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 $^{123}$I-MIBG scintigraphies were done, one at diagnosis and another after 10 weeks of rest, 370 Mbq of $^{123}$I-MIBG was administered intravenously, and planar anterior thorax images acquired at 4 h. The uptake of $^{123}$I-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. 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. A definitive diagnosis is difficult due to the variety of signs and symptoms described.

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. 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.

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.

$^{123}$I-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. Previous studies show a decrease in total myocardial uptake with reduced HMR in athletes, associated with physical exercise due to increased vagal modulation. Estorch et al. described a reduction in $^{123}$I-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 $^{123}$I-MIBG scintigraphy can be a useful method for diagnosing and controlling overtraining syndrome in the sportsperson.

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123I-MIBG Myocardial Scintigraphy in Overtraining Syndrome

Figure 1. $^{123}$I-MIBG scintigraphy at diagnosis (arrow: cardiac region).

Figure 2. $^{123}$I-MIBG scintigraphy after rest (arrow: cardiac region).
REFERENCES