Endurance Training on Congenital Valvular Regurgitation: An Athlete Case Series

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ABSTRACT

HOYT, Jr., W. J., P. N. DEAN, A. S. JOHN, L. W. GIMPLE, D. J. MISTRY, and R. W. BATTLE. Endurance Training on Congenital Valvular Regurgitation: An Athlete Case Series. *Med. Sci. Sports Exerc.*, Vol. 48, No. 1, pp. 16–19, 2016. **Background**: Both intense endurance training and valvular regurgitation place a volume load on the right and left ventricles, potentially leading to dilation, but their effects in combination are not well-known. **Purpose**: The purpose of this case series is to describe the combined volume load of intense endurance athletic training and regurgitant valvular disease as well as the challenging assessment of each component's cardiovascular effect. **Methods**: In this article, the clinical course of three elite endurance athletes with congenital valvular disease were reviewed. **Results**: A swimmer with aortic regurgitation, a cyclist with aortic regurgitation, and a cyclist with pulmonary regurgitation were found to have severe dilation of the associated ventricles despite continuing to train at an elite level without symptoms. **Conclusions**: Because of the cumulative effects of endurance training and valvular regurgitation, each athlete manifested ventricular dilation out of proportion to their valvular disease and symptoms. Although the effects of congenital valvular disease and athletic remodeling on ventricular dilation have been thoroughly studied individually, their cumulative effect is not well understood. This complicates the assessment of athletes with valvular regurgitation and underscores the need for athlete-specific recommendations for valve replacement. **Key Words:** CONGENITAL HEART DISEASE, SPORTS CARDIOLOGY, VALVULAR DISEASE, ENDURANCE TRAINING

The heart adapts to intense athletic training, depending on the regimen's degree of static and dynamic components, with hypertrophy and dilation—or some degrees of both (3,4,9,10,12,14,16,17,21–23,26,27,31–33). The degree of cardiovascular change has been documented well and generally thought to be a benign adaptation to the rigors of exercise (3,12,23). Valvular regurgitation places a volume load on the associated ventricle and can produce chamber dilation and, ultimately, symptoms of heart failure (5,8,24,25,28,30,34). The effects of endurance training superimposed upon preexisting valvular regurgitation is not known. In this article, three elite athletes with congenital valvular regurgitation present with ventricular dilation out of proportion to both their symptoms and severity of valvular regurgitation. The purpose of this case series is to describe the combined volume load of intense endurance athletic training and regurgitant valvular disease as well as the challenging assessment of each component's cardiovascular effect.

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CASE REPORTS

Case 1. An 18-yr-old male collegiate swimmer was diagnosed with bicuspid aortic valve after evaluation of abnormal ECG findings. His initial transthoracic echocardiogram (TTE) at that time revealed fusion of the right and left coronary cusps of the aortic valve, moderate regurgitation without stenosis, a normal aortic root size of 3.3 cm, a normal left ventricular (LV) size with LV internal diameter in diastole (LVIDd) of 5.8 cm, and an ejection fraction of 50%. Subsequent exercise stress test with stress echocardiogram showed a peak blood pressure of 172/93 mm Hg; no clinical, ECG, or echocardiographic evidence of inducible ischemia or arrhythmia; no worsening of aortic regurgitation with exercise; and improvement of ejection fraction with exercise. He has since moved to attend college, which has taken him from sea level to around 5000-ft elevation. His weekly training regimen since the move consists of 5 d of swimming with 4500- to 5500-m distance each morning and 5500–6500 m each evening as well as one weight training workout.

His echocardiogram 1 yr after diagnosis showed mild dilation of the aortic root to 3.6 cm, an increase in LVIDd to

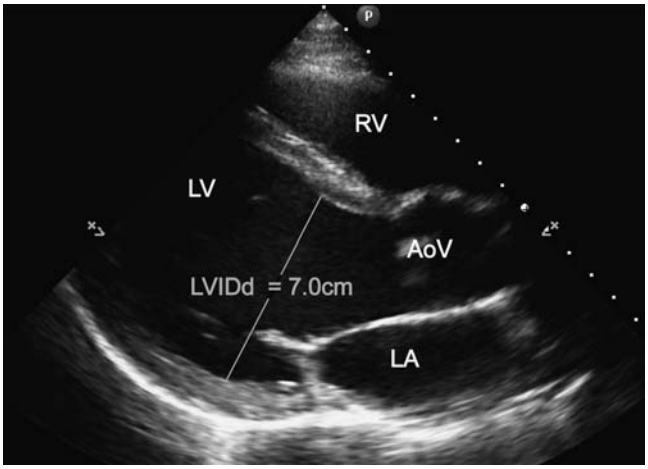

FIGURE 1—Echocardiogram illustrating the dilated LV in an athlete with bicuspid aortic valve and moderate regurgitation without stenosis. AoV, aortic valve; LA, left atrium; RV, right ventricle.

7.0 cm, a similar ejection fraction of 50%, and moderate aortic valve regurgitation without stenosis (Fig. 1). Subsequent TTE yielded similar results without additional dilation of the LV or aortic root. Other than a II/VI decrescendo diastolic murmur, his examination result was normal, with normal pulse pressure of 108/68 mm Hg. He continues to swim at the intercollegiate level without any symptoms of exercise intolerance as well as setting personal-best times in his event, the 100-m individual medley.

Case 2. A 36-yr-old male elite recreational cyclist was diagnosed at 22 yr of age with bicuspid aortic valve and severe aortic regurgitation. His first TTE at age 24 yr showed a severely dilated LV with LVIDd of 7.2 cm and LV internal diameter in systole of 5.0 cm, bicuspid aortic valve with severe aortic regurgitation. Despite these findings, he maintained a vigorous training regimen throughout his adult life, which he documented closely for training purposes. His weekly fitness schedule consisted of approximately three bicycle rides of 30–40 miles each that averaged 16.6 mph according to his cycling computer. In addition, he ran 4 $d \cdot wk^{-1}$ on a 4-mile course with average mile split times of 8 min. He repeatedly declined aortic valve replacement because he noticed no limiting symptoms.

The degree of left-ventricular dilation was monitored with left-ventricular diameters by echocardiogram and LV volumes by cardiac magnetic resonance imaging (MRI) (Table 1). Coincidently, an MRI was obtained after a 2-month period of deconditioning after hospitalization for meningitis. Compared with studies obtained during training, his deconditioned LV diastolic volume had declined from 345 to 303 mL. After another several years of training, LV diastolic volume again increased to 359 mL. At age 35, after repeated recommendation from his cardiologist, he decided to undergo aortic valve replacement although he still denied symptoms. After aortic valve replacement, the patient resumed biking at a low level after several months of general convalescence. Within 6 months, he was again riding his bike without limitation at similar distances as he was preoperatively. His LV diastolic volume had declined significantly to 178 mL by 6 months postoperatively. Although his noninvasive studies showed a dramatic improvement in LV dilation, at his most recent clinic evaluation, he denied subjective improvement during his workouts (Table 1).

Case 3. A 20-yr-old male with history of congenital critical pulmonary valve stenosis joined the university club cycling team at age 18 yr when he started college. His medical history is notable for pulmonary balloon valvuloplasty at 1 d of age, subsequent severe pulmonary regurgitation, and pulmonary valve repair at 15 yr of age due to severe right ventricular dilation with a right ventricular end-diastolic volume (RVEDV) of 182 $mL \cdot m^{-2}$ by MRI. That repair consisted of annular plication, posterior commissurotomy, and aneurysmorrhaphy of the dilated main pulmonary artery. At 16 yr of age, approximately 7 months postoperatively, he had mild pulmonary regurgitation by MRI and TTE, his RVEDV had improved to 126 $mL \cdot m^{-2}$, and his right ventricular ejection fraction (RVEF) was unchanged at 41%.

He joined this competitive cycling team during his first year of college at age 18 yr, starting with recreational cycling and progressing to an elite level, increasing his exercise training accordingly. Weekly training included running 7–8 miles daily, weightlifting of 50–100 lb, and cycling practice for 3–4 h. During training, he notes to be among the fastest members of that team, particularly during climbing sections. After about 5 months of this training regimen, a cardiac MRI demonstrated a RVEDV of 177 $mL \cdot m^{-2}$, no significant change in his pulmonary regurgitant fraction, and increased LV end-diastolic volume to 94 $mL \cdot m^{-2}$. His cardiopulmonary exercise stress test around that time showed a $\dot{V}O_{2max}$ of 46.4 $mL \cdot kg^{-1} \cdot min^{-1}$ (96% predicted).

After a prolonged discussion with his cardiologist, he decided to reduce his activity level. He reduced his running to 20–30 $min \cdot d^{-1}$, weightlifting to less than 50 lb, and stopped participating on the cycling team. Repeat MRI demonstrated an RVEDV of 165 and 155 $mL \cdot m^{-2}$ after 6 months and 1 yr of activity restrictions, respectively.

DISCUSSION

Case 1. Swimming places a high dynamic load on the heart, and some compensatory ventricular dilation would be

TABLE 1. Cyclist with aortic regurgitation.

		TTE		MRI	
Patient Age (yr)	Pertinent Event	LVIDd (cm)	LVIDs (cm)	LV-Vd (mL)	LV-Vs (mL)
28	40 miles per workout, averaging 17.0 mph	7.4	5.0	345	156
29	After 2 months of deconditioning	7.3	4.4	303	111
34	40 miles per workout, averaging 17.5 mph	8.5	5.0	359	161
35	6 months after aortic valve replacement	5.4	3.9	178	77

LVIDs, end-systolic LV inner;diameter, LV-Vd, LV end-diastolic volume; LV-Vs, LV end-systolic volume.

expected (3,15,21,22). The high altitude and training intensity would certainly enhance these cardiopulmonary demands (6,7,35). Aortic regurgitation is known to cause LV dilation, and his progressive LV dilation could understandably cause concern for resultant ventricular dysfunction (24,34). It has been shown that in male athletes without valvular regurgitation, LVIDd was commonly elevated (above 55 mm) in as much as 45% of athletes, but values higher than 65 mm were rare (19). However, the combined loads of aortic regurgitation and exercise confound established echocardiographic parameters for severity. In addition, low-to-normal ejection fraction at rest can occur among elite athletes (1,19,22). Nevertheless, continued improvement in his event times, a rigorously documented parameter in this athlete, further mitigates against LV dilation signifying underlying cardiac dysfunction, particularly after moving to high altitude.

Case 2. Compared with high school and college athletics, the physician has no absolute limiting control on recreational exercise. This cyclist and runner showed that intense levels of training can be achieved even at the recreational level. With his autonomy, he also illustrates the natural course of the aortic regurgitation with decades of endurance training (Table 1). He had long since met indications for aortic valve replacement by noninvasive measurements, but he had refused surgery as he felt asymptomatic (2,18). The clinical utility of his medical history was enhanced by thorough documentation of his training and close follow-up with echocardiograms and MRI. The volumetric analysis of his sequential training, detraining due to meningitis, return to training, and aortic valve replacement display an interesting data set of the individual contributions of aortic regurgitation and exercise to his LV dilation. Normalization of LV volume with detraining may be incomplete, as it was here, even without the presence of concurrent valvular disease (20). However, the small decrease in LV volume after detraining compared with the dramatic decrease after aortic valve replacement suggests aortic insufficiency contributed the most to LV dilation in this patient (20). Despite this dramatic improvement in MRI parameters after aortic valve replacement, he reported no improvement of his subjective exercise tolerance or training speeds. This highlights the multifactorial contributions to a subjective sense of athletic performance, suggesting that objective monitoring exercise capacity over time may more accurately evaluate his functional capacity. Correlation of this information to the patient's history and imaging data likely may further assist in the complex determination of the timing of valve replacement.

Case 3. Pulmonary regurgitation places an additional volume load on the right ventricle, which can cause right ventricular dilation and dysfunction that can manifest as exertional dyspnea and exercise intolerance (5,24,25,28,34). Competitive cycling, which places both a high dynamic and static load on the heart, may magnify the effect of this congenital valvular disease on ventricular dilation (15,22). His exercise stress test results were within normal limits but could be considered suboptimal in a rigorously trained athlete. The compounded influences of his valvular disease and athletic training further complicate the interpretation of his exercise stress test. Despite his valvular regurgitation, this athlete was able to train alongside his other teammates and perform competitively. Reduction in training intensity was only modestly effective at reducing right-ventricular volume. Additional concern is raised at the dramatically disproportionate dilation of his right ventricle relative to his left, suggesting that pulmonary regurgitation contributes more significantly to the right ventricular dilation than the expected proportional dilation of endurance training in this patient (26). Ultimately, the decision to reduce activity was reached only after thorough discussion between the physician and the patient, taking the athletic goals of the patient and accepted potential need for surgery into consideration (11).

CONCLUSIONS

The purpose of this case series is to describe the cumulative volume load of intense dynamic athletic training and regurgitant valvular disease as well as the challenging assessment of each component's cardiovascular effect. Because of these cumulative effects, each athlete manifested ventricular dilation out of proportion to their valvular disease and symptoms. While the effects of congenital valvular disease and athletic remodeling on ventricular dilation and hypertrophy have been thoroughly studied individually, their cumulative effect is not well understood (3,8,22,28). Although athletes with mild-to-moderate valvular regurgitation are generally not greatly disadvantaged in cardiopulmonary capacity, determining parameters for valve replacement in asymptomatic athletes with more severe valve regurgitation has not been delineated (13,29). This complicates the assessment of athletes with valvular regurgitation and underscores the need for athlete-specific recommendations for valve replacement.

As a case series, conclusions regarding safety of exercise or alternative timing of valve replacement in athletes with congenital valve disease cannot be definitively drawn from this descriptive study, and this subject deserves further investigation. Nevertheless, current clinical guidelines in patients with congenital valvular regurgitation likely do not apply to the elite athlete. As such, using these parameters to determine the need for valve replacement in athletes may subject the athlete to premature or unnecessary surgery. This complicates the assessment of athletes with valvular regurgitation and underscores the need for athlete-specific recommendations for valve replacement. These cases also highlight the importance of a obtaining a detailed training history, understanding the static and dynamic burdens of intense athletic training, and creatively assessing symptomatology when burdens of exercise and valvular regurgitation have cumulative effects on cardiac parameters.

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