterly Medicare payments than those who were not tested (Figure 4). The average Medicare payment per quarter was lowest in subjects who were tested, diagnosed, and treated (\$5,758) and highest in subjects who were not tested, clinically diagnosed, and not treated (\$10,759).

TABLE 1. PATIENT CHARACTERISTICS STRATIFIED BY SLEEP APNEA TESTING AND DIAGNOSIS

Patient Characteristics	Tested, Diagnosed (N = 553)	Tested, Not Diagnosed (N = 19)	Not Tested, Clinically Diagnosed (N = 710)	Not Tested, Not Clinically Diagnosed (N = 29,437)	<i>P</i> Value
Age, yr, mean (SD)	67.1 (12.1)	72.6 (11.7)	69.4 (12.3)	76.5 (10.9)	<0.001
Sex, %, male/female	56.6/43.4	51.1/48.9	50.6/49.4	42.4/57.6	<0.001
Charlson Comorbidity Index, N (%)					
<1	138 (25.0)	3 (15.8)	178 (25.1)	7,175 (24.4)	0.7917
≥1	415 (75.1)	16 (84.2)	532 (74.9)	22,262 (75.6)	
Treated for SA in 2004 and 2005, N (%)	290 (52.4)	1 (5.3)	110 (15.5)	144 (0.5)	
Treatment type in 2004 and 2005, N (%)					
CPAP	278 (50.3)	1 (5.3)	97 (13.7)	136 (0.5)	<0.001
Oxygen	107 (19.4)	0 (0)	40 (5.6)	17 (0.0)	<0.001

Definition of abbreviations: CPAP = continuous positive airway pressure; SA = sleep apnea.

TABLE 2. ALL-CAUSE MORTALITY AND HOSPITALIZATION STRATIFIED BY SLEEP APNEA TESTING, DIAGNOSIS, AND TREATMENT STATUS

	Tested, Diagnosed, Not Treated (N = 295)	Tested, Diagnosed, Treated (N = 258)	Tested, Not Diagnosed, Not Treated (N = 19)	Not Tested, Clinically Diagnosed, Not Treated (N = 630)	Not Tested, Clinically Diagnosed, Treated (N = 80)	Not Tested, Not Diagnosed, Not Treated (N = 29,437)	P Value
Mortality 2004, N (%)	21 (7.1)	9 (3.5)	2 (10.5)	122 (19.4)	14 (17.5)	5,892 (20.0)	<0.001
Mortality 2005, N (%)	23 (8.4)	11 (4.4)	0 (0)	63 (12.4)	7 (10.6)	2,950 (12.5)	0.0006
Overall mortality, N (%)	44 (14.9)	20 (7.8)	2 (10.5)	185 (29.4)	21 (26.3)	8,842 (30.0)	<0.001
No. of patients hospitalized (%)	227 (76.9)	192 (74.4)	16 (84.2)	570 (90.5)	61 (76.2)	22,254 (75.6)	<0.0001

DISCUSSION

From the results of this study, we draw two main conclusions: (1) in Medicare beneficiaries with HF, SA is very much underdiagnosed; and (2) when SA is diagnosed via testing, treated patients have better survival compared with those who are not treated. These results affirm the role of SA testing and treatment in improving outcome in Medicare patients with new-onset HF and raise important implications on the need to properly test, diagnose, and treat patients with SA with HF.

To our knowledge, this is the first study to analyze SA diagnosis and testing in subjects with HF using a large population-based data set. Although the prevalence of SA in HF is well documented in the range of 40 to 60% (2–4, 6–10, 33), our results indicate that far fewer patients with HF (about 2%) are tested for SA in clinical practice, demonstrating that SA is underdiagnosed in this population. Our data raise the possibility that underdiagnosing SA in HF by health care providers in the United States is associated with excess mortality and health care resource use.

The undertesting for SA in the Medicare HF population studied contributed to the underdiagnosis of SA. As noted, of 572 subjects tested, 97% were diagnosed with SA. Based on multiple studies (2–7) of consecutive patients with HF, we estimate that 50% of this Medicare population of 30,719 might suffer from SA, yet only 2% of all subjects were tested for SA. Although the true prevalence of SA in this population is unknown for a number of reasons, 2% could considerably be an underestimation; the discrepancy is large and was unexpected. There are likely multiple reasons for this low rate of testing. It is not clear what is guiding the physician in terms of a decision to test for SA. A lack of availability of resources and

patients not agreeing to be tested are possible. Another factor that likely contributes to underdiagnosis is that subjects with HF often do not have the classic SA symptoms, such as excessive daytime sleepiness (3, 4, 34, 35). Furthermore, patients with HF with central sleep apnea (CSA) do not snore much, which is why their condition remains occult (3). Despite this, there are a number of predictors that, when present, should increase suspicion for sleep apnea and testing for SA diagnosis (4, 6, 34). Improved physician awareness of the relatively close association between HF and SA should encourage more testing of the HF population for SA to ensure that patients with comorbid SA and HF are properly diagnosed and treated. In the current findings, only 3% of patients tested did not have the disease, suggesting a high prevalence of SA in these patients. We suspect that most patients tested and diagnosed suffered from obstructive SA because the hallmarks of obstructive sleep apnea (OSA) (i.e., snoring and obesity) are apparent in such patients compared with those having CSA (3, 4, 6, 34).

When considering survival and after accounting for a number of variables and comorbidities (age, sex, and CCI), we found that subjects with HF who were tested, diagnosed, and treated for SA had a 2-year probability of survival of about 90% compared with 70% for those who were not tested (Figure 2). Although multiple other factors might have contributed to the difference in survival between the two groups (e.g., those tested were younger, were more compliant with medications and had better quality of medical care than those who were not tested, etc.), these results are consistent with evidence that effective treatment of SA in HF improves outcomes. In this regard, treatment of OSA with nasal CPAP significantly improves

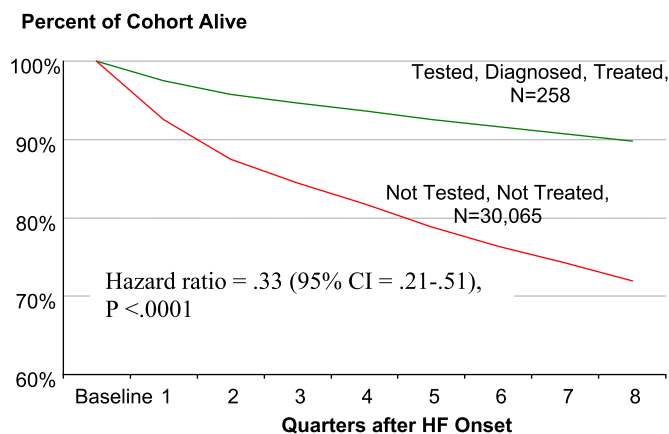


Figure 2. Kaplan-Meier survival curves for the tested, diagnosed, and treated subjects versus not tested and not treated subjects, adjusted by age, sex, and Charlson Comorbidity Index, 2004 through 2005. CI = confidence interval; HF = heart failure.

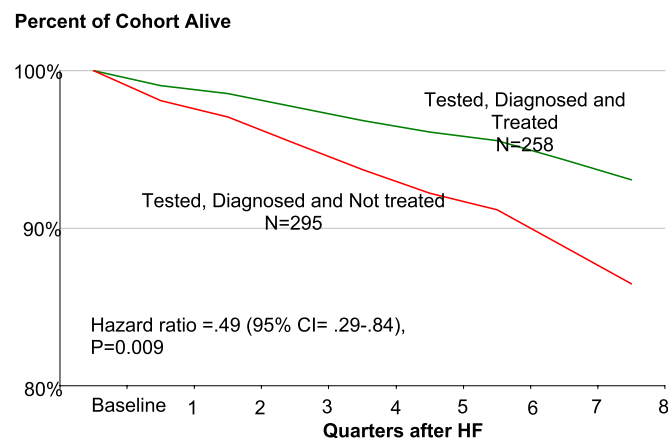


Figure 3. Kaplan-Meier survival curves for tested, diagnosed, and treated subjects versus tested, diagnosed, and not treated subjects, adjusted by age, sex, and Charlson Comorbidity Index, 2004 through 2005. CI = confidence interval; HF = heart failure.

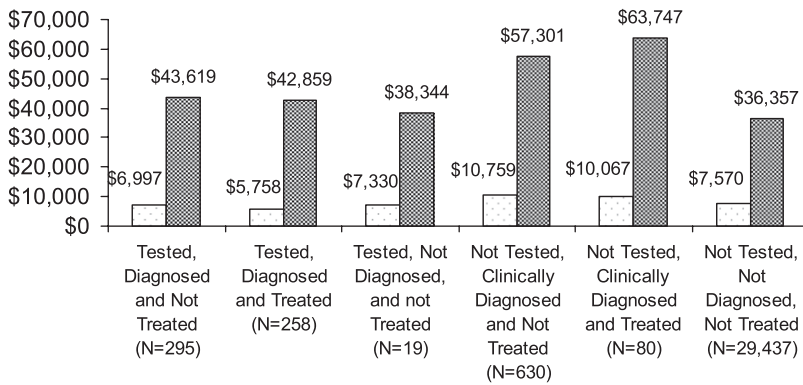


Figure 4. Mean 2-year Medicare payments per patient (shaded bars) and mean quarterly Medicare payments per patient (open bars).

cardiac function and survival of patients with HF (16–21), and as noted, we suspect that most of the Medicare patients had OSA. Similarly, effective CPAP therapy decreases nocturnal ventricular arrhythmias (36) and improves left ventricular ejection fraction and survival of patients with HF with CSA, even though adherence to CPAP is limited (22). Furthermore, oxygen therapy attenuates frequency of disordered breathing events (37), decreases sympathetic activity (38, 39) and brain natriuretic peptide (40), and increases left ventricular ejection fraction in subjects with CSA (41). Meanwhile, as noted above, the subjects with HF not tested, not diagnosed, and not treated ($n = 30,065$) had the highest mortality. We acknowledge that the true contribution of SA to mortality in this Medicare cohort is unknown and our data may overestimate it, yet based on the available literature, underdiagnosed and untreated SA contributed in part to excess mortality (12–14, 20, 21, 35, 42).

There were also a number of subjects in whom SA was diagnosed, but they were not treated for it. The mortality of this group of subjects was twice as high as those who were treated (Table 2, Figure 3). Meanwhile, we speculate that this group of subjects had mild SA, and therefore a decision was made not to treat the SA, and perhaps to more aggressively treat the HF, which has been shown to improve SA (43). Although there are several other possible reasons that these subjects were not treated, including refusal to be treated, our aforementioned speculation is consistent with better survival of these subjects compared with those who were not tested, many of whom suffered from SA. Because only 2% of all subjects with HF were tested for SA, we also speculate that the quality of medical care of the subjects who were tested for SA was superior to that of the remaining subjects who were not tested.

As noted above, there were also a small number of subjects ($n = 80$) who were not tested but were treated for SA. Although we do not know the circumstances for this surprising finding, this group of subjects had a high mortality rate (Table 2); possible reasons for this include poor therapy choice (CPAP versus oxygen), poor administration of therapy (such as inappropriately low or high CPAP levels), poor compliance, or incorrect diagnosis. Therefore, any treatment for undiagnosed SA is unwarranted.

Limitations

Our study has a number of limitations. Because this was an observational study, there was no control over treatment assignment (e.g., SA testing). Therefore, treatment effects may have been biased by covariates. Although we adjusted for the CCI, age, and sex, adjustment for other patient characteristics was not performed. It is possible that patients had to be healthy and mobile enough to undergo an overnight PSG. It is also possible that the SA-tested patients had better access to care or received

better quality of care than those not-tested patients in the first place. An alternative method (44) of using a propensity score in the analysis might strengthen the statistical rigor; however, such analysis of this data set is difficult to generate for a number of reasons. First, it is unclear which criteria guide a physician’s decision to test a patient for SA. Although certain predictors may trigger suspicion of SA, it is not clear to what extent they impact the decision to test for SA. Often resource constraints and patient preferences (e.g., a patient’s refusal to undergo testing) influence the decision to test someone for SA. The extent to which these factors impact decision making is not known. Therefore, it is not possible to generate an accurate or complete list of covariates that would allow us to predict the likelihood of undergoing SA testing and of receiving diagnosis of SA. Second, although some SA risk factors (e.g., body mass index) and symptoms (e.g., snoring, periods of not breathing, and daytime sleepiness) may prompt a physician to test a patient for SA, they are not tracked in the Medicare claims databases. Symptoms for CSA usually remain occult, and when they manifest themselves, they are not coded on medical bills or claims. Last, Medicare prescription drug information was not available at the time of this analysis, restricting our ability to track patients’ medication history, which may be predicative of their health status. Given these limitations, it is not feasible to construct a logistic model with clinically meaningful covariates to predict diagnosis or treatment distribution. In light of the potential impact of covariates, the magnitude and significance of the benefit associated with SA testing may be overestimated.

Our primary interest was subjects with newly diagnosed HF without previous diagnosis of SA based on claims history. Whether our observations apply to a population of patients with prevalent HF is uncertain. In this study, subjects with HF were grouped based on SA testing and treatment status. The database provides no insight as to why some subjects were tested and others with a clinical diagnosis of SA were not tested. Similarly, there is no method of determining whether subjects were compliant with treatment when prescribed, but improved survival of these subjects suggests adherence to therapy. As the accuracy of the mortality analysis depends on the timeliness of notification of a beneficiary’s death by caregivers, a margin of error is possible but should be small because financial and benefits transactions of many stakeholders are dependent upon this information being furnished. Last, our analysis was based on subjects’ medical claims, which were collected for the purpose of making health care payments instead of clinical research; therefore, the diagnosis of HF, diagnosis of SA, and resource use (i.e., testing and treatment) were subject to miscoding. The classification of SA testing and treatment was based on procedure codes as identified in payer policies, clinical guidelines, and through physician consultation. This has been

used accurately in the past (30, 31). There are strong financial incentives for appropriate claims filing for SA testing and treatment, so it is unlikely that the data set grossly underestimates the true rates. Because sleep testing is expensive, few Medicare beneficiaries are likely to pay out of pocket. Sleep laboratories and durable medical equipment providers likewise have strong financial incentives to report claims. Even if these data underestimate the true rate of testing and treatment by 100%, the gap between what is being reported and what we know the prevalence of sleep-disordered breathing in congestive HF to be is enormous. Last, but not least, we could not tell exactly if the diagnosis of SA was CSA or OSA, although we speculate that it was mostly patients with obvious risks for OSA who were screened and treated. However, as noted above, in patients with HF, both forms of SA are associated with excess mortality, and effective treatment of both CSA and OSA is associated with improved survival.

Conclusions

Our study demonstrates that among the Medicare population with newly diagnosed HF, those who were tested, diagnosed, and treated for SA had better survival compared with those who were not tested, not diagnosed, and consequently not treated. Meanwhile, only 2% of subjects with new-onset HF were tested for SA over a 2-year period, yet the prevalence of SA in HF has been reported to be about 50%. Although more studies are needed to determine the relationship between SA, HF, and treatment on outcomes, our results contribute to emerging data in support of SA testing of patients newly diagnosed with HF.

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APPENDIX A: LIST OF CODES USED FOR ANALYSIS

ICD9 PROCEDURES CODES

Item	ICD-9 Code	Descriptor
Sleep apnea	786.03	Apnea (under symptoms involving respiratory system and other chest symptoms)
	786.04	Cheyne-Stokes respiration
	780.51	Insomnia with sleep apnea
	780.53	Hypersomnia with sleep apnea
	780.57	Other and unspecified sleep apnea
	327.2x	Organic sleep apnea
		327.2x was added in 2006 and includes:
		327.20 Organic sleep apnea, unspecified
	327.21 Primary central sleep apnea	
	327.23 Obstructive sleep apnea (adult) (pediatric)	
	327.25 Congenital central alveolar hypoventilation	
	327.27 Central sleep apnea in conditions classified elsewhere	
	327.29 Other organic sleep apnea	

ICD9 PROCEDURES CODES

Item	ICD-9 Code	Descriptor
Sleep testing	89.17	Polysomnogram (specifies recording)
CPAP and related	93.90	Continuous positive airway pressure (CPAP) (includes bi-level airway pressure and noninvasive positive pressure [NIPPV])
Uvulopalatopharyngoplasty	27.69	Other plastic repair of palate
Jaw alignment and soft tissue reconstruction	76.62	Open osteoplasty (osteotomy) of mandibular ramus
	76.63	Osteoplasty (osteotomy) of body of mandible
	76.64	Other orthognathic surgery on mandible

CPT PROCEDURES CODES

Item	CPT/HCPCS Code	Descriptor
Sleep testing	95806	Sleep study, simultaneous recording of ventilation, respiratory effort, ECG or heart rate, and oxygen saturation, unattended by a technologist
	95807	Sleep study, simultaneous recording of ventilation, respiratory effort, ECG or heart rate, and oxygen saturation, attended by a technologist
	95808	Polysomnography; sleep staging with 1–3 additional parameters of sleep, attended by a technologist
	95810	Polysomnography; sleep staging with 4 or more additional parameters of sleep, attended by a technologist
	95811	Polysomnography; sleep staging with 4 or more additional parameters of sleep, with initiation of continuous positive airway pressure therapy or bilevel ventilation, attended by a technologist
Surgical treatments	21193	Reconstruction of mandibular rami, horizontal, vertical, C, or L osteotomy; without bone graft
	21194	Reconstruction of mandibular rami, horizontal, vertical, C, or L osteotomy; with bone graft
	21195	Reconstruction of mandibular rami and/or body, sagittal split; without internal rigid fixation
	21196	Reconstruction of mandibular rami and/or body, sagittal split; with internal rigid fixation
	21198	Osteotomy, mandible, segmental;
	21199	Osteotomy, mandible, segmental; with genioglossus advancement