Ethnic differences in ventricular hypertrabeculation on cardiac MRI in elite football players

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Abstract

Purpose Left ventricular (LV) trabeculation may be more pronounced in ethnic African than in Caucasian (European) athletes, leading to possible incorrect diagnosis of left ventricular non-compaction cardiomyopathy (LVNC). This study investigates ethnic differences in LV hypertrabeculation amongst elite athletes with cardiac magnetic resonance (CMR) and electrocardiography (ECG).

Methods 38 elite male football (soccer) players (mean age 23.0, range 19–34 years, 28/38 European, 10/38 African)

underwent CMR and ECG. Hypertrabeculation was assessed using the ratio of non-compacted to compacted myocardium (NC/C ratio) on long-axis and short-axis segments. ECGs were systematically rated.

Results No significant differences were seen in ventricular volumes, wall mass or E/A ratio, whereas biventricular ejection fraction (EF) was significantly lower in African athletes (European/African athletes LVEF 55/50 %, $p=0.02$; RVEF 51/48 %, $p=0.05$). Average NC/C ratio was greater in African athletes but only significantly at mid-ventricular level (European/African athletes: apical 0.91/1.00, $p=0.65$; mid-ventricular 0.89/1.45, $p<0.05$; basal 0.40/0.46, $p=0.67$). ECG readings demonstrated no significant group differences, and no correlation between ECG anomalies and hypertrabeculation.

Conclusions A greater degree of LV hypertrabeculation is seen in healthy African athletes, combined with biventricular EF reduction at rest. Recognition of this phenomenon is necessary to avoid misdiagnosis of LVNC.

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Introduction

The hearts of healthy athletes of African descent demonstrate various features overlapping with cardiac pathology. More abnormal ECG patterns are observed than in European athletes, most frequently left ventricular (LV) hypertrophy, early repolarisation, ST-segment elevation and T-wave inversions [1–4]. On average, LV mass is greater, and wall thickness more often exceeds generally accepted cut-offs for hypertrophy in African athletes [1, 5, 6]. Whilst this normal ethnic variation may cause unjustified diagnosis of cardiomyopathy

leading to exclusion from sports [7], sudden cardiac death attributed to hypertrophic cardiomyopathy is also more frequent in African athletes [8]. Moreover, recent studies indicate that ethnicity may be an important determinant of hypertrabeculation or non-compaction, with more pronounced LV trabeculation in African individuals [9–11]. This may lead to misdiagnosis of left ventricular non-compaction cardiomyopathy (LVNC) [7]. LVNC, characterised by multiple trabeculations and intertrabecular recesses, is associated with arrhythmias, sudden cardiac death, heart failure, and thromboembolic events [12]. In the absence of clear diagnostic criteria, LVNC is usually diagnosed by measuring the ratio of non-compacted to compacted myocardium with either echocardiography or cardiac magnetic resonance (CMR) [13, 14]. As intensive physical training is associated with morphological and functional cardiac changes [15, 16], the differentiation with cardiac pathology is already challenging [17]. Since the majority of available data are based on European athletes, imaging investigations in both clinical and pre-participation screening settings are more likely to be misinterpreted in African athletes [6, 7].

The aim of this study is to investigate the CMR and ECG differences in a group of African and European elite football (soccer) players focusing on the presence of hypertrabeculation.

Methods

Ethics statement

All subjects gave written informed consent and the study was approved by the medical ethics committee of the University Medical Center Utrecht.

Study population

Thirty-eight elite male football players were included (mean 23.0, range 19–34 years): 28 of Caucasian (European) ethnicity, and 10 of sub-Sahara African descent. All subjects played in the highest division of Dutch football (all the players of the first and second team of a single football club) and trained for a minimum of 9 h a week. None of the subjects' clinical records (football club's sports physician/sports cardiologist) showed evidence of hypertension or cardiovascular, pulmonary or metabolic disease and none reported any related signs or symptoms on a questionnaire completed for this study.

Study protocol

CMR imaging was performed with a 1.5 T MRI scanner (Achieva, Philips, Best, the Netherlands). The protocol

included ECG-gated breathhold steady-state free precession (SSFP) cine images (2-chamber LV and right ventricle (RV), 4-chamber, LV and RV outflow tract and short-axis) and quantitative flow measurements of the valves [15]. The short axis covered both ventricles completely using 10 mm-slice breathhold cines without interslice gap, 50 frames per cardiac cycle, matrix 256×256 and field of view 350–400 mm.

All subjects were interviewed, completed a questionnaire on training history, past medical and family history and underwent blood pressure, height and weight measurement, followed by a 12-lead electrocardiography immediately prior to the CMR.

CMR analysis

One of two blinded observers, experienced in a robust contour tracing protocol [18], traced endocardial and epicardial contours of both ventricles on a workstation (View Forum cardiac package version R5.1V1L2.SP3, Philips, Best, the Netherlands). All results were checked by an experienced blinded observer. End-diastolic (ED) and end-systolic (ES) endocardial contours were used to calculate end-diastolic (EDV) and end-systolic (ESV) volumes, ejection fraction (EF), stroke volume (SV) and ED wall mass. LV and RV outflow tracts (LVOT/RVOT) were included in the endocardial borders. Both papillary muscles and trabeculae were excluded from the endocardial contours and included in the blood volume [18]. End-diastolic ventricular diameters and septal wall thickness were measured on the short-axis images. An experienced observer analysed flow velocity measurements over the mitral and tricuspid valves, for calculation of early (E peak) and late (A peak) diastolic filling velocities. E/A ratios were used as an indirect measure of diastolic function [19].

Assessment of hypertrabeculation

Ratios of non-compacted to compacted myocardium (NC/C ratio) were measured in the end-diastolic phase on 16 short-axis segments (American Heart Association) to obtain segmental information on the presence and distribution of hypertrabeculation (Fig. 1) [13, 14]. Image slices with measurements were crosschecked with the 4-chamber image to ensure appropriate representation of the apical, mid-ventricular and basal levels. The first slice showing a clear ventricular lumen was considered the apical level. The mid-ventricular level was positioned just apical to the papillary muscle insertion to avoid measuring the papillary muscle as part of the hypertrabeculation, whereas the basal level was positioned just basal to the papillary muscle, defined as the first slice not showing the papillary muscle. Two observers first measured 10 subjects for training purposes. Measurements were repeated in a randomised

Fig. 1 Cardiac segmentation on CMR short-axis view. Short-axis midventricular view illustrating borders (dotted lines) of AHA segments. The ratios of non-compacted (trabeculation) and compacted myocardium (myocardial wall) were measured in the mid-portion of each segment. At the apical level there was only one measurement on the lateral and septal aspect

fashion in these subjects with at least a 1-week interval to calculate interobserver variability. The remaining subjects were subsequently measured by one observer.

The short-axis measurements were also related to long-axis view measurements, as described by Petersen et al. [14]. Maximum NC/C ratio was measured in the end-diastolic phase on three long-axis images (2-chamber, 4-chamber and LVOT) perpendicular to the compacted myocardium and excluding the apex (segment 17). The maximum NC/C ratio of these three long-axis measurements was used for analysis. Subsequently, the correlation of maximum NC/C ratio of long-axis and short-axis images was calculated.

ECG analysis

ECG analysis was performed by three experienced sports cardiologists using a web-based programme, according to the Lausanne Criteria for ECG evaluation (as recommended by study group of sport cardiology of the European Society of Cardiology, see Table 1 for specifications) [20]. Discrepancies were solved by consensus decision.

Statistical analysis

Data are presented as the mean $\pm$ standard deviation. CMR volumes and wall mass were indexed to body surface area (BSA), with BSA calculated using the Du Bois and Du Bois

Table 1 ECG parameters and consensus ratings

	European		African	
Quantitative parameters	<i>n</i> =28		<i>n</i> =10	
Heart rate (bpm)	55 $\pm$ 9.4		53 $\pm$ 11	
PQ interval (ms)	161 $\pm$ 21		171 $\pm$ 31	
QRS duration (ms)	100 $\pm$ 13		98 $\pm$ 9.2	
QT interval (ms)	411 $\pm$ 27		400 $\pm$ 13	
QTc ^a	392 $\pm$ 27		375 $\pm$ 37	
Qualitative parameters	%	n	%	n
Overall judgment: abnormal ECG?	43	12	40	4
ST segment elevation ^b	36	10	40	4
LV hypertrophy ^c	29	8	50	5
Early repolarisation	21	6	20	2
First-degree AV block/AV dissociation	14	4	20	2
T-peak abnormalities	11	3	20	2
RBBB ^d	7	2	0	0
Fractionated QRS complex	7	2	0	0
Heart rhythm	4	1	0	0
LA enlargement	4	1	0	0
Other anomalies*	0	0	0	0

AV atrioventricular; RBBB right bundle branch block; LA left atrial; LV left ventricular

^a QTc, corrected QT interval according to Bazett's formula: $QT/\sqrt{RR}$

^b ST-segment elevation [25]: ≥ 1 mm ST elevation in I/II/III/aVL/aVF/V5/V6 or ≥ 2 mm in V1/V2/V3/V4 at 0.06–0.08 s from J-point

^c LV hypertrophy [25]: R wave in aVL ≥ 1.1 mV or sum amplitude of ≥ 3.5 mV in V1 and either V5 or V6 [23]

^d RBBB: >120 ms

*heart axis abnormality (deviating from -30 to 120° range), right atrial enlargement, RV hypertrophy, left bundle branch block (>120 ms), left anterior hemiblock, extrasystole, ST-segment depression, Wolff-Parkinson-White syndrome, Brugada syndrome, pericarditis, microvoltages, epsilon wave, pre-existing infarction

formula [21]. Differences between groups were assessed using Student's *t*-test and adjusted for multiple testing using Bonferroni post-hoc correction. All tests were two-sided and a *p*-value of 0.05 was considered statistically significant. The interrater reliability of average NC/C ratios for each ventricular level was investigated using the two-way intra-class coefficient (ICC), calculated over the second series of measurements of each observer [22].

Results

Baseline characteristics of all subjects show no significant differences between groups (Table 2). Table 3 shows the cardiac dimensions and function measurements for both ventricles. Ventricular diameters, volumes and wall mass were in the same range for all subjects. Although no signs

Table 2 Baseline characteristics

	European	African	<i>p</i> -value
n	28	10	
Age (years)	23±4.6	23±2.8	NS
Height (m)	183±5.9	179±4.8	NS
Weight (kg)	78±8.3	77±6.5	NS
BSA (m ²)	2.00±0.13	1.95±0.10	NS
SBP (mmHg)	125±11	128±7.0	NS
DBP (mmHg)	67±8.5	74±5.5	NS
Resting heart rate	54±9.3	52±9.9	NS
Training/week (hours)	16±4.4	14±2.8	NS

mean ± SD; *BSA* body surface area; *SBP/DBP* systolic/diastolic blood pressure; *NS* non-significant at 5 % significance level and after Bonferroni correction for multiple testing

of diastolic dysfunction were found, as illustrated indirectly by equal E/A ratios, biventricular resting EF was lower in African athletes (European/African athletes LV EF 55/50 %, RV EF 51/48 %). The mean interventricular septal wall thickness was marginally greater in African athletes (European/African athletes 9.1/9.7 mm), with none exceeding 12 mm.

The ICC (intrater reliability) for average short-axis NC/C ratio measurements was 0.695 for the apical, 0.822 for the mid-ventricular, and 0.936 for the average basal NC/C ratio,

Table 3 Quantitative ventricular parameters adjusted for body surface area

	European	African	<i>p</i> -value
n	28	10	
LV			
EDV index (ml/m ²)	125±17	120±11	NS
ESV index (ml/m ²)	57±11	61±9.6	NS
EDM index (ml/m ²)	55±8.2	56±6.3	NS
EDD SA (mm)	61±4.5	60±4.3	NS
EF (%)	55±5.8	49±4.8	0.02
MV E/A ratio	2.1±0.40	2.1±0.74	NS
RV			
EDV index (ml/m ²)	133±19	127±15	NS
ESV index (ml/m ²)	65±13	67±13	NS
EDM index (ml/m ²)	13±2.0	13±1.7	NS
EDD SA (mm)	51±5.1	47±6.9	NS
EF (%)	51±5.2	48±4.2	0.05
TV E/A ratio	1.6±0.27	1.6±0.49	NS
Septal wall SA (mm)	9.1±1.0	9.7±1.1	NS

mean ± SD; *EDV* end-diastolic volume; *ESV* end-systolic volume; *EDM* end-diastolic mass; *EDD* end-diastolic diameter; *SA* short-axis view; *EF* ejection fraction; *MV/TV* mitral/tricuspid valve; *NS* non-significant at 5 % significance level and after Bonferroni correction for multiple testing

with an ICC >0.80 generally considered to indicate strong agreement. NC/C ratios (Fig. 2) more often exceeded the clinical CMR non-compaction cut-off value of 2.3 in African (short/long-axis measurements 60/50 %) than in European football players (short/long-axis measurements 18/11 %). The percentages of European/African athletes exceeding an NC/C ratio of 2.3 in at least one segment (short-axis measurements) by ventricular level were: 4/10 % (apical), 11/50 % (midventricular) and 4/10 % (basal). Although the mean NC/C ratios were higher in African athletes in almost all segments, differences were most pronounced at the mid-ventricular level (European/African athletes 0.89/1.45, *p*<0.05) and small at the apical and basal level (European/African athletes: apical 0.91/1.00, *p*=0.65; basal 0.40/0.46, *p*=0.67) (Fig. 3). The maximum NC/C ratio between the short-axis and long-axis view measurements correlated very well (*r*=0.86).

ECG analysis demonstrated no significant relevant differences between the two groups (Table 1). The most frequently observed anomalies were ST-segment elevation (European/African athletes 36/40 %), LV hypertrophy (European/African athletes 29/50 %), early repolarisation (European/African athletes 21/20 %) and T-peak abnormalities (European/African athletes 11/20 %). Observed ECG anomalies were not significantly correlated to the extent of hypertrabeculation as measured by the mean mid-ventricular NC/C ratio.

Discussion

This study, investigating CMR differences in cardiac morphology and function amongst healthy African and European athletes, demonstrates more myocardial hypertrabeculation combined with reduced biventricular EF in African athletes. It is important for clinicians to realise that the combination of a mildly reduced ejection fraction and hypertrabeculation or non-compaction may be a physiological process in these healthy athletes.

Earlier research has highlighted ethnic differences in both ECG findings and cardiac morphology, more frequently showing LV wall thickness and mass exceeding accepted cut-offs and abnormal ECG patterns in African athletes [1–4, 6]. The higher prevalence of LV hypertrophy in African athletes is clinically relevant since the prevalence of hypertrophic cardiomyopathy (HCM), the leading cause of sudden cardiac death in athletes, is higher in the Afro-Caribbean, both in the general population and in athletes [8, 23]. Moreover, uncertainty remains if left ventricular non-compaction is a distinct cardiomyopathy or merely a phenotype of different underlying diseases, possibly sharing a common pathogenic basis with HCM [24].

Fig. 2 Non-compaction/compaction ratios by ventricular segment

CMR provides a higher spatial resolution than echocardiography and can detect hypertrabeculation better [25]. The recommended end-diastolic (>2.3) NC/C ratio cut-off value for CMR [14] differs from the end-systolic ratio (>2.0) used with echocardiography [26, 27]. Although the cut-off value of >2.3 was originally established using long-axis CMR measurements, the assessment of all short-axis segments allows a better overview of the segmental distribution of hypertrabeculation and correlates well with the long-axis method ($r=0.86$).

Since the trabecular layer of the developing ventricular walls is known to become compacted from base to apex and from septal to lateral wall [28], less trabeculation will be found at the basal level. Petersen et al. observed hypertrabeculation/

non-compaction, defined as presence of two myocardial layers with different degrees of tissue compaction, in 91 %, 78 % and 21 % of healthy non-athletic and athletic volunteers at apical, mid-ventricular and basal levels, respectively [14]. These findings suggest that the apical and basal levels are less suitable for differentiation, since the apex is affected in most cases, whereas the basal level will often be unaffected. Indeed, differences at apical and basal level were small in our study, whereas mid-ventricular measurements demonstrated significant differences.

Previously reported ethnic differences on ECG could not be replicated in this study. The trend of more frequent LV hypertrophy and T-wave abnormalities seen in our African athletes did not reach statistical significance in our relatively

Fig. 3 Variation in myocardial hypertrabeculation. Short-axis CMR views of an athlete with little trabeculation (ABC), and another athlete with more pronounced trabeculation (DEF). Grey lines with arrowheads represent non-compacted layer of myocardium, white lines with arrowheads represent compacted layer of myocardium

small study population, as compared with significant differences in larger study populations [1, 3, 29]. The observed ECG anomalies in trained subjects, and particularly in African athletes, are generally considered physiological and benign [30].

The reduction in resting state EF observed in this study does not result in LV dilation or clinical symptoms; all subjects functioned well at the highest levels of the Dutch football league. It remains to be seen if the hypertrabeculation and lower EF could eventually lead to LV dilation and further EF reduction with overtraining or in later life.

Study limitations

The small sample size of our African athletes and absence of female athletes and non-athletic controls makes it difficult to generalise our findings to the general population. Our preliminary findings require validation in larger more diverse populations. Nevertheless, the reported *p*-values are exceedingly small, suggesting that the probability of our findings being coincidental (alpha error) is very small, despite the relatively small sample size. The cross-sectional nature of this study without follow-up precludes any speculation about possible long-term effects of hypertrabeculation.

Conclusion

This study demonstrates a greater degree of myocardial hypertrabeculation in healthy African athletes, combined with biventricular EF reduction at rest. Recognition of a mildly reduced ejection fraction with hypertrabeculation as a physiological process in these healthy athletes is necessary to avoid confusion with left ventricular non-compaction cardiomyopathy.

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