Lecompte Procedure in a Case of Anatomically Corrected L-Malposition of the Great Arteries

To the Editor:

We present the case of a 12-month-old patient, echocardiographically diagnosed with tetralogy of Fallot, with indications for surgical treatment due to cyanosis. In the hemodynamic study, subaortic interventricular communication (IVC) was found as well as aortic override, infundibular, valvular, and supravalvular pulmonary stenosis, together with L-malposition of the great arteries and the right coronary artery that crosses the pulmonary infundibulum very close to the valvular plane (Figure).

Interventricular communication was closed using a polytetrafluoroethylene (PTFE) patch via the atrial route, the pulmonary artery was sectioned and directly reimplanted onto the right ventricle, with infundibular resection and enlargement of the anterior wall of the pulmonary outflow tract using an autologous pericardial patch without prosthetic conduit (Lecompte procedure or REV). A Lecompte maneuver was not necessary. After correction, the RV/LV pressure ratio was 0.4, without residual pulmonary gradient. There were no complications at 12-month follow-up.

The anatomically corrected malposition of great arteries is an infrequent type of conotruncal malformation where there is ventricular/arterial concordance despite presenting great vessel malposition. Thus, if there is atrioventricular concordance, the circulatory physiology is normal and surgical treatment consists in correcting the associated lesions, usually interventricular communication with infundibular pulmonary stenosis. It can also be associated with tricuspid hypoplasia/atria with right ventricular hypoplasia, right aortic arch, juxtaposition of atrial appendages and dextrocardia.1

In this malformation, the correction of the pulmonary stenosis is determined by the position of the right coronary artery in the right ventricular infundibulum, which usually entails using an extracardiac conduit. This involves successive reinterventions in order to replace this conduit. Monta et al2 describe a transannular enlargement technique via patch plasty of the right aortic valve, applicable in patients where the coronary artery is displaced away from this. In our case, given the age of the patient and the position of the coronary arteries, we chose the Lecompte procedure, connecting the pulmonary artery directly to a right ventriculotomy. This technique preserves the growth-potential of RV-pulmonary artery continuity and avoids the reinterventions due to the extracardiac conduit, making it especially attractive for use in young children. Recent studies comparing both procedures demonstrate significantly greater late morbidity with the conventional technique, which is associated with reintervention due to conduit obstruction.3,4

In the Lecompte procedure, most reinterventions due to pulmonary restenosis are associated with the use of patches with a pericardial monocusp valve which calcifies and induces obstruction.5,6 The use of a patch without a monocusp valve, as in our case, bypasses this problem, but it produces a pulmonary regurgitation that can cause late right ventricular dysfunction.

In the anatomically corrected L-malposition of great vessels with pulmonary stenosis, the Lecompte procedure offers an excellent alternative to RV-pulmonary artery conduit, since it enables earlier correction and reduces the need for reinterventions.

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REFERENCES


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Letters to the Editor

We present the case of a 21-year-old athlete, with no pathological or family history of note, who trains regularly and intensively and reports feeling run-down, with underperformance (physical and mental) and dizziness. Exploration showed sinus bradycardia <40 beats/min, due to which echocardiography was done (normal left ventricular function), a stress test (normal increase in blood pressure) and blood analysis (urea, serum ferritin and liver enzymes within normal limits). Overtraining syndrome was diagnosed and rest indicated.

Two 123I-MIBG scintigraphies were done, one at diagnosis and another after 10 weeks of rest, 370 Mbq of 123I-MIBG was administered intravenously, and planar anterior thorax images acquired at 4 h. The uptake of 123I-MIBG was quantified via the heart/mediastinum ratio (HMR) which, at the time of diagnosis, was slightly reduced (HMR, 1.71; normal >1.8) (Figure 1) but which normalized after rest (HMR, 2.12) (Figure 2).

Overtraining syndrome is defined as a state of prolonged fatigue and physical underperformance due to intense training with inadequate rest periods.1 This leads to the autonomous nervous system failing to adapt giving rise to decreased pituitary ACTH release and cortisol response, demonstrating reduction in intrinsic sympathetic activity and sensitivity to catecholamines.2,3 A definitive diagnosis is difficult due to the variety of signs and symptoms described.1,2

The first sign presented is underperformance associated with sensations of physical and mental fatigue, that generally accompany competition or recent intense training, unexplained muscle-tendon injury, increased irritability, apathy, sleep disturbance, weight-loss, changes in appetite, etc. Physical exploration demonstrates decreased heart rate and blood pressure.1 The determination of different enzymes and hormones during physical training can aid diagnosis and help prevent overtraining. The only treatment is rest for 6-12 weeks.4

In the case presented, overtraining was diagnosed due to physical and mental underperformance; the only sign found was sinus bradycardia, with all the normal complementary explorations. A meta-analysis was recently published, where a significant effect of physical training on the resting RR interval was shown in healthy individuals, where sinus bradycardia was due to an increase in vagal modulation.5

123I-MIBG is a guanethidine analogue, similar in structure to norepinephrine, that acts as a false neurotransmitter and is captured actively by presynaptic neurons. Its cardiac uptake is correlated with norepinephrine content and, thus, with the presence of sympathetic myocardial tissue.6 Previous studies show a decrease in total myocardial uptake with reduced HMR in athletes, associated with physical exercise due to increased vagal modulation.7 Estorch et al 8 described a reduction in 123I-MIBG uptake in marathon runners after prolonged exercise that normalized while resting. In the case described, myocardial uptake decreased during clinical testing but returned to normal after rest, indicating recovery of the sympathetic nervous system.

In conclusion, cardiac 123I-MIBG scintigraphy can be a useful method for diagnosing and controlling overtraining syndrome in the sportsperson.