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Prevalence and clinical significance of late gadolinium enhancement in children and adolescents with hypertrophic cardiomyopathy: a systematic review and meta-analysis

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Abstract

Objectives: Hypertrophic cardiomyopathy is the leading cause of sudden cardiac death among the paediatric population. The aim of this study is to investigate the prevalence and clinical significance of late gadolinium enhancement, as assessed by cardiac MRI, in paediatric hypertrophic cardiomyopathy. *Methods:* A systematic literature search was conducted in PubMed, SCOPUS, and Ovid SP to identify relevant studies. Pooled estimates with a 95% confidence interval were calculated using the random-effects generic inverse variance model. Statistical analysis was performed using Review Manager v5.4 and R programming. *Results:* Seventeen studies were included in this meta-analysis, encompassing a total of 778 patients. Late gadolinium enhancement was highly prevalent in paediatric hypertrophic cardiomyopathy, with a pooled prevalence of 51% (95% confidence interval, 40–62%). The estimated extent of focal fibrosis expressed as a percentage of left ventricular mass was 4.70% (95% confidence interval, 2.11–7.30%). The presence of late gadolinium enhancement was associated with an increased risk of adverse cardiac events (pooled odds ratio 3.49, 95% confidence interval 1.10–11.09). The left ventricular mass index of late gadolinium enhancement-positive group was higher than the negative group, with a standardised mean difference of 0.91 (95% confidence interval, 0.42–1.41). *Conclusion:* This meta-analysis demonstrates that prevalence of late gadolinium enhancement in paediatric hypertrophic cardiomyopathy is similar to that in the adult population. The presence and extent of late gadolinium enhancement are independent predictors of adverse cardiac events, underscoring their prognostic significance among the paediatric population.

Hypertrophic cardiomyopathy is a myocardial disorder characterised by autosomal dominant inheritance patterns resulting from genetic mutations affecting the cardiac sarcomere genes.[1] This condition exhibits clinical variability in the paediatric population, spanning from asymptomatic incidental findings to progression to end-stage congestive heart failure, or sudden cardiac death.[2] It stands as a leading cause of sudden cardiac death among paediatric population, with an estimated annual risk ranging from 1 to 7%.[3] Histologically, hypertrophic cardiomyopathy is distinguished by the presence of myocardial fibrosis, myocardial hypertrophy, and disarrayed myocytes.[4] These changes are primarily attributable to an increased extracellular collagen content resulting from mutations in sarcomere genes.[4] Patients with hypertrophy cardiomyopathy often manifest coronary microvascular dysfunction, which results in a diminished coronary flow reserve and ultimately predisposing them to a spectrum of adverse cardiac complications.[5]

Cardiac MRI with delayed contrast is a standard non-invasive imaging for detection, quantification, and differentiation of interstitial myocardial fibrosis in cardiomyopathies, while also enabling the evaluation of left ventricular thickness and mass.[6,7] Furthermore, cardiac MRI with gadolinium contrast facilitates the assessment of myocardial hypertrophy and changes, aiding in the differentiation from alternative diagnoses involving left ventricular hypertrophy.[8] Late gadolinium enhancement effectively reveals macroscopic myocardial scarring in patients with hypertrophic cardiomyopathy, serving as a predictive indicator for poor cardiac outcomes.[9] The presence of myocardial scarring in hypertrophic cardiomyopathy patients is associated with diastolic dysfunction leading to heart failure and arrhythmias.[10] Late gadolinium enhancement is detected in approximately 60% of adult populations with overt hypertrophic cardiomyopathy with its prevalence and progression showing a propensity to increase with time.[9,11,12] Similarly, the presence of late gadolinium enhancement is also reported in paediatric patients and this presence tends to progress with time.[2,13]

The extent of myocardial fibrosis detected through late gadolinium enhancement in cardiac MRI has been identified in adult population as a significant risk factor for adverse cardiac events, which include ventricular fibrillation, ventricular tachycardia, implantable

cardioverter-defibrillator discharges, hospitalisations, and sudden cardiac death.[11,14] The demographic profiles, clinical presentations, and risk factors for mortality in paediatric hypertrophic cardiomyopathy differ from those observed in the adult population, underscoring the distinct and unique characteristics of this cardiac condition in the younger age group.[15] However, there is a paucity of data regarding the prevalence of late gadolinium enhancement in paediatric cases and its predictors for cardiac complications.[16] Therefore, this study aims to investigate the prevalence of late gadolinium enhancement in paediatric hypertrophic cardiomyopathy and its clinical significance.

Materials and methods

Search strategies

A systematic literature search was conducted via PubMed, SCOPUS, and Ovid SP through September 2023 using the following search strategy: ((hypertrophic cardiomyopathy) OR (HCM)) AND ((pediatric) OR (paediatric) OR (children) OR (adolescent) OR (young adult)) AND ((late gadolinium enhancement) OR (late gadolinium enhancement) OR LGE). The authors also retrieved additional papers of interest from the reference lists of selected articles and reviews to optimise the search. The literature search was limited to studies published in English language and peer-reviewed journals. Abstracts and case reports were excluded from the search strategy.

Eligibility criteria

The criteria for inclusion for this meta-analysis include (i) observational cohort studies (prospective or retrospective), (ii) studies involving paediatric patients, (iii) diagnosis of hypertrophic cardiomyopathy, (iv) evaluation of myocardial fibrosis using cardiac MRI, and (v) reporting the presence or absence of late gadolinium enhancement. The criteria for exclusion for this meta-analysis include (i) studies involving adult patients, (ii) studies related to other forms of cardiomyopathy such as dilated cardiomyopathy, ischaemic cardiomyopathy, infiltrative cardiomyopathy or acute myocarditis, and (iii) studies that did not report relevant data, outcomes, and variables between late gadolinium enhancement positive and negative groups.

Study selection and data extraction

The authors independently screened all the titles and abstracts of the articles for eligibility to be included in the meta-analysis. When the eligibility for inclusion based on titles and abstracts was inconclusive, full-text of the article was used for review. Any discrepancies or disagreements regarding the inclusion criteria were resolved through consensus and discussion between both authors. Relevant data from the included studies were independently extracted by both authors into a standardised electronic form created in Excel. The following data were extracted from the included studies: name of first author, year of publication, country of the study, study design, age, prevalence of late gadolinium enhancement, late gadolinium extent, prevalence of adverse event, left ventricular ejection fraction, and left ventricular mass index.

Quality assessment

The methodological quality and the risk of bias of the included studies were evaluated according to the Newcastle–Ottawa Scale.[17] The quality of the selected observational studies was determined based on study selection (representativeness of the exposed cohort, selection of the non-exposed cohort, ascertainment of exposure, demonstration that outcome of interest was not present at start of study), comparability (comparability of cohorts on the basis of the design and analysis), and outcome (assessment of outcome, was follow-up long enough for outcomes to occur, adequacy of follow-up of cohorts). Each study was allocated a score of one for each criterion, with a potential maximum score of two for comparability when the studies fulfilled the decision rule for each criterion, culminating in a possible maximum total score of nine.

Data analysis

The primary endpoints of this study were the prevalence and extent of late gadolinium enhancement. The secondary endpoints of this study were the prevalence and odds ratio of adverse events in patients with late gadolinium enhancement, left ventricular ejection fraction, and left ventricular mass index in both late gadolinium enhancement positive and negative groups. Pooled estimates with a 95% confidence interval were calculated using the random-effects generic inverse variance model.[18] Prevalence was expressed as percentage. The standard error for prevalence was calculated using the formula of standard error $= \sqrt{p\,(1\text{-}p)/\,n}$, 95% confidence interval $= p \pm 1.96 \times$ standard error; where, $p =$ prevalence and $n =$ sample size. Standardised mean differences were used for continuous variables presented as mean ± standard deviation. Heterogeneity between studies was determined using the chi-squared test, with the degree of heterogeneity quantified by I^2.[19] I^2 values of 25, 50, and 75% correspond to low, moderate, and high heterogeneity effects, respectively.[19] Begg's funnel plot and Egger's test were used to assess the possibility of publication bias.[20] Publication bias was considered significant if the Begg's funnel plot was asymmetric and Egger's test had a $P < 0.05$. Statistical analysis was conducted using the Cochrane Review Manager v5.4[21] and R programming language[22] with metafor package.[23] The findings of this meta-analysis were reported according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[24]

Results

Search results and eligible studies

The systematic literature search identified a total of 480 articles, with an additional two articles were identified through the reference list of the included articles. After eliminating duplicates, 130 articles were screened based on title and abstract. A comprehensive assessment was conducted on 20 studies for eligibility based on a full-text review. Out of the 20 articles reviewed in detail, three were excluded: two did not provide data regarding the prevalence of late gadolinium enhancement, and one did not specify the type of cardiomyopathy. Consequently, 17 eligible studies were included in this meta-analysis. A detailed flow chart illustrating the study selection process details following Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow diagram is depicted in Figure 1. All 17 studies were included in the analysis of prevalence of late gadolinium enhancement. Prevalence and odds ratio for adverse cardiac events were based on the analysis of four and five studies, respectively. Finally, seven studies were included for the calculations of standardised mean differences

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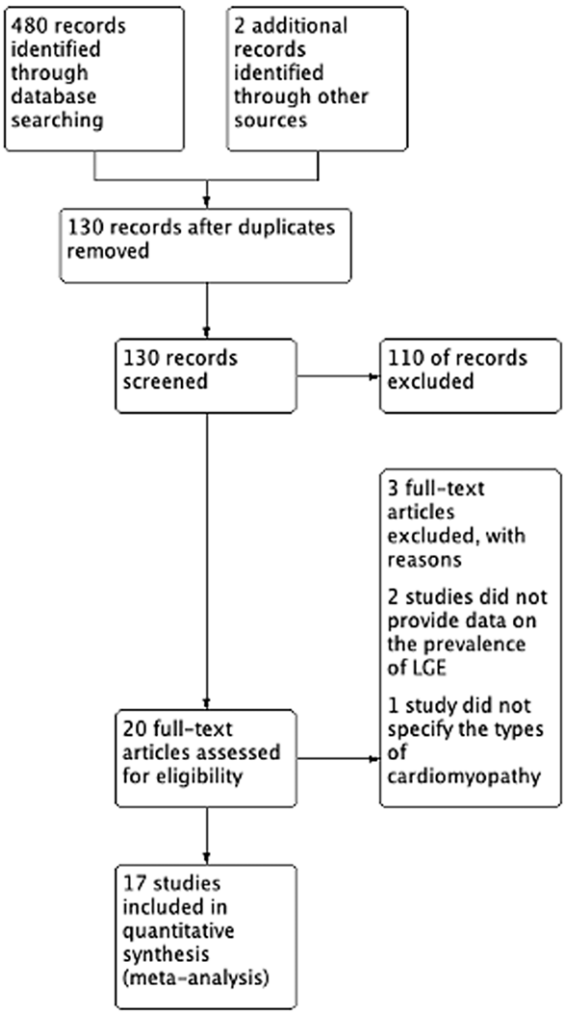

Figure 1. PRISMA flow chart of study selection. LGE, late gadolinium enhancement.

of left ventricular ejection fraction and left ventricular mass index.

Study characteristics

A total of 778 patients derived from 17 studies were included in this meta-analysis.[13,16,25–39] The sample size of the included studies ranged from 13 to 152 participants. All studies included in this analysis were retrospective cohort studies. The average age of the population under study was approximately 14.3 ± 2.3. Sixteen of the studies reported male participants as the majority, whereas only one study reported a majority of female participants. A majority of the studies were conducted in North America with a total of 11 studies (eight United States and three Canada). Four studies were conducted in Asian countries (China, Egypt, Turkey, and India) and two in European nations (Sweden and Poland). An overview of the characteristics of the included studies is summarised in Table 1.

Critical appraisal of studies

The study quality according to the Newcastle-Ottawa Scale is shown in Table 2. The scores of the studies ranged from five to nine. The majority of the studies achieved scores of six or higher, and four studies obtained the maximum score of nine. In total, only six studies were classified as high quality. Eleven studies did not report on the duration of the follow-up and therefore adequacy of follow-up was unable to be assessed. The average score of the studies included was 6.9 ± 1.39.

Prevalence of late gadolinium enhancement

The prevalence of late gadolinium enhancement in paediatric hypertrophic cardiomyopathy displayed significant variability, ranging from 18 to 92%. The overall pooled prevalence of late gadolinium enhancement was 51% (95% CI, 40–62%) (Fig. 2). Furthermore, eight studies reported the extent of late gadolinium enhancement as a percentage of left ventricular mass, which ranged from 2.18% to 11.5%. The overall pooled extent of late gadolinium enhancement was 4.70% (95% confidence interval, 2.11–7.30%) (Fig. 3). Among these, seven reported septal hypertrophy as the most common pattern for late gadolinium enhancement, four studies reported myocardial hypertrophy, and one study reported mid-wall hypertrophy.

Adverse cardiac events in late gadolinium enhancement-positive patients

Adverse cardiac events reported in these studies include ventricular arrhythmias, atrial fibrillation, congestive heart failure, implantable cardioverter-defibrillator discharges, heart transplantation, and aborted sudden cardiac death. The prevalence of adverse cardiac events ranged from 20% to 35.3%. The overall prevalence of adverse cardiac events was 22% (95% confidence interval, 15–29%) (Fig. 4). Furthermore, the presence of late gadolinium enhancement was associated with an increased risk of adverse cardiac events compared to patients without late gadolinium enhancement (pooled odds ratio 3.49, 95% confidence interval 1.10–11.09) (Fig. 5).

Left ventricular ejection fraction and mass index

Seven studies reported data on the left ventricular ejection fraction and left ventricular mass index. The analysis indicated no significant difference in left ventricular ejection fraction between the late gadolinium positive and negative groups (standardised mean difference −0.53, 95% confidence interval −3.68, 2.62) (Fig. 6). However, the left ventricular mass index of late gadolinium enhancement-positive group was slightly higher than the negative group, with a standardised mean difference of 0.91 (95% confidence interval, 0.42–1.41) (Fig. 7).

Publication bias

The publication bias of the meta-analysis was assessed using Begg's funnel plot and Egger's test. Visual assessment of Begg's funnel plot for prevalence of late gadolinium enhancement showed a symmetrical distribution of studies around the overall estimate (Fig. 8). However, Egger's test indicated the presence of publication bias ($P = 0.0003$). Regarding the odds ratio of adverse cardiac events, the visual assessment of Begg's funnel plot displayed a symmetrical distribution (Fig. 9). Furthermore, Egger's test also suggested the absence of publication bias ($P = 0.05142$).

Discussion

Studies have reported that late gadolinium enhancement is present in 55–67% of adult patients with hypertrophic cardiomyopathy.[9] This meta-analysis found that the prevalence of late gadolinium

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Table 1. Description of the included studies

Study	Country	Study design	Sample Size	Age	Male (%)	Female (%)	LGE positive, n (%)	LGE evaluation method
Chaowu 2013[16]	China	Retrospective cohort	71	12.8 ± 4.1	65	35	52 (73.24)	SI ≥ 6 SDs of the signal of non-enhanced myocardium
Smith 2014[24]	United States	Retrospective cohort	30	14.1 ± 3.2	57	43	17 (56.67)	Semi-automated
Windram 2015[25]	Canada	Retrospective cohort	38	12.7 ± 3.3	79	21	7 (18.42)	SI ≥ 6 SDs above the mean of normal myocardium
Hussain 2015[26]	Canada	Retrospective cohort	28	12.8 ± 2.2	75	25	8 (28.57)	Semi-automated
Spinner 2016[27]	United States	Retrospective cohort	33	13.2 ± 5.0	88	12	17 (51.52)	Visual assessment
Bogarapu 2016[28]	United States	Retrospective cohort	29	13.5 ± 6.1	52	48	11 (37.93)	Semi-automated
Compton 2016[29]	Canada	Retrospective cohort	56	12 ± 3	82	18	15 (26.79)	Visual assessment
Raja 2018[13]	United States	Retrospective cohort	152	14.3 ± 4.5	72	28	70 (46.05)	SI > 6 SDs of remote myocardium, FWHM
Hernandez 2018[30]	United States	Retrospective cohort	13	15.38 ± 1.93	92	8	7 (53.85)	NR
Sunthankar 2019[31]	United States	Retrospective cohort	30	15.8 ± 2.2	63	37	18 (60)	Visual assessment
Elfadl 2019[32]	Egypt	Retrospective cohort	14	9.8 ± 5.6	41	59	4 (28.57)	Visual assessment
Bonura 2020[33]	United States	Retrospective cohort	126	19 ± 5.93	62	38	81 (64.29)	SI > 6 SDs above the mean signal intensity
Alis 2020[34]	Turkey	Retrospective cohort	26	13.8 ± 2.5	73	27	16 (61.54)	Semi-automated
Österberg 2021[35]	Sweden	Retrospective cohort	26	16 ± 5.19	77	23	14 (53.85)	Semi-automated
Petryka-Mazurkiewicz 2021[36]	Poland	Retrospective cohort	54	12.03 ± 4.71	69	31	28 (53.85)	SI > 6 SDs of remote myocardium
Kirmani 2023[37]	United States	Retrospective cohort	52	13.8 ± 3.11	78	22	48 (92.31)	Visual assessment
Mukhtar 2023[38]	India	Retrospective cohort	28	12.9 ± 6.03	60	40	17 (60.71)	NR

FWHM = full width at half maximum; NR = not reported; SI = signal intensity.

enhancement in paediatric hypertrophic cardiomyopathy was 51%, which is similar to the adult population. However, assessing late gadolinium enhancement in paediatric patients can be challenging due to potential patient cooperation issues, leading to motion artefacts or respiration interference.[25] Furthermore, the extent of late gadolinium enhancement tends to increase over time in both paediatric and adult populations as hypertrophic cardiomyopathy is a progressive condition.[26,40,41] On average, studies have shown an increase of approximately 6–7 grams of late gadolinium enhancement in adult and paediatric populations over a span of about 2 years of follow-up.[12,13] Late gadolinium enhancement has a strong correlation with hypertrophy, as individuals without late gadolinium enhancement generally have normal wall thickness.[25] The presence of late gadolinium enhancement in patients with hypertrophic cardiomyopathy is influenced by several factors including connective tissue deposition and fibrosis of the myocardium, microvascular ischaemia and left ventricular wall thickness, and mass.[42,43] In hypertrophic cardiomyopathy, diastolic dysfunction is an early manifestation attributed to elevated myocardial stiffness resulting from increased fibrosis.[34] Studies have linked late gadolinium enhancement, an indicative of fibrosis, with increased left ventricular filling pressures and abnormal myocardial relaxation, leading to diastolic remodelling.[34]

Cardiac MRI offers high-resolution spatial images for assessing wall thickness and identifying localised pattern of hypertrophy, being particularly valuable in detecting challenging-to-visualise apical wall and basal anteroseptal thickening when compared to echocardiography.[44,45] Late gadolinium enhancement in adults often displays a mid-wall and midmyocardial pattern.[1,46,47] However, the majority of studies included in this meta-analysis demonstrated that the late gadolinium enhancement in paediatric

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Table 2. Newcastle-Ottawa-scale scores for the quality assessment of including studies

	Selection				Comparability	Outcome			
Studies	Representativeness of the exposed cohort	Selection of the non-exposed cohort	Ascertainment of exposure	Demonstration that the outcome of interest was not present at the start of the study	Comparability cohorts on the basis of the design or analysis	Assessment of the outcome	Was follow-up enough for outcomes to occur	Adequacy of follow-up	Total (9)
Chaowu 2013[16]	*	*	*	*	**	*	*	*	9
Smith 2014[24]	*	*	*	*	**	*	*	*	9
Windram 2015[25]	*	*	*	*	**	*			7
Hussain 2015[26]	*	*	*	*	*	*			6
Spinner 2016[27]	*	*	*	*	*	*	*	*	8
Bogarapu 2016[28]	*	*	*	*	*	*			6
Compton 2016[29]	*	*	*	*	*	*			6
Raja 2018[13]	*	*	*	*	**	*	*	*	9
Hernandez 2018[30]	*	*	*	*		*			5
Sunthankar 2019[31]	*	*	*	*	*	*			6
Elfadl 2019[32]	*	*	*	*	*	*			6
Bonura 2020[33]	*	*	*	*	**	*	*	*	9
Alis 2020[34]	*	*	*	*	*	*			6
Österberg 2021[35]	*	*	*	*	*	*			6
Petryka-Mazurkie wicz 2021[36]	*	*	*	*	*	*			6
Kirmani 2023[37]	*	*	*	*	*	*	*	*	8
Mukhtar 202[38]	*	*	*	*	*	*			6

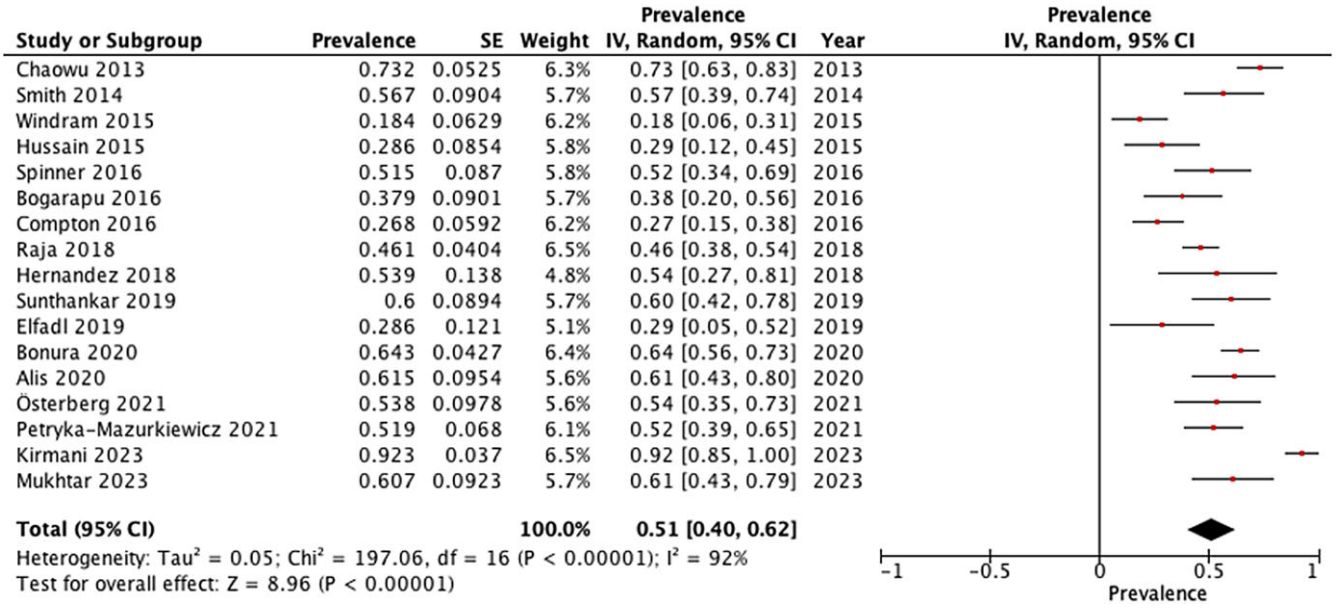

Figure 2. Prevalence of late gadolinium enhancement in paediatric patients with hypertrophic cardiomyopathy.

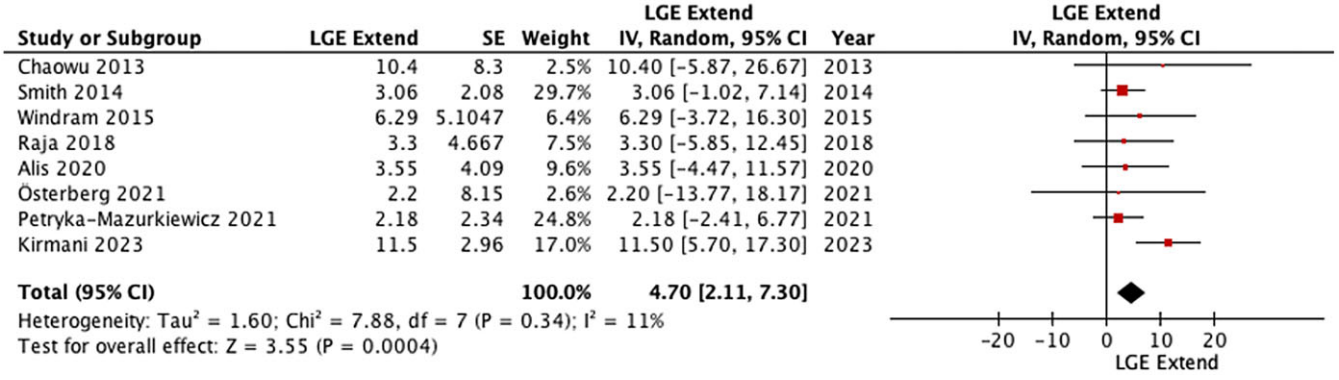

Figure 3. Late gadolinium enhancement extent expressed in percentage of left ventricular mass.

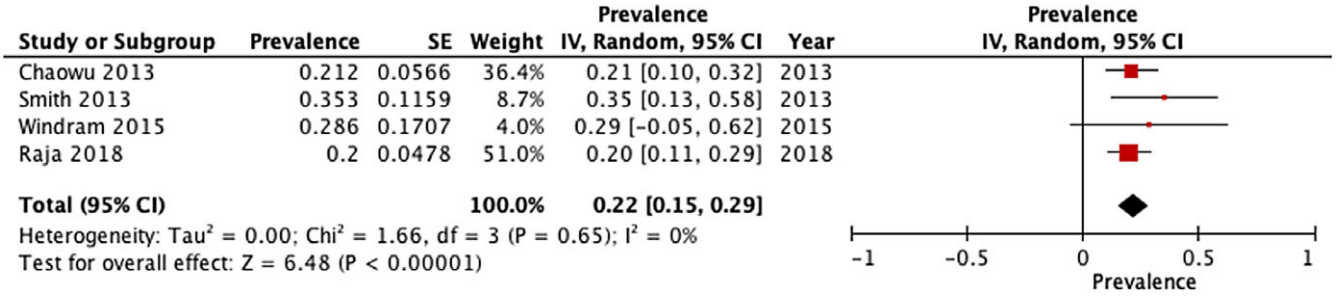

Figure 4. Prevalence of adverse cardiac events in late gadolinium enhancement-positive patients.

population has a septal hypertrophy pattern. Majority of children with positive late gadolinium enhancement exhibited myocardial scarring and pronounced hypertrophic involvement in the interventricular septum.[26,29] The extent, severity, and distribution of hypertrophy may serve as the primary substrates for adverse outcomes, including ventricular arrhythmias or sudden cardiac death.[48] The extent of late gadolinium enhancement has been shown to be associated with an increased risk of ventricular tachycardia and sudden cardiac death in both adult and paediatric populations.[25,28] Left ventricular apical aneurysms are more frequently observed in older children, although they remain rare, yet they carry a substantial annual risk of adverse clinical outcomes, estimated at around 11%.[16,49,50] Furthermore, paediatric patients with a concentric hypertrophy pattern demonstrate a higher prevalence compared to adults and are also associated with poor outcomes such as end-stage hypertrophic cardiomyopathy,

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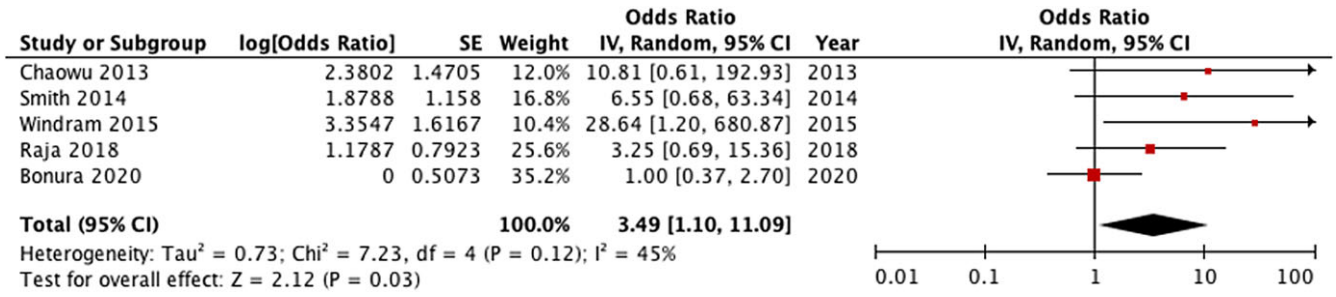

Figure 5. Pooled odds ratio for adverse cardiac events.

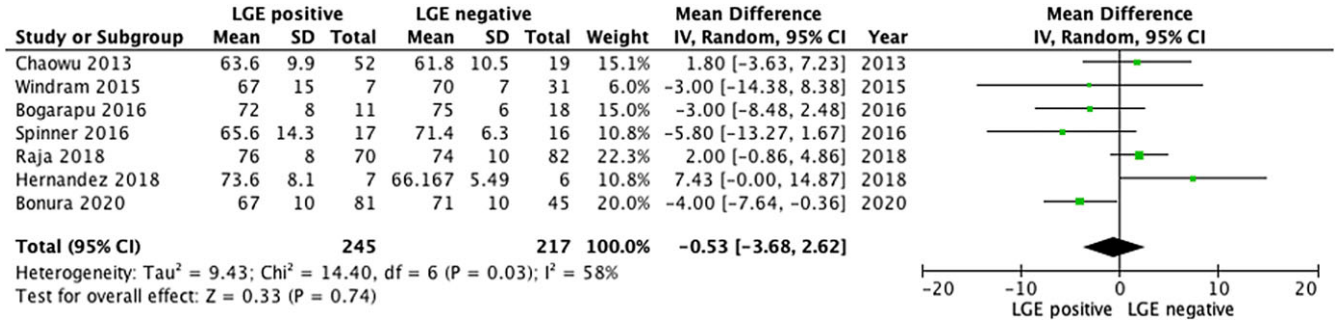

Figure 6. Standardised mean difference for left ventricular ejection fraction.

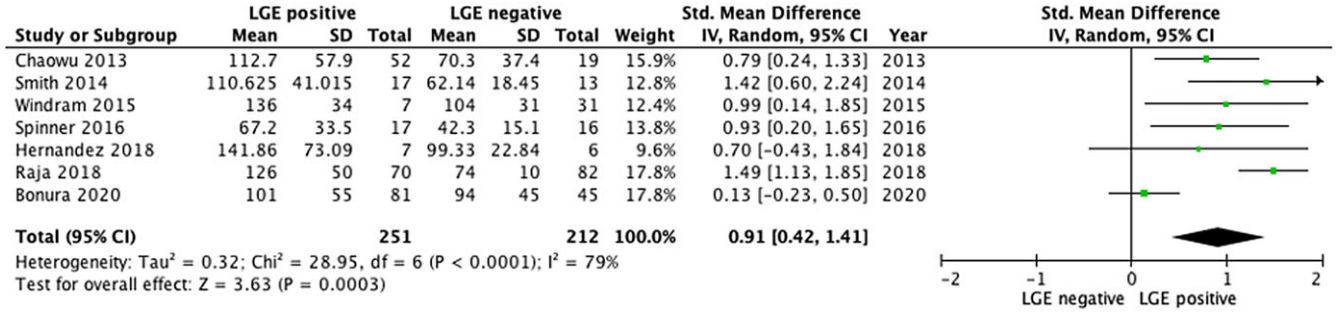

Figure 7. Standardised mean difference for left ventricular mass index.

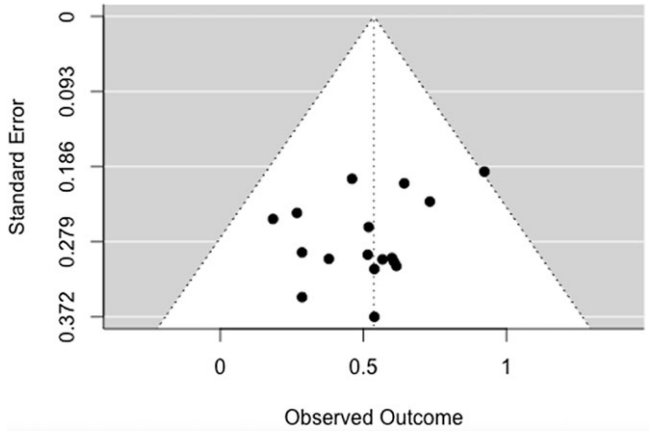

Figure 8. Begg's funnel plot for prevalence of late gadolinium enhancement.

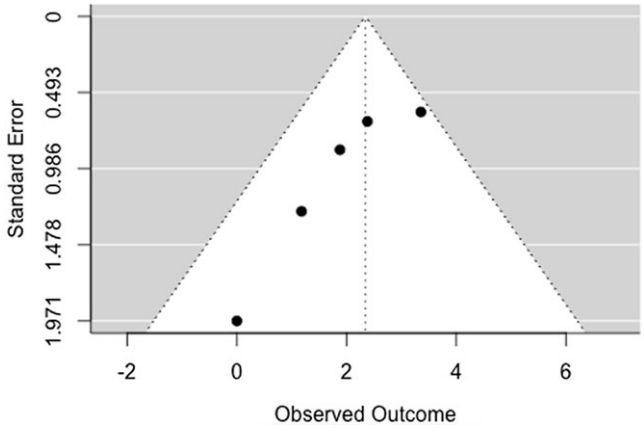

Figure 9. Begg's funnel plot for odds ratio for adverse cardiac events.

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congestive heart failure, and heart transplantation.[16] In cases where paediatric patients exhibit extensive left ventricular hypertrophy extending beyond the interventricular septum, this condition could potentially worsen myocardial ischaemia, potentially leading to the progression to end-stage hypertrophic cardiomyopathy.[51]

Hypertrophic cardiomyopathy represents a primary contributing factor to the occurrence of sudden cardiac death among adolescents, primarily attributed to ventricular tachycardia or ventricular fibrillation, resulting in haemodynamic instability.[3,49] Studies have shown that late gadolinium enhancement in adult populations is an independent risk factor for ventricular arrhythmias, implantable cardioverter-defibrillator discharge, and an increased relative risk of all-cause and cardiovascular mortality.[52] The findings of this meta-analysis also indicated that the presence of late gadolinium enhancement was associated with a higher risk of adverse cardiac events in paediatric population. Furthermore, studies also suggested that the presence of late gadolinium enhancement in paediatric hypertrophic cardiomyopathy increases the likelihood of requiring an implantable cardioverter-defibrillator.[25] However, the role of late gadolinium enhancement as an indicator for implantable cardioverter-defibrillator placement in the paediatric population is limited due to the high incidence of complications, ranging from 32 to 41% during follow-up.[53,54] In paediatric hypertrophic cardiomyopathy, left ventricular hypertrophy generally becomes apparent during late childhood or adolescence and earlier presentation in childhood is often associated with complex syndromes and a less favourable prognosis.[34]

The findings of this meta-analysis suggested that there was no significant difference in left ventricular ejection fraction between those with and without late gadolinium enhancement. Therefore, the role of late gadolinium enhancement as an indicator of left ventricular functional decline remains uncertain.[26] Peak strain analysis has been suggested as a more sensitive indicator of systolic dysfunction, especially when compared to ejection fraction, which can be paradoxically increased in patients with hypertrophic cardiomyopathy.[55–57] Additionally, T1 mapping of cardiac MRI, which quantifies diffuse interstitial fibrosis, emerges as a more useful indicator for left ventricular functional decline.[26,58] Study by Sunthankar et al demonstrated that patient patients with hypertrophic cardiomyopathy demonstrated increased native T1 but not synthetic extracellular volume.[32] Furthermore, the global native T1 is inversely proportional to the left ventricular ejection fraction.[32] Moreover, this meta-analysis showed a slight increase in the left ventricular mass index in patients with late gadolinium enhancement. However, some studies suggest that there is no direct correlation between left ventricular mass and left atrial function.[59] Nevertheless, an increased left ventricular mass may disrupt the electrophysiological processes of the myocardium leading to initiation of fatal arrhythmias.[60] Thus, in turn, establishes that increase in left ventricular mass in patients with hypertrophic cardiomyopathy is an independent risk factor for sudden cardiac death.[61]

There were several limitations of this meta-analysis. Some studies were excluded from this meta-analysis due to the absence of raw data regarding the presence of late gadolinium enhancement. Furthermore, many studies relied on binary classification for presence of late gadolinium enhancement and did not provide data regarding its extent and quantification, which are known to be more informative for prognostication. Furthermore, we are unable to analyse the association between quantified late gadolinium enhancement and left ventricular mass index or ejection fraction. The studies did not report incidence of life-threatening and non-life-threatening cardiac adverse effects separately given the distinct management strategies required for each category. Subgroup analysis across various age cohorts was unfeasible due to the predominant absence of patient categorisation into distinct age groups within the majority of studies. In addition, the meta-analysis of prevalence of late gadolinium enhancement revealed a highly significant heterogeneity possibly attributed to variations in methodology, low sample sizes, lack of population representativeness, and incomplete reporting of results. Nevertheless, the use of random-effect models aimed to minimise the influence of heterogeneity on the outcomes. Additionally, all the studies included in this meta-analysis were retrospective observational studies, and the pooled prevalence was not adjusted for potential confounding variables.

Conclusion

In conclusion, this meta-analysis reveals important insights into late gadolinium enhancement in children and adolescents with hypertrophic cardiomyopathy. The prevalence of late gadolinium enhancement in this population is found to be similar to what is observed in adults. Furthermore, this meta-analysis highlights that the presence and extent of late gadolinium enhancement serve as independent predictors for adverse cardiac events, underlining its significance as a valuable tool for prognostication. This implies that children and adolescents with late gadolinium enhancement-positive hypertrophic cardiomyopathy should undergo regular follow-up assessments, including electrocardiograms, echocardiograms, or cardiac MRIs, to monitor their condition. Further large-scale prospective and longitudinal studies should be conducted to further assess the prognostic value of late gadolinium enhancement in paediatric hypertrophic cardiomyopathy. Such studies will provide a more comprehensive understanding of this condition in the younger population and help refine treatment and management strategies.

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Competing interests. None.

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