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# Cardiovascular Magnetic Resonance Imaging's Prognostic Utility in the Evaluation of Hypertrophic Cardiomyopathy Patients' Long-Term Outcomes and Mortality: A Review

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## ABSTRACT

**Background:** As an additional imaging modality, cardiac magnetic resonance imaging (CMR) is valuable in the evaluation of patients with suspected or diagnosed hypertrophic cardiomyopathy (HCM). **Objective:** Aim was to determine the effectiveness of cardiac magnetic resonance imaging to diagnose long term outcomes among patients of hypertrophic cardiomayopathy.

Given that HCM in children is linked to a higher risk of mortality and worse long-term outcomes, the prognosis and quality of life for these children are greatly affected by the age at which symptoms first appear and the underlying cause of their condition. It is crucial to correctly assess the heart and determine the HCM phenotype in order to make a diagnosis, stratify the prognosis, and do follow-up. The anatomy and function of the heart can be studied by cardiac magnetic resonance (CMR) scans, which can characterize tissues and evaluate blood flow and perfusion. Using CMR, minor anomalies in the myocardial composition can be detected and the various phenotypic expression of HCM can be characterized. The capacity to assess the severity and breadth of cardiac fibrosis using parametric mapping or late-gadolinium enhanced sequences is exclusive to CMR and is especially helpful for clinical assessment and prognostic stratification of children with HCM. Furthermore, HCM in children may worsen with time. Since the pace, timing, and severity of disease progression differ from patient to patient, it is crucial to closely monitor diagnosed patients' hearts and do serial follow-up throughout their lifetimes. Focusing on its clinical function in diagnosis, prognosis, and serial follow-up, this review provides an updated overview of CMR's usage in childhood HCM.

## INTRODUCTION

Cardiac magnetic resonance imaging (CMR) is superior to traditional imaging methods for patients with hypertrophic cardiomyopathy (HCM), whether the condition is suspected or proven.

Cardiomyopathy caused by genetics usually shows up as hypertrophic cardiomyopathy (HCM). Approximately one in 500 people experience it in the general population, according to several studies conducted across the world [1,2]. There have been more than a thousand mutations described in eleven genes that code for the cardiac sarcomere [2]. Among the defining anatomical characteristics of hypertrophic cardiomyopathy are main myocardial hypertrophy, myocyte disarray, and myocardial fibrosis [4]. Its symptoms might range from asymptomatic atrial and ventricular arrhythmias to acute heart failure and sudden cardiac death, among many others.

Cardio MR allows for three-dimensional heart tomographic imaging without ionizing radiation. With high spatial and temporal resolution, the images can be taken from any angle. The unique characteristics of CMR make it an excellent tool for providing detailed phenotypic characterization of HCM, which aids in diagnosis and may even yield prognostic information [5]. In addition, the gold standard for gauging ventricular size and function is computed tomography (CMR).

Additionally, CMR has the ability to describe heart tissue. There has been a lot of research into the correlation between clinical outcomes and measuring gadolinium retention by the myocardium in fibrotic areas. This can be improved with the use of newly developed T1 mapping techniques, which provide additional information about diffuse fibrosis [6,7].

Numerous diseases can impact the heart muscle; these conditions are referred to as cardiomyopathies. The European Society of Cardiology (ESC) currently uses the following classifications: hypertrophic, dilated, arrhythmogenic, limited, and others [8]. Another way to classify them is as either genetic or non-familial. More specifically, we feel obliged to note how contentious this classification is [9].

It is crucial to routinely check in with cardiac imaging because cardiomyopathies can cause symptoms to seem different and evolve over time. These methods greatly improve diagnosis, therapy prescription, and optimize prognosis.

Evaluations consist of electrocardiograms (ECGs), physical examinations, and reviews of medical history. Transthoracic echocardiography (TTE) is a tool that doctors use to diagnose cardiomyopathy. In order to obtain valuable insights into prognosis and to improve the quality of functional and anatomical assessments, cardiovascular magnetic resonance imaging (CMR) is frequently utilized. In some cases, it may be necessary to do testing using nuclear medicine or cardiovascular computed tomography.

Although CMR has many potential benefits as a diagnostic tool, it is important to weigh those benefits against the risks of sedation and anesthesia, which are often required for children under the age of 10 and are more severe in cases with HCM.[10] Thus, every imaging modality has its own set of pros and cons. When used correctly, CMR may improve early identification and survival rates by helping with risk classification for ventricular arrhythmias and SCD, both of which can be fatal.the twelfth The optimal imaging strategy for children with HCM would involve integrating the two modalities.

This study aims to provide a comprehensive overview of the current knowledge on the diagnostic, prognostic, and follow-up functions of CMR in pediatric HCM.

**Prognostic value of CMR**

For the reasons already mentioned, SCD is the leading killer of infants with HCM.Similar to SCD, HCM that starts in childhood is associated with a worse prognosis and an increased risk of mortality compared to HCM that starts in adults. [12,13] Accurate prognostic classification is crucial because the prognosis varies greatly depending on the underlying etiology and the age of diagnosis. Reason being, the result can have far-reaching effects on the child's standard of living.

Cardiovascular fibrosis (CF) and left ventricular hypertrophy (LVH) can be defined and assessed using cardiac magnetic resonance imaging (CMR), which has prognostic significance for risk stratification evaluation in adults and, potentially, children with HCM.[14] There is an inverse relationship between the severity of LVH and the risk of SCD in children with HCM. Heart failure (LVH) is a major clinical risk factor for potentially deadly ventricular arrhythmias; myocardial areas six standard deviations above the predicted mean for body surface area suggest severe LVH.Works Cited References [15,16]``.

Several studies have looked at the predictive value of LGE (on CMR) as a major risk factor for the occurrence of ventricular arrhythmias and SCD in childhood HCM.[17] Their research demonstrated that the presence of LGE enhances the chance of unfavorable outcomes in HCM patients compared to those without LGE. This is because patches of fibrosis frequently provide the substrate for the development of ventricular arrhythmias at the foundation of SCD. The severity of LGE is an additional determinant of ventricular arrhythmias. We state that the left ventricular end-systolic volume is extensive or severe when it surpasses 15% of the total left ventricular myocardial mass. Greater hypertrophy in children with LVH is associated with a higher prevalence of LGE in pediatric HCM, demonstrating a close link between the two.pp. pp. 18–20

In heart failure patients, myocardial perfusion is reduced in the endocardium, a region with the highest wall stress.[21] The amount of reduced blood flow is proportional to the enlargement's size and the scar's thickness. Negative left ventricular remodeling is associated with microvascular dysfunction, which happens when myocardial perfusion is diminished and is a risk factor for myocardial ischemia. Microvascular dysfunction and myocardial ischaemia are associated with worse clinical outcomes in patients with advanced HCM disease. Furthermore, both hypertrophied and non-hypertrophied myocardial regions have shown abnormal perfusion. This indicates that it may occur at an early stage of illness progression and may serve as an accurate prognostic indicator.[22] is a Little is known about the predictive power of T1 mapping in cases of pediatric HCM at this time.T1 mapping has the potential to be a predictive tool in adult HCM, particularly for individuals with increased ECV, a risk factor in and of itself, by stratifying the probability of diffuse myocardial fibrosis.[24] Consequently, T1 mapping could potentially be a fascinating outcome predictor for pediatric HCM patients; further research is needed to establish its therapeutic utility.

**Clinical follow-up of CMR**

The 2020 AHA/ACC guidelines recommend that adults with HCM get repeat CMR imaging every three to five years to better stratify their risk of SCD [25]. The Unfortunately, the topic of repeat CT scans for children with HCM is left out. Currently, the only strategy that is indicated is to screen juvenile HCM patients during follow-up with echocardiography [26]. Because severe HCM development and progression are known to occur during childhood, the varying diagnostic CMR results likely reflect a changing cardiac phenotype over time [27]. It is widely recognized that children with HCM are more likely to develop and experience worsening left ventricular myocardial hypertrophy. Axelsson-Raja et al. and Ali et al. have described the progressive nature of myocardial fibrosis in pediatric HCM patients using LGE in follow-up CMR.on page 28, within by utilizing repeated CMR scans, they were able to detect an increase in both the occurrence and

severity of LGE in patients. Since myocardial fibrosis worsens with time, maintaining CMR imaging may be advantageous for pediatric HCM patients, especially those with LGE evident upon diagnosis. Serial follow-up, according to Axelsson-Raja et al., should include screenings every 1–3 years to catch changes in clinical status as they occur [29].

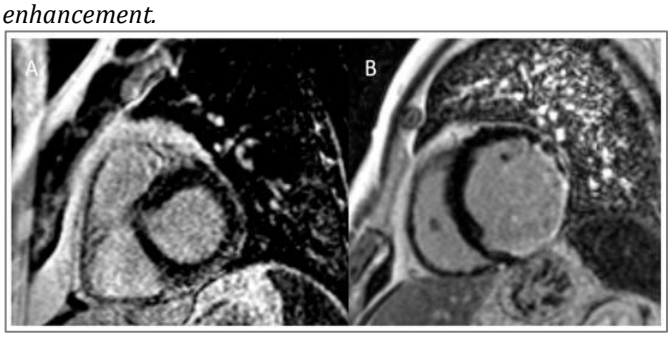
**Risk Prediction of CMR**

Three different methods for calculating LGE—two-standard deviation (SD), full-width half maximum, and three-standard deviation (SD)—each have their own unique impact on risk stratification for cardiovascular disease by assessing cardiac fibrosis and scarring [30]. Because of its sensitivity in detecting moderate, low-density fibrosis, the 2-SD technique is useful for early disease identification, especially in cases of hypertrophic cardiomyopathy (HCM) or diffuse coronary fibrosis (DCM), where even slight fibrotic abnormalities indicate disease progression [56]. But it also has the potential to pick up noise and other distortions, which might cause the fibrosis estimate to pop. There is an elevated risk of arrhythmias and SCD, however the 3-SD method more precisely identifies noticeable scarring. Distributed fibrosis may go undetected, yet it helps patients with larger scars, like those following a myocardial infarction. FWHM achieves a happy medium between sensitivity and specificity by establishing a cutoff at half of the peak signal intensity. This is especially helpful in the detection of transmural infarcts, which are associated with poor outcomes and arrhythmias [31]. However, FWHM may not work in cases of myocarditis or other types of diffuse fibrosis. Arrhythmias, HF, and increased mortality are all more likely to occur in patients with larger scar burdens; however, the method of assessment changes the risk assessment.

Figure 1 shows LGE patterns, which can teach us a lot about heart disease [32]. A mid-wall LGE in the interventricular septum is common in nonischemic cardiomyopathies like DCM or myocarditis, but a subendocardial or transmural LGE pattern shows ischemia damage in myocardial infarction and other ischemic cardiomyopathies. Inflammatory illnesses such as sarcoidosis and myocarditis can lead to epicardial LGE. A widespread or patchy LGE could be an indication of an infiltrative disease such amyloidosis or sarcoidosis, while a common cause of LGE at the right ventricular insertion locations is pulmonary hypertension or HCM [32].

**Figure 1**

*Myocarditis patient (A) with a subepicardial late gadolinium pattern, and myocardial infarction patient (B) with a subendocardial/transmural late gadolinium enhancement.*




Clinical magnetic resonance imaging (CMR) can assist find the etiology of potentially fatal arrhythmias [33]. Whether the left ventricular ejection fraction (LVEF) was 35% or lower or 35% or higher, ventricular arrhythmias were linked to LGE in both groups of individuals. Classification of cardiovascular risk is one potential benefit of CMR for cancer patients [34]. Several clinical situations could benefit from better risk assessment if LGE is assessed during CMR [35]. Patients with cardiomyopathies, whether caused by ischemia or non-ischemia, can benefit from knowing whether LGE is present and how severe it is [36]. There is an increased risk of heart failure mortality or hospitalization for patients with multiple areas of left ventricular enlargement (LGE) [37]. This risk increases in the event of deteriorating heart failure, sudden cardiac death (SCD), or aborted SCD, which is defined as non-fatal ventricular fibrillation (VF), sustained ventricular tachycardia (VT), or adequate implantable cardioverter-defibrillator therapies. Guidelines for 2020 HCM have been proposed by the American College of Cardiol (ACC) and the American Heart Association (AHA), which include evaluation of LGE in the risk assessment for SCD.

Quantitative LGE Cem analysis can aid in the prediction of SCD in HCM patients regardless of their baseline features [38]. This group of patients is more likely to develop SCD if they have large patches of LGE, particularly if those regions include at least 15% of LVM. For a subset of HCM patients, a higher LGE level may be more helpful in predicting the chance of SCD than either the European HCM Risk-SCD rating or the American College of Cardiology Foundations (ACCF)/AHA algorithm [39].

**Perspectives for Future**

Most models used for diagnosing, predicting, and classifying children at risk of HCM using CMR data are currently based on criteria for adult HCM.[40] To establish reference values for youngsters, learn more about their distinct traits, and make sure CMR is consistently applied to this group, more research is needed. At the moment, there is a dearth of pediatric normative datasets like parametric mapping and tissue characterization. T1 mapping results may vary among models and suppliers of CMR scanners due to variables such as magnetic field strength and manufacturer. To identify abnormal myocardium, thus, various institutions typically use T1 values within their own jurisdiction.[41] The Because comparing and understanding paediatric CMR studies is so challenging, there must be standardization. Additionally, state-of-the-art CMR methods like atria appraisal, strain measures, and 4D flow and diffusion tensor imaging may be helpful for children with HCM.[42] Before we can put our faith in these strategies, however, we must further investigate their therapeutic use and applicability to children. Therefore, deep learning-based quantitative methods have the potential to greatly improve LGE and perfusion imaging methodologies. Quantification has the potential to improve the consistency and accuracy of clinical follow-up and decision-making.

The development of similar imaging modalities and the establishment of common clinical definitions of diagnostic and prognostic criteria of CMR findings are

critical steps toward facilitating the combining and comparison of CMR data in juvenile HCM. In addition, the pediatric features make it possible to incorporate CMR into clinical follow-up, which helps with risk prediction and reassessment taking the changing cardiac phenotype into account. Obtaining a clinical CMR baseline for all patients with pediatric HCM would be beneficial due to the fact that every child's heart and body are unique. As a result, tailored clinical evaluation, treatment, and investigation can now take place. Those patients who showed worse disease characteristics on the first CMR or who developed worse HCM over time might benefit from a second CMR.

## CONCLUSION

Whether a child has suspected or proven HCM, cardiovascular magnetic resonance imaging is a useful tool. Comprehensive magnetic resonance imaging (CMR) scans the heart efficiently and identifies prognostic risk factors because of its many aspects. When it comes to evaluating the structure, function, perfusion, and composition of the myocardium, CMR is unparalleled. CMR imaging and quantification are made possible by LGE sequences for parametric mapping of diffuse myocardial-replacement fibrosis and localized fibrosis. Cardiovascular risk stratification and patient-specific cardiac information provided by CMR improves diagnosis and survival rates. In children, HCM progresses more rapidly and poses a greater risk of potentially fatal ventricular arrhythmias and sudden cardiac death (SCD) compared to adults. Diagnostics, prognoses, and serial follow-up for pediatric HCM can be facilitated using CMR.

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