A diagnostic dilemma in non-compaction, resulting in near expulsion from the football world cup

Claudia Stöllberger1* and Josef Finsterer2

12nd Medical Department, Krankenanstalt Rudolfstiftung, Juchgasse 25, 1030 Wien, Austria; and 2Medizinische Abteilung, Krankenanstalt Rudolfstiftung, Juchgasse 25, 1030 Wien, Austria

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Left ventricular non-compaction is a cardiac abnormality whose echocardiographic diagnostic criteria are still controversial. The distinction between normal and pathologic is additionally impeded by the fact that the left ventricular myocardium is more intensively trabeculated in African blacks than in Caucasians. The impact of these uncertainties and unresolved issues in the diagnosis of non-compaction is illustrated by a 23-year-old professional African footballer in whom an aberrant left ventricular band was misinterpreted as non-compaction. The diagnosis of non-compaction resulted in the immediate withdrawal of the playing licence and impending deportation of the young man from Germany to his home country. Organized by the footballer’s lawyer, repeated echocardiographies and cardiac magnetic resonance imaging failed to diagnose non-compaction. After several months, the young man regained his playing licence and played as a striker in the national team of his country of origin in the football world cup. Uncertainties and unresolved issues may result in misdiagnosis of non-compaction, thus promoting discrimination and degradation. This case highlights the urgent need for standardization of diagnostic criteria for left ventricular non-compaction and to assess if they need to be different for African blacks and Caucasians.

Keywords

Echocardiography • Cardiomyopathy • Africans • Non-compaction • Football

Left ventricular non-compaction is a cardiac abnormality of unknown aetiology, associated with arrhythmias, heart failure, thromboembolic events, cardiomyopathy, and neuromuscular disorders.1 Left ventricular hypertrabeculation was initially diagnosed by ventriculography and echocardiography.2,3 The diagnostic criteria for LVHT are still controversial.3–5 The distinction between normal and pathologic findings is additionally impeded by findings in patients which show that the left ventricular myocardium is more intensively trabeculated in African blacks than in Caucasians.6 The impact of these uncertainties and unresolved issues is illustrated by the following case of an African footballer.

A 23-year-old black African male of excellent health, a professional footballer in top leagues, was transferred from a Saudi-Arabian to a German club, with associated profound changes in environment, climate, training conditions, and culture. During that time of acclimatisation, a sport-medical investigation was carried out which disclosed repolarization abnormalities in the electrocardiogram and increased trabeculations of the right and left ventricle, leading to the surprising diagnosis of LVHT.

The diagnosis of LVHT resulted in the immediate withdrawal of the playing licence and impending deportation of the young man. The young man’s manager and lawyer organized a further cardiac examination in order to confirm or exclude the diagnosis. On echocardiography, a normal systolic function was found with no signs of left ventricular wall thickening. Within the cardiac apex 2 prominent trabeculations were seen and an abnormal band crossed the left ventricular cavity, with the initial appearance of abnormal trabeculations (Figures 1 and 2). There were neither two-layered myocardial structures nor intertrabecular spaces suggesting LVHT. Thus, the echocardiographic examination did not confirm LVHT. Also, repeated cardiac magnetic resonance investigations showed accentuation of the papillary muscles, a homogenous left ventricular myocardium, and failed to diagnose LVHT. After several months and repeated checks, the young man regained his playing licence and played as a striker in the national team of his country of origin at the football world cup.

Electrocardiographic abnormalities are a frequent finding in young African sportsmen. In 155 young African football players, they were found in 26%, most frequently, like in our patient,
T-wave abnormalities. However, in these series, none of the cases with an abnormal electrocardiogram showed pathologic echocardiographic findings. Thus, the relevance of an abnormal electrocardiogram in an African sportsman is undetermined.

Like in our case, electrocardiographic abnormalities may lead to the referral for echocardiography which suggested LVHT. Unfortunately, diagnosing LVHT by echocardiography is not easy because historically, different attempts to characterize and quantify LVHT resulted in different diagnostic criteria. According to Chin’s criteria, the distance between the epicardial surface and trough of the recess ($x$), and the distance between the epicardial surface and the peak of the trabeculation ($y$), has to be measured. A smaller $x/y$ ratio indicates increased depth of intertrabecular recesses.

According to Oechslin’s criteria, LVHT is present if the left ventricular myocardium shows a two-layered structure at end-systole, if the ratio between the non-compacted and compacted layer is $>2.5$, and if the intertrabecular spaces are perfused from the ventricular side. According to Stöllberger’s criteria, LVHT is present if the left ventricular myocardium shows more than three trabeculations apically from the papillary muscles, if these trabeculations move synchronously with the myocardium and if the intertrabecular spaces are perfused from the ventricular side. Most probably, a combination of these three echocardiographic criteria will be the most useful tool to differentiate LVHT from normal cardiac structures. Three-dimensional and contrast echocardiography may be of additional help to visualize the myocardial structures. Left ventricular hypertrabeculation is also diagnosed by cardiac-computed tomography and magnetic resonance imaging, and various diagnostic criteria exist for these imaging methods. However, there is generally a lack of comparative studies between pathoanatomic and imaging studies in order to assess the sensitivity and specificity of the various diagnostic criteria for LVHT. As in our case, however, cardiac magnetic resonance imaging might be useful to exclude LVHT in echocardiographically uncertain cases.

Left ventricular hypertrabeculation may be erroneously diagnosed if LVHT is not well distinguished from prominent papillary muscles, aberrant bands, false tendons, tumours, thrombi, abscesses, or intramural haematomas. Whether this distinction is more difficult in African blacks than in Caucasians has not been assessed. Furthermore, it is unknown if aberrant bands, false tendons and prominent papillary muscles are more frequent in Africans than in Caucasians.

Uncertainties and unsolved issues may result in misdiagnosis of LVHT thus promoting discrimination and degradation as in the presented case. This case confirms the urgent need for standardization of diagnostic criteria for LVHT and to assess if they need to be different for African blacks and Caucasians.

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References