THE PREVALENCE OF INCREASED LEFT VENTRICULAR TRABECULATION IN INDIVIDUALS WITH SICKLE CELL ANAEMIA?

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Introduction  Left ventricular non compaction (LVNC) cardiomyopathy is rare amongst Caucasians but studies in African/Afro-Caribbean origin (black) heart failure patients demonstrate that a high proportion fulfil criteria for LVNC (30%). Recent observations in elite athletes, have also demonstrated a 3-fold greater prevalence of increased LV trabeculations amongst black athletes compared with Caucasian athletes, with almost 15% fulfilling
echocardiographic criteria for LVNC. We postulate that the increased LV trabeculations observed in black individuals represents an ethnically determined cardiac response to increase preload. Sickle cell anaemia is associated with an increased preload and a high cardiac output. The aim of this study was to evaluate the prevalence of increased LV trabeculation amongst homozygous sickle cell disease patients.

Methods Between 2005 and 2012, 99 consecutive normotensive sickle cell patients (53% male) underwent echocardiography. Echocardiograms were analysed for trabeculations defined as localised protrusions of the ventricular wall ≥3 mm in thickness associated with intertrabecular recesses filled with blood from the left ventricular cavity as assessed by colour Doppler and previously published criteria for LVNC. The results were compared with 132 healthy black individuals (55% male).

Results Sickle cell patients were older compared with controls (33 ±11 years vs 21±6 years; p<0.0001) with no difference in systolic blood pressure (118±11 mm Hg vs 120±15 mm Hg; p 0.165) in either group. Sickle cell patients had a mean haemoglobin level of 8.6±1.2 g/dl (range 5.5–11.6 g/dl). Sickle cell patients displayed a higher prevalence of increased LV trabeculations compared with controls (28.3% vs 12.1%; p0.0002). Of the sickle cell patients, 20.8% fulfilled conventional Chin et al and 10% Jenni et al criteria for LVNC. None of the controls fulfilled the published LVNC criteria. Sickle cell patients with LV trabeculations exhibited a larger LV cavity size compared to controls with LV trabeculations (51.7 mm ±6.0 mm; range 44–66 mm vs 47.1 mm±6.0 mm; range 38–54 mm; p<0.0001) but showed no difference in LV diastolic dimension compared with Sickles cell patients without LV trabeculations (mean LVDd was 51.1 mm±5.6 mm vs 51.9 mm±6.1 mm; p0.604). There were no differences in LV systolic or diastolic function in sickle cell patients with or without increased LV trabeculations (EF by Simpsons method was 61%±8.1% vs 61%±8.4%; p 0.985; E/A ratio was 2.0±0.8 vs 1.7±0.6; p 0.111 and MV deceleration was 191 ms±36 ms vs 194±51 ms; p 0.792).

Conclusions The high prevalence of increased LV trabeculations in sickle cell patients compared with black controls further reinforces the likelihood of this morphological anomaly representing a physiological response to increased cardiac preload and endorses the need for robust criteria for diagnosing LVNC in black individuals.