

# Implantable cardioverter defibrillator and competitive sport participation

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## Fact

On 7 June 2009, the young soccer player Anthony Van Loo, during the match opposing his team KSV Roeselare to the R Antwerp FC in Belgian First Division, unexpectedly and suddenly collapsed while jogging slowly at 43th minute of the first half. Having fallen unnaturally, he landed on his back and remained there still and unconsciousness for a few seconds.

When his teammates realized that something horrible was occurring, and started running over to him, the 'miracle' occurred: some arm and legs shocks rattled the lying body of the athlete, and he regained consciousness. The implantable cardioverter defibrillator (ICD) previously implanted in his chest had recognized the ominous arrhythmia which was terminating his life, and timely and appropriately discharged the shocks that restored normal cardiac rhythm (see the event on <http://www.youtube.com/watch?v=UjZK4HgoY2Q>).

## Opinion

Another life saved by the ICD, appropriately implanted and working nicely. Another clear demonstration of the efficiency of this device to preserve, and even extend, the life of a young athlete. In general, a further confirmation that human scientific progress can modify the natural course of our lives by offering unexpected opportunities, particularly to individuals with inherited cardiac disease. In this case, a young athlete willing to participate in an otherwise prohibited experience, such as a competitive soccer career.

As a human being, I enjoyed watching a young athlete, having died, who resuscitated and, as a scientist, I felt the enthusiasm for the unlimited chances that research and investigation might bring to our lives. However, as a sports physician, certain worrying questions started coming to my mind.

Was this athlete affected by an inherited cardiac disease at the risk of cardiac arrest, as he unequivocally appeared to be? If so, don't we already know that sport participation, particularly

competitive soccer, is associated with an increased risk of adverse events, namely cardiac arrest?<sup>1,2</sup> Why, my mind was arguing, was this athlete, at risk for sudden cardiac death, advised (or better cleared) to participate in competitive soccer? This question had already found itself in the midst of largely implemented international guidelines specifically designed to manage athletes with cardiovascular disease, which unanimously state that patient-athletes with ICD (implanted for primary or secondary prevention in an arrhythmogenic cardiac disease) should avoid competitive sport participation.<sup>3,4</sup>

As physicians, should we uniquely meet the patient's aspirations for unrestricted lifestyle by providing the most updated technical solution, or should we be fully responsible for the patient's health? Then, shouldn't the physician consider with prudence all available scientific information describing the advantages and limitations of the ICD, as well as the risk of competitive sport participation?

From this perspective, it would appear questionable to allow this patient-athlete to continue participating in sports based only on the possible protection offered by ICD. This consideration stems not only from the awareness of complications (although rare) and inefficiencies of the ICD, but also from the consideration of the physician's ethical purpose to protect the young individual's life, by avoiding all possible (although often unpredictable) hazards for cardiac arrest such as, in this case, competitive sport participation.

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## CARDIOVASCULAR FLASHLIGHT

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### Sixty-four-slice computed tomography for the detection of multiple intra-thoracic thrombi in Trousseau syndrome

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A 57-year-old man was referred to our intensive care unit 48 h after the onset of an anterior ST-elevation myocardial infarction. Reports of previous hospitalization revealed that he was treated for pulmonary adenocarcinoma complicated with pulmonary embolism. He received chronically fluindione, and INR were constantly between 2 and 3.

The ECG showed sinus rhythm and Q-waves with no persistent ST elevation in anterior leads. Echocardiography visualized a large anterior akinesia without mural thrombus, and ejection fraction was 40%.

To complete work up, a 64-slice cardiac computed tomography was performed to precise coronary artery lesions. There was no lesion in right coronary artery and left circumflex. The left anterior descending artery was patent, but a large thrombus was observed in its proximal segment.

With the same acquisition, multi-slice computed tomography (MSCT) showed multiple thrombi in left and right ventricles (Panel B), left atrium appendage (Panel D), and pulmonary arteries (Panel B).

During hospitalization, the patient presented a deep right arm venous thrombosis.

Our case illustrates multiple venous and arterial thrombotic localizations in a patient suffering from a severe form of Trousseau syndrome (TS). The TS was first described in 1865. Some tumours, especially adenocarcinomas of the lung and the pancreas, are associated with hypercoagulability related to pro-thrombotic factors secreted by these tumours. On a pathophysiological point of view, TS is poorly understood, but high levels of tissue factors are generally associated. As the patient presented recurrent thrombosis with vitamine K antagonist, he was definitively treated with low-molecular-weight heparin.

In our case, MSCT had a better accuracy than echocardiography to visualize intra-ventricular mural thrombus. Two-dimensional echocardiography is routinely used in clinical practice to detect left ventricular thrombus. However, problem of the near-field artefact is an important drawback of 2-D echocardiography in the evaluation of left ventricular apical thrombi.

MSCT could be a tool to detect intraventricular thrombi, especially in patients with poor echogenicity. Left ventricle should be systematically analysed in order to detect thrombi in patients with history of myocardial infarction who underwent cardiac MSCT.

Panel A. Axial contrast-enhanced computed tomography showing a spiculated left lower lobe nodule.

Panel B. Cardiac multi-slice computed tomography. Four-chamber view depicting mural thrombus (arrows) in left and right ventricles (LV and RV) and thrombus (arrows) in right lower lobe segmental pulmonary artery (PA).

Panel C. Curved-MPR CT angiography showing a patent left anterior descending artery with thrombus in its proximal segment (arrow).

Panel D. Coronal oblique CT view: thrombus (arrow) in left atrial appendage (LAA).

