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APICAL HYPERTROPHIC CARDIOMYOPATHY RELEVANCE, DIAGNOSIS, AND MANAGEMENT: A NIGERIA AND WEST AFRICA NARRATIVE

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ABSTRACT

Apical hypertrophic cardiomyopathy (ApHCM) is a distinct and often under-recognized phenotype of hypertrophic cardiomyopathy characterized by disproportionate left ventricular apical hypertrophy, giant T-wave inversion, and, in some patients, apical aneurysm, arrhythmia, or thromboembolic risk. Although ApHCM has been studied extensively in Asian and selected Western cohorts, evidence from West Africa remains sparse, and the condition is frequently obscured by overlapping phenotypes such as hypertensive heart disease and endomyocardial fibrosis. This review synthesizes current evidence on the epidemiology, clinical presentation, imaging phenotype, differential diagnosis, risk stratification, and management of ApHCM, with attention to diagnostic barriers in Nigeria and the broader West African region. We highlight the added value of focused echocardiographic protocols, contrast imaging, and cardiac magnetic resonance in improving case detection and risk assessment, and we discuss how regional infrastructure constraints shape real-world practice. By integrating global evidence with local diagnostic realities, this review aims to provide a practical framework for earlier recognition, more accurate phenotyping, and context-appropriate management of ApHCM in under-resourced settings.

Keywords: Apical hypertrophic cardiomyopathy; Yamaguchi syndrome; Giant T-wave inversion; Cardiac magnetic resonance; Echocardiography; Endomyocardial fibrosis; West Africa; Nigeria; Sudden cardiac death; Apical aneurysm

1 INTRODUCTION

The second leading cause of sudden cardiac death (SCD) is Hypertrophic cardiomyopathy (HCM) which is the most common inherited heart disease with a variation: Apical hypertrophic cardiomyopathy(ApHCM) represents a distinct morphological variant of hypertrophic cardiomyopathy, but its prevalence is under-characterized in current literature[1],[2]. A giant T wave on electro-cardiogram shows ApHCM and is more frequent in the Japanese population than in Western populations. This diagnostic ambiguity is further compounded by the localized clinical overlap with prevalent endomyocardial conditions, which often mimic or mask the distinct apical morphology characteristic of ApHCM [3], [4], [5]. Furthermore, access to advanced image modality creates a constraint toward extreme examination of “ace of spade” configuration especially in a resource limited environment [6].

One diagnostic challenge is high rate of under-detection of echocardiographic associated apical aneurysms, which necessitate cardiac magnetic resonance imaging for accurate identification [7]. Consequently, regional clinicians must maintain a high index of suspicion for this phenotype, particularly when electrocardiograms exhibit giant T-wave inversions, a hallmark often misinterpreted in the presence of more common hypertensive heart diseases [5], [8]. Given that current guidelines offer limited ApHCM-specific recommendations, adapting international diagnostic criteria to the West African clinical landscape remains a significant hurdle for practitioners. Furthermore, the high incidence of coronary artery disease mimics in this population risks misdiagnosis and the implementation of unnecessary therapeutic interventions [9], [10].

Establishing a formal diagnostic framework is therefore essential to distinguish ApHCM-related microvascular ischemia from traditional ischemic heart disease, as the latter remains a prevalent concern in the region. [11], [12] To address these challenges, future efforts must prioritize epidemiological mapping and the standardization of imaging protocols to detect the characteristic apical distribution of hypertrophy effectively.[1], [13] Moreover, the integration of cardiac magnetic resonance imaging is critical, as it overcomes the limitations of two-dimensional echocardiography in visualizing subtle LV wall thickening patterns that are often missed in routine assessments [14]. Beyond imaging limitations, the scarcity of specialized diagnostic infrastructure including CMR and genetic screening often leads to significant delays in identifying the disease, further complicating the management of patients in low- and middle-income healthcare settings[15], [16]. Addressing these systemic barriers requires international collaboration to bridge the infrastructure gap, as evidenced by recent initiatives to establish nuclear imaging and cardiac centers of excellence within the region. This narrative review aims to synthesise current evidence on the epidemiology, clinical presentation, multimodality imaging, differential diagnosis, and management of ApHCM, with particular emphasis on the diagnostic challenges unique to Nigeria and West Africa, and to propose context-adapted strategies for clinical practice in resource-limited settings.

2 METHOD

This paper is a narrative review conducted in accordance with established standards for qualitative synthesis of clinical and epidemiological literature. The review was not registered prospectively, consistent with its narrative rather than systematic design.

2.1 Literature Search Strategy

A comprehensive search of three electronic databases was performed: PubMed, Google Scholar, and Scopus. The search spanned publications from January 2015 to June 2026, capturing a decade of contemporary evidence while encompassing the most significant advances in multimodality cardiac imaging and hypertrophic cardiomyopathy (HCM) risk stratification. Only articles published in the English language were included.

Search terms were applied in varying combinations using Boolean operators (AND, OR, NOT) and included: "apical hypertrophic cardiomyopathy", "Yamaguchi syndrome", "apical HCM", "hypertrophic cardiomyopathy", "giant T-wave inversion", "cardiac magnetic resonance", "left ventricular apical aneurysm", "echocardiography HCM", "apical hypertrophic cardiomyopathy Africa", "hypertrophic cardiomyopathy West Africa", "hypertrophic cardiomyopathy Nigeria". Reference lists of all retrieved articles were manually screened to identify additional publications not captured by the primary electronic search, including landmark pre-2015 studies cited for essential context.

2.2 Inclusion Criteria

Articles were considered eligible for inclusion if they met the following criteria: (i) original research articles, case reports, case series, systematic reviews, meta-analyses, or previously published narrative reviews addressing the epidemiology, clinical presentation, diagnostic evaluation, imaging characterisation, risk stratification, or management of apical hypertrophic cardiomyopathy; (ii) studies reporting on the broader HCM spectrum where apical phenotype data were extractable or discussed; (iii) publications specifically addressing cardiovascular disease patterns, diagnostic infrastructure, or HCM in sub-Saharan Africa or West Africa; and (iv) publications addressing conditions relevant to the differential diagnosis of ApHCM in the West African context, including hypertensive heart disease, endomyocardial fibrosis, and Takotsubo cardiomyopathy.

2.3 Exclusion Criteria

Articles were excluded if they: (i) were published before January 2015, except where a foundational or landmark publication predating this period was deemed essential for contextualisation in such cases, the article is explicitly cited with its historical significance noted; (ii) were not available in the English language; (iii) addressed HCM variants without specific relevance to the apical phenotype or its differential diagnosis; or (iv) were conference abstracts, editorials, or letters without substantive data.

2.4 Study Selection and Data Extraction

Article titles and abstracts were screened independently by the lead author for relevance. Full texts of potentially eligible articles were retrieved and reviewed. Given the narrative design of this review, no formal risk-of-bias assessment tool was applied; however, the quality and source of evidence informing each clinical statement are indicated throughout the text, with preference given to prospective studies, multicentre registries, and guideline documents over single-centre retrospective series where available.

2.5 Synthesis Approach

Data were synthesised narratively, organised thematically around the major clinical domains of ApHCM: epidemiology and global prevalence, electrocardiographic features, multimodality imaging, differential diagnosis, risk stratification, and management. Given the scarcity of West African-specific data, evidence from Asian and European cohorts where ApHCM is comparatively better characterised is contextualised against the clinical and infrastructural realities of the West African setting throughout the review.

2.6 Epidemiology and Global Perspective

There are ethnic and geographical variation as it concerns the Epidemiological studies of ApHCM (see Table 1). The prevalence among hypertrophic cardiomyopathy cases, ranging from 1-3% in North American and European cohorts to 13-41% in Asian populations, with evidence of elevated rates in Afro-Caribbean and African descent groups relative to Caucasians [1], [6], [17], [18]. This disparity underscores the potential under-recognition of ApHCM in West Africa, where isolated case reports suggest its occurrence amid a broader spectrum of cardiomyopathies, yet comprehensive regional data remain scarce [19]. Male predominance is consistent globally (Table 1), with diagnosis typically in midlife, though the genetic underpinnings show lower sarcomeric mutation rates in ApHCM compared to other variants [1],[2]. A histological evaluation reveals distinct structural abnormalities such as myocardial disarray and replacement fibrosis that necessitate advanced tissue characterization techniques for accurate differential diagnosis [20].

Table 1: Epidemiology of ApHCM in published studies

Authors	Publication year	Mean age (SD)	Male, n%	Country/Ethnicity	Prevalence
Lee et al. [21]	2015	54.0± 12.0	278 (79.4%)	South Korean (Asian)	94/350(27%)
Vucicevic et al.[22]	2016	62.8 (range 38–89	22 (70.9)	USA	31/143 (21.6%)
Kim et al.[23]	2016	56 (49–64)	75 (88%)	Korean (Asian)	85/350 (24%)
Neubauer et al.[24]	2019	51.0 ± 10.0	175 (78%)	North America and Europe	224/2,755 (8.1%)
Aoki et al.[25]	2016	NR	NR	Japanese (Asian)	6/12 (50%)
Dastidar et al[26]	2016	60(54-70)	NR	United Kingdom (European)	31/104(29.3%)
Alexandra[27]	2021	54.9 ± 16.9	527 (65%)	Australia (Multiethnic HCM cohort)	East Asians: 17/75 (23%); Europeans: 37/611 (6%)
Pavlos Rouskas[28]	2023	50.7 ± 18.6	93 (65%)	NR	143/ 856 (16.7%)
Anand, et al.,[29]	2024	59.4 ± 16.6	233 (56%)	United States	NR
Tong & Bilgrami[30]	2025	47.5 ± 12.3	30(76.9%)	Bruneian (Southeast Asian)	15/39 (38.5%)

Recent investigations also indicate that ventricular diastolic dysfunction often precedes overt morphological remodeling in these segments, suggesting that early detection of these functional changes could serve as a vital marker for disease progression and risk stratification [31]. Beyond morphologic and functional assessment, the incorporation of cardiac magnetic resonance imaging has proven instrumental in distinguishing ApHCM from other conditions, such as eosinophilic heart disease or amyloidosis, which frequently present with similar phenotypic features [29]. Martens and colleagues evaluated different phenotypic features of HCM, suggesting that establishing a multidisciplinary

framework that integrates genetic screening with these imaging modalities remains essential to differentiate sarcomeric hypertrophic cardiomyopathy from phenocopies such as amyloidosis [32]. Such diagnostic precision is particularly vital given that access to targeted therapies, including cardiac myosin inhibitors and gene-silencing agents, is severely restricted across most African healthcare even in epidemiological data (See Table 1) systems [33].

2.7 ApHCM Evidence from West Africa and Nigeria

In West Africa, data on ApHCM remain sparse and largely confined to case reports and small echocardiographic series, such as a study from southwestern Nigeria identifying hypertrophic cardiomyopathy in 2% of referrals with giant T-wave inversions in 42.9% suggestive of apical involvement, alongside isolated instances of pure apical phenotypes in patients presenting with chest pain and confirmed by speckle-tracking echocardiography [19], [34], [35]. These reports underscore frequent diagnostic overlap with endomyocardial fibrosis, as illustrated by multimodality imaging in a dyspneic. West African patient revealing coexistent apical hypertrophy, cavity obliteration, and calcification [4]. Limited regional genetic analyses further complicate phenotyping, with familial screening revealing septal hypertrophy in relatives of Cameroonian cases harboring MYBPC3 mutations, emphasizing the need for expanded genomic studies amid infrastructural constraints [36].

Such diagnostic complexity is further compounded by the clinical need to distinguish ApHCM from secondary hypertrophy, as evidenced by echocardiographic indices such as the apical-basal wall thickness ratio and relative apical longitudinal strain which are essential for differentiating primary cardiomyopathy from hypertensive heart disease [37]. Korthals and colleagues opined that, the diagnostic utility of cardiovascular magnetic resonance remains superior for identifying apical myocardial disarray and characterizing late gadolinium enhancement patterns that are pathognomonic for hypertrophic cardiomyopathy versus amyloidosis [38].

Also, with phenotypic heterogeneity inherent in these cases necessitates a more granular classification, distinguishing between pure-apical and distal-dominant forms, which exhibit unique prognostic trajectories and distinct genetic backgrounds [39]. Moreover, understanding these structural nuances is vital, as ApHCM typically follows a more favorable clinical course with lower rates of all-cause mortality and heart failure hospitalizations compared to non-apical hypertrophic cardiomyopathy variants [40]. However, the increased risk of apical aneurysm formation and subsequent thromboembolic complications in specific subsets necessitates rigorous monitoring, even in patients who remain asymptomatic [41].

In Ghanaian tertiary centers, transthoracic echocardiography with speckle-tracking has emerged as a key modality for detecting apical strain reduction and aneurysm formation in ApHCM cases among West African patients, facilitating earlier risk stratification despite limited access to advanced imaging [19], [42]. Good standard protocol combined with feature-tracking could enhance early identification of apical fibrosis and strain abnormalities in ApHCM beyond echocardiographic limits [33], [43].

Furthermore, synthesizing existing Nigerian study on the clinical presentation of ApHCM particularly the observed prevalence of giant T-wave inversions in symptomatic patients and on 2% are being examined with echocardiographic [34] is essential for constructing regional prognostic models that robustly incorporate independent predictors such as age and left ventricular ejection fraction in the context of overlapping cardiovascular comorbidities.

However, A related and more structured effort to map hypertrophic cardiomyopathy (HCM) in Nigeria is already underway. Ogah and colleagues have registered a PROSPERO systematic review protocol (CRD420251020441) to synthesize the prevalence, clinical features, and outcomes of HCM across Nigerian cohorts from 1960 to 2024[44]. Ogah et al. review will provide systematic, pooled epidemiological estimates for HCM broadly, this narrative synthesis focuses specifically on the apical phenotype, offering the imaging, diagnostic-pathway and management context needed to interpret ApHCM cases once they are identified. Together, the two efforts point to a growing regional appetite and infrastructure for coordinated HCM research in Nigeria.

3 CLINICAL MANIFESTATION

Patients with apical hypertrophic cardiomyopathy often report a variable symptom complex, with approximately 54% of cases manifesting clinically through chest pain, palpitations, dyspnea, or syncope[45]. While these symptoms are common, the heterogeneous nature of the condition can also manifest in sudden cardiac events, necessitating a high index of suspicion in patients who do not fit typical hypertensive profiles [46]. In West African, ApHCM often presents with a deceptive clinical profile, characterized by exertional dyspnea and atypical chest pain that frequently mimics hypertensive heart disease or endemic inflammatory cardiomyopathies[47]. Specifically, the presence of these symptoms necessitates a careful exclusion of hypertensive heart disease and secondary cardiomyopathies, which often present with similar morphological features that require advanced imaging for definitive differentiation [48]. Even with

series of symptoms that are common, complications could be unspecified cardiac manifestation and cerebrovascular events.

3.1 Management Strategies

The management of apical hypertrophic cardiomyopathy requires a risk-stratified approach, focusing on the prevention of sudden cardiac death and the mitigation of heart failure symptoms. Risk stratification follows the 2024 AHA/ACC Hypertrophic Cardiomyopathy Guidelines, incorporating clinical markers including maximal wall thickness, family history of sudden cardiac death, unexplained syncope, non-sustained ventricular tachycardia on Holter monitoring, apical aneurysm presence on imaging, and late gadolinium enhancement burden on CMR where available. Patients with one or more major risk factors should be evaluated for ICD implantation through shared decision-making[49], [50]. Nevertheless, 2023 ESC Guidelines for the Management of Cardiomyopathies[51] has shown to cover a broader spectrum and can offer complementary framework for training west African cardiologist practice.

3.2 Pharmacological Intervention

Beta-blockers and calcium channel blockers serve as the primary therapeutic agents to alleviate symptoms by reducing heart rate and enhancing diastolic filling, thereby addressing the hemodynamic consequences of outflow tract or mid-ventricular gradients[8]. In cases of refractory angina or severe obstructive features, judicious titration of disopyramide may be considered to mitigate high-pressure gradients and secondary apical cavity obliteration[52]. Furthermore, long-term longitudinal monitoring must include regular risk assessments for arrhythmias, particularly in patients exhibiting intramural late gadolinium enhancement, which has been significantly associated with an increased incidence of ventricular tachyarrhythmia.

Cardiac myosin inhibitors mavacamten and the more recently approved aficamten are an important pharmacological advance not otherwise addressed in this section. Both agents stabilize the super-relaxed state of cardiac myosin, reducing hypercontractility and improving diastolic filling independent of outflow-tract obstruction, and both are now approved for obstructive HCM on the strength of EXPLORER-HCM, VALOR-HCM and SEQUOIA-HCM. Their extension to non-obstructive phenotypes, and the category most ApHCM falls into has been less uniform: mavacamten failed to meet its primary endpoints in the dedicated non-obstructive trial (ODYSSEY-HCM), whereas aficamten met both primary endpoints (symptom burden and exercise capacity) in the analogous ACACIA-HCM trial, suggesting the class effect may not generalize evenly across agents in non-obstructive disease. Apical-specific evidence remains limited to case-level reports, including a recent case of symptomatic improvement and reduced intraventricular gradient with mavacamten in mid-ventricular and apical HCM[53], [54] rather than trial-level data. Cardiac myosin inhibitors are not yet available in most West African health systems.

3.3 Surgical Considerations

According to Bărboi et al., and Rodríguez et al., patients who progress to refractory heart failure or develop significant left ventricular apical aneurysms, surgical interventions such as apical myectomy or, in extreme cases, cardiac transplantation, may be indicated to alleviate mechanical obstruction and improve long-term outcomes [55], [56]. Additionally, the presence of apical thrombi, which can occur even in the absence of overt ventricular aneurysms or persistent atrial fibrillation, necessitates the consideration of chronic anticoagulation to minimize embolic risk [57]. Given the variable clinical progression of this condition, clinicians should also maintain vigilance for the development of life-threatening arrhythmias, as patients may transition from an asymptomatic state to presenting with ventricular fibrillation or sustained tachycardia [58].

3.4 Counseling

Currently, no counseling recommendations exist specifically for apical hypertrophic cardiomyopathy (ApHCM); therefore, patient management generally follows recommendations established for hypertrophic cardiomyopathy (HCM) as a whole. Contemporary guidelines emphasize the importance of maintaining a healthy lifestyle and participating in regular physical activity. However, exercise prescriptions should be individualized based on symptom burden, arrhythmic risk, the presence of left ventricular outflow tract obstruction (LVOTO), and other risk factors for sudden cardiac death (SCD)[50]. As by Lampert etal. rather than universally prohibiting vigorous activity, current recommendations advocate shared decision-making between patients and clinicians to determine appropriate exercise intensity and participation in recreational or competitive sports[59].

Following diagnosis, most individuals with ApHCM can continue their usual employment and daily activities. Nevertheless, occupations involving substantial physical exertion should be carefully discussed because of the potential hemodynamic and arrhythmic risks associated with intense activity. Likewise, individuals employed in specialized professions such as aviation, military service, and emergency response occupations are generally required to comply with occupation-specific cardiovascular fitness standards established by regulatory bodies and employers.

Genetic and reproductive counseling remains an essential component of care for women of childbearing age with ApHCM. Patients should receive counseling regarding the hereditary nature of the disease and the potential risk of transmission to offspring. Available evidence indicates that most women with HCM, including those with ApHCM, tolerate pregnancy well because the hypertrophied ventricle can usually accommodate the physiological increase in blood volume during gestation. However, women with pre-existing significant outflow gradients or symptomatic obstruction may experience worsening hemodynamic compromise and are at greater risk of pregnancy-related complications. Consequently, pre-conception risk stratification and multidisciplinary management are strongly recommended.

Functional assessment before conception and during pregnancy may include exercise testing in asymptomatic women or those with mild symptoms to evaluate exercise tolerance, heart rate response, and the occurrence of arrhythmias. Such assessments contribute to individualized risk evaluation and facilitate appropriate counseling regarding maternal and fetal outcomes.

3.5 Emerging Diagnostic and Prognostic Approaches for Apical HCM: Opportunities and Challenges in West Africa

Emerging data suggest that leveraging cardiac MRI, particularly feature-tracking technology, could further enhance these prognostic efforts by identifying early-stage myocardial changes and distinguishing true apical hypertrophy from conditions like endomyocardial fibrosis [42], [43]. Integrating these advanced diagnostic protocols is essential to address the high rates of mutation-negative cases observed in apical phenotypes, where genetic heterogeneity often complicates clinical decision-making [60].

These models must account for the limited availability of genetic testing and late gadolinium enhancement imaging, which are currently inaccessible to most patients and hinder the accurate identification of HCM phenocopies [61]. Consequently, clinicians must prioritize the detection of markers such as apical aneurysms, which serve as critical indicators of potential sudden cardiac death and adverse clinical events in resource-limited settings [62], [63].

The scarcity of specialized cardiovascular imaging equipment and trained cardiac radiologists in West Africa severely limits the routine application of Cardiac Magnetic Resonance in clinical practice, which is essential for differentiating ApHCM from hypertensive heart disease [15], [64] . Furthermore, the reliance on routine echocardiography, often the only accessible tool in these settings, frequently leads to the underreporting of apical structural changes that would otherwise be visualized through superior imaging techniques. Moreover, the high cost of contrast-enhanced echocardiography acts as an additional barrier, further hindering the detection of apical aneurysms and thrombi that carry significant morbidity [65]. Consequently, fostering collaborations to improve regional access to advanced imaging and dedicated genetic counseling is essential to bridge the current diagnostic divide [66]. Considering diagnostic infrastructure, clinicians must establish robust longitudinal surveillance programs for high-risk patients, as regional data on sudden cardiac death risk factors remain critically underdeveloped [67]. Addressing these systemic gaps requires a dual approach: integrating established cardiovascular screening protocols into primary care to mitigate the risk of misdiagnosis and launching regional registries to better delineate the phenotype's natural history [68], [69]. Ultimately, comprehensive evaluation involving precise morphological assessment and tissue characterization remains the cornerstone for overcoming diagnostic ambiguity and ensuring optimal therapeutic outcomes for this often-neglected patient population [70].

3.6 Diagnostic considerations and proposed solutions

By optimizing imaging workflows, better characterization of apical thinning and "ace of spades" morphology will provide vital differential insights when evaluating patients with myocardial infiltration or restrictive physiology [71], [72]. These workflows are particularly critical given the high prevalence of hypertension in the region, which frequently mimics hypertrophic remodeling and complicates the clinical diagnosis of true ApHCM [73].

3.7 Electrocardiographic Patterns

The presence of giant negative T-waves in the precordial leads, often exceeding 10 mm in depth, remains a hallmark diagnostic clue for apical hypertrophic cardiomyopathy that warrants further investigation when encountered in the clinical setting [74]. Complementary to these repolarization abnormalities, echocardiographic assessment should be rigorously evaluated for the "spade-like" cavity configuration at end-diastole, although contrast-enhanced imaging is frequently required to bypass the technical limitations of conventional transthoracic windows [5], [75]. A representative 12-lead ECG is shown in Figure 1, showing a characteristics of ApHCM

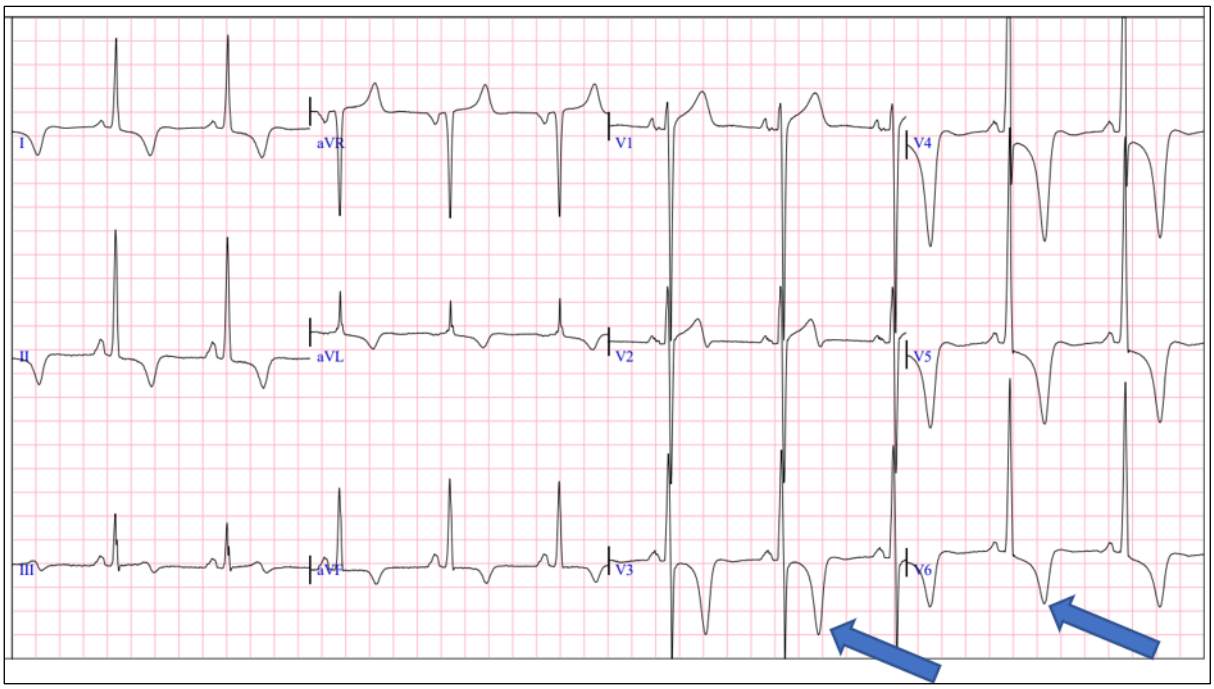

Figure 1: 12-lead ECG demonstrating deep symmetric T-wave inversions in leads V3–V6 exceeding 10 mm in depth, characteristic of apical hypertrophic cardiomyopathy

3.7.1 Echocardiographic Imaging

In this context, the integration of cardiac magnetic resonance imaging is essential to overcome the inherent constraints of echocardiographic visualization, particularly when defining segmental wall thickness and identifying apical aneurysms[76], [77]. However, in resource-constrained environments where cardiac magnetic resonance imaging remains inaccessible, practitioners should maximize the diagnostic potential of transthoracic echocardiography by utilizing contrast-enhanced agents to achieve definitive endocardial border definition[5], [65]. Complementary techniques, including the adoption of specialized off-axis views and speckle-tracking echocardiography, have demonstrated utility in revealing the pathognomonic apical structural nuances that conventional imaging often overlooks [78], [79].

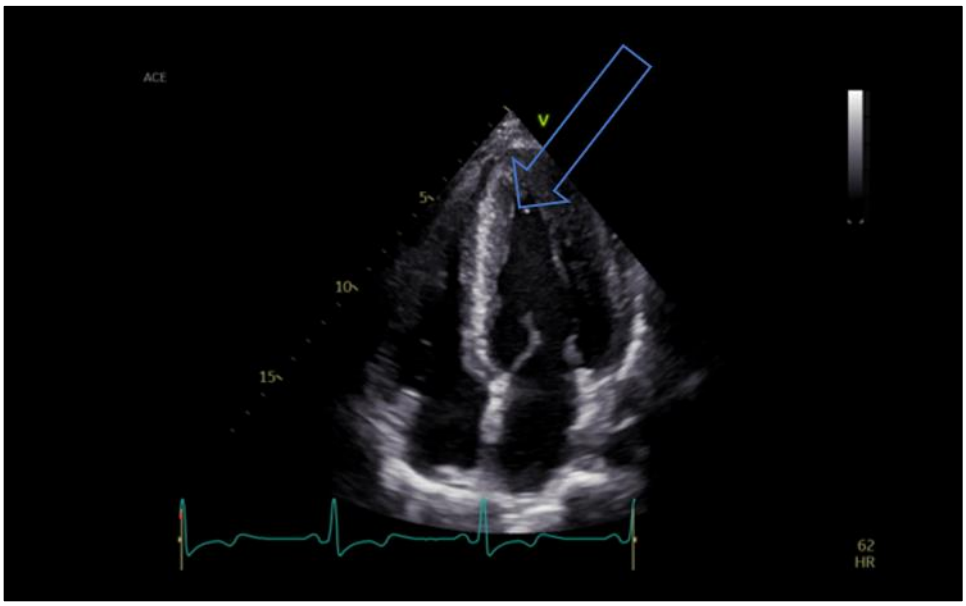

Figure 2: 4-chamber view, demonstrating systolic cavity obliteration at the LV apex, with a "spade-like" configuration.

Consequently, a heightened diagnostic index of suspicion combined with these focused echocardiographic refinements remains paramount for identifying the apical phenotype, especially when the characteristic "ace of spades"

configuration is not immediately apparent. Furthermore, clinicians should routinely assess for supportive criteria, such as apical cavity obliteration greater than 20 mm or left atrial dilatation, to facilitate early detection of non-spade phenotypes that may represent precursors to the classic anatomical presentation.[80] Figure 2 illustrates a representative contrast-enhanced echocardiogram demonstrating this appearance.

3.7.2 Cardiac Magnetic Resonance

As the gold standard for structural characterization, CMR provides superior tissue characterization through late gadolinium enhancement, which is crucial for identifying myocardial fibrosis an independent risk factor for adverse outcomes and sudden cardiac death [17]. Furthermore, CMR is instrumental in diagnosing apical ventricular aneurysms that may be missed by echocardiography, which are frequently composed of replacement fibrosis and represent a high-risk nidus for ventricular tachyarrhythmias[14], [20]. Beyond morphological assessment, having a good modality will give a precise quantification of left ventricular volumes and apical wall thickness.[72], [81] Representative CMR images demonstrating this apical pattern are shown in Figure 3.

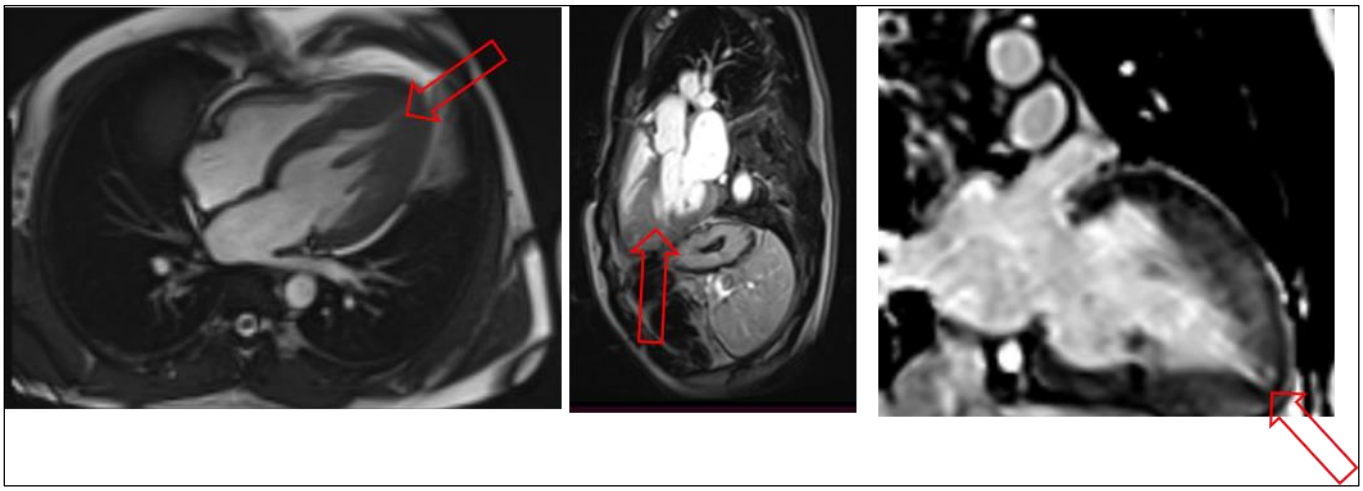

Figure 3: (A) Cardiac MRI, 4-chamber magnitude image (early gadolinium enhancement sequence) demonstrating marked apical myocardial hypertrophy with near-obliteration of the left ventricular apical cavity, producing the characteristic "ace-of-spades" configuration. Basal and mid-ventricular segments are relatively spared. (B) Three-chamber magnitude image confirming disproportionate apical wall thickening relative to the basal septum and LVOT, without evidence of dynamic outflow tract obstruction. (C) image showing patchy mid-wall hyperenhancement localized to the hypertrophied apical segment, consistent with focal myocardial fibrosis a recognized substrate for ventricular arrhythmia in ApHCM.

3.7.3 Diagnostic Challenges of Apical Hypertrophic Cardiomyopathy in West African Populations

In west Africa, diagnostic burden is extensive: often faced with inaccessible imaging in scenarios where echocardiographic resolution remains insufficient. To further enhance diagnostic efficacy, it is important to establish structured clinical pathways that systematically incorporate accessible tools, such as focused speckle-tracking and comprehensive ECG analysis [19], [82] alongside the development of regional registries to better capture the unique phenotypic and genetic nuances of ApHCM in West African populations. Furthermore, these efforts must be supported by multi-center collaborations aimed at optimizing referral systems and standardizing diagnostic protocols to bridge the persistent gap in early cardiomyopathy identification and personalized management.

Despite its clinical importance, the identification of ApHCM in West African populations is frequently compromised by an over-reliance on conventional echocardiography, which often fails to capture the nuanced apical thinning and morphological changes required for definitive diagnosis [5]. This diagnostic bottleneck is exacerbated by the frequent misattribution of apical hypertrophy to hypertensive heart disease, given the high regional prevalence of hypertension, combined with the limited accessibility of confirmatory genetic testing and advanced cardiac magnetic resonance imaging [33], [40], [83]. To mitigate these systematic challenges, leveraging large-scale, digitized cardiovascular datasets through automated interpretation of phenotypic patterns offers a promising framework for clinician decision support, potentially addressing these deep-seated disparities in the diagnosis and risk stratification of cardiomyopathies across the region. Furthermore, fostering institutional partnerships to integrate emerging technologies like echocardiography-based deep learning could provide cost-effective alternatives to late gadolinium enhancement, which remains largely unavailable for fibrosis quantification in this region [84]. Such interventions are essential, as the current diagnostic ambiguity surrounding cardiomyopathies in low-resource environments is further compounded by overlapping infectious diseases and undiagnosed genetic drivers [85].

Consequently, establishing specialized tertiary centers equipped with both advanced imaging modalities and expert personnel is vital to transition from symptomatic management to precision-based therapeutic interventions. These specialized centers must also champion the standardization of longitudinal follow-up protocols, as current gaps in data collection frequently obscure the long-term clinical trajectory of ApHCM patients within these diverse, underserved populations [86], [87].

3.7.4 Complications of Apical Hypertrophic Cardiomyopathy

3.7.4.1 Cardiac Arrhythmias and Sudden Cardiac Death

Cardiac arrhythmias are among the most frequently reported complications of ApHCM. Patients may experience palpitations due to atrial or ventricular arrhythmias. In a reported case by Jordan et al., the patient presented with palpitations and exertional dyspnea, highlighting the arrhythmogenic potential of the disease[88]. Some other complications are ventricular tachycardia and ventricular fibrillation which occurs in high-risk individuals and can predispose to sudden cardiac death (SCD). Atrial fibrillation may also develop secondary to left atrial enlargement and diastolic dysfunction, increasing the risk of thromboembolic events. The authors further noted that some patients with ApHCM develop arrhythmias severe enough to require antiarrhythmic therapy or implantation of a cardioverter-defibrillator [88].

Cardiac arrhythmias represent one of the most important complications of apical hypertrophic cardiomyopathy (ApHCM) and may contribute to sudden cardiac death in susceptible individuals. Although the exact mechanisms remain incompletely understood, progressive myocardial fibrosis and structural remodeling appear to play central roles in arrhythmogenesis. Jeong et al. demonstrated a significant association between abnormal circumferential transmural strain difference (cTSD) and late gadolinium enhancement, suggesting that myocardial fibrosis contributes to abnormal markers in ApHCM [89]. However, Fibrotic tissue and myocyte disarray can disrupt normal electrical conduction pathways, creating regions of conduction delay and electrical heterogeneity that predispose patients to atrial and ventricular arrhythmias[90], while progressive myocardial remodeling creates an arrhythmogenic substrate that may culminate in ventricular tachyarrhythmias and sudden cardiac death.

3.7.4.2 Heart Failure

Heart failure may develop because of progressive myocardial hypertrophy, impaired ventricular relaxation, and diastolic dysfunction. Although most patients initially presented in New York Heart Association (NYHA) functional classes I and II, heart failure remained an important cause of mortality during follow-up. Stringer and colleagues have reported that Heart failure is another recognized complication of ApHCM and is primarily related to progressive myocardial hypertrophy and impaired ventricular relaxation [91] with a secondary cause as AF [92]. These causes can be further revealed with early imaging and anticoagulation medications.

Yin et al., In a long-term cohort study, suggested that progressive heart failure was among the major adverse clinical events, with age ≥55 years, increased left atrial volume index, reduced systolic mitral annular velocity (S'), non-sustained ventricular tachycardia, and late gadolinium enhancement identified as independent predictors of poor prognosis [93].

3.7.4.3 Thrombolic complications and Stroke

In differentiation of ApHCM cerebrovascular embolic events might occur, which are not sole backed by sufficient data but should put into consideration [94]. ApHCM patients are at increased risk of thromboembolic complications, particularly when atrial fibrillation develops. In a cohort study, thromboembolic complications, including ischemic stroke, may occur in patients with ApHCM, particularly in the presence of atrial fibrillation [91]. Progressive myocardial remodeling and fibrosis contribute to left atrial enlargement and abnormalities in atrial electrical conduction, thereby increasing the likelihood of atrial fibrillation. The resulting blood stasis within the atria promotes thrombus formation, which may subsequently embolize to the cerebral circulation and cause ischemic stroke. Therefore, structural, and functional myocardial abnormalities not only contribute to arrhythmogenesis but may also indirectly increase the risk of thromboembolic complications [91].

3.7.4.4 Imaging pitfall

A critical diagnostic pitfall in this clinical landscape involves the clinician's tendency to prioritize exclusionary testing, such as repeated coronary angiography to rule out acute coronary syndrome, rather than proactively pursuing definitive structural assessment when apical hypertrophy is suspected [69], [95]. This reactive approach often delays the recognition of ApHCM, as practitioners may dismiss minor echocardiographic irregularities as benign consequences of chronic hypertension, thereby missing the opportunity to intervene before the development of progressive myocardial fibrosis [67].

Consequently, patients frequently undergo multiple unnecessary evaluations that fail to address the underlying structural pathology, ultimately increasing the burden of morbidity associated with this condition in resource-constrained environments. Moreover, the failure to identify apical aneurysms which are missed by up to 40% of echocardiograms but detectable via cardiac magnetic resonance precludes timely management of associated thromboembolic risks and lethal ventricular arrhythmias [71]. Furthermore, emerging evidence suggests that integrating explainable advanced electrocardiography with these imaging findings can provide superior diagnostic accuracy for ApHCM, especially in cases where hypertrophy is relative or traditional imaging remains inconclusive [96].

3.8 Follow-Up Variations in Apical Hypertrophic Cardiomyopathy (ApHCM)

The natural history of ApHCM is far from uniform due to morphological, arrhythmic, thromboembolic, fibrotic, and outcomes. In the event of ApHCM the follow up protocol has not been fully established.

Table 2: Follow-Up Periods, Type of Apical Hypertrophic Cardiomyopathy, Imaging Modality, Outcomes, and Follow-Up Variation

S/N	Author(s) / Year	Follow-Up Period	Type of ApHCM	Modality Used	Outcome(s)	Follow-Up Variation
1	Kim[23]	**Median 37 months** (Prospective)	Pure Apical (15.3%) Septal-Apical (9.1%) Concentric (17.9%) ASH (16.8%)	CMR (cine + LGE) TTE (contrast) Tissue Doppler	AAn prevalence 14.3% by CMR Adverse events (cardiac death, HF, CVA) similar with/without AAn at 37 mo AAn likely a long-range problem	**Apical Aneurysm Formation** Underdetected by echo; CMR prevalence 3–5%; 6-fold ↑ thromboembolic risk
2	**Rubiś et al.**[97]	**Mean 12.3 months ± 1.4** (Prospective longitudinal)	All HCM phenotypes; apical segments specifically assessed	Serial CMR (T1 mapping, T2, ECV, LGE, post-contrast T1)	Global fibrosis stable over 1 yr Significant ECV↑ specifically in apical segments Apical fibrosis progresses earlier and preferentially	**Progressive Apical Fibrosis** Interstitial fibrosis progresses preferentially in apical segments over 1 year
3	**Raza et al.**[98]	**3 months** (Anticoagulation course) Case Report	Pure ApHCM with apical aneurysm	TTE TEE Cardiac CT angiography CMR (LGE) PET-CT	Giant 4.8×3.4 cm LV thrombus in sinus rhythm. Initial suspicion of cardiac malignancy Near-complete resolution after 3 months warfarin.	**LV Thrombus Formation** Can occur in sinus rhythm; may mimic cancer; responds to anticoagulation
4	**Rana et al.**[99]	**1 year** (Anticoagulation trial) Case Report	ApHCM / EMF overlap (47-yr-old female)	TTE (Definity contrast) CMR (LGE, subendocardia	Apical thrombus persisted after 1-year apixaban	**Endomyocardial Fibrosis Overlap** Particularly problematic in

				l enhancement) Stress TTE	Persistent thrombus on CMR diagnosis revised to EMF LV thrombus did not resolve: key differentiator from ApHCM	African populations
5	**Shah et al.**[100]	**In-hospital only** (Case Report)	Pure ApHCM / Yamaguchi Syndrome in non-Asian (Hispanic) patient	TTE (perflutren contrast) Left heart catheterisation 12-lead ECG	Admitted as NSTEMI; no obstructive CAD on catheterisation Ace-of-spades LV morphology confirmed EKG: T-wave inversions I, II, V2–V6 misread as ACS	**ACS Mimicry at Follow-Up** Giant T-wave inversions misinterpreted as ischemia at repeat presentations
6	**Wicaksono et al.**[101]	**Short-term in-hospital only** (Case Report)	Pure ApHCM / Yamaguchi Syndrome (64-yr male)	TTE (apical 4-ch, PLAX, 3-ch) CMR (cine + LGE) Coronary angiography 12-lead ECG	COVID-19 co-infection elevated hs-TnI diagnosed as AMI DCA: no significant CAD (MINOCA) CMR: apical wall 22 mm, mid-apical obliteration, fibrosis 7.4 g Antiplatelet stopped; conservative management with bisoprolol	**COVID-19 Myocardial Injury** Exacerbates pre-existing ApHCM; mimics AMI
7	**Eltawansy et al.**[75]	**In-hospital only** (Case Report)	Pure ApHCM (91-yr-old female)	TTE (contrast LVO) Left heart catheterisation Telemetry (NSVT monitoring)	Recurrent syncope + NSVT documented on telemetry Left ventriculogram: spade-like cavity, no LVOT gradient Dual-chamber ICD implanted for secondary prevention Metoprolol succinate initiated	**Ventricular Tachycardia / SCD** Predominantly monomorphic VT from scar reentry; SCD rate 1.8–4.7%/year with aneurysm

8	**Tong &** Bilgrami [30]	**Not reported** (Retrospective 2011–2017)	Pure ApHCM 38.5% Asymmetric Septal HCM 41% Focal/Concentric 20.5%	CMR (LGE, 1.5T/3T SSFP) TTE Holter monitoring EP testing (selected cases)	Myocardial fibrosis more prevalent in ApHCM (83.3%) vs ASH (60%) on CMR No SCD outcomes data by subtype 51.3% patients asymptomatic at diagnosis Palpitations most common symptom (71.8%)	**Progressive Apical Fibrosis** Interstitial fibrosis progresses preferentially in apical segments over 1 year
9	**Hussain**[102]	**Planned annual TTE +** Holter Case Report	ApHCM + Subaortic Membrane (SAS co-existing)	TTE CMR (LGE, LVOT flow velocity) 12-lead ECG Holter (planned)	Coexistence of ApHCM + SAS in 1.7% of HCM cases CMR: no LGE; EF 67%; RV EF 54%; flow acceleration at LVOT 2.1 m/s Conservative management: metoprolol + verapamil 180 mg/day Surgical SAS resection deferred pending gradient >50 mmHg	**ACS Mimicry at Follow-Up** Giant T-wave inversions misinterpreted as ischemia at repeat presentations
10	**Dzirlo et al.**[103]	**3 months** (Cardiomyopathy recovery) Case Report	Transient apical wall thickening (TAWT);Takotsubo mimic	TTE (serial) CMR (T2 STIR, LGE)	Complete regression of TAWT at 3 months Absence of LGE + resolution distinguishes from true ApHCM T2 signal elevation resolved; no fibrosis residue	**Transient Apical Thickening** Can mimic ApHCM during recovery from Takotsubo cardiomyopathy

ApHCM = apical hypertrophic cardiomyopathy; CMR = cardiac magnetic resonance; LGE = late gadolinium enhancement; ECV = extracellular volume fraction; TTE = transthoracic echocardiography; TEE = transesophageal echocardiography; SCD = sudden cardiac death; ICD = implantable cardioverter-defibrillator; VT = ventricular tachycardia; NSVT = non-sustained VT; ASH = asymmetric septal hypertrophy; AAn = apical aneurysm; EMF = endomyocardial fibrosis; HF = heart failure; CVA = cerebrovascular accident; LVOT = left ventricular outflow tract; SAS = subaortic membrane stenosis; MINOCA = myocardial infarction with non-obstructive coronary arteries

4 DISCUSSION

The evidence synthesized in this review suggests that the major challenge in recognizing apical hypertrophic cardiomyopathy (ApHCM) in West Africa may be related not only to the underlying frequency of the disease but also to

limitations in its detection and phenotypic characterization[92]. ApHCM is characterized predominantly by hypertrophy of the left ventricular apical segments and can be difficult to identify when the apex is inadequately visualized on routine transthoracic echocardiography (TTE) [104]. Contemporary reviews describe ApHCM as an under-recognized phenotype and emphasize that apical foreshortening, poor endocardial definition, and limited acoustic windows can lead to diagnostic error. This limitation is particularly important in resource-constrained settings where TTE may represent the principal cardiac imaging modality.

In a study specifically examining patients with ApHCM, Moon and coworkers. found that cardiac magnetic resonance (CMR) detected apical pouches in 17 of 31 patients (54.8%), whereas echocardiography correctly identified only 47.1% of the apical pouches detected by CMR. CMR also identified two apical thrombi that were not detected by echocardiography. Importantly, the authors found that the addition of contrast to echocardiography improved diagnostic yield [22]. These findings provide a plausible explanation for why reliance on conventional echocardiography alone may result in incomplete recognition of the ApHCM phenotype. However, the evidence does not support viewing CMR as simply a replacement for echocardiography. Rather, the two modalities provide complementary information. TTE remains the first-line imaging investigation because of its accessibility, portability and ability to assess cardiac morphology and function in real time. The 2024 AHA/ACC guideline recommends CMR when echocardiography is inconclusive, when an alternative diagnosis is suspected, and when additional assessment of apical aneurysm, ventricular function or myocardial fibrosis is required [50]. herefore, in West African settings where universal access to CMR is unrealistic, an important objective should be to improve the diagnostic yield of the echocardiographic examinations that are already available while establishing referral pathways for patients requiring CMR.

The importance of this approach is illustrated particularly well by the detection of left ventricular apical aneurysms. Apical aneurysm represents an important adverse phenotype in HCM because it is associated with ventricular arrhythmias, thromboembolic complications, and other adverse cardiovascular outcomes. In a large CMR-based cohort of 1,332 patients with ApHCM, Yang et al. identified left ventricular apical aneurysms in 2.3% of patients and demonstrated their association with adverse clinical characteristics[105]. Furthermore, the 2024 AHA/ACC guideline specifically identifies LV apical aneurysm as an established clinical risk factor that should be considered during sudden cardiac death risk assessment [50].

The difficulty of detecting apical aneurysms using conventional echocardiography further emphasizes the importance of optimizing imaging techniques. In a comparative study of patients with HCM, conventional non-contrast echocardiography had substantially lower sensitivity for detecting LV apical aneurysms than contrast echocardiography. Contrast echocardiography demonstrated a sensitivity of approximately 98%, comparable to CMR after expert image review, whereas non-contrast echocardiography had a sensitivity of approximately 67%[106]. These findings have particular relevance for West Africa because they suggest that some of the diagnostic limitations attributed to the absence of CMR may be partially mitigated by relatively accessible modifications to echocardiographic practice, particularly contrast-enhanced LV opacification and careful acquisition of dedicated apical views, The potential diagnostic value of ECG should also be considered within this resource-adapted framework. Deep or giant T-wave inversion is a well-recognized electrocardiographic feature of the apical phenotype and can provide an important clinical clue prompting targeted assessment of the LV apex. Nevertheless, ECG abnormalities should be regarded as a trigger for further investigation rather than as a diagnostic substitute for cardiac imaging. The morphological diagnosis of ApHCM requires demonstration of the characteristic apical hypertrophy, and echocardiography or CMR remains necessary for phenotypic confirmation[2], [92]

An important implication of these findings for West Africa is therefore the development of a tiered diagnostic strategy rather than an expectation that all suspected cases undergo CMR. Patients with suggestive clinical or ECG findings could first undergo carefully performed TTE with dedicated visualization of the apex. Where endocardial definition is inadequate, contrast echocardiography should be considered. Patients with equivocal findings, suspected apical aneurysm or pouch, suspected alternative myocardial disease, or a need for tissue characterization and fibrosis assessment should subsequently be referred for CMR. This approach is consistent with contemporary HCM guidance, which positions CMR as an important complementary investigation when echocardiography is inconclusive or when additional phenotypic and risk information is required [50].

This distinction is particularly important when interpreting the apparently low prevalence of ApHCM reported in African cohorts. A low observed prevalence should not automatically be interpreted as evidence that the phenotype is intrinsically uncommon in African populations (Table 2). At the same time, the available evidence is insufficient to conclude that ApHCM is definitively underdiagnosed across West Africa. The more defensible interpretation is that the true epidemiological burden remains uncertain because the available African evidence is limited and diagnostic ascertainment may vary according to imaging availability, image quality, diagnostic expertise and access to CMR. This represents an important knowledge gap that should be addressed through prospective, multicentre African studies using standardized diagnostic criteria and, where feasible, multimodality imaging.

Although no ApHCM-specific risk stratification algorithm has been validated, high-risk features warrant systematic clinical application: apical aneurysm (conferring a six-fold increase in thromboembolic risk), unexplained syncope, documented non-sustained ventricular tachycardia, LGE burden exceeding 15% of LV mass, and progressive left atrial dilatation. The 2024 AHA/ACC guidelines endorse shared decision-making for ICD implantation in patients with one or more major risk factors, which are applicable to patients with ApHCM who have apical aneurysms or documented ventricular arrhythmias[67]. In settings where CMR is unavailable, non-sustained VT on ambulatory recording should serve as a mandatory trigger for CMR referral. Anticoagulation should be initiated in all ApHCM patients with apical aneurysm and atrial fibrillation, and considered strongly in those with aneurysm in sinus rhythm given the thromboembolic case data [56].

4.1 Recommendation and Future route

- Future research efforts should prioritize longitudinal cohorts to better delineate the prognostic significance of these structural abnormalities, potentially offering a more cost-effective framework for risk stratification that circumvents the current reliance on inaccessible diagnostic modalities.
- For a Nigeria setting, the successful implementation of the PEACE registry by Karaye, demonstrates the feasibility of large multicenter cardiomyopathy research in Nigeria [107]. However, comparable data for ApHCM are lacking across Nigeria and West Africa. Future research should focus on establishing regional ApHCM registries to determine disease prevalence, demographic and clinical characteristics, imaging phenotypes, genetic profiles, and long-term outcomes. Prospective studies incorporating echocardiography, cardiac magnetic resonance imaging, and genomic analyses would help clarify the burden of disease, identify population-specific risk factors, and support the development of context-specific diagnostic and management strategies for the region [107]. In a study to come we propose to study a single private hospital system in Nigeria to observe the prevalence.
- Another aspect is the machine learning algorithm which has come to stay. By Integrating machine learning algorithms and for risk stratification could provide a scalable solution by identifying subtle, non-linear patterns in myocardial architecture that indicate higher risk profiles [2], [108], [109]. Such computational frameworks, by leveraging features like T1 mapping and ECV fraction, might eventually mitigate existing technical constraints and enhance the systematic assessment of diffuse fibrosis even in centers with limited cardiovascular magnetic resonance expertise [110].

4.2 Limitation

The present study as a narrative and synthesis is susceptible to selection bias and does not employ formal quality appraisal tools such as GRADE; findings represent qualitative syntheses rather than definitive effect estimates. However, West African evidence on ApHCM is critically limited, consisting predominantly of case reports and small observational series none of which constitute population-level prevalence data constraining the generalisability of epidemiological claims. Also, restriction of the search to English-language publications may have excluded relevant Language studies. However, the rapidly evolving landscape of CMR feature-tracking, machine learning phenotyping, and guideline updates means that some recommendations may be superseded by emerging evidence. Finally, the absence of primary institutional data from the authoring institution limits the paper's original clinical observations.

5 CONCLUSION

ApHCM remains an underappreciated and diagnostically challenging variant of HCM in West Africa, where infrastructural constraints and phenotypic overlap with endomyocardial fibrosis and hypertensive heart disease compound the risk of misdiagnosis. While its natural history is generally more favourable than other HCM subtypes, patients who develop apical aneurysms, progressive fibrosis, or ventricular arrhythmias carry substantial morbidity and mortality risk demanding timely identification.

Clinicians across Nigeria must maintain a heightened index of suspicion, leveraging accessible tools particularly giant T-wave inversion recognition on ECG, contrast echocardiography, and speckle-tracking strain analysis while advocating for greater CMR access through regional collaborations. The establishment of dedicated regional registries modelled on the PEACE registry experience, adaptation of international guidelines to the West African context, and integration of scalable machine learning-driven phenotyping represent the most impactful strategies to reduce the diagnostic burden and improve long-term outcomes for this neglected patient population. Closing the ApHCM diagnostic gap in Nigeria is not merely a technical challenge but a health equity imperative demanding coordinated institutional, governmental, and international commitment.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflict of interest.

Author Contributions

T.M. contributed to the conception and design of the Manuscript, performed the literature review, and critically revised the manuscript for important intellectual content. M.C.N. was responsible for data acquisition of literature papers, drafted the initial manuscript, and coordinated revisions based on co-author feedback and contributed to supervision of the overall preparation of the Review.

All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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