

generator. Because of the mechanism involved in both twiddler and reel syndrome, all leads would be affected to some extent, in case of several leads.

Ratchet syndrome: Ratchet syndrome can be defined as lead retraction and dislodgement due to progressive lead displacement through its fixation parts or protector sleeves, facilitated by movements of the ipsilateral arm and due to incomplete lead fixation to the protector sleeve, but without generator rotation over any of its axes. In this case, the problem could involve all system leads in a patient or, more commonly, only one of the leads, with all others in normal position; this is a key finding for identifying ratchet syndrome and distinguishing it from the other 2 lead macro-dislodgement syndromes.

Prior to surgical revision of the system in the case of lead dislodgement, simple visualization of the lead(s) and radiographic visualization of the generator position, along with a comparison to the original implant position, will provide an approximate identification of the mechanism involved. For cases in which the evidence is not definitive, either because the system uses only 1 lead and there are no signs of any mechanism (eg, twisting) or because the generator is in its normal position and there are no lead abnormalities, it would be preferable to use the term “lead macro-dislodgement” alone.

We believe that the classification and management of the proposed definitions may be helpful to the clinician and to the physician implanting the device and would allow better characterization of this rather common complication in patients with implantable electronic cardiac devices.

Miguel A. Arias,* Marta Pachón, Alberto Puchol, Jesús Jiménez-López, Blanca Rodríguez-Picón, and Luis Rodríguez-Padial

Unidad de Arritmias y Electrofisiología Cardíaca, Servicio de Cardiología, Hospital Virgen de la Salud, Toledo, Spain

*Corresponding author:

E-mail address: maapalomares@secardiologia.es (M.A. Arias).

Available online 11 January 2012

REFERENCES

1. Feltz Guzmán GI, Vivas Balcones D, Pérez de Isla L, Zamorano Gómez JL. Malposición de catéter de marcapasos durante 9 años. Rol del ecocardiograma. *Rev Esp Cardiol.* 2011;64:250.
2. Carnero-Varo A, Pérez-Paredes M, Ruiz-Ros JA, Giménez-Cervantes D, Martínez-Corbalán FR, Cubero-López T, et al. “Reel Syndrome”: a new form of Twiddler's syndrome? *Circulation.* 1999;100:e45–6.
3. Von Bergen NH, Atkins DL, Gingerich JC, Law IH. “Ratchet” syndrome, another etiology for pacemaker lead dislodgement: a case report. *Heart Rhythm.* 2007;4:788–9.
4. Benezet-Mazuecos J, Benezet J, Ortega-Carnicer J. Pacemaker twiddler syndrome. *Eur Heart J.* 2007;28:2000.
5. Bracke F, Van Gelder B, Dijkman B, Meijer A. Lead system causing twiddler's syndrome in patients with an implantable cardioverter-defibrillator. *J Thorac Cardiovasc Surg.* 2005;129:231–2.
6. Higgins SL, Suh BD, Stein JB, Meyer DB, Jons J, Willis D. Recurrent twiddler's syndrome in a nonthoracotomy ICD system despite a Dacron pouch. *Pacing Clin Electrophysiol.* 1998;21:130–3.

doi: 10.1016/j.rec.2011.09.025

Acute Myocardial Infarction for Thrombotic Occlusion in Patient With Elevated Coagulation Factor VIII

Infarto agudo de miocardio por oclusión trombótica en paciente con elevación del factor VIII de la coagulación

To the Editor,

We read with interest the article in the July issue of the *Revista Española de Cardiología* about hematologic diseases and the heart. We believe it would be appropriate to add elevated factor VIII (FVIII) as a risk factor for venous and arterial thrombotic events. For example, we present the case of a 45-year-old man with no cardiovascular risk factors who played sports regularly and had a body mass index of 23. He attended our hospital because of severe chest pain after cycling for 20 min. The electrocardiogram (ECG) showed ST elevation in leads V₂ to V₆, I, and aVL, and complete right bundle branch block. Fibrinolytic therapy was administered (7000 U of tenecteplase) for the first hour after onset of pain and he was transferred to our hospital. On arrival, he was asymptomatic and hemodynamically stable; the ECG showed normal ST segment with normal QS in leads V₂ to V₃ and negative T-waves in the precordial leads. Transthoracic echocardiography on admission revealed a dilated left ventricle, with a slightly depressed left ventricular ejection fraction (LVEF) of 38% and anteroseptal akinesia. At 23 h after onset, coronary angiography was performed, showing the presence of thrombotic material in the proximal segment of the left anterior descending artery, responsible for 40% obstruction with distal TIMI III flow. The remaining vessels were free of disease (Fig. 1). The physical examination was normal; laboratory tests showed troponin I levels of 57 ng/mL and peak creatine kinase of 2440 IU/L. The lipid profile, blood count,

and coagulation parameters were all normal. Given the lack of cardiovascular risk factors and the presence of arterial thrombosis, a hypercoagulation study was performed 48 h after admission to hospital. Fibrinogen, protein S, protein C, and antithrombin III levels were normal. Neither mutation of Leiden factor V nor lupus anticoagulant was detected. FVIII levels were elevated (234.2%; normal range, 50%–140%). The patient did not have a history of



Figure 1. Coronary angiography. Anteroposterior cranial view: thrombus in proximal segment of the left anterior descending artery.



Figure 2. Follow-up coronary angiogram at 7 days. The intracoronary thrombus has disappeared.

venous or arterial thrombosis. The case was discussed with members of the Hematology department, who indicated starting anticoagulation therapy with acenocumarol. Seven days after the infarction, the coronary angiography was repeated and it was confirmed that the thrombus had disappeared (Fig. 2). Three months after the infarction, the patient remained asymptomatic and with improved ventricular function (LVEF, 50%) in the follow-up echocardiography but FVIII levels were still high (210%).

Although the main cause of myocardial infarction is atherosclerotic disease, other causes such as coronary artery thrombosis should be kept in mind, particularly in younger patients. Study of coronary artery disease in younger individuals includes patients younger than 45 years. Our patient was 45 years old, the cutoff age, with no cardiovascular risk factors, and he presented with acute anterior myocardial infarction caused by a thrombus in the left anterior descending artery, with no angiographic evidence of coronary artery disease.

Among coagulation system disorders traditionally associated with arterial thrombosis are hyperfibrinogenemia; protein C, protein S, and antithrombin factor III deficiencies; increased thrombin activity; and mutation of Leiden factor V.^{1,2} For several

years, FVIII elevation has been included in the list of prothrombotic factors. FVIII is a glycoprotein that acts as a cofactor for factor IX and is essential for thrombus formation. Several studies have shown that elevated FVIII is an independent risk factor for arterial thrombosis (myocardial infarction, ischemic stroke, and peripheral arterial thrombosis), venous thrombosis, and thrombotic recurrence.³⁻⁵ Most patients included in these studies had their first event at a younger age than our patient. It is also known that patients with acute myocardial infarction with elevated FVIII have a worse prognosis. However, the underlying mechanism for this FVIII elevation is not known,⁶ and there is little indication in the literature about management of patients who have suffered thrombotic events and how long anticoagulation therapy should be maintained in the absence of a recurrence. We followed the indications of colleagues from the Hematology department, who recommended oral anticoagulation for 6 months.

In summary, FVIII should be included in the study of hypercoagulation of a patient with myocardial infarction and no arteriosclerotic disease.

Verónica Hernández, Nuria Muñoz, M. Antonia Montero, Agustín Camacho, Fernando Lozano, and Vicente Hernández*

Servicio de Cardiología, Hospital General de Ciudad Real, Ciudad Real, Spain

*Corresponding author:

E-mail address: veronicahernandez@hotmail.com (V. Hernández).

Available online 12 January 2012

REFERENCES

1. Gorog DA, Rakhit R, Paramus D, Laffan M, Davies GJ. Raised factor VIII is associated with coronary thrombotic events. *Heart*. 1998;80:415-7.
2. Xu W, Wang TY, Becker RC. Enfermedades hematológicas: desde dentro del corazón. *Rev Esp Cardiol*. 2011;64:606-13.
3. Bank I, Libourel EJ, Middelkoop S, Hamulyak K, Van Pampus EC, Buller HR, et al. Elevated levels of FVIII: C within families are associated with an increased risk for venous and arterial thrombosis. *J Thromb Haemost*. 2005;3:79-84.
4. O'Donnell J, Laffan M. Elevated plasma factor VIII levels a novel risk factor for venous thromboembolism. *Clin Lab*. 2001;47:1-6.
5. Bosma J, Rijbroek A, Rauwerda JA. A rare case of thromboembolism in a 21-year old female with elevated factor VIII. *Eur J Endovasc Surg*. 2007;34:592-4.
6. Hernández-Jerónimo J, Pérez-Campos E, Matadamas C, Majluf-Cruz A. Un nuevo factor de riesgo trombofílico: el aumento del factor VIII plasmático. *Rev Invest Clin*. 2003;55:448-57.

doi: 10.1016/j.rec.2011.10.013

Diagnostic Implication of the Percentage Change in Troponin I in Normal Range in Patients With Suspected Unstable Angina

Implicación diagnóstica de la variación porcentual de troponina I en el rango de normalidad en pacientes con sospecha de angina inestable

To the Editor,

Unstable angina (UA) is a frequent cause of hospital admission. Risk stratification should be the first step in managing diagnosis and treatment.¹ Nevertheless, despite the existing diagnostic resources, a substantial percentage of the patients that are at high risk according to the most widely used prognostic scoring systems (PSS) still show no evidence of coronary artery disease (CAD) in the angiographic study performed during the hospital stay.²

With the aim of providing information to complement electrocardiography (ECG) and PSS in the management of these patients in the scenario described here, we studied whether variations within normal range (NR) in the levels of troponin I (TnI), measured serially in the emergency service using a conventional (as opposed to ultrasensitive) method, predicted the detection of CAD in the hemodynamic study.

For this purpose, we evaluated all the patients admitted to our service with UA in 2010. We included prospectively all the patients with no history of ischemic heart disease and a negative result in serial testing for enzymes indicative of myocardial damage, defined as levels below the 99th percentile of the upper reference limit (URL), associated with intermediate or high risk according to the Thrombolysis in Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) risk scores, who had undergone coronary angiography during the hospital stay.