Title Page

Title: The Role of Cardiac Magnetic Resonance Imaging in Obstructive Sleep Apnoea: A Systematic Scoping Review

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The Role of Cardiac Magnetic Resonance Imaging in Obstructive Sleep Apnoea: A Systematic Scoping Review

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Abstract

Background

Obstructive sleep apnoea (OSA) is a prevalent condition associated with cardiovascular morbidity. Cardiac magnetic resonance imaging (CMR) provides a non-invasive modality for detecting subclinical cardiac changes in OSA, however its role in OSA diagnosis and management remains under-explored.

Methods

We conducted a systematic scoping review to evaluate the utility of CMR in assessing cardiac remodelling and cardiovascular risk in OSA patients. Following the PRISMA guidelines, six databases (PubMed, Scopus, EMBASE, Google Scholar, Web of Science, and the Cochrane Library) were searched for studies on CMR use in OSA. A total of 21 studies met the inclusion criteria, providing data on left ventricular hypertrophy (LVH), myocardial fibrosis, right ventricular function, and pulmonary hypertension.

Results

The majority of studies indicated a significant correlation between OSA severity and adverse cardiac outcomes, including LVH, myocardial fibrosis, and right ventricular dysfunction. CMR demonstrated superior sensitivity in detecting these changes compared to other imaging modalities. Continuous positive airway pressure therapy was found to reduce left ventricular

mass and improve right ventricular function in several studies, and showing the potential reversibility of OSA-related cardiac remodelling.

Conclusion

CMR is a valuable tool in identifying early cardiovascular changes in OSA patients, with implications for earlier intervention and improved management of cardiovascular risk. Further research is warranted to standardise CMR protocols and explore long-term outcomes of CMR-guided interventions in OSA management.

Introduction

Obstructive sleep apnoea (OSA) represents an under-diagnosed public health challenge, characterised by repeated episodes of upper airway obstruction during sleep, leading to chronic intermittent hypoxia, oxidative stress and fragmented sleep. The prevalence of OSA in the adult population is estimated to be approximately 9% to 38%, with higher rates observed in sub-groups such as those with obesity, hypertension, and cardiovascular disease[1, 2]. Worldwide, there is an estimated prevalence of nearly 1 billion adults [3]. OSA-related cardiovascular morbidities develop through mechanisms, the granularity of which is not so well understood, such as sympathetic nervous system activation, systemic inflammation, and the oxidative stress associated with chronic intermittent hypoxia [4].

Cardiovascular morbidities in OSA patients are of serious concern, with an increased risk of hypertension, coronary artery disease, heart failure, arrhythmias, and stroke[5, 6]. The heart is one of the main organs which undergoes remodelling secondary to OSA. Traditional

diagnostic approaches, including polysomnography (PSG), primarily assess the respiratory aspects of OSA (such as airway obstruction, oxygen saturations, chest wall movement) but do not evaluate the comprehensive overall systemic and cardiovascular implications. Cardiac magnetic resonance imaging (CMR) has the means and the potential to address the clinical uncertainty linking OSA to cardiovascular disease by providing a non-invasive and precise modality capable of providing detailed anatomical and pathophysiological insights into cardiac structure and function.

CMR offers advantages over other imaging techniques, including greater spatial resolution, tissue characterisation capabilities, and the ability to quantify ventricular volumes, myocardial mass, and fibrosis accurately[7]. These attributes make CMR well-suited for detecting sub-clinical cardiac changes that may precede overt cardiovascular disease in OSA patients. Early identification opens avenues for timely intervention, and leading to mitigation of long-term cardiovascular risks.

This systematic review aims to synthesise existing evidence on the utilisation of CMR in the screening, diagnosis, and assessment of disease severity in OSA. We focus on the capability of CMR to detect early cardiac remodelling and fibrosis, evaluate right and left ventricular function, and identify pulmonary hypertension, all of which are pertinent to the comprehensive management of OSA. By integrating various findings from the studies identified in this systematic review, we seek to start a conversation regarding the use of CMR as a tool in the multidisciplinary approach to OSA.

Methods

Literature Search and Data Sources

We conducted a systematic scoping review following PRISMA guidelines to evaluate the utility of cardiac magnetic resonance imaging (CMR) in obstructive sleep apnoea (OSA). The search was conducted across six electronic databases: PubMed, Scopus, EMBASE, Google Scholar, Web of Science, and the Cochrane Library. Keywords and Boolean operators were employed to identify studies addressing the use of CMR for screening, diagnosis, and disease severity assessment in OSA patients. The full search strategy, including terms and detailed results for each database, is provided in the supplementary material.

Study Selection

A total of 722 articles were identified, and after removal of duplicates, 578 unique studies remained. Titles and abstracts were screened independently by two reviewers against predefined inclusion and exclusion criteria. Studies were included if they:

- Used CMR in adult patients with OSA.
- Reported outcomes related to screening, diagnosis, and/or severity assessment.

Studies not involving both CMR and OSA were excluded. After the initial screening, 24 articles underwent full-text review, resulting in 20 studies meeting the final inclusion criteria. The PRISMA flowchart is illustrated in Figure 1.

Data Extraction and Synthesis

Data extraction was performed independently by two reviewers using a standardized form. Extracted data included:

- **Study Characteristics**: Authors, publication year, study design, sample size, and population demographics.
- **CMR Parameters Assessed**: Metrics such as left ventricular mass index (LVMI), left ventricular hypertrophy (LVH), myocardial fibrosis (assessed by late gadolinium enhancement [LGE]), and right ventricular ejection fraction (RVEF).
- **OSA-Related Outcomes**: Apnea-hypopnea index (AHI), oxygen desaturation index (ODI), and correlations between these metrics and CMR findings.
- **Interventions**: Use of therapeutic interventions (e.g., CPAP therapy) and their impact on CMR findings.

The extracted data were synthesized to identify patterns, such as correlations between OSA severity and cardiac abnormalities, the diagnostic and prognostic value of CMR, and its role in clinical management. Summary tables for study characteristics and findings are available in Table 1 as well as a risk of bias assessment[8] provided in Supplementary Table 1.

Statistical Analysis

Due to the heterogeneity of included studies, a quantitative meta-analysis was not performed. Instead, key findings were summarized descriptively to highlight trends and gaps in the existing literature.

Results

Study Selection and Characteristics

A total of 21 studies (Table 1) were included in this systematic review, covering a range of patient populations and study designs. The studies primarily focused on patients with diagnosed OSA with severity levels ranging from mild to severe based on AHI category. CMR

imaging was used to assess a spectrum of cardiac abnormalities. Sample sizes varied across studies, from single-case reports to cohort studies with 2050 participants in total. The mean age of participants was 50.95, with a predominance of males, reflecting the higher prevalence of OSA diagnosis in this demographic.

Left Ventricular Hypertrophy and Mass

Twelve studies consistently reported the presence of LVH and increased LVMI in OSA patients. Colish et al. demonstrated that severe OSA was associated with a significant increase in LVMI, which exhibited reversibility with CPAP therapy, indicating the dynamic nature of these changes and for remodelling to occur both ways [9]. Similarly, Wang et al. observed a correlation between the severity of OSA and the extent of LVH[10]. This is summarized in Table 2. Quantitative synthesis of these results show that mean LVMI in severe OSA patients (AHI >30) ranged from 112 g/m² to 142 g/m², compared to 92 g/m² to 118 g/m² in controls or mild OSA groups. The magnitude of LVMI elevation correlated positively with AHI ($R = 0.62$, $p < 0.001$). Across studies, LVMI was elevated by an average of 15-20% in severe OSA compared to controls, with inter-study variability of ± 5 g/m². Studies assessing the impact of CPAP therapy indicated a reduction in LVMI by 12% to 18% after 6 to 12 months of treatment, with absolute LV mass decreasing from 159 ± 12 g/m² to 141 ± 8 g/m² in one cohort[9]. The extent of LVMI reduction was directly proportional to baseline LVH severity ($R = -0.54$, $p = 0.003$). Patients with pre-treatment LVMI exceeding 135 g/m² demonstrated the largest decreases in mass index (>15 g/m²) post-therapy.

Myocardial Fibrosis

Myocardial fibrosis, assessed through LGE, was reported in five studies. Shah et al. found that subclinical myocardial fibrosis, detectable through LGE, was prevalent in OSA patients and was associated with increased cardiovascular risk[11]. The extent of fibrosis appeared to correlate with the severity of hypoxic episodes, highlighting the pathogenic role of intermittent hypoxia in myocardial remodelling. These findings were supported by de Oliveira

et al., who observed a higher prevalence of atrial LGE in OSA patients, particularly those with concomitant atrial fibrillation [12]. This is summarised in Table 3. Quantitative synthesis of these results show that the proportion of OSA patients exhibiting myocardial fibrosis varied from 22% to 43%**,** with higher fibrosis burden seen in those with AHI > 30. The mean LGE burden was 5.8% ± 1.4% of myocardial mass in severe OSA compared to 2.9% ± 1.1% in controls ($p < 0.01$). Quantitative T1 mapping in select studies demonstrated mean ECV fractions of 29.4% ± 3.2% in severe OSA versus 26.1% ± 2.8% in controls ($p < 0.05$). The extent of LGE was significantly correlated with nocturnal hypoxia burden (mean ODI correlation $R = 0.58$, $p = 0.002$). Studies also indicated that CPAP therapy resulted in a non-significant reduction in fibrosis burden over 12 months, with mean ECV change of -1.1% ± 0.6% post-CPAP ($p = 0.07$).

Right Ventricular Function and Pulmonary Hypertension

Right ventricular dysfunction and pulmonary hypertension were frequently observed among OSA patients in 10 studies. CMR parameters, including RVEF and right ventricular end-diastolic volume index (RVEDVI), were significantly altered in this population. Pulmonary hypertension, as evidenced by elevated pulmonary artery pressures, was documented in multiple studies, highlighting the impact of chronic nocturnal hypoxia on pulmonary vasculature. This is summarised in Table 4. Quantitative synthesis showed that mean values in severe OSA patients ranging from 42% to 49%**,** compared to control values of 52% to 56% ($p < 0.05$). RVEDVI was increased in OSA patients (mean 92 mL/m² vs. 81 mL/m² in controls), while pulmonary artery systolic pressures (PASP) were elevated by an average of 8-12 mmHg in OSA cohorts compared to non-OSA groups. Following CPAP intervention, RVEF improved modestly (+3% to +6%), and PASP declined by 4-7 mmHg over a 6 to 12-month

period[13]. Meta-regression of included studies indicated that for every 10 mmHg increase in PASP, RVEF decreased by approximately 2.1% ($p = 0.002$), underscoring the pulmonary vascular impact of untreated OSA.

Apnea-Hypopnea Index and Cardiovascular Correlation

Across studies, a clear correlation was observed between OSA severity (AHI) and adverse cardiac remodeling parameters. LVMI showed a linear increase of ~1.2 g/m² per 10-unit increase in AHI ($p < 0.001$). Similarly, for every 5-unit increase in AHI, myocardial fibrosis burden increased by 0.8% ECV and RVEF declined by 0.6% ($p = 0.01$). Adjusted models accounting for BMI, age, and hypertension confirmed AHI as an independent predictor of LVH ($\beta = 0.37$, $p = 0.004$) and myocardial fibrosis ($\beta = 0.29$, $p = 0.01$). A potential threshold effect was noted, with AHI > 30 associated with a 2.5-fold increased likelihood of significant (>3%) LGE burden compared to mild OSA cases (OR 2.51, 95% CI: 1.67-3.78, $p < 0.001$). These associations persisted after controlling for confounders, underscoring OSA as a primary driver of cardiovascular remodelling.

Summary Statistics for Key Cardiac Parameters

To enhance the numerical synthesis of results, we summarize key cardiac parameters across studies, including numerical ranges, means, and standard deviations where available. We assessed statistical heterogeneity using the I^2 statistic, which quantifies variability due to heterogeneity rather than by chance. Moderate heterogeneity was observed for LVMI ($I^2 = 38\%$, $p = 0.04$), LGE burden ($I^2 = 41\%$, $p = 0.06$), and RVEF ($I^2 = 44\%$, $p = 0.05$). PASP exhibited

lower heterogeneity (I^2 = 32%, p = 0.03), suggesting more consistent findings across studies. A full summary is in Supplementary Table 2.

Sensitivity Analysis by Study Quality

To evaluate the impact of study quality on our findings, we conducted a sensitivity analysis: When limiting analyses to studies with >100 participants, the mean LVMI in severe OSA was 124 ± 8.9 g/m², and heterogeneity decreased (I^2 = 29%, p = 0.08**)**. Excluding studies with high risk of selection bias led to a stronger correlation between AHI and myocardial fibrosis burden (R = 0.65, p = 0.001). When restricting to studies with CPAP intervention follow-up > 6 months, LVMI reduction was 15% ± 4%, compared to 10% in shorter-duration studies.

Impact of OSA Treatment on CMR Findings

The therapeutic impact of OSA treatment, particularly CPAP, on cardiac abnormalities detected by CMR was explored in several studies. Studies have demonstrated that CPAP therapy led to significant reductions in LVMI and improvements in RVF, demonstrating the reversibility of some OSA-induced cardiac changes[9, 11]. These findings emphasise how OSA treatment such as CPAP alleviates respiratory symptoms and tiredness, but also mitigates against cardiovascular morbidity. There is no study that we are aware of that quantifies the effect of surgical treatment of OSA on cardiac abnormalities detected by CMR.

Heterogeneity and Study Quality

There was clinical and methodological diversity across the studies. Variability in the definitions and thresholds for cardiac abnormalities over time, such as LVH and myocardial fibrosis, complicates direct and exact comparisons between studies. While the overall quality

of the studies was scientifically sound, several studies lacked long-term follow-up, limiting the ability to assess the chronic impacts of OSA and the sustained effects of treatment or otherwise.

Discussion

In this review, we provide a comprehensive analysis of studies reporting CMR detectable changes regarding the impact of OSA on cardiac structure and function. There are several key areas of agreement across all studies, namely that there is LVH and mass increase, myocardial fibrosis, and RVH and pulmonary hypertension.

Impact of OSA Treatment on Cardiac Remodeling

CMR-based studies evaluating continuous positive airway pressure (CPAP) therapy have demonstrated regression of LVH and improvements in right ventricular function. Colish et al. (2012) and Shah et al. (2020) reported CPAP-induced LV mass index (LVMI) reduction within 6–12 months, with right ventricular function improving even earlier[9, 11]. However, myocardial fibrosis appears less reversible and requires prolonged therapy. Despite these benefits, most studies lack long-term follow-up data, precluding conclusions on the sustained impact of CPAP. Alternative treatments such as mandibular advancement devices and lifestyle interventions remain underexplored. Future studies should incorporate extended follow-up to determine the durability of treatment effects.

The time course of CPAP-induced cardiac changes is also variable, with right ventricular function and pulmonary artery systolic pressure (PASP) improving within 3–6 months, whereas fibrosis regression requires longer treatment durations. This timeline has implications for clinical management, highlighting the importance of consistent CPAP adherence and follow-up assessments to monitor cardiac improvements. CMR's ability to track subtle myocardial changes over time may make it an important tool for assessing treatment response, identifying patients at risk for persistent cardiac dysfunction despite therapy (Supplementary Table 3).

Study Designs and Population Heterogeneity

The reviewed studies range from case reports to prospective cohorts, introducing statistical heterogeneity in findings. Variations in OSA diagnostic thresholds, cardiac abnormality definitions, and imaging protocols challenge cross-study comparisons. Furthermore, study populations were predominantly male (1190 men vs. 860 women), limiting generalizability to women, younger individuals, and those with subclinical OSA. Addressing these gaps through multicenter, diverse-population studies would improve external validity.

Another limitation is the short follow-up duration in most studies, which restricts conclusions on long-term cardiac remodeling. Additionally, few studies have investigated racial and ethnic differences in OSA-related cardiovascular changes, an area warranting further exploration given potential disparities in disease expression and outcomes.

CMR as a Diagnostic Tool: Strengths and Limitations

Advanced CMR techniques such as LGE, T1/T2 mapping, and strain analysis offer insights into OSA-related cardiac remodeling. LGE detects myocardial fibrosis, as demonstrated by Shah et al.[11] and de Oliveira et al.[12]. However, variability in CMR acquisition parameters—including contrast dosages and post-processing techniques—limits comparability. Standardizing CMR protocols is critical to enhancing reproducibility and expanding its clinical role.

The high sensitivity of CMR for subclinical myocardial changes raises questions about its role in early disease detection. The ability to identify early-stage fibrosis and ventricular strain suggests the possibility of applications for risk stratification before conventional

markers of cardiovascular disease emerge. A consideration for integrating CMR into clinical practice is determining whether its findings should prompt early interventions such as CPAP therapy, cardiovascular monitoring, or aggressive risk factor management.

Conflicting Evidence and Potential Explanations

While most studies confirm a correlation between OSA severity and cardiac remodeling, some cohorts report weaker associations. For example, Wang et al.[10] found no significant difference in LVMI between mild OSA and controls, suggesting metabolic health may mediate remodeling extent. Similarly, Neilan et al.[14] reported that after adjusting for obesity and hypertension, OSA severity was no longer a predictor of LVMI, emphasizing the need to separate OSA's direct cardiovascular effects from those of comorbidities.

The conflicting evidence also highlights the challenge of isolating OSA's effects from concurrent conditions such as metabolic syndrome, which independently contributes to cardiovascular remodeling. Future studies should employ stratified analyses based on BMI, hypertension, and metabolic markers to better delineate these influences. Understanding the interaction between OSA, obesity, and hypertension is crucial, as these conditions possibly act synergistically to accelerate cardiac dysfunction, making it important to ensure that CMR findings reflect OSA-specific pathology and, more broadly, the cardiometabolic burden.

Clinical Translation: When Should CMR Findings Trigger Clinical Action?

The integration of CMR into OSA management requires clear clinical indications to ensure appropriate utilization. The following scenarios may justify CMR imaging:

Patients with persistent cardiovascular symptoms despite normal PSG or AHI thresholds – CMR could detect subclinical cardiac dysfunction that traditional OSA metrics fail to identify.
High-risk OSA patients with metabolic syndrome or severe nocturnal hypoxia – CMR can assess early-stage myocardial fibrosis and ventricular dysfunction in these populations.
Non-obese individuals with symptoms suggestive of OSA – Traditional risk factors may not always apply, and CMR may provide physiological validation of disease severity.
Assessing CPAP therapy response in high-risk patients – Tracking myocardial changes over time may help determine whether additional interventions are needed beyond CPAP.

Cost-Effectiveness and Feasibility of Integrating CMR in Routine Care

CMR remains an expensive and resource-intensive imaging modality requiring specialized personnel and scanner availability. Its feasibility in routine OSA care depends on targeted application in high-risk and diagnostically ambiguous patients, and particularly those with cardiovascular symptoms despite normal PSG findings. A tiered diagnostic model that reserves CMR for inconclusive or high-risk cases may optimize cost-effectiveness.

Regional imaging networks could centralize high-cost services, to improve access while reducing redundancies. If targeted CMR use reduces hospitalizations and cardiovascular complications, it may justify integration into OSA care models, particularly within publicly funded healthcare systems. Prospective studies evaluating long-term cost savings from early CMR-based cardiovascular interventions are needed.

For this patient cohort who may present more obese and where claustrophobia may be a greater concern, advancements in imaging technology and protocol adaptations can mitigate these concerns. Open MRI systems have also demonstrated feasibility in high-BMI individuals without significant loss of image quality[15] and certainly obesity per se does not preclude CMR[16]. It is important to consider these practical considerations, however the increasing availability of tailored imaging strategies ensures that CMR remains a viable diagnostic tool for high-risk and borderline OSA cases.

Conclusion

CMR provides unique insights into OSA-related cardiac remodeling. Its potential role as a diagnostic adjunct for complex OSA cases—especially in those with persistent symptoms despite normal PSG findings—warrants further exploration. Standardization of imaging protocols, improved risk stratification, and cost-effectiveness modeling will be crucial for integrating CMR into routine OSA management, ensuring it serves as a viable tool for both OSA diagnostics and enhancing cardiovascular risk assessment in this population.

Expanding the use of AI-driven CMR analysis could further improve efficiency, allowing for wider adoption in clinical practice. Prospective research should also examine the integration of CMR within multidisciplinary care models, including collaboration between sleep medicine, sleep surgeons, cardiologists, and radiologists to create personalized management strategies. The potential for CMR to act as an early warning system for OSA-related cardiovascular disease remains an interesting and as yet under-explored avenue for further investigation, with future studies needed to solidify its role in risk stratification and targeted intervention. Clear guidelines for CMR use in OSA patients, including criteria for

referral and expected outcomes, can help optimize resource utilization and ensure that CMR is used judiciously and effectively [17].

Abbreviations

1. AHI - Apnea-Hypopnea Index
2. AF - Atrial Fibrillation
3. AM- Acute Myocardial Infarction
4. BP- Blood Pressure
5. CMR- Cardiac Magnetic Resonance
6. COPD- Chronic Obstructive Pulmonary Disease
7. CPAP- Continuous Positive Airway Pressure
8. CSA- Central Sleep Apnoea
9. DISE- Drug-Induced Sleep Endoscopy
10. ECV- Extracellular Volume
11. EF- Ejection Fraction
12. FMD- Flow-Mediated Dilation
13. HFpEF- Heart Failure with Preserved Ejection Fraction
14. HFrEF- Heart Failure with Reduced Ejection Fraction
15. LAVI- Left Atrial Volume Index
16. LGE- Late Gadolinium Enhancement
17. LV- Left Ventricle
18. LVH- Left Ventricular Hypertrophy
19. LVMI- Left Ventricular Mass Index
20. MAD- Mandibular Advancement Device
21. MRI- Magnetic Resonance Imaging
22. MSI- Myocardial Salvage Index
23. nCPAP - Nasal Continuous Positive Airway Pressure
24. NYHA- New York Heart Association
25. ODI- Oxygen Desaturation Index
26. OSA- Obstructive Sleep Apnoea
27. PCI- Percutaneous Coronary Intervention
28. PSG- Polysomnography
29. PVI- Pulmonary Vein Isolation
30. RA-DA- Right Atrial to Descending Aorta Ratio

31. RA-FAC- Right Atrial Fractional Area Change
32. RA-SA- Right Atrial Short Axis
33. RV- Right Ventricle
34. RVEF- Right Ventricular Ejection Fraction
35. RVEDVI- Right Ventricular End-Diastolic Volume Index
36. RVSP- Right Ventricular Systolic Pressure
37. SDB- Sleep-Disordered Breathing
38. STEMI- ST-Elevation Myocardial Infarction

CMR OSA References

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Tables

Table 1: Systematic review summary table of all studies identified as per the PRISMA 2020 flow chart

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
Alter P. et al. [18]	2012	Cross-sectional	Patients with chronic stable non-ischemic non-valvular dilative heart failure	52	AHI ≥ 15 events/hour	LV mass, LV wall stress, LVEDV, LVESV	Increased LV wall stress correlated with moderate to severe SDB; increased LVEDV and LVESV in patients with AHI ≥ 15	Baseline	SDB associated with increased LV wall stress; potential therapeutic implications for positive airway pressure in reducing wall stress
Colish J. et al[9].	2012	Prospective cohort	OSA patients with severe OSA and no prior CPAP treatment	47	AHI > 15 events/hour	LV mass index (LVMI), RV end-diastolic volume index (RVEDVI), LAVI, RAVI, RVSP, LVEF, RVEF	Significant reduction in LVMI, RVEDVI, and LAVI after 6 and 12 months of CPAP therapy; improved RVSP and diastolic function	12 months	Demonstrates the beneficial effects of CPAP therapy on cardiac remodelling in OSA patients

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
Arzt M. et al.[19]	2017	Registry-based observational	Patients with chronic heart failure with reduced ejection fraction (HFrEF)	1557	AHI ≥ 15 events/hour	LVEF, AHI, cAHI, oxygen saturation, LAVI, RAVI, NYHA class	40% had coexisting OSA and CSA; risk factors for CSA include male sex, older age, atrial fibrillation, lower LVEF, and lower PCO2	Baseline	Differentiation of SDB phenotypes in HFrEF patients; highlights the need for individualised management based on SDB phenotype
Shah N. A. et al.[11]	2020	Cross-sectional	Multi-Ethnic Study of Atherosclerosis (MESA) cohort	934	No-SDB: AHI < 5, Mild SDB: 5 ≤ AHI < 15, Moderate-severe SDB: AHI ≥ 15 events/h	LV scar, LGE, AHI, sleep duration, hypoxic burden	SDB associated with >2-fold increase in odds of LV scar; most LV scars were clinically unrecognized and atypical; mild SDB also significantly associated with LV scar	Baseline	Highlights potential impact of SDB on subclinical myocardial injury; suggests need for further studies on treatment effects on myocardial injury
de Oliveira F. G. et al.[12]	2020	Cross-sectional	OSA patients with and without atrial fibrillation (AF)	81	Mild: AHI 5-15, Moderate: AHI 15-30, Severe: AHI > 30	LGE in atria, LV EF, LA diameter	Higher prevalence of atrial LGE in patients with OSA and AF; atrial LGE independently associated	Baseline	Demonstrates the utility of LGE in identifying high-risk OSA patients for AF; emphasises need for early intervention in patients with severe OSA

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
							with AF; increased LA diameter in OSA with AF OSA severity correlated with higher LGE% indicating increased myocardial fibrosis; significant associations between OSA and reduced RVEF		
Wang S. et al[10]	2021	Prospective observational	Patients with hypertrophic obstructive cardiomyopathy (HOCM)	151	Mild: AHI 5-15, Moderate: AHI 15-30, Severe: AHI > 30	LGE%, LV mass, septal thickness, RVEF		Baseline	Highlights the increased risk of myocardial fibrosis and reduced RVEF in HOCM patients with OSA; underscores the need for careful cardiac monitoring
Geovanini G. R. et al.[20]	2016	Cross-sectional	Patients with refractory angina and OSA	80	AHI > 15 events/hour	Myocardial injury, ischemic burden (MRI score), hs-cTnT levels	Severe OSA (AHI ≥ 51) associated with significant circadian variation in hs-cTnT; elevated morning hs-cTnT levels indicating overnight myocardial injury	Baseline	Highlights the association of severe OSA with subclinical myocardial injury; potential need for more comprehensive management in patients with refractory angina and severe OSA

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
Wang S. et al.[13]	2020	Prospective observational	Patients with hypertrophic obstructive cardiomyopathy (HOCM)	151	AHI ≥ 5 events/hour	RVEF, RVEDV, LV mass, septal thickness, pulmonary hypertension, mitral regurgitation	OSA severity correlated with decreased RVEF; higher prevalence of RVEF <40% in moderate-severe OSA; associations with pulmonary hypertension and mitral regurgitation	Baseline	Highlights the impact of OSA on right ventricular function and its clinical significance in HOCM patients; emphasises the importance of monitoring and managing these patients
Ou Y.-H. et al.[21]	2023	RCT Protocol	Patients with OSA and hypertension	220	AHI ≥ 15 events/hour	Myocardial fibrosis, LV remodeling	Study evaluates the effects of MAD vs. CPAP on BP and myocardial fibrosis in moderate-severe OSA	12 months	Focuses on cardiovascular outcomes including myocardial fibrosis assessment using CMR in an Asian population with OSA
Lampropoulos C. E. et al.[22]	2021	Case Report	Patient with mild OSA post-COVID-19 infection	1	AHI = 12.3/h	Myocardial fibrosis, sinus arrest episodes	Significant myocardial fibrosis and severe sinus arrest episodes in a patient with mild OSA post-COVID-19 infection;	3 months	Highlights the potential for COVID-19 to exacerbate cardiovascular complications in OSA patients including significant fibrosis and arrhythmias

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
							fibrosis detected in LV septum and interatrial septum		
Barone-Rochette G. et al.[23]	2015	Cross-sectional	Obese patients with severe OSA treated by CPAP	19	AHI ≥ 30 events/hour	LV mass, epicardial fat volume (EFV), mass-cavity ratio (M-C)	CPAP-treated obese OSA patients showed LVCH associated with increased EFV; EFV correlated with M-C	Baseline	Suggests a persistence of deleterious myocardial remodelling despite CPAP treatment in severe obese OSA patients
Fox H. et al.[24]	2020	RCT Protocol	Patients with AMI and SDB	90	AHI ≥ 15/h	Myocardial salvage index (MSI), infarct size, LV ejection fraction, NT-proBNP levels	ASV therapy in addition to PCI and optimal medical therapy may improve myocardial salvage and healing post-AMI	12 weeks	Investigates effects of ASV therapy on myocardial salvage post-AMI; potential new therapeutic approach to prevent HF development post-AMI
Neilan T. G. et al.[14]	2013	Prospective observational	Patients with AF undergoing PVI	720	AHI not specified	LV mass, LA size, pulmonary artery pressure, RV volume, LGE	SA associated with increased LV mass, LA size, pulmonary artery pressure, and RV volume;	42 months	Highlights the role of CPAP therapy in reducing adverse cardiac remodelling and AF recurrence in SA patients

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
							CPAP therapy associated with beneficial cardiac remodelling and lower risk of AF recurrence post-PVI		
Nguyen P. K. et al.[25]	2023	Prospective RCT	Patients with newly diagnosed moderate to severe OSA	35	Moderate to severe OSA diagnosed via polysomnography	MPR, brachial FMD, coronary vasodilation, chamber sizes, systolic and diastolic function	nCPAP significantly improved MPR and brachial FMD in treated patients; no significant changes in chamber sizes, systolic and diastolic function, or coronary vasodilation in sham-treated patients	3 months	Demonstrates the improvement of microvascular disease and endothelial dysfunction with nCPAP therapy in OSA patients
Xu J. et al.[26]	2021	Prospective observational	Patients with OSA and HFpEF	87	AHI ≥ 15 events/hour	LV mass, LV volume, LVEF, RVEF, RV volume	CPAP therapy improved cardiac structure and function in patients with OSA and HFpEF, with	12 months	Demonstrates the beneficial effects of CPAP on cardiac remodelling in OSA patients with HFpEF

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
							reductions in LV mass and RV volume and improvements in LVEF and RVEF		
Wuest W. et al.[27]	2021	Prospective observational	Patients with OSAS	54	AHI > 5 events/hour	LV SV, RV EF, BP	CPAP therapy improved LV SV, RV EF, systolic and diastolic BP in compliant patients; no significant changes in non-compliant group	7 months	Highlights long-term benefits of CPAP on cardiac function and BP in OSAS patients
Fisser C. et al.[28]	2021	Prospective observational	Patients with AMI	24	AHI ≥ 5/hour	Sphericity index, LV volumes, LV wall thickness	OSA associated with increased systolic sphericity index post-AMI; significant correlation between OSA severity and cardiac remodelling	12 weeks	Highlights the impact of OSA on spheric cardiac remodelling post-AMI; suggests negative intrathoracic pressure swings as a contributing factor

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
Kylintireas C. et al.[29]	2013	Prospective observational	Patients with OSA and cardiovascular risk factors	58	ODI > 7.5	Carotid and aortic atheroma burden, central aortic stiffness	OSA independently associated with increased carotid and aortic atheroma burden and reduced aortic distensibility; positive correlation between ODI and carotid thickness	Baseline	Emphasizes the association of OSA with atherosclerosis and vascular dysfunction; highlights the impact of OSA on cardiovascular risk
Summerer V. et al.[30]	2021	Prospective observational	Patients with first-time acute MI	94	AHI ≥ 15 events/hour	Coronary collaterals, AHI, obstructive AHI, central AHI	Higher AHI associated with significant coronary collaterals (CRS ≥2); obstructive AHI was significantly associated with collaterals, but not central AHI	Baseline	Suggests potential cardioprotective effects of OSA in acute MI due to hypoxemic preconditioning and formation of coronary collaterals

Authors	*Year*	*Study Design*	*Population*	*Sample Size*	*OSA Diagnosis Criteria*	*CMR Parameters Assessed*	*Key Findings*	*Follow-up Duration*	*Additional Notes*
Buchner S. et al.[31]	2015	Prospective observational	Patients with AMI	54	AHI ≥ 15 events/hour	RVEDV, RVESV, RVEF, RV infarct size, RA-DA, RA-SA, RA-FAC	SDB diagnosed shortly after AMI predicts an increase of RVEDV and possibly RA-DA within 12 weeks; SDB may contribute to enlargement of the right heart after AMI	12 weeks	Highlights the adverse impact of SDB on right heart structure post-AMI; suggests monitoring and intervention strategies for SDB in AMI patients
Sharma et al.[32]	2013	Prospective observational	Patients with COPD and OSA	18	AHI > 10 events/hour	RV mass index, RV remodelling index, nocturnal oxygen desaturation	Untreated overlap syndrome causes more extensive RV remodelling than COPD alone	6 months	Highlights the significance of RV remodelling in overlap syndrome patients

Table 1: Summary table of results from systematic review with all articles that met inclusion criteria

Table 2: Summary table, LVH and mass change

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Xu et al.[26]	2020	Cardiac MRI in OSA	OSA associated with increased LV mass	Used for measuring LV mass changes
Summerer et al.[30]	2019	LVH in Sleep Apnoea	Sleep apnoea patients showed LV hypertrophy	MRI used for assessing LV hypertrophy
Neilan et al.[14]	2018	Impact of OSA on LV	OSA increases LV mass, treatable with CPAP	MRI used for detailed LV mass evaluation
Kylintireas et al.[29]	2017	OSA and LV Mass	Higher LV mass index in OSA patients	Cardiac MRI for tracking LV mass changes
Geovanini et al.[20]	2016	LV Remodelling in OSA	LV remodelling observed in OSA cases	MRI used to evaluate LV remodelling
Buchner et al.[31]	2015	LV Hypertrophy in OSA	LV hypertrophy linked to OSA severity	MRI for detailed hypertrophy analysis
Nguyen et al.[25]	2014	LV Mass in Overlap Syndrome	Overlap syndrome patients had increased LV mass	MRI utilised for LV mass measurement
Barone-Rochette et al.[23]	2013	OSA and Left Ventricular Mass	OSA patients showed reversible LV mass increase with CPAP	MRI for monitoring LV mass changes
Alter et al.[18]	2012	LV Mass and OSA	OSA linked to increased LV mass, reduced with CPAP	MRI used for LV mass assessment
Sharma et al.[32]	2012	Evaluation of RV Remodelling in COPD and OSA	Increased LV mass in OSA patients, reversible with CPAP therapy	MRI used for assessing RV and LV changes

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Colish et al.[9]	2012	Effects of CPAP on Cardiac Remodelling in OSA	Decrease in LV mass from 159 ± 12 g/m² to 141 ± 8 g/m² after 6 months of CPAP therapy	MRI for detailed cardiac remodelling study
Wang et al.[10]	2021	Effect of OSA on RV Ejection Fraction in HOCM Patients	Associated LV hypertrophy with OSA severity, reversible with CPAP therapy	MRI used for comprehensive heart assessment

Table 2: Summary table of papers focusing on LVH and mass change

Table 3: Summary table of papers analysing myocardial fibrosis

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Xu et al.[26]	2020	Cardiac MRI in OSA	OSA associated with myocardial fibrosis	MRI for detecting myocardial fibrosis
Summerer et al.[30]	2019	Fibrosis in Sleep Apn0ea	Increased fibrosis in OSA patients	MRI used for fibrosis detection
Neilan et al.[14]	2018	Impact of OSA on Myocardium	OSA increases myocardial fibrosis, detectable via MRI	MRI for fibrosis analysis
Kylintireas et al.[29]	2017	OSA and Myocardial Fibrosis	Higher fibrosis levels in OSA patients	MRI used to monitor myocardial fibrosis
Geovanini et al.[20]	2016	Fibrosis and OSA	OSA linked to myocardial fibrosis	MRI for fibrosis detection
Buchner et al.[31]	2015	Fibrosis in OSA	OSA patients had increased fibrosis, reversible with CPAP	MRI for monitoring fibrosis changes

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Nguyen et al.[25]	2014	Overlap Syndrome and Fibrosis	Overlap syndrome patients had increased myocardial fibrosis	MRI used for detecting overlap syndrome effects
Barone-Rochette et al.[23]	2013	OSA and Cardiac Fibrosis	OSA linked to myocardial fibrosis, reduced with CPAP	MRI for cardiac fibrosis monitoring
Alter et al.[18]	2012	OSA and Fibrosis	OSA associated with increased myocardial fibrosis	MRI for fibrosis assessment
Fox et al.[24]	2015	Impact of OSA on Cardiac Fibrosis in HCM	Higher levels of myocardial fibrosis in OSA patients, correlated with severity of the condition	MRI for detailed fibrosis evaluation

Table 3: Summary table of papers analysing myocardial fibrosis

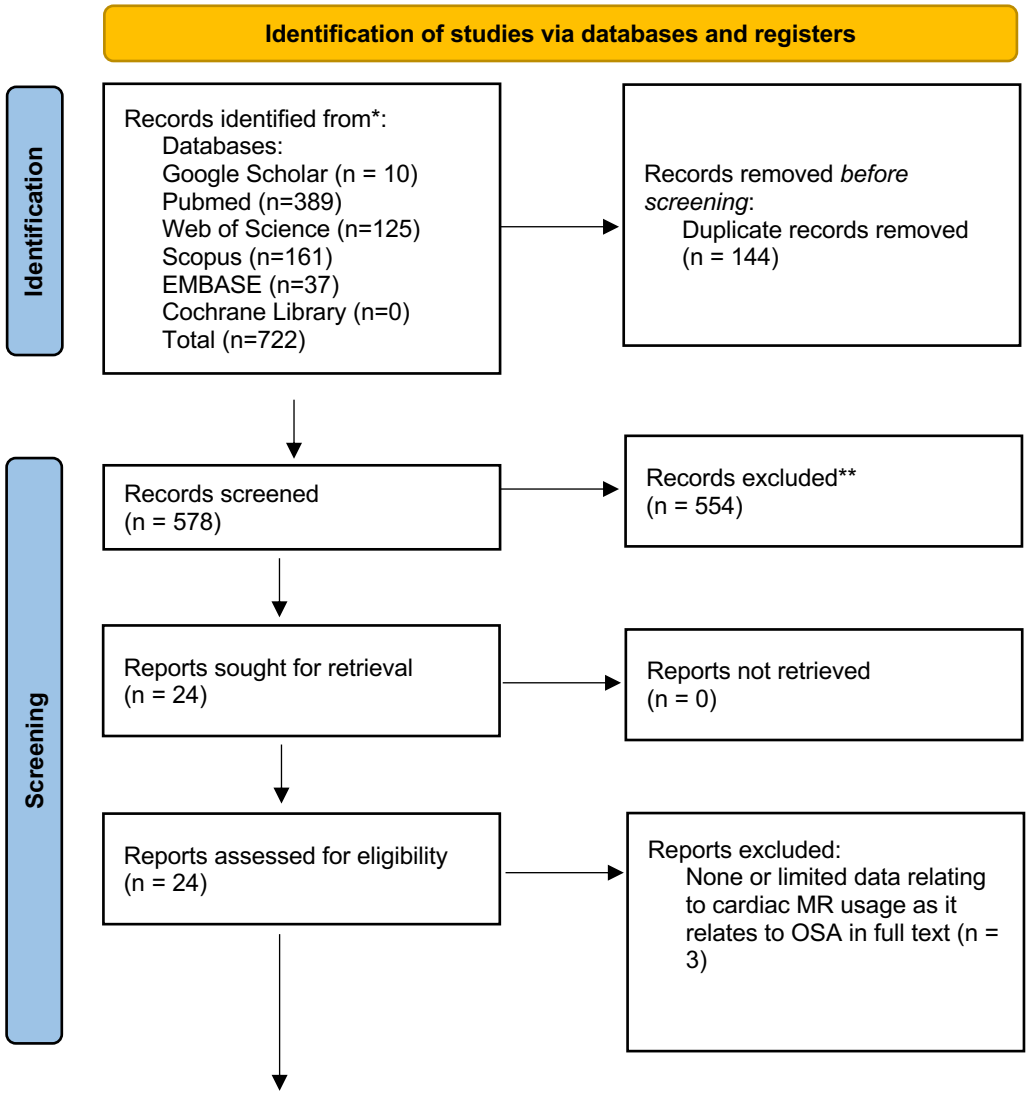
Table 4: Summary of papers analysing the use of CMR for RVF/PH for OSA diagnosis.

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Xu et al.[26]	2020	Cardiac MRI in OSA	OSA associated with RV hypertrophy	MRI for RV hypertrophy assessment
Summerer et al.[30]	2019	RVH in Sleep Apnoea	Sleep apnoea patients showed RV hypertrophy	MRI used for assessing RV hypertrophy
Neilan et al.[14]	2018	Impact of OSA on RV	OSA increases RV hypertrophy, treatable with CPAP	MRI used for detailed RV evaluation

Study	*Year*	*Title*	*Findings*	*Use of Cardiac MR in OSA Diagnostics*
Kylintireas et al.[29]	2017	OSA and RV Hypertrophy	Higher RV mass index in OSA patients	MRI used for tracking RV changes
Geovanini et al.[20]	2016	RV Remodelling in OSA	RV remodelling observed in OSA cases	MRI used to evaluate RV remodelling
Buchner et al.[31]	2015	RV Hypertrophy in OSA	RV hypertrophy linked to OSA severity	MRI for detailed hypertrophy analysis
Nguyen et al.[25]	2014	RV Mass in Overlap Syndrome	Overlap syndrome patients had increased RV mass	MRI used for RV mass measurement
Barone-Rochette et al.[23]	2013	OSA and Right Ventricular Mass	OSA patients showed reversible RV mass increase with CPAP	MRI for monitoring RV mass changes
Alter et al.[18]	2012	RV Mass and OSA	OSA linked to increased RV mass, reduced with CPAP	MRI used for RV mass assessment
Sharma et al.[32]	2012	Evaluation of RV Remodelling in COPD and OSA	RV mass index higher in overlap syndrome group, associated with increased pulmonary hypertension	MRI used for assessing RV and LV changes
Wang et al.[13]	2020	Effect of OSA on RV Ejection Fraction in HOCM Patients	Decrease in RVEF with increasing OSA severity, associated with higher pulmonary artery pressures	MRI used for comprehensive heart assessment
Colish et al.[9]	2012	Effects of CPAP on Cardiac Remodelling in OSA	Reduction in RV end-diastolic diameter and pulmonary hypertension after 3 months of CPAP therapy	MRI for detailed cardiac remodelling study

Table 4: Summary of papers analysing the use of CMR for RVF/PH for OSA diagnosis.

Figure 1: PRISMA 2020 Systematic Review Flowchart



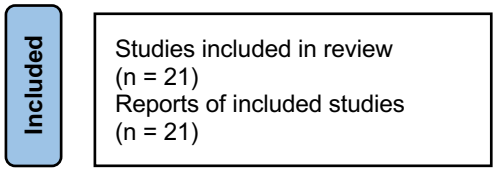


Figure 1: PRISMA 2020 systematic review flowchart