Case study

Athlete’s heart or dilated cardiomyopathy?

Courtesy of Prof. Sanjay Sharma, St. George’s Hospital, London

Patient history/ pathology

A 23-year-old asymptomatic male underwent cardiac evaluation after his father was diagnosed with cardiomyopathy. The patient participated in the triathlon at the regional and national level. His echocardiogram revealed an enlarged left ventricle (LV) of 61 mm with mildly depressed left ventricular function (left ventricular ejection fraction (LVEF) 52%) and reduced global left ventricular longitudinal strain (-12.5%).

Challenges

A proportion of endurance male athletes reveal an enlarged LV with a mildly depressed LVEF of 48-52%, raising the differential diagnosis of dilated cardiomyopathy (DCM). The distinction between physiological cardiac enlargement with mildly depressed LVEF and DCM is crucial since DCM is implicated in exercise-related sudden cardiac death and may be exacerbated by vigorous exercise.[1] The diagnosis of DCM has further implications because it requires the initiation of prognostically important medications and regular surveillance. Dilated cardiomyopathy may also be inherited in up to 40% of cases. Given his family history, the findings on the echocardiogram could not be entirely attributed to physiological left ventricular remodelling (athlete’s heart). In such cases, exercise echocardiography is important in facilitating the differentiation. An increase in LVEF <10% or a peak LVEF <63% favour DCM.

System, probe & device used

We used a Vivid E95 machine with a 4 dimensional probe (4Vc-D) to acquire LVEF using 4 dimensional full left ventricular volume analysis. The 3-4 multi-beat method was used to permit the highest possible volume rate for accurate analysis. Automated Functional Imaging (AFI) was used to measure global left ventricular longitudinal strain. Tissue Doppler was used to assess diastolic function. The scans were performed at baseline and during exercise on a supine bicycle.

Step-by-step procedure

A baseline echocardiogram, including 4D volume analysis, tissue Doppler imaging, and speckle tracking, was performed. Exercise echocardiography was conducted on a supine bicycle with an incremental ramp protocol of 20 Watts per minute. The tests revealed an increase in LVEF from 45% at baseline to 57% at peak exercise.

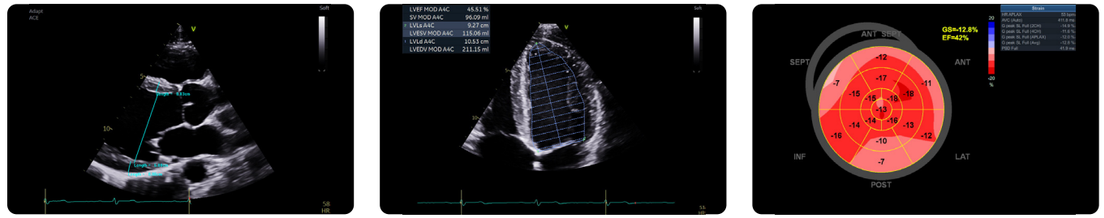
Cardiovascular Magnetic Resonance (CMR) imaging revealed enlargement of all 4 chambers and an LVEF of 52%. There were no regional wall motion abnormalities or myocardial scar.

Conclusion

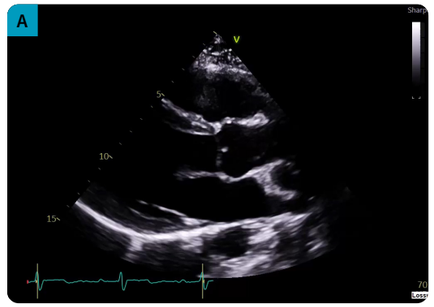
Although our athlete showed an increase in LVEF, our experience suggests that a failure to increase LVEF >10% or an increase in peak LVEF > 63% predicts DCM with a sensitivity of >80% and a specificity of >90%. We are less reliant on global left ventricular longitudinal strain (GLS) until more data is available, but our preliminary observations suggest that>70% of athletes with baseline low LVEF also have a GLS >-19%.

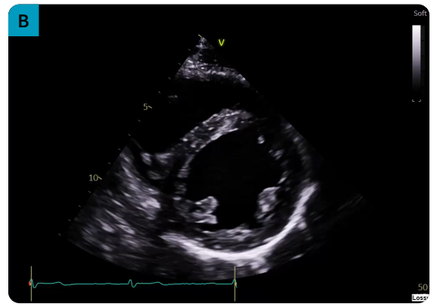
Imaging follow-up

The patient had genetic testing, revealing a pathogenic variant in the gene encoding titin, a recognised cause of DCM.[1] Based on the absence of symptoms, high level of fitness, only mildly depressed left ventricular function, lack of scar on the CMR, and absence of exercise-induced arrhythmias on an exercise test, he has opted to continue competitive triathlon. He is under 6 monthly assessments with resting echocardiograms, which have not revealed any deterioration in function over the past 18 months.

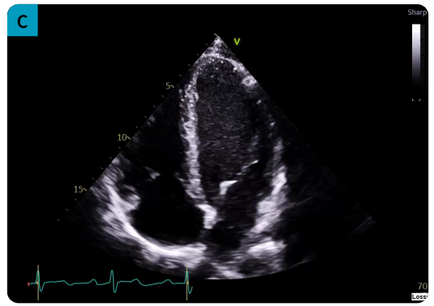


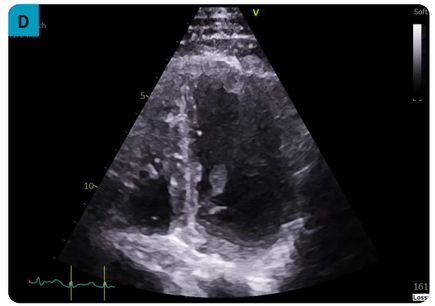
Static images reveal a dilated left ventricle with mildly reduced LVEF and reduced global longitudinal strain.





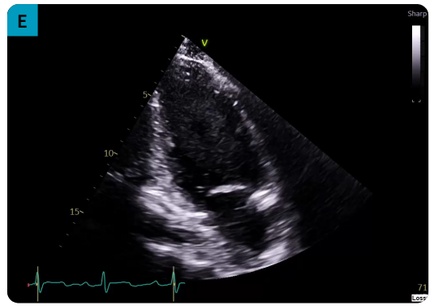
A-B) Enlarged LV with mild global hypokinesia.

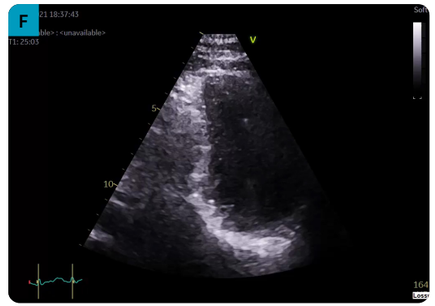




C) Baseline 4-chamber left ventricular view demonstrating mild global hypokinesia.

D) Peak exercise echocardiographic 4-chamber view.





E) Baseline 2-chamber left ventricular view demonstrating mild global hypokinesia.

F) Peak exercise echocardiographic 2-chamber view.

1 Millar LM, Fanton Z, Finocchiaro G, Sanchez-Fernandez G, Dhutia H, Malhotra A, Merghani A, Papadakis M, Behr ER, Bunce N, Oxborough D, Reed M, O’Driscoll J, Tome Esteban MT, D’Silva A, Carr-White G, Webb J, Sharma R, Sharma S. Differentiation between athlete’s heart and dilated cardiomyopathy in athletic individuals. Heart. 2020 Jul;106(14):1059-1065. doi: 10.1136/heartjnl-2019-316147. Epub 2020 Apr 27. PMID: 32341137.

Prof. Sanjay Sharma is a paid consultant for GE HealthCare and was compensated for his participation in this testimonial/Case study. The statements by Prof. Sanjay Sharma described here are based on his own opinions and on results that were achieved in his unique setting. Since there is no “typical” hospital/clinical setting and many variables exist, i.e. hospital size, case mix, staff expertise, etc. there can be no guarantee that others will achieve the same results.

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