

Received: 2013.06.06
Accepted: 2013.07.10
Published: 2013.09.23

A teenager with tetralogy of fallot becomes a soccer player

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ACDE **Massimo Bolognesi**
BF **Diletta Bolognesi**

Sports Cardiology Center Medicine, Cesena Italy

Corresponding Author: Massimo Bolognesi, e-mail: massbolo1@tin.it

Patient: Male, 0
Final Diagnosis: Tetralogy of Fallot
Symptoms: —
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual or unexpected effect of treatment

Background: Tetralogy of Fallot (ToF) is the most common form of cyanotic congenital defect. Adult subjects with results of repair of tetralogy of Fallot may present post-surgical consequences that limit their physical capacity and thus their ability to compete in sports. Conversely, adults with excellent repair of congenital heart disease may have a chance to participate in competitive sports.

Case Report: This case report illustrates the clinical course of a teenager with an outcome of surgical repair for TOF and demonstrates the boy's excellent physical capacity that ensures his ability to play soccer.

Conclusions: This case report raises the question of the possible revision of the criteria of the Italian COCIS protocol in terms of corrected congenital heart disease.

Key words: tetralogy of fallot • congenital heart disease • sports/ pre-participation screening • cardiorespiratory fitness • teenager

Full-text PDF: <http://www.amjcaserep.com/download/index/idArt/889440>



1168



—



4



16

Background

Tetralogy of Fallot (ToF) is the most common form of cyanotic congenital defect [1]. This anomaly has 4 anatomic features: 1) anterior and rightward displacement of the aortic root; 2) ventricular septal defect; 3) right ventricular outflow tract obstruction; and 4) right ventricular hypertrophy. Surgical repair is achieved by patch closure of the ventricular septal defect and by enlarging the RV outflow tract through an infundibular patch [2].

This leads to 3 important long-term consequences of repaired ToF: the presence of significant RV outflow tract restenosis and/or significant pulmonary regurgitation, as well as the occurrence of cardiac arrhythmias, predominantly of ventricular origin related to re-entry in the area of the infundibular patch. Several reports have assessed the exercise capacity in ToF patients [3]. These reports emphasize that several residual lesions can cause a reduced exercise tolerance in patients with repaired ToF. Residual PS after direct or conduit repair of ToF has been associated with reduced exercise capacity and increased ventilatory response to exercise, as a consequence of increased afterload to the RV with abnormal cardiac output response to exercise [4]. Presence of residual pulmonary artery branch stenosis and presence of aortopulmonary collateral vessels have also been associated with abnormal ventilatory response to exercise, as a consequence of ventilation/perfusion mismatch [5,6]. There is evidence that, in the presence of normal RV systolic function, the effect of isolated significant pulmonary regurgitation on exercise capacity is limited [7]. However, patients with significant pulmonary regurgitation who also have a reduced RV systolic function have the lowest exercise capacity, particularly if they also have a reduced

LV systolic function. Recent observations also confirm the predominant impact of reduced RV function of exercise capacity in repaired ToF [8]. Finally, a subgroup of patients with repaired ToF will present, or will be at risk of, sustained potentially life-threatening ventricular arrhythmias. Factors known to be associated with an increased risk of ventricular arrhythmias are: older age at repair, increased RV systolic pressure, the presence of RV dilation and dysfunction (usually as a consequence of significant pulmonary regurgitation), the presence of LV systolic dysfunction, and the presence of a broad QRS (duration >180 msec) [9,10]. Patients with an excellent repair who have a normal or near-normal RV pressure, no or only mild RV volume overload, no significant residual shunt, and no atrial or ventricular arrhythmias on ambulatory ECG monitoring or exercise testing can participate in all sports of any intensity [11].

Case Report

This case report describes the history of a 13-year-old soccer player, born with a congenital heart malformation complex without cyanosis (Fallot Tetralogy of Fallot-Pink). This congenital heart disease was surgically corrected at the age of 10 months by the classic repair in Tetralogy of Fallot – an outflow patch of pericardium was used to relieve subpulmonic obstruction, and the VSD was closed with a pericardial or Dacron patch. The cardiac surgery led to an excellent result. After the radical heart surgery, the child had normal development, showing a natural inclination to physical activity. Since birth, he was followed by our pediatric cardiology unit, where he performed a regular clinical and instrumental follow-up, from which it is documented that he never showed any kind of cardiac complication. He always practiced in recreational sports activity, but at

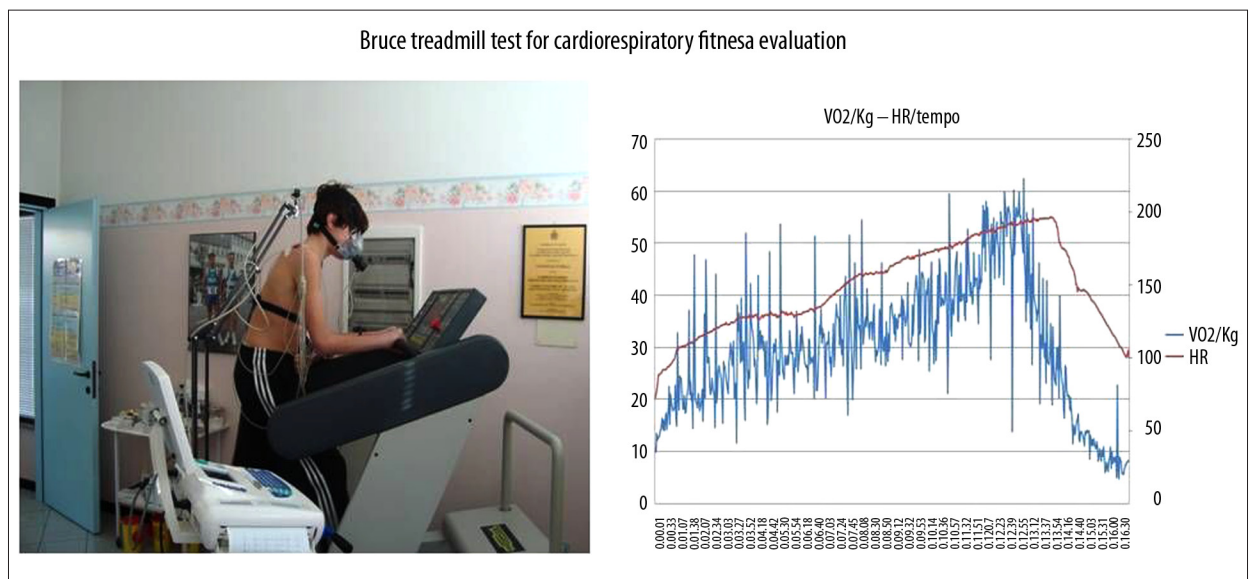


Figure 1. The athlete undergoes cardiorespiratory exercise treadmill testing for estimate VO2 max and anaerobic threshold.

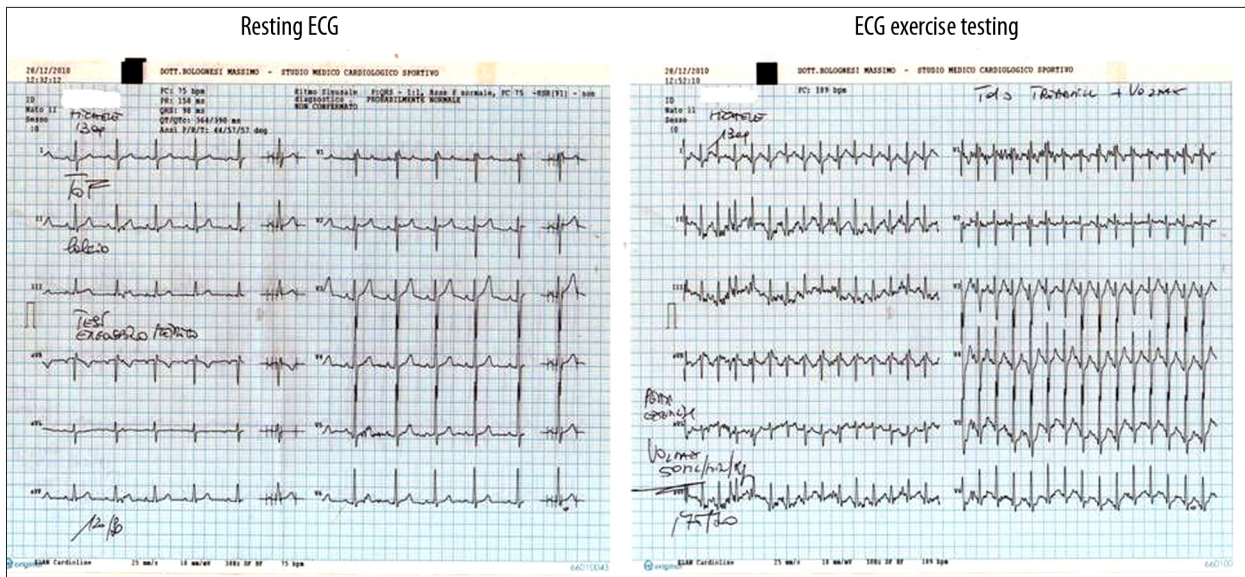


Figure 2. Resting ECG and during exercise testing show normal pattern.

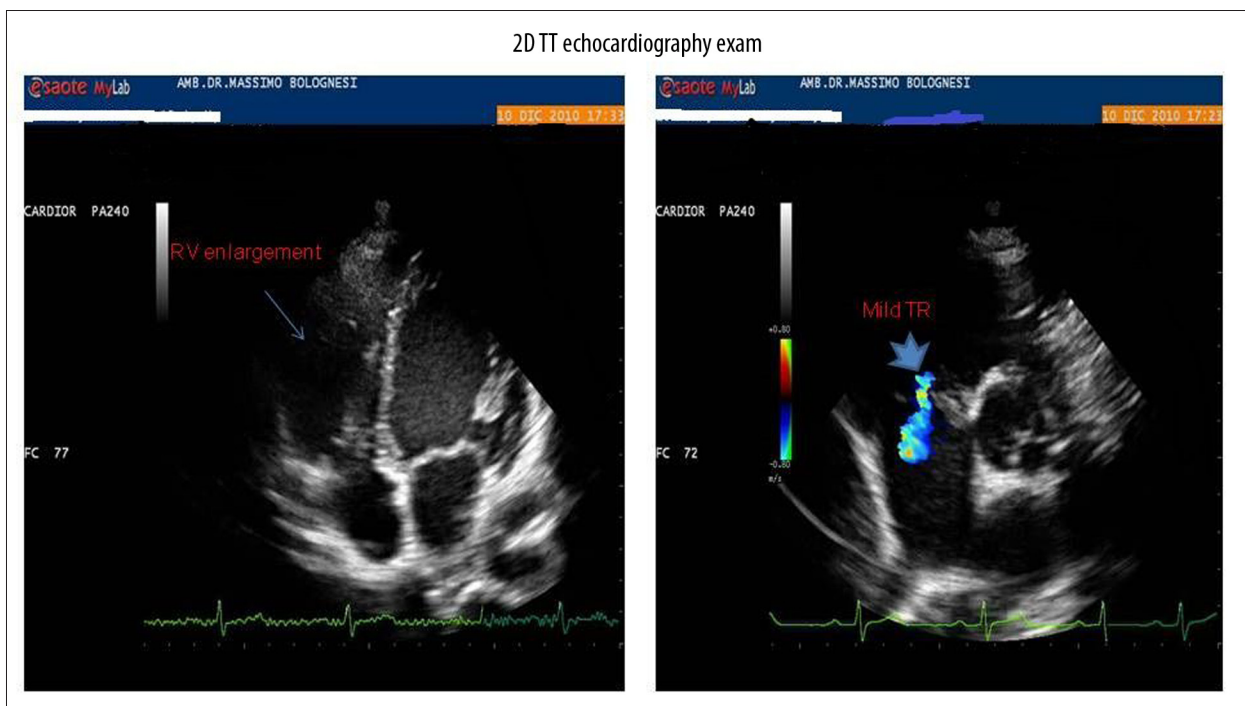


Figure 3. 2D Trans-thoracic echocardiography show mild right ventricular enlargement and mild tricuspid regurgitation.

some point he asked to engage in competitive soccer. Indeed, the aspiring young footballer has come into our medical center to undergo pre-participation screening and showed a better cardiorespiratory fitness than many of his peers with indices of optimal recovery. During the cardiorespiratory exercise testing, the athlete has achieved a maximum heart rate of 195 bpm at the height of the effort to a VO_2 max >50 ml/min/kg and a ventilatory anaerobic threshold corresponding to 82% of VO_2 max (Figure 1). Resting ECG showed sinus rhythm, normal

AV and intraventricular conduction (QRS <100 ms), with normal repolarization. ECG during exercise testing did not detect arrhythmias (Figure 2). Subsequent transthoracic bidimensional echocardiography (Figure 3) showed excellent repair of the VSD and normal size and normal morphology of the left ventricle, with preserved systolic and diastolic function. Mild right ventricular enlargement and mild tricuspid regurgitation were present; no abnormalities of the RV outflow tract were found other than a trivial pulmonary regurgitation. The aortic root was

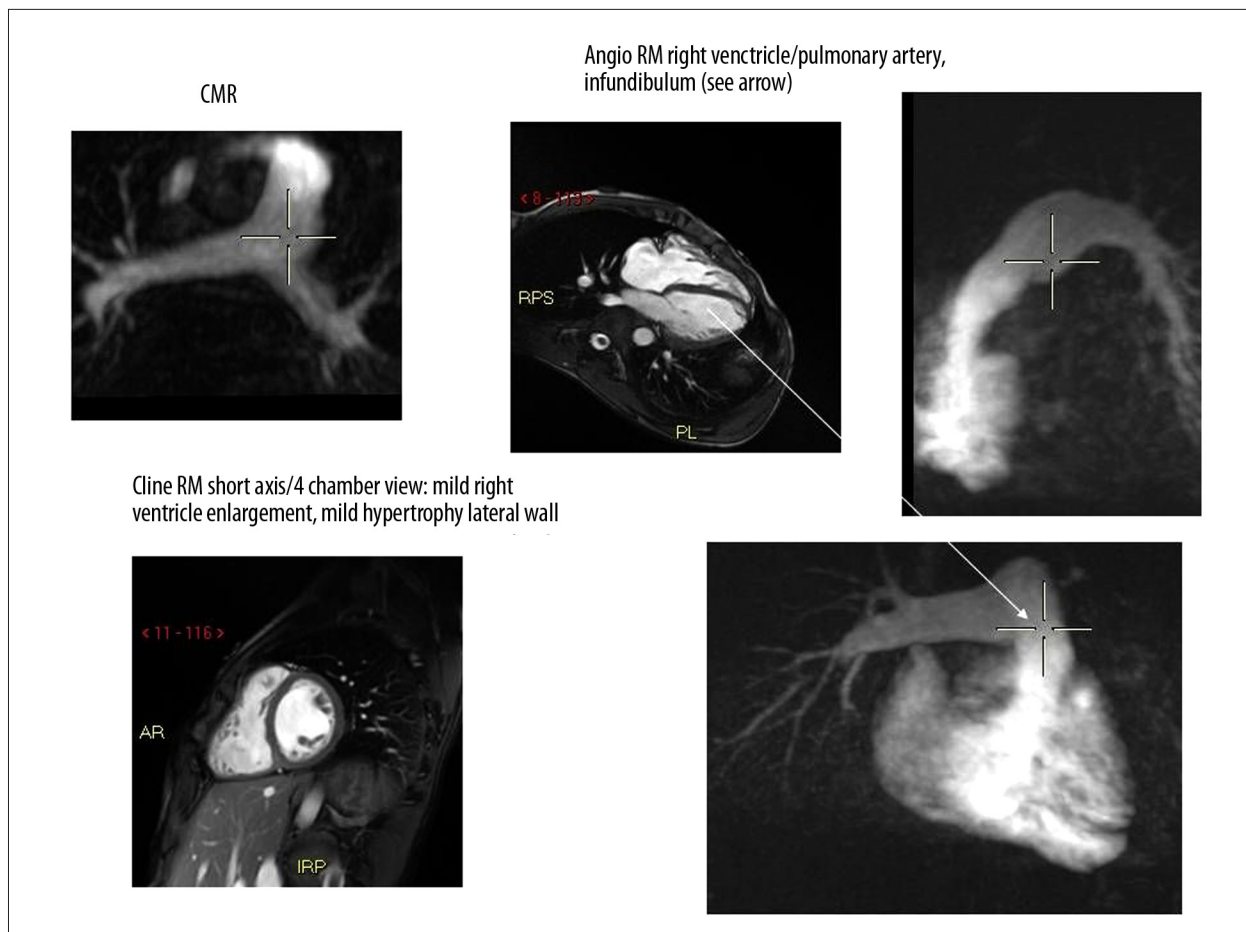


Figure 4. Cardiac MRI reveals slight dilation with hypertrophy of the right ventricle and shows the infundibular region of the right ventricular outflow tract.

normal size, without aortic valve regurgitation. Cardiac MRI was also performed, showing a slight dilatation and mild hypertrophy of the right ventricle, with normal biventricular contractile function and normal size of the aortic root and the pulmonary arteries, and without significant alterations of diameter at the level of the pulmonary valve (trivial regurgitation <5%) (Figure 4). In light of this evidence, in agreement with the pediatric cardiologist referent, he has been qualified as a competitive player, overcoming the restrictions of Italian Protocol COCIS 2009.

Discussion

In TOF, early surgical intervention is recommended. It has been suggested that sudden cardiac death is related to right ventricular hypertrophy or dilatation. The risk of sudden cardiac death increases with time, suggesting that “long-term follow-up by specialized cardiologists or pediatricians should be intensified” [12]. In Italy, the eligibility criteria in competitive sports are set by Protocol COCIS 2009, which recommends that adult competitive athletes with TOF corrected

surgically only participate in sports of low cardiorespiratory commitment, mainly neurogenic type, with the exception of soccer. However, some authors suggest that patients who have good surgical results can generally participate in athletic competition without restriction [13]. Others have stated that more athletes with excellent repair should be allowed to participate in all sports, providing that the following criteria are met: normal or near-normal right heart pressure; no or only mild right ventricular volume overload; no evidence of a significant residual shunt; and no atrial or ventricular tachyarrhythmia abnormality on ambulatory ECG monitoring or exercise testing [14]. Statistical analysis demonstrated a significant relationship between reduced work performance and residual disease, notably cardiac enlargement, increased peak systolic right ventricular pressure, pulmonic valve incompetence, residual ventricular septal defect, pulmonary hypertension, and cardiac rhythm disturbances [15].

This case raises possibility of revising the criteria of the COCIS 2009 [16] in terms of corrected congenital heart disease. In light of these findings, and in agreement with the pediatric

cardiologist referent, this athlete has been considered as qualified and suitable for competitive soccer, overcoming the restrictions of COCIS 2009.

Conclusions

Individuals with congenital heart disease are relatively rare but are valuable members of society, thanks in part to the progress of medicine and cardiac surgery over the past 50 years. The increased survival of this population and their progressive integration into social life, work, and sports requires collaboration between clinics in the suburbs and centers of excellence in the assessment of a patient's ability to safely

participate in sports. This presents an exciting challenge for modern sports medicine.

Consent

Informed consent was obtained from the patient for the procedures performed. Consent for data publication was also obtained from the patient. Written informed consent was obtained from the patient for publication of this manuscript and accompanying images.

Competing interests

The authors declare that they have no competing interests.

References:

1. Breitbart R, Fyler D: Tetralogy of Fallot. In Nadas' Pediatric Cardiology, 2nd ed. Keane, Locke, & Fyler, Philadelphia: Saunders-Elsevier, 2006; 559
2. Fraser CD Jr, McKenzie ED, Cooley DA: Tetralogy of Fallot: surgical management individualized to the patient. *Ann Thorac Surg*, 2001; 71: 1556–63
3. Sarubbi B, Pacileo G, Pisacane C et al: Exercise capacity in young patients after total repair of Tetralogy of Fallot. *Pediatr Cardiol*, 2000; 21(3): 211–15
4. Lurz P, Giardini A, Taylor AM et al: Effect of altering pathologic right ventricular loading conditions by percutaneous pulmonary valve implantation on exercise capacity. *Am J Cardiol*, 2010; 105(5): 721–26
5. Rhodes J, Dave A, Pulling MC et al: Effect of pulmonary artery stenoses on the cardiopulmonary response to exercise following repair of tetralogy of Fallot. *Am J Cardiol*, 1998; 81(10): 1217–19
6. Sutton NJ, Peng L, Lock JE et al: Effect of pulmonary artery angioplasty on exercise function after repair of tetralogy of Fallot. *Am Heart J*, 2008; 155(1): 182–86
7. Giardini A, Specchia S, Coutsoumbas G et al: Impact of pulmonary regurgitation and right ventricular dysfunction on oxygen uptake recovery kinetics in repaired tetralogy of Fallot. *Eur J Heart Fail*, 2006; 8(7): 736–43
8. Meadows J, Powell AJ, Geva T et al: Cardiac magnetic resonance imaging correlates of exercise capacity in patients with surgically repaired tetralogy of Fallot. *Am J Cardiol*, 2007; 100(9): 1446–50
9. Gatzoulis MA, Balaji S, Webber SA et al: Risk factors for arrhythmia and sudden cardiac death late after repair of tetralogy of Fallot: a multicentre study. *Lancet*, 2000; 356(9234): 975–81
10. Knauth AL, Gauvreau K, Powell AJ et al: Ventricular size and function assessed by cardiac MRI predict major adverse clinical outcomes late after tetralogy of Fallot repair. *Heart*, 2008; 94(2): 211–16
11. Graham J, Thomas P, Driscoll DJ et al: 36th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task Force 2: Congenital heart disease. *J Am Coll Cardiol*, 2005; 45(8): 1326–33
12. Nollert GD, Däbritz SH, Schmoekel M: Risk factors for sudden death after repair of tetralogy of Fallot. *Ann Thorac Surg*, 2003; 76(6): 1901–5
13. Bashore TM: Adult Congenital Heart Disease: Right Ventricular Outflow Tract Lesion. *Circulation*, 2007; 115: 1933–47
14. Graham TP Jr, Driscoll DJ, Gersony WM et al: Task Force 2: Congenital Heart Disease. *JACC*, 2005; 45(8): 1326–33
15. Wessel HU, Cunningham WJ, Paul MH et al: Exercise performance in tetralogy of Fallot after intracardiac repair. *J Thorac Cardiovasc Surg*, 1980; 80(4): 582–93
16. COCIS (Italian Cardiovascular Guidelines for eligibility in competitive sports). 2009; 53–54