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The Athlete's Right Heart

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Abstract

The athlete's heart is a known entity with morphological changes and a proportional physiological increase in chamber size. Even though, in the literature, the emphasis has been on the left heart, recent studies have described the effect of an intense exercise regimen on the right heart. Out-of-proportion, isolated, severe right atrium enlargement in athletes has not been described in the literature. This article reports 2 athletes with out-of-proportion, severe right atrium enlargement and discusses their presentations, baseline characteristics, and echocardiographic and cardiac magnetic resonance findings.

Introduction

A high-intensity training program imposes morphophysiologic effects on the left and right ventricles and atria. The physiological changes increase cardiac output in order to meet the increased metabolic demands associated with high-intensity exercise. The effect of intense training on the left heart includes increased Left Ventricle (LV) chamber size and left ventricular mass and increased Left atrial (LA) volume[1-3]. Recent studies have shown increased dimension of the Right Ventricle (RV) and Right Atrium (RA) in athletes compared to normal subjects[4-6]. Since the right heart has thin walls, it is very sensitive to volume overload and susceptible to afterload increase; therefore, even though the chambers' size may increase, function remains preserved. In the setting of continuous RV volume overload RV function can decrease[7].Most studies have shown proportional RV and RA chamber size enlargement[8]. Out-of-proportion, isolated, severe RA enlargement in athletes has not been described in the literature. This report describes findings in 2 athletes who demonstrated severe RA enlargement, out of proportion to the rest of the chambers.

Abbreviations: LV: Left Ventricle; RV: Right Ventricle; RA: Right Atrium

Patients Studied

Patient #1 is a 36-year-old Caucasian male who presented after having an abnormal electrocardiogram during a life insurance physical exam. The electrocardiogram Figure 1Ashowed sinus bradycardia, incomplete right bundle branch block, and T wave inversions in the inferior and anterior leads. The patient had no known medical history and did not take any prescribed, or over-the-counter medications, or recreational drugs. He has an intense exercise regimen (6 days a week and at least 2 hours per day) and has participated in several marathons and Ironman Triathlons in the past 10 years. Patient #2, a 65-year-old Caucasian male with a medical history of hyperlipidemia treated with simvastatin. He presented to our cardiology clinic after a routine echocardiogram showed a dilated RA and RV and raised concern about intracardiac shunt. He had completed several marathon competitions in the past and has had an intense exercise regimen for more than 20 years. He currently runs about 30 miles per week. An electrocardiogram Figure 1B showed sinus bradycardia and T wave inversions in the inferior and anterolateral leads. He underwent a transesophageal echocardiogram, which did not show any obvious evidence of intracardiac shunts, including patent foramen ovale or atrial septal defect.

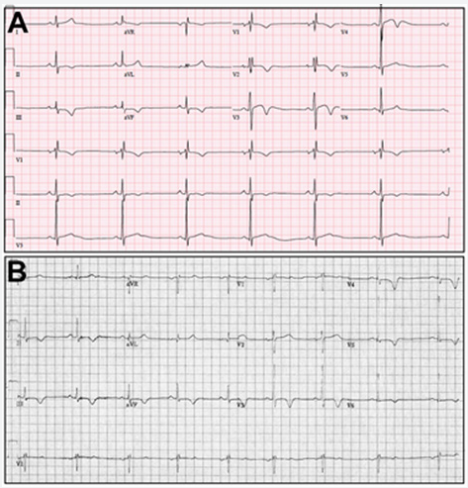

Figure 1: Electrocardiogram findings in patient #1(A) and patient #2(B).

Pertinent baseline, echocardiographic, and cardiac magnetic resonance characteristics of these patients are summarized in Table 1. Both patients' echocardiograms showed normal LV cavity size, normal LV function, normal global longitudinal systolic strain (Figure 2), and a mildly enlarged LA. Patient #1 had normal LV diastolic function, and Patient #2, being older, had Grade 1 diastolic dysfunction. The echocardiograms showed both patients had mildly increased RV size with normal function (Figure 3), normal RV tissue Doppler (Figure 4), and normal RV speckle tracking (Figure 5). Patient #1 had moderate tricuspid valve regurgitation with a pulmonary artery systolic pressure of 25mmHg (Figure 6A), and Patient #2 had trace tricuspid valve regurgitation with a pulmonary artery systolic pressure of 21 mmHg (Figure 6B). Both patients had a severely enlarged RA (Figure 3), which was out of proportion to the other chambers, as well as a dilated inferior vena cava, measured at 3.0 cm in Patient #1 and 2.4cm in Patient #2.

Table 1: Baseline, echocardiographic and cardiac magnetic resonance characteristics of patients.

Variables	Case	
	Patient #1	Patient #2
Age	36	65
Gender	Male	Male
Race	Caucasian	Caucasian
Blood pressure	100/66	124/70
Heart rate	40	52
BSA (m^2)	1.9	1.8
Echocardiogram		
Left heart		
LVEF	62%	65%
LV diameter (cm)	5.1	4.5
IVSW thickness (cm)	1.2	1.0
LVPW thickness (cm)	1.3	1.0
Stroke volume (ml)	110	85
E to A ratio	2.3	1.0
Lateral E' velocity (cm/s)	16.8	9.4
Septal E' velocity (cm/s)	12.4	7.1
LVGLS	-24.2%	-18.0%
Right Heart		
RV systolic tissue velocity (cm/s)	12.9	13.0
TR peak velocity (m/s)	2.2	2.0
RVGLS	-21.8%	-27.2%
CMR		
LVEF	66%	72%
LA volume index (ml/m^2)	67.4	41.7
LVED volume index (ml/m^2)	86.0	62.0
RVEF	60%	60%
RA volume index (ml/m^2)	123.7	70.0
RVED volume index (ml/m^2)	103.0	80.0

Note: BSA = Body surface area; EF = Ejection fraction; IVSW = Interventricular septal wall; LVPW = Left ventricular posterior wall; GLS = Global longitudinal strain; LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle; TR = Tricuspid valve regurgitation; ED = End diastolic; CAD = Coronary artery disease.

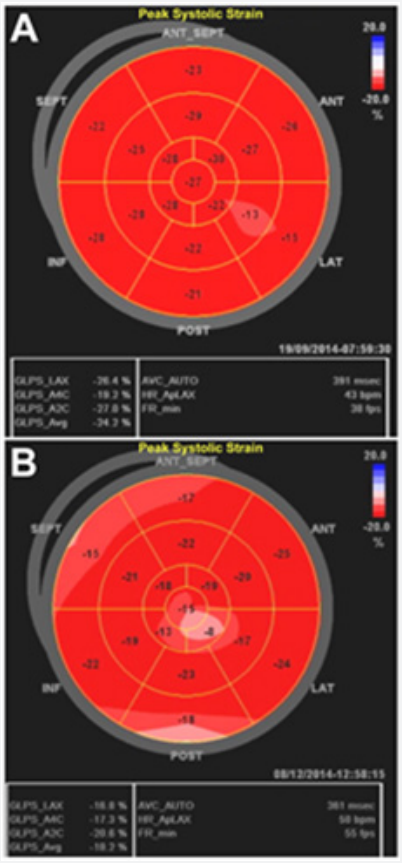

Figure 2: Global longitudinal systolic strain of the left ventricle in patient #1(A) and patient #2(B).

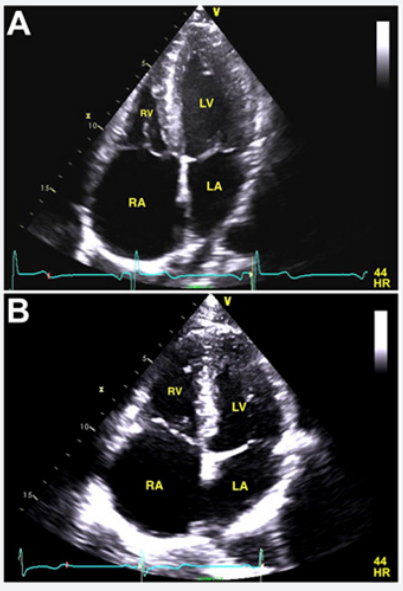

Figure 3: Echocardiographic apical four-chamber view in patient #1(A) and patient #2(B).

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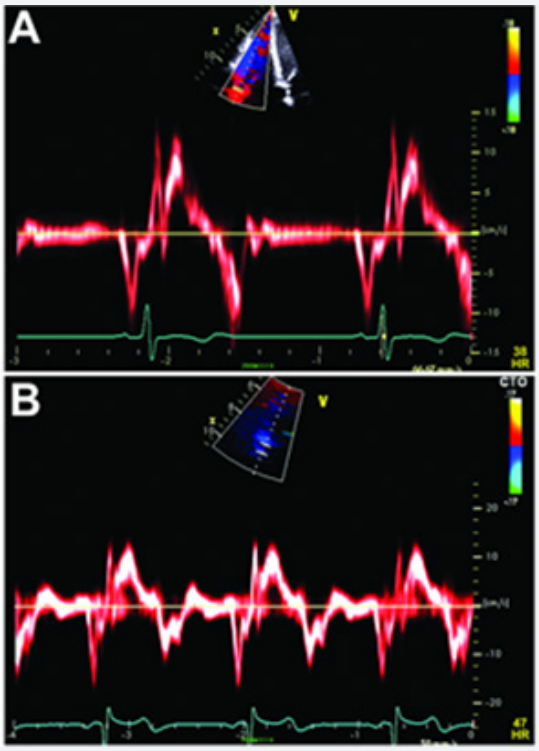

Figure 4: Right ventricular tissue Doppler in patient #1(A) and patient #2(B).

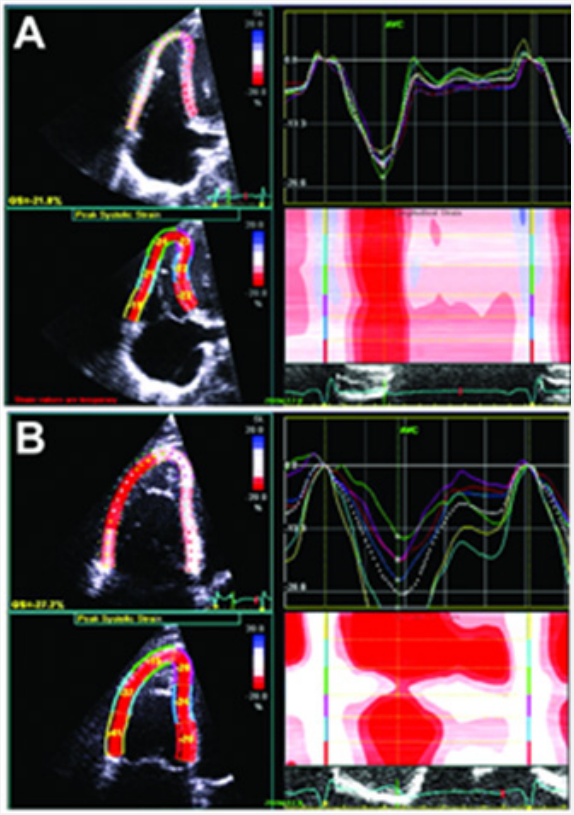

Figure 5: Right ventricular speckle tracking in patient #1(A) and patient #2(B).

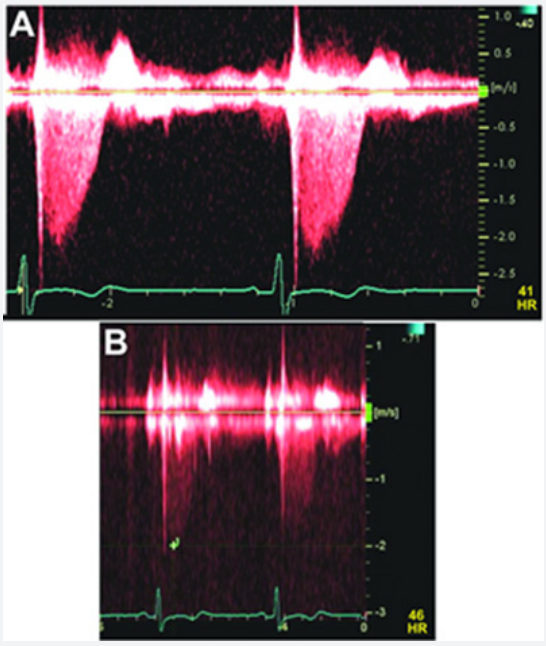

Figure 6: Continuous wave Doppler of tricuspid valve in patient #1(A) and patient #2(B).

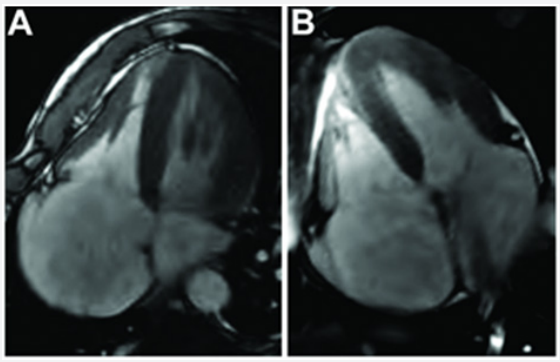

Figure 7: Cardiac magnetic resonance four-chamber view in patient #1(A) and patient #2(B).

Both patients underwent cardiac magnetic resonance imaging, which showed no evidence of anomalous pulmonary veins, sinus venosus atrial septal defect, or arrhythmogenic right ventricular dysplasia, and confirmed the diagnoses of severely enlarged RA (Figure 7). Cardiac magnetic resonance imaging was negative for delayed enhancement in both patients.Patient #1 had a Holter monitor placed to rule out silent arrhythmias; the results were negative for any arrhythmias. Due to a dilated inferior vena cava, he underwent a computed tomography scan of the abdomen and pelvis, which was negative for intra-abdominal shunt. A computed tomography angiogram showed normal coronary arteries. Right heart catheterization showed an RA pressure of 9 mmHg, RV pressure of 25/11mmHg, pulmonary artery pressure of 23/11 with a mean pressure of 15mmHg, pulmonary capillary pressure of 11mmHg, and pulmonary vascular resistance of 0.9 Woods units, and an intracardiac shunt

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study was negative for left-to-right shunt. He also was consulted for and underwent genetic testing, which showed mutation in the ILK and SOS1 genes, which are of unknown significance. Both patients remain active with no symptoms(Table 2).

Table 2: Baseline, echocardiographic and cardiac magnetic resonance characteristics of patients.

Variables	Case	
	Patient #1	Patient #2
Age	36	65
Gender	Male	Male
Race	Caucasian	Caucasian
Blood pressure	100/66	124/70
Heart rate	40	52
BSA (m^2)	1.9	1.8
Echocardiogram		
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Discussion

The athlete's heart is frequently identified to have morphological remodeling secondary to intense exercise. The medical literature has emphasized the changes affecting the left heart chambers[1-3]. Only recently have a few studies proposed the effect of intense exercise on the right heart with proportional increase in RA and RV chamber sizes[4-6]. I present two patients with athlete's heart and severely enlarged RA, out of proportion to other chambers. In both patients, other etiologies of RA enlargement - including intra- and extracardiac shunts, arrhythmogenic right ventricular dysplasia, Epstein's anomaly, and anomalous pulmonary veins - were excluded. It has been hypothesized that disproportionate hemodynamic stress on the right heart during exercise causes a mismatch between wall stresses of the left and right heart[9]. Although both sides of the heart are exposed to the same increase in cardiac output, vascular resistance in the pulmonary vascular bed can be reduced by 30-50% during exercise whereas vascular resistance in the systemic bed can be reduced 75%[10]. This can lead to a disproportional increase of stroke work on the right side compared with the left side (217% vs. 70%)[11-12]. In these studies, increased physiological stress on athletes' right heart caused proportional right chamber enlargement;however severe RA has not been reported in athlete's heart. It is unclear if these athletes had isolated RA enlargement12 as an underlying condition that was made worse by intense exercise. The long-term outcome of these two patients remains enigmatic remains uncertain.

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