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CASE REPORT

J. Scharhag
T. Meyer
I. Kindermann
G. Schneider
A. Urhausen
W. Kindermann

Bicuspid aortic valve

Evaluation of the ability to participate in competitive sports: case reports of two soccer players

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Dr. med. Jürgen Scharhag (✉)
Priv.-Doz. Dr. med. T. Meyer
Prof. Dr. med. W. Kindermann
Institut für Sport- and Präventivmedizin
(Leiter: Prof. Dr. med. W. Kindermann)
Universität des Saarlandes
66123 Saarbrücken, Germany
Tel.: 06 81 / 3 02-37 50
Fax: 06 81 / 3 02-42 96
E-Mail: j.scharhag@mx.uni-saarland.de

Dr. med. I. Kindermann
Klinik für Innere Medizin III –
Kardiologie, Angiologie
und Internistische Intensivmedizin
(Leiter: Prof. Dr. med. M. Böhm)
Univeritätskliniken des Saarlandes,
Homburg

Dr. med. G. Schneider
Radiologische Klinik
(Leiter: Prof. Dr. med. B. Kramann)
Univeritätskliniken des Saarlandes,
Homburg

Prof. Dr. med. A. Urhausen
Abteilung für Sportmedizin
(Leiter: Prof. Dr. med. A. Urhausen)
Centre Hospitalier, Clinique d'Eich
Luxemburg

Summary Two competitive soccer players aged 23 and 17 years with known bicuspid aortic valve presented for sports-medical preparticipation screening. Both athletes were well trained and had a maximal oxygen uptake of 61 and 60 ml/min/kg, respectively. Echocardiography of the first athlete revealed an eccentric hypertrophy of the left ventricle (end-diastolic diameter 58–59 mm, septal and posterior myocardial wall thickness 12–13 mm) with good systolic and diastolic function and a functional bicuspid aortic valve with mild regurgitation. In the second athlete, echocardiography showed a bicuspid aortic valve with moderate regurgitation and a relative stenosis, a hypertrophied left ventricle (end-diastolic diameter 62–63 mm, myocardial wall thickness 13–16 mm) and dilation of the ascending aorta of 46 mm, which was confirmed by magnetic resonance imaging. According to international guidelines, the first athlete was allowed to participate in competitive soccer. Nevertheless, regular cardiologic examinations in intervals of 6 months were recommended. In the second case, the athlete was not allowed to take part in competitive sports due to the extended ectasy of the ascending aorta and the concomitant risk of an aortic rupture. In addition, the left ventricular hypertrophy has to be considered as pathologic. Therefore, the athlete was only allowed to exercise in recreational sports with low and easily controllable intensities. *Conclusion* In athletes with bicuspid aortic valve, besides the evaluation of the aortic valve, physiologic adaptations of the heart have to be differentiated from pathological changes. Furthermore, the aorta deserves special attention, because in the case of a (probably genetically determined) dilated ascending aorta, an elevated risk for aortic rupture is present during intensive and competitive exercise. A general judgement in athletes with bicuspid aortic valves on their ability to participate in competitive sports is, therefore, not possible.

Key words Preparticipation screening – valvular heart disease – football

Introduction

In the literature, 0.5 to 7 athletes per 100 000 athletes and year are reported to suffer from sudden cardiac death in sports [12, 14, 15, 28]. In most of the cases, unknown cardiovascular disease is the underlying cause, which can further be differentiated between athletes under and over 35–40 years of age [2, 5, 12, 14, 15, 28]. In contrast to athletes aged over 35–40 years, in which the main cause of sudden cardiac death during sports is atherosclerotic coronary artery disease [6, 19, 20, 27, 31, 32], most common causes in younger athletes are cardiomyopathies, coronary anomalies, myocarditis, aortic stenosis and ruptured aortic aneurysms [15]. Therefore, preparticipation screening of athletes is of outstanding importance. Besides the differentiation of physiological adaptations and pathological changes of the heart, exercise physiology-related as well as sports discipline-related aspects have to be considered in individual risk stratification. By the following case reports on two competitive soccer players, the evaluation of the ability to participate in competitive sports in athletes with bicuspid aortic valve is described.

Case 1

Medical history

A 23 year old football player from a foreign first league team presented for sports-medical preparticipation screening with the question whether he can participate in competitive sports with bicuspid aortic valve and regurgitation, because of which he was previously declared as medically unfit. In addition, the athlete reported a heart rate of 224/min as displayed on his heart rate monitor towards the end of his training session during skippings with maximal intensity a month ago. Only a "fast heart rate" was noticed, symptoms of dyspnea, angina pectoris, dizziness, nausea, etc. were absent. For safety, the athlete discontinued the training, and within the next 5–10 min the heart rate had been normalized without any interventions. An ECG immediately registered after his training session was without any abnormalities.

Apart from elevated blood cholesterol levels, no cardiovascular or other diseases were known, and the family history was free of cardiovascular diseases. Nicotine abuse was negated, alcohol was consumed occasionally. The athlete took fish oil capsules and occasionally magnesium supplements. Soccer training was 6×per week, with 1 soccer match per week.

Physical examination

Good general health and nutritional status (weight: 76 kg; height: 174 cm). Regular heart rate of 60/min. Resting blood pressure 130/65 mmHg. 2/6 early systolic murmur and 1/6 early diastolic murmor with p.m. over the aortic valve. No other abnormalities.

Clinical examination

Total blood cholesterol 256 mg/dl, HDL-cholesterol 62 mg/dl, LDL-cholesterol 174 mg/dl, triglycerides 88 mg/dl, uric acid 6.8 mg/dl. Creatine kinase (CK) 448 U/l due to prior sportive activity (last training session one day before examination), normal CK-MB. Normal values for red and white blood cell counts, potassium, sodium, magnesium, creatinin, urea, glucose, GOT, GPT, gamma-GT, TSH, C-reactive protein.

Resting and exercise ECG

Resting ECG QRS axis between +30 and +60°, sinus rhythm of 60/min. Incomplete right bundle branch block, pronounced R waves in the left precordial leads. No other abnormalities.

Exercise ECG (Increamental multi-stage treadmill exercise test with 0.5% elevation; initial speed 2.5 m/s; speed was increased every 3 minutes by 0.5 m/s.) Linear increase in heart rate up to a maximal heart rate of 198/min and a maximal lactate concentration of 11.7 mmol/l. Termination due to exhaustion. ECG without abnormalities. Maximal systolic blood pressure 230 mmHg. Six minutes after test cessation, the heart rate was 118/min, blood pressure 145/65 mmHg. Maximal oxygen uptake 61 ml/min/kg.

Echocardiography

Functional bicuspid aortic valve with commisural fusion of the noncoronary and right coronary leaflets (Fig. 1b and 1c, arrow), but good opening aortic valve area (planimetered aortic valve area 4.3 cm^2). Aortic root diameter 31 mm, ascending aorta without abnormalities. Mild regurgitation of the aortic valve (V. contracta 2.5 mm; Pressure-Half-Time 860 ms; regurgitation fraction 14%). Maximal and mean systolic gradient 17 and 8 mmHg, respectively. Left ventricular end-diastolic diameter 58–59 mm, end-systolic diameter 33 mm, thickness of the interventricular septum and posterior wall 13 and 12 mm, respectively. Left ventri-

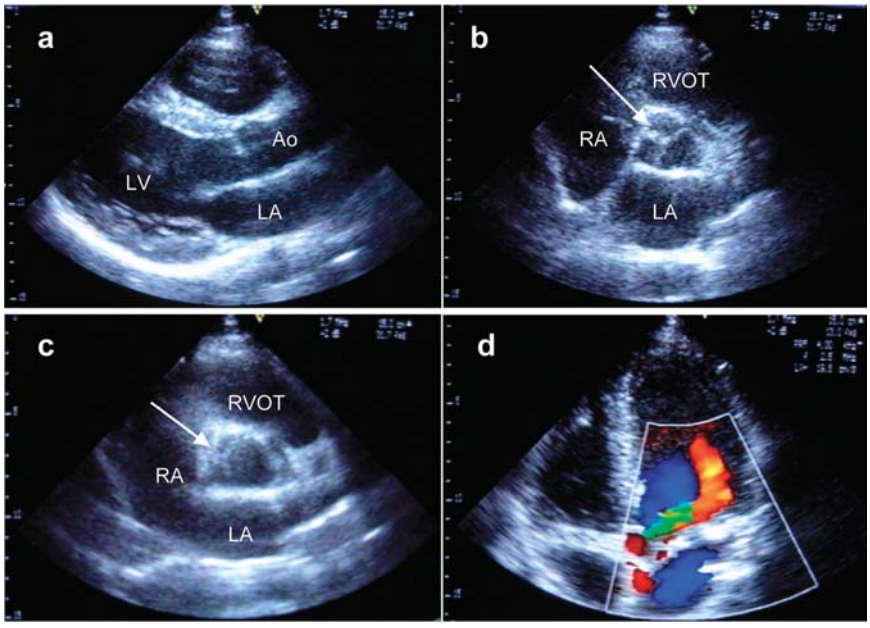

Fig. 1 **a** End-diastolic parasternal long axis view showing a slightly dilated left ventricle (LV) and a normal ascending aorta (Ao). **b** Diastolic parasternal short axis view: atypical configured aortic leaflets with commisural fusion of the noncoronary and right coronary leaflets (arrow). **c** Systolic parasternal short axis view without seperation of the noncoronary and right coronary leaflets (arrow). **d** Diastolic apical long axis view with aortic regurgitation. *Ao* aorta; *LA* left atrium; *LV* left ventricle; *RA* right atrium; *RVOT* right ventricular outflow tract

cular mass 167 g/m^2 according to the formula of Devereux [3], and 131 g/m^2 according to the formula of Dickhuth [4], respectively. Ejection fraction 65% (enddiastolic volume 178 ml; endsystolic volume 62 ml). Mitral valve without abnormalities, distance mitral valve E point to interventricular septum 6 mm. Mitral inflow velocities: E-wave 107 cm/s, A-wave 43 cm/s. Right ventricular end-diastolic diameter 25 mm. Pulmonary valve, tricuspid valve, left and right atrium without abnormalities.

Clinical judgement

The athlete was allowed to participate in competitive sports with the recommendation for regular cardiologic and echocardiographic examinations at 6 months intervals. In addition, antibiotic prophylaxis was recommended prior to interventions with increased risk for bacteraemia.

Case 2

Medical history

A 17-year-old soccer player from a junior team of a club of the Deutsche Bundesliga presented for preparticipation screening. No complaints were reported. However, a valvular heart disease was mentioned, which would be examined regularly by a cardiologist at 6 months intervals but did not restrict participation in competitive sports. No cardiovascular risk factors were known, the family history was free of cardiovascular diseases. Soccer training was 3× per week, with 1 soccer match per week.

Physical examination

Good general status of health and nutrition (weight: 62 kg; height: 170 cm). Regular heart rate of 53/min. Resting blood pressure 110/70 mmHg. 2/6 systolic murmor and 2/6 early diastolic murmur with p.m. at Erb. No other abnormalities.

Clinical examination

Creatine kinase (CK) 424 U/l due to prior sportive activity (last training session one day before examination), CK-MB 17 U/l. GOT 35 U/l, GPT 34 U/l. Triglycerides postprandial 212 mg/dl. Normal values for red and white blood cell counts, potassium, sodium, magnesium, calcium, total cholesterol, HDL- and LDL-cholesterol, bilirubin, LDH, cholinesterase, amylase, lipase, alkaline phosphatase, gamma-GT, total protein, creatinin, urea, uric acid, fT3, fT4, glucose, TSH, C-reactive protein.

Resting and exercise ECG

Resting ECG QRS axis between +60 and +90°, sinus bradycardia of 53/min. Distinct S-wave in

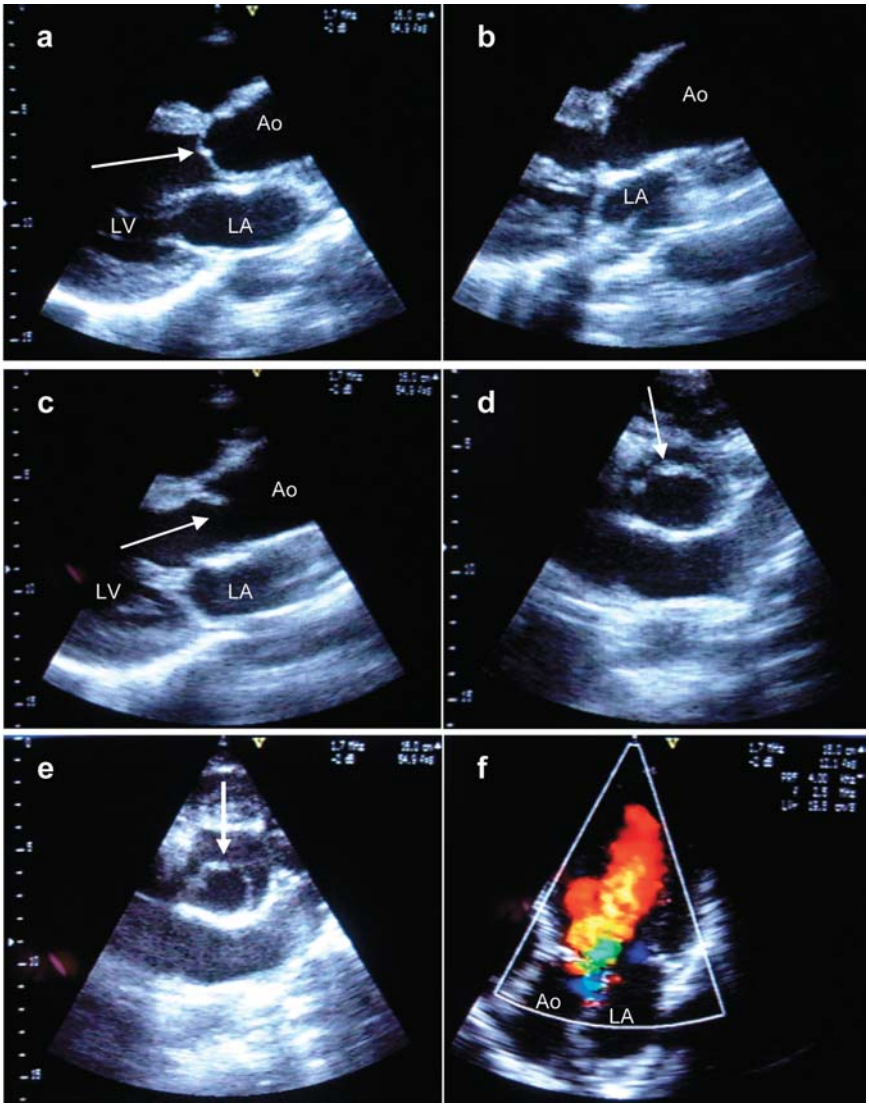

Fig. 2 **a** End-diastolic parasternal long axis view showing a prolaps of the aortic leaflets (arrow). **b** Parasternal long axis view showing the ectasy of the ascending aorta (Ao) up to 46 mm. **c–e** Systolic parasternal long axis (Fig. 2 c) and short axis views (Fig. 2 d and 2 e) demonstrating the reduced opening motion of the aortic valve (arrows) and a reduced eccentric aortic valve area (Fig. 2 e). **f** Diastolic apical long axis view with aortic regurgitation. *Ao* aorta; *LA* left atrium; *LV* left ventricle; *RA* right atrium; *RVOT* right ventricular outflow tract

leads II, III and aVF, negative T wave in lead III, high peak T-waves and increased R-wave in the left precordial leads. No other abnormalities.

Exercise ECG (Incremental multi-stage cycle-ergometry; start at 100 W, increase by 50 W every 3 min.) Athlete accomplished 1.5 min at 300 W, maximal heart rate 201/min. Termination due to fatigue of the leg muscles. Apart from two ectopic atrial beats at a heart rate of 125/min, no other abnormalities were observed. Maximal blood pressure 175/80 mmHg at the end of test. Six minutes after cessation, the heart rate was 111/min and the blood pressure 110/70 mmHg. Maximal oxygen uptake 60 ml/min/kg.

Echocardiography

Bicuspid aortic valve with diastolic prolapse (Fig. 2 a) and reduced systolic motion and opening (Fig. 2 c, d, e). In the M-mode eccentric closing line of the aortic cusps, aortic root diameter 32 mm. Ectasy of the ascending aorta up to 46 mm (Fig. 2 b). Moderate aortic regurgitation (V. contracta 6 mm, Pressure-Half-Time 320 ms; Fig. 2 f). Maximal and mean systolic gradient 67 and 37 mmHg, respectively. Planimetered opening aortic valve area 2.9 cm^2. Left ventricular end-diastolic diameter 62–63 mm, end-systolic diameter 38 mm, interventricular septum and posterior wall thicknesses 13–14 and 15–16 mm, respectively. Left ventricular mass according to the formula of Devereux [3] 252 g/m^2, ac-

cording to the formula of Dickhuth [4] 147 g/m^2. Ejection fraction 65% (end-diastolic volume 225 ml; endsystolic volume 79 ml). Mitral inflow velocities: E-wave 107 cm/s, A-wave 62 cm/s. Mitral valve without abnormalities, distance mitral valve E point to interventricular septum 8 mm. Right ventricular end-diastolic diameter 19 mm. Pulmonary valve, tricuspid valve, left and right atrium without abnormalities.

Magnetic resonance imaging

Slightly dilated left ventricle with pronounced muscular structure, ejection fraction 70% (enddiastolic volume 229 ml; end-systolic volume 69 ml). Bicuspid aortic valve with moderate regurgitation and functional stenosis, planimetered aortic valve area 3.4 cm^2. Maximal flow velocity at the aortic valve 4.9 m/s, corresponding to a pressure gradient of 95 mmHg. Diameter of the ascending aorta up to 46 mm, of the aortic arch 30 mm, and 25 mm of the descending aorta. Normal right ventricle with normal systolic function. No wall motion abnormalities.

Clinical judgement

The athlete was not allowed to participate in competitive sports. A detailed explanation of the risks involved in participating in competitive sports was given to both the athlete and his parents. Only recreational sportive activities with low and good controllable intensities, i.e. slow jogging or cycling were allowed. The importance of blood pressure was also stressed. In addition, three- to six-monthly cardiological check-ups were recommended as well as an antibiotic prophylaxis prior to interventions with increased risk for bacteraemia.

Discussion

In approximately 1–2% of the population, a bicuspid aortic valve is present. Apart from aortic regurgitation at young ages, there is a high risk for developing an aortic stenosis by infective endocarditis as well as by degenerative changes. Furthermore, an increased risk exists for aortic dilation, aortic aneurysms and aortic dissections [7, 9]. In about 8% of athletes up to the age of 40 years, sudden cardiac death during sports is caused by aortic stenosis and ruptured aortic aneurysms [15]. Given this, the preparticipation screening in athletes with a bicuspid aortic valve is of utmost importance.

Both presented cases report on young and well-trained competitive soccer players with congenital bicuspid aortic valves. In the first case, a mild aortic regurgitation existed. Besides a physiological hypertrophy induced by six professional training sessions and one game per week, also a pathologic adaptation caused from a slightly elevated volume load due to the aortic regurgitation has to be considered. Nevertheless, when interpreting the abovementioned findings, it has to be considered that left ventricular end-diastolic diameters of this extent are to be found amongst competitive soccer players (in [21] 16% of soccer players presented a left ventricular end-diastolic diameter of $\geq$60 mm) and athletes of other sports which are comparable to soccer [11, 21, 22, 30, 33], and therefore, can not generally be classified as pathologic. In addition, the left ventricular end-diastolic volume, interventricular septum and posterior wall thicknesses and left ventricular myocardial mass were also within normal ranges for competitive and well-trained athletes [21–24, 29, 30, 33].

However, the evaluation of the ability to participate in competitive sports in the first case is complicated by the reported heart rate of 224/min during training. But is has to be noticed that no cardiac symptoms were felt and that the ECG, which was registered directly after training cessation, remained without abnormalities. Moreover, incorrect measurements by artifacts caused from muscular activities during skippings as well as by interferences with transmitters of heart rate monitor's electrodes from other athletes have to be considered. Furthermore, by an increased self-observation especially in the first time of use of heart rate monitors, athletes often report on unexpected high heart rates during exercise, and thereby leads to the feeling of insecurity. Since the exercise test at a maximal heart rate of 201/min showed no ECG abnormalities in the first case, it can be assumed that the heart rate recorded during training was an incorrect measure.

In the second case, a dilation of the left ventricle was also present. But in contrast to the first case, the second athlete already had a moderate aortic regurgitation, which was accompanied with a pathologically elevated end-diastolic volume [23]. In addition, an aortic stenosis was present. The end-diastolic diameter of 62–63 mm was within the upper range for normal values in adult athletes (95 percentile in [21]: 63 mm) [21, 30, 33], but exceeded the upper reference limit for younger athletes aged 14 to 18 years (up to 60 mm in [25]). Also the myocardial wall thicknesses of the left ventricular posterior wall and the interventricular septum, which approximates to 15–16 and 13–14 mm, respectively, have to be classified as pathologic for young athletes aged 14 to 18 years [25] and even for adult athletes [22, 30, 33].

Only in a few healthy adult competitive rowers and canoeists have myocardial wall thicknesses between 13 and 16 mm been reported [22, 30, 33]. The left ventricular myocardial mass also exceeded the upper reference limit for athlete's heart [22, 24, 25, 30]. The most important finding, which strongly forces to forbid the participation in competitive sports, is the dilation of the ascending aorta of 46 mm [17]. It is well known that a bicuspid aortic valve, regardless of the degree of a possible stenosis, is a risk factor for proximal aortic dissections [9, 13, 26]. Accelerated degenerative processes of the aortic media are assumed to be the main cause of dilation of the ascending aorta in congenital bicuspid aortic valves [6–8, 10, 18]. At least in some patients, it appears as if these above mentioned degenerative processes may result from a genetically determined lack in fibrillin-1 and other intracellular matrix proteins of vascular smooth muscle cells (i.e. fibronectin and tenascin). Along with increased concentrations of metalloproteinase-2, which is related to an increase 11 in apoptosis of vascular smooth muscle cells [18], the increased activity of metalloproteinase-2 correlates with the aortic diameter [7]. An increase in the aortic dilatation in turn results in an increasing aortic regurgitation [1].

Consequently, in the case of the second athlete, a much more pronounced finding is obvious, which goes along with an increased risk for an aortic rupture. Furthermore, the risk of dying from an aortic rupture is independent from age [9]. In addition, the pathological elevated myocardial mass represents a further risk factor for sudden cardiac death in sports [15].

Therefore, in the second case, the athlete was not allowed to participate in competitive soccer. Moreover, because of the foreseeable indication for a mechanical aortic valve replacement and replacement of the ascending aorta [2], a career in professional soccer could not be recommended. In accordance to the eligibility recommendations for competitive athletes with cardiovascular abnormalities of the 36th Bethesda Conference [17], only recreational activities with low and easily controllable intensities have been allowed, as during competition often phases of high intensities occur, which can induce relevant increases in arterial blood pressure and therefore would increase the risk of an aortic rupture. An antihypertensive medication was not prescribed in the first instance in this case due to the low to normal resting blood pressure.

Conclusion

Apart from the evaluation of the aortic valve in athletes with bicuspid aortic valves, physiologic and pathologic adaptations of the heart have to be differentiated and special attention should be directed to the aorta, because in the case of a (probably genetically determined) aortic dilation, intensive and competitive exercise hold an elevated risk for an aortic rupture. A general judgement in athletes with bicuspid aortic valves on their ability for competitive sports is therefore not possible.

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