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Brief Report

Predominantly Structural Cardiac Differences in Untreated Normal-Weight Obstructive Sleep Apnea: A Comprehensive Echocardiographic Study

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Abstract

Background and Objectives: Obstructive sleep apnea (OSA) is associated with cardiac remodeling, although the frequent coexistence of obesity complicates interpretation of cardiovascular abnormalities. We investigated the distribution of structural and functional echocardiographic differences across cardiac domains in untreated normal-weight adults with OSA. *Material and Methods*: In this prospective, single-center, cross-sectional study, 29 untreated patients with OSA and 15 controls without OSA, all with body mass index < 25 kg/m^2, underwent respiratory polygraphy and comprehensive transthoracic echocardiography. Left ventricular (LV) mass index (LVMI) was the primary outcome. *Results*: Most patients with OSA had mild disease (21/29, 72%). Patients with OSA had higher LVMI than controls (79.3 ± 17.1 vs. 64.5 ± 9.1 g/m^2; $p < 0.001$), together with greater interventricular septal and posterior wall thickness and higher LV end-diastolic volume index. In contrast, no consistent between-group differences were observed across conventional indices of LV systolic and diastolic function, atrial structure, or right ventricular function. Standardized effect sizes were largest for LV structural measurements, whereas estimates across functional, atrial, and right ventricular domains were generally smaller and less precise. After adjustment for age, sex, body mass index, and arterial hypertension, the association between OSA and higher LVMI persisted in the primary model (B = 10.56 g/m^2; $p = 0.047$; bias-corrected and accelerated (BCa) bootstrap 95% CI, 1.71–21.16), although sensitivity analysis indicated limited robustness. *Conclusions*: In untreated normal-weight adults with predominantly mild OSA, comprehensive echocardiographic assessment revealed findings suggestive of a predominantly structural pattern of cardiac differences, centered on the LV, without a consistent parallel signal across conventional functional, atrial, or right ventricular indices. These findings provide a multidomain characterization of cardiac involvement in OSA in the absence of excess body weight and warrant confirmation in larger longitudinal studies.

Keywords: obstructive sleep apnea; echocardiography; cardiac remodeling; left ventricular structure; normal weight; cardiovascular phenotyping

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1. Introduction

Obstructive sleep apnea (OSA) has been associated with left ventricular (LV) hypertrophy, altered cardiac geometry, and myocardial dysfunction [1,2]. However, the cardiac phenotype reported across studies is heterogeneous, reflecting differences in OSA severity, cardiovascular comorbidities, imaging methodology, and, importantly, the frequent coexistence of obesity [2,3]. Obesity independently promotes LV remodelling and may therefore obscure the cardiac abnormalities associated with OSA [3,4].

Beyond cardiac remodeling, OSA has also been investigated in relation to coronary artery disease and cardiovascular prognosis. Recent studies have explored its association with coronary anatomical complexity, including the SYNTAX score, while prospective data in patients with unstable angina have linked OSA to a higher risk of major adverse cardiovascular and cerebrovascular events in selected metabolic subgroups [5,6]. These observations highlight the broader cardiovascular relevance of OSA, although coronary atherosclerotic burden, cardiovascular outcomes, and echocardiographic remodeling represent distinct aspects of cardiac involvement. Previous studies have addressed the interaction between obesity and cardiac remodelling using statistical adjustment, comparisons across obesity strata, or imaging studies in non-obese populations [3,4,7,8]. Cho et al. evaluated myocardial function in patients with OSA in comparison with normal-weight and obese controls, whereas cardiac magnetic resonance studies have demonstrated adverse LV remodelling in non-obese OSA [7,8]. Nevertheless, relatively limited data are available from untreated adults who are strictly normal weight (body mass index [BMI] < 25 kg/m^2), particularly when structural, systolic, diastolic, atrial, and right ventricular echocardiographic domains are assessed within the same study. Characterizing the distribution of cardiac differences across these domains may provide a more integrated description of cardiac involvement in this selected OSA phenotype.

We therefore performed comprehensive echocardiographic phenotyping in untreated normal-weight adults with OSA and prospectively selected normal-weight controls without OSA, with LV mass index as the primary structural outcome, to characterize the distribution of between-group differences across cardiac domains.

2. Materials and Methods

Between November 2025 and June 2026, in this prospective, single-center, cross-sectional study, we consecutively screened adults referred to the Respiratory Medicine Unit of the Azienda Ospedaliera Universitaria Integrata of Verona (Italy) for suspected OSA. During the period, 307 individuals underwent overnight respiratory polygraphy, of whom 215 were diagnosed with OSA. Patients with untreated OSA and normal weight (BMI < 25 kg/m^2) were eligible. Exclusion criteria were age < 18 years, previous OSA treatment, heart failure, significant valvular heart disease, cardiomyopathy, atrial fibrillation, previous myocardial infarction, or inadequate echocardiographic image quality.

Normal-weight adults without OSA (apnea–hypopnea index [AHI] < 5 events/h), evaluated during the same study period, were prospectively selected as controls before echocardiographic assessment to obtain groups with broadly comparable age, sex, and BMI distributions. Selection was based on demographic comparability rather than predefined individual matching criteria. Controls underwent the same respiratory polygraphy, comprehensive echocardiographic assessment, and cardiovascular eligibility screening as patients with OSA and fulfilled the same exclusion criteria. Control selection was completed before echocardiographic data were reviewed to minimize selection bias.

One patient with OSA was excluded because inadequate echocardiographic image quality prevented reliable measurements. The final study population comprised 29 patients

with untreated OSA and 15 controls without OSA. No echocardiographic measurements were missing among the 44 participants included in the final analysis.

Demographic and clinical variables included age, sex, BMI, smoking status, arterial hypertension, diabetes mellitus, and chronic obstructive pulmonary disease (COPD). Respiratory polygraphy was performed using a portable Nox T3s™ device (Nox Medical, Reykjavík, Iceland). Only recordings with adequate signal quality were included. Recorded signals included thoracic and abdominal respiratory inductance plethysmography (RIP), peripheral pulse oximetry oxygen saturation (SpO_2), snoring, and body position. Respiratory flow was estimated from thoracic and abdominal RIP signals using the Nox calibrated RIP flow (cRIP Flow), without a nasal pressure cannula. Recordings were analyzed using the automated respiratory event-detection algorithm implemented in Noxturnal™ software version 6.1 (Nox Medical, Reykjavík, Iceland). Automatically detected respiratory events were subsequently reviewed and, when necessary, manually corrected by an experienced pulmonologist specialized in sleep medicine. Respiratory events were scored according to the American Academy of Sleep Medicine criteria [9]. Apnea was defined as a ≥90% reduction in airflow for ≥10 s, and hypopnea as a ≥30% reduction in airflow associated with ≥3% oxygen desaturation. The apnea–hypopnea index (AHI), oxygen desaturation index (ODI), oxygen saturation nadir, percentage of recording time spent with oxygen saturation < 90% (T_{90}), and the Epworth Sleepiness Scale (ESS) score [9–11] were recorded.

Comprehensive transthoracic echocardiography was performed using a Vivid 7 ultrasound system (GE Vingmed Ultrasound, Horten, Norway) by an experienced cardiologist blinded to respiratory polygraphy results, according to established echocardiographic recommendations [12,13]. The echocardiographic assessment encompassed LV structure, LV systolic and diastolic function, atrial structure, and right ventricular indices. Measurements included LV end-diastolic diameter (LVEDd), interventricular septal thickness in diastole (IVSd), posterior wall thickness in diastole (PWd), LV mass index (LVMI), global longitudinal strain (GLS), left atrial volume index measured from the apical four-chamber view (4Ch-LAVI), and indices of diastolic and right ventricular function. LV end-diastolic and end-systolic volume indices (EDVI and ESVI, respectively) and LV ejection fraction (LVEF) were obtained using the biplane method of Simpson (or biplane disk summation method) from apical four- and two-chamber views according to established recommendations. Left ventricular mass was calculated from linear echocardiographic measurements using the ASE-recommended corrected cube formula (Devereux formula): LV mass (g) = $0.8 \times 1.04 \times [(\text{LVEDd} + \text{IVSd} + \text{PWd})^3 - \text{LVEDd}^3] + 0.6$, with linear dimensions expressed in centimeters. LV mass was indexed to body surface area (LVMI, g/m^2), according to ASE/EACVI recommendations [12]. Left ventricular hypertrophy was defined using sex-specific ASE/EACVI reference thresholds for linearly derived LVMI (>115 g/m^2 in men and >95 g/m^2 in women) [12].

No formal a priori sample-size calculation was performed because of the exploratory nature of the study and the limited availability of untreated normal-weight patients with OSA; the final analytical sample included participants selected according to the study eligibility and control selection procedures during the prespecified recruitment period. Continuous variables were assessed for normality using the Shapiro–Wilk test and are presented as mean ± standard deviation (SD) or median [interquartile range (IQR)], as appropriate. Between-group comparisons were performed using Welch's *t*-test for normally distributed continuous variables, Mann–Whitney *U* test for non-normally distributed continuous variables, or Fisher's exact test for categorical variables.

LV mass index was the prespecified primary outcome and principal measure of LV structure. All other echocardiographic measurements were considered secondary exploratory outcomes and were examined across structural, systolic, diastolic, atrial, and right ventricu-

lar domains. To complement between-group hypothesis testing and describe the magnitude and precision of differences across echocardiographic domains, standardized effect sizes were expressed as Hedges' g with 95% confidence intervals (CIs). Hedges' g was calculated from the original continuous measurements, with 95% CIs derived using the standard small-sample variance estimator.

Multivariable linear regression was prespecified to evaluate the association between OSA and LVMI after adjustment for age, sex, BMI, and diagnosed arterial hypertension. Given the limited sample size, bias-corrected and accelerated (BCa) bootstrap 95% CIs based on 2000 resamples were calculated to characterize the uncertainty of the regression coefficient estimates. Multicollinearity was assessed using variance inflation factors, and influential observations using Cook's distance and leverage values. Regression assumptions were assessed by visual inspection of residual histograms, normal P-P plots, and standardized residuals versus predicted values. A sensitivity analysis was performed by refitting the regression model after excluding the observation with the highest Cook's distance.

All tests were two-sided, and $p < 0.05$ was considered statistically significant. Analyses were performed using IBM SPSS Statistics version 30 (IBM Corp., Armonk, NY, USA).

3. Results

Forty-four normal-weight participants were included: 29 patients with untreated OSA and 15 prospectively selected normal-weight controls without OSA. Patients with OSA were numerically older and more frequently male than controls, while BMI distributions were relatively similar (Table 1). Diagnosed arterial hypertension was more frequent among participants with OSA (48% vs. 20%), although the precision of this comparison was limited by the small sample size. As expected, participants with OSA had higher AHI and ODI, lower nadir SpO_2, and greater T_{90}, whereas daytime sleepiness was similar between groups. OSA was predominantly mild: 21 patients (72%) had mild, 6 (21%) moderate, and 2 (7%) severe disease (Tables 1 and 2).

For the prespecified primary outcome, LVMI was higher in participants with OSA than in controls (79.3 ± 17.1 vs. 64.5 ± 9.1 g/m^2; $p < 0.001$). Other LV structural measures showed differences in the same general domain: participants with OSA had greater IVSd and PWd and higher EDVI, whereas LVEDd was numerically greater but estimated imprecisely (Table 1). Despite the higher mean LVMI observed in participants with OSA, only one patient with OSA (3.4%) and no controls exceeded the sex-specific reference thresholds for left ventricular hypertrophy (Fisher's exact test, $p = 1.000$).

In contrast, no consistent between-group differences were observed across conventional LV systolic or diastolic functional indices, atrial measures, or most right ventricular indices. LVEF, GLS, mitral E velocity, E/A ratio, average e′ velocity, E/e′ ratio, 4Ch-LAVI, RAAI, tricuspid S′, and RVOT diameter were broadly similar between groups. RVOT VTI was modestly lower in participants with OSA; given the exploratory nature and number of secondary comparisons, this isolated finding should be interpreted cautiously (Table 1 and Supplementary Table S1).

The distribution of standardized between-group differences across echocardiographic domains is shown in Figure 1. The largest effect-size estimates were concentrated among LV structural measures, whereas estimates for systolic, diastolic, atrial, and right ventricular indices were generally smaller and less precise, with most 95% confidence intervals crossing zero.

Table 1. Clinical characteristics and echocardiographic findings across cardiac domains.

Variables	Controls (N = 15)	OSA (N = 29)	*p*-Value
Demographics			
Age, years	57.5 ± 10.4	62.6 ± 9.4	0.127
Male sex, n (%)	8 (53)	21 (72)	0.315
Body mass index, kg/m^2	22.6 ± 1.9	23.4 ± 1.3	0.168
Former or current smokers, n (%)	8 (53)	12 (41)	0.532
Comorbidities			
Arterial hypertension, n (%)	3 (20)	14 (48)	0.104
Diabetes, n (%)	1 (6.7)	3 (10)	>0.999
COPD, n (%)	0 (0)	3 (10)	0.540
Left ventricular structure			
LVEDd, mm	44.8 ± 4.7	47.6 ± 4.2	0.063
IVSd, mm	8 [1]	9 [2]	0.006
PWd, mm	8 [0]	9 [2]	0.013
LVMI, g/m^2	64.5 ± 9.1	79.3 ± 17.1	<0.001
RWT	0.34 [0.07]	0.36 [0.10]	0.414
EDVI, mL/m^2	47.9 ± 13.8	57.1 ± 13	0.043
Left ventricular systolic function			
LVEF, %	55.6 ± 3.6	55.3 ± 4.7	0.853
GLS, %	−19.3 ± 2.8	−18.4 ± 3.1	0.299
Left ventricular diastolic function			
Mitral E velocity, cm/s	66 [15]	59 [20]	0.683
DT, ms	214.9 ± 40	249.9 ± 81	0.061
E/A ratio	0.93 [0.41]	0.84 [0.40]	0.586
Average e' velocity, cm/s	10.1 ± 2.1	9.2 ± 2.3	0.197
Average E/e' ratio	6.2 [1.6]	6.8 [3.1]	0.450
Atrial structure			
4Ch-LAVI, mL/m^2	24.6 [10]	25.7 [9.4]	0.379
RAAI, cm^2/m^2	8.1 [3.5]	8.7 [3.1]	0.612
Right ventricular indices			
Tricuspid S', cm/s	16 [3]	15 [2]	0.153
RVOT diameter, mm	29.7 ± 4.3	31.3 ± 4.6	0.282
RVOT VTI, cm	15.6 ± 2	14.1 ± 2.6	0.044

Data are reported as the number of patients (percentage), mean ± standard deviation or median [interquartile range], as appropriate. Between-group comparisons were performed using Welch's *t*-test, Mann–Whitney U test, or Fisher's exact test, as appropriate. Abbreviations: COPD indicates chronic obstructive pulmonary disease; LVEDd, left ventricular end-diastolic diameter; IVSd, interventricular septal thickness in diastole; PWd, posterior wall thickness in diastole; LVMI, left ventricular mass index; RWT, relative wall thickness; EDVI, left ventricular end-diastolic volume index measured from the apical biplane view; LVEF, left ventricular ejection fraction; GLS, global longitudinal strain; DT, E-wave deceleration time, 4Ch-LAVI, left atrial volume indexed measured from the apical four-chamber view; RAAI, right atrial area indexed; Tricuspid S', tricuspid annular systolic velocity; RVOT, right ventricular outflow tract; RVOT VTI, right ventricular outflow tract velocity time integral.

Table 2. Sleep study and daytime sleepiness characteristics.

Variables	Controls (N = 15)	OSA (N = 29)	*p*-Value
Polygraphy variables			
AHI, events/h	2.3 [3.2]	12.9 [6.5]	<0.001
OSA severity *, n (%)			
Mild	-	21 (72)	
Moderate	-	6 (21)	
Severe	-	2 (7)	
ODI, events/h	2.2 [3.6]	12.4 [10.3]	<0.001
Nadir SpO_2, %	87.8 ± 4	80.7 ± 6.7	<0.001
T_{90}, %	0.1 [1.2]	3.5 [9.8]	0.001
Daytime Sleepiness			
ESS **	5.5 [5]	6 [4]	0.376

Data are reported as numbers (%), mean ± standard deviation, or median [interquartile range], as appropriate. * OSA severity was classified as mild (5–14.9 events/h), moderate (15–29.9 events/h), or severe (≥30 events/h) in OSA patients only; ** ESS was available in 14 controls and 27 patients with OSA. Abbreviations: AHI, indicates apnea–hypopnoea index; OSA, obstructive sleep apnea; ODI, oxygen desaturation index; T_{90}, percentage of recording time spent with pulse oximetry oxygen saturation (SpO_2) below 90%; ESS, Epworth sleepiness scale.

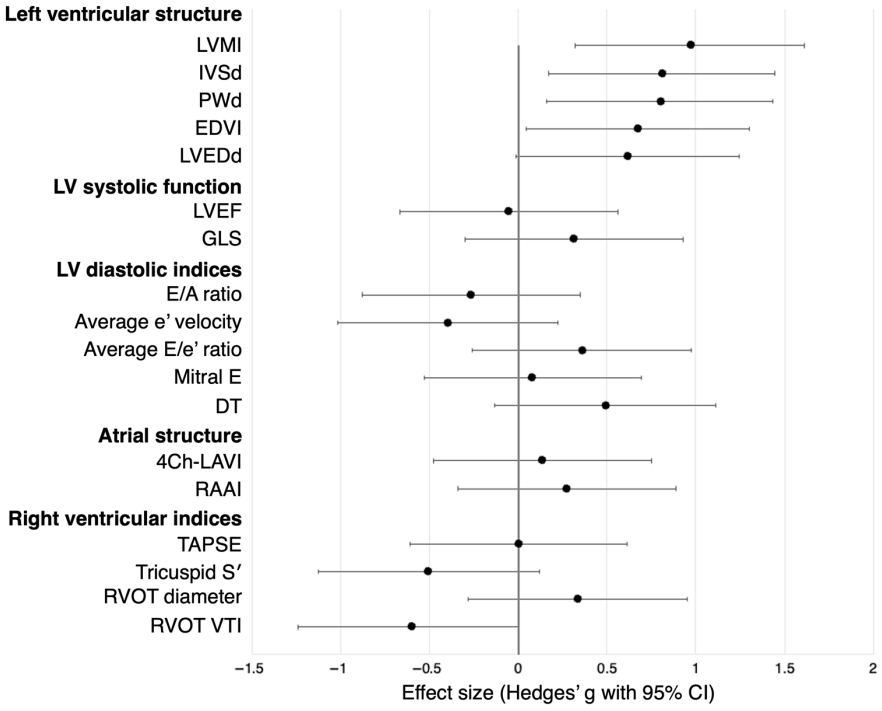


Figure 1. Distribution of standardized echocardiographic differences across cardiac domains in untreated normal-weight obstructive sleep apnea. Hedges' g effect sizes with 95% confidence intervals comparing untreated normal-weight participants with obstructive sleep apnea (OSA) and prospectively selected normal-weight controls without OSA. Effect-size estimates were largest for left ventricular structural measures, whereas estimates across systolic, diastolic, atrial, and right ventricular domains were generally smaller and less precise, with most confidence intervals crossing zero. Positive

values indicate higher measurements in participants with OSA; because the clinical meaning of higher values differs among echocardiographic variables, effect direction should be interpreted according to the individual parameter rather than as uniformly favorable or unfavorable. Abbreviations: LVMI, left ventricular mass index; IVSd, interventricular septal thickness in diastole; PWd, posterior wall thickness in diastole; EDVI, left ventricular end-diastolic volume index measured from the apical biplane view; LVEDd, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; GLS, global longitudinal strain; DT, E wave deceleration time; 4Ch-LAVI, left atrial volume indexed measured from the apical four-chamber view; RAAI, right atrial area indexed; TAPSE, tricuspid annular plane systolic excursion; Tricuspid S′, tricuspid annular systolic velocity; RVOT, right ventricular outflow tract; RVOT VTI, right ventricular outflow tract velocity time integral.

In multivariable linear regression, OSA remained associated with higher LVMI after adjustment for age, sex, BMI, and diagnosed arterial hypertension (B = 10.56 g/m^2; $p = 0.047$). BCa bootstrap resampling yielded a similar estimate, with a 95% CI of 1.71 to 21.16 g/m^2 (Table 3). Visual inspection of regression diagnostics did not reveal major departures from residual normality, linearity, or homoscedasticity. The maximum Cook's distance was 0.239. In a sensitivity analysis excluding the observation with the highest Cook's distance, the association between OSA and LVMI remained positive but was attenuated (B = 7.88 g/m^2, 95% CI −1.56 to 17.33; $p = 0.099$).

Table 3. Multivariable association between obstructive sleep apnea and left ventricular mass index in normal-weight participants.

Independent Variables	B (Unstandardized)	SE	95% CI for B	*p*-Value	Bootstrap (BCa) 95% CI
OSA	10.56	5.14	0.17 to 20.96	0.047	1.71 to 21.16
Age	0.32	0.24	−0.17 to 0.80	0.194	
Male sex	7.42	5.23	−3.16 to 18.0	0.164	
BMI	1.25	1.60	−1.98 to 4.49	0.437	
Arterial hypertension	0.64	4.85	−9.19 to 10.47	0.896	

Dependent variable: LVMI. Model $R^2 = 0.289$; adjusted $R^2 = 0.195$; overall model $p = 0.020$. No evidence of multicollinearity was observed (all VIF < 1.3). The maximum Cook's distance was 0.239. Bias-corrected and accelerated (BCa) bootstrap 95% confidence intervals based on 2000 resamples were calculated to characterize the uncertainty of the regression coefficient estimates. Abbreviations: OSA indicates obstructive sleep apnea; LVMI, left ventricular mass index; BMI, body mass index; SE, standard error; BCa, bias-corrected and accelerated; VIF, variance inflation factor; CI, confidence interval.

4. Discussion

The main finding of this study is that comprehensive echocardiographic assessment of untreated normal-weight adults with predominantly mild OSA revealed a pattern of between-group differences concentrated primarily in LV structural measures. LVMI, the prespecified primary outcome, was higher in participants with OSA, accompanied by greater interventricular septal and posterior wall thickness and higher EDVI. In contrast, between-group differences were generally smaller and less precise across conventional indices of LV systolic and diastolic function, atrial structure, and right ventricular function. In the primary multivariable model, the association between OSA and higher LVMI persisted after adjustment for age, sex, BMI, and diagnosed arterial hypertension, although sensitivity analysis indicated that the estimate was influenced by an individual observation.

An association between OSA and LV remodelling is not novel in itself. Previous echocardiographic studies and meta-analyses have reported greater LV mass, altered geometry, and

variable degrees of systolic and diastolic dysfunction in OSA [2]. Moreover, the contribution of obesity to this association has been specifically addressed. In the Wisconsin Sleep Cohort, Korcarz et al. demonstrated the complex interaction between OSA, obesity, and cardiac remodelling [3], while more recent work has further emphasized the contribution of BMI and blood pressure to LV structural abnormalities in OSA [4]. Thus, our findings should not be interpreted as establishing an obesity-independent effect of OSA on the myocardium. Rather, restricting both groups to BMI < 25 kg/m^2 provides a clinically selected setting in which the contribution of excess body weight is substantially reduced.

Studies specifically examining cardiac involvement in the absence of obesity further contextualize our findings. Cho et al. reported impaired myocardial performance, including reduced longitudinal deformation, in patients with OSA compared with normal-weight controls [7]. Using cardiac magnetic resonance, Xu et al. subsequently demonstrated adverse LV remodelling in non-obese adults with OSA [8]. Our study differs from these reports in several respects. Both OSA participants and controls were strictly normal weight; controls underwent the same respiratory and cardiovascular assessment and were selected prospectively before echocardiographic data were reviewed, and echocardiography systematically covered structural, systolic, diastolic, atrial, and right ventricular domains. In addition, almost three quarters of our OSA cohort had mild disease. Accordingly, the present findings extend rather than replace previous evidence by showing that, in this selected population, the most pronounced echocardiographic differences were concentrated in LV structural measurements rather than being distributed consistently across cardiac domains.

The predominance of structural differences should be interpreted cautiously. Our cross-sectional design cannot establish that structural remodelling precedes functional impairment, and the absence of statistically significant differences in functional indices does not demonstrate equivalence between groups. Nevertheless, the consistency of the structural findings across LVMI, wall thickness, and EDVI, together with the distribution of standardized effect sizes across echocardiographic domains, supports a predominantly structural echocardiographic pattern in this cohort. Importantly, the higher LVMI observed in participants with OSA predominantly reflected differences within the conventional reference range rather than established left ventricular hypertrophy. Therefore, these findings should be interpreted as structural echocardiographic differences rather than evidence of overt pathological hypertrophy. This observation may also help explain differences from studies reporting impaired GLS or diastolic function, because cardiac manifestations of OSA are likely influenced by disease severity, obesity, hypertension, comorbidity burden, and imaging methodology [2,7,8].

The relatively mild OSA burden in our cohort is also relevant. Twenty-one of twenty-nine participants with OSA had mild disease, yet structural differences were detectable compared with normal-weight controls. This observation should not be interpreted as evidence that mild OSA causes LV remodelling, nor can our data establish a dose–response relationship. Rather, it suggests that structural cardiac differences may be present even in selected normal-weight patients without predominantly moderate-to-severe OSA. Previous studies linking nocturnal hypoxaemia and OSA severity to LV remodelling have generally included broader clinical populations [14]. Larger studies spanning the full spectrum of OSA severity will be required to determine whether the multidomain echocardiographic pattern observed here varies with respiratory or hypoxic burden.

From a clinical perspective, our findings do not support routine echocardiographic screening of normal-weight patients with OSA on the basis of the present data alone. Instead, they highlight a subgroup in which cardiac structural differences may be detectable despite the absence of excess body weight and without a parallel signal across conventional functional indices. Whether this pattern identifies patients at increased cardiovascular

risk, progresses over time, or is modified by continuous positive airway pressure treatment remains unknown and requires longitudinal investigation [15].

The principal strengths of this study are the prospective enrolment of a strictly normal-weight population, the prospective selection of controls before echocardiographic assessment, identical respiratory and cardiovascular eligibility procedures in both groups, and comprehensive echocardiographic assessment performed by a cardiologist blinded to respiratory polygraphy findings. Standardized effect sizes and bootstrap confidence intervals were additionally used to characterize the magnitude and precision of the observed differences.

This study has several limitations. First, the single-center, cross-sectional design and the small sample size limit the precision and generalizability of the findings and preclude causal or temporal inference. The exploratory nature of the study and the absence of an a priori sample-size calculation should also be acknowledged. Second, the number of covariates relative to the sample size raises the possibility of model overfitting. The adjusted association between OSA and LVMI was attenuated after exclusion of one potentially influential observation and should therefore be interpreted cautiously. Bootstrap confidence intervals were used to characterize estimation uncertainty rather than to provide independent validation of the regression model. Third, OSA severity was assessed using a single overnight respiratory polygraphy recording rather than repeated measurements or full polysomnography. Therefore, night-to-night variability could not be evaluated, and the apnea–hypopnea index may have been underestimated because sleep time was not directly measured. Most participants had mild OSA, limiting generalizability to patients with more severe disease. Fourth, although diagnosed arterial hypertension was included in the multivariable model, adjustment for hypertension status alone cannot fully account for differences in blood pressure levels, cumulative blood pressure exposure, hypertension duration, or the potential effects of antihypertensive treatment. Residual confounding related to blood pressure cannot be excluded. Furthermore, although all participants had a BMI < 25 kg/m^2, normal body weight does not exclude differences in regional adiposity, including epicardial adipose tissue. Previous imaging studies have highlighted the potential cardiovascular relevance of inflammatory characteristics of pericoronary adipose tissue in other clinical populations [16]. However, epicardial adiposity was not assessed in our cohort, and its potential contribution to the observed LV structural differences remains unknown. Finally, secondary echocardiographic outcomes were not adjusted for multiple comparisons and should be interpreted as exploratory. The absence of longitudinal cardiovascular follow-up prevents assessment of the prognostic significance or progression of the observed echocardiographic differences.

5. Conclusions

In this small cohort of untreated normal-weight adults with predominantly mild OSA, comprehensive echocardiographic assessment revealed findings suggestive of a predominantly structural pattern of cardiac differences, primarily involving the left ventricle. These exploratory observations require confirmation in larger longitudinal studies before their clinical and prognostic significance can be established.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/medicina62112195/s1, Supplementary Table S1: Additional echocardiographic measurements across cardiac domains.

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draft preparation, M.C., L.G.D.C., E.C.; writing—review and editing, M.C., L.G.D.C., E.C. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and was approved by the South-West Veneto Area Ethics Committee (approval code 623CET; 6 June 2025).

Informed Consent Statement: Written informed consent for participation in the study was obtained from all subjects included in the study.

Data Availability Statement: The data supporting the findings of this study are not publicly available due to privacy and confidentiality considerations related to the small study population and the detailed individual-level clinical information collected. De-identified data may be made available by the corresponding author upon reasonable request, subject to applicable ethical and institutional requirements.

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