Reticular Telangiectatic Erythema Associated With an Implantable Cardioverter Defibrillator

Gerard Pitarch, MD; Pedro Mercader, MD; Arantxa Torrijos, MD; Teresa Martinez-Menchón, MD; Jose M. Fortea, MD, PhD

Reticular telangiectatic erythema (RTE) is a skin reaction associated with implantable cardiac devices (ie, pacemakers and cardioverter defibrillators). We present a patient who developed an erythematous patch over the implantable cardioverter defibrillator site. We discuss the clinical features, histologic findings, and patch testing of this entity.

Cutis. 2006;78:329-331.

Case Report
A 69-year-old man with a history of arterial hypertension and chronic bronchitis received an implantable cardioverter defibrillator in November 2002 because of ventricular tachycardia and sinus node dysfunction secondary to myocardial infarction. In February 2004, the patient noticed an erythematous patch on the skin overlying the implantable cardioverter defibrillator, with no associated symptoms.

Results of a physical examination showed blanchable and reticulate macular erythema with poorly defined margins and superficial telangiectases (Figure). Palpation revealed no induration, temperature increase, or fluctuation. The patient presented without fever or malaise. Results of laboratory tests were within reference range. Patch testing using metal allergens, as well as the standard battery of allergens of the Spanish Contact Dermatitis Research Group, proved negative. The clinical presentation, absence of signs that suggest infection, and negative patch test results suggested the diagnosis of reticular telangiectatic erythema (RTE); therefore, no histologic study was conducted. After 6 months of follow-up, the skin plaque disappeared spontaneously, leaving slight residual hyperpigmentation.

Comment
In recent years, subcutaneously implanted cardiac devices (ie, pacemakers and cardioverter defibrillators) frequently have been used. Skin complications associated with these metallic devices are infrequent. Although infections are the most common problems, implant extrusion also may occur as well as contact dermatitis.

RTE was first described by Gensch and Schmitt in 1981 in a patient with a pacemaker implantation. Since 1981, the MEDLINE database documented 19 patients with this type of lesion, referred to under different names, including RTE, circumscripted RTE, erythema, circumscripted erythema, persistent telangiectatic erythema, annular erythema, erythema with telangiectases, telangiectatic pacemaker erythema, and telangiectatic erythematous cutaneous reaction.

In all reported cases of RTE, the clinical picture was similar; the lesions, in the form of erythematous maculae with poorly defined margins and telangiectases of varying sizes in the skin overlying the implant site, appeared throughout a variable period of time (weeks to several years) after implantation of the metallic device. No edema or vesiculation was noted. The lesions may cause mild itching or a burning sensation, though RTE typically is asymptomatic, and the general condition of the patient is not affected. The clinical condition is so characteristic that Herbst and Weiss suggested eliminating the need for both histologic evaluation and patch testing in patients with a clinical diagnosis of RTE but no signs of infection and without overlying dermatitis.
The histology of RTE also is characteristic, though nonspecific. Dilated vessels are seen in the superficial dermis, with a perivascular lymphohistiocytic infiltrate and no involvement of the epidermis. Similar findings may be recorded in lupus tumidus, polymorphous light eruption, or erythema chronicum migrans, though the clinical history discards these diagnoses.

Patch testing is not essential for establishing the diagnosis of RTE, though it may help to rule out possible contact dermatitis in response to some component of the implant. In those cases where patch testing was performed, no relevant allergens were identified.

The evolution of patients with RTE is variable. In some cases, the lesions persist for years, which has led Kopera et al to suggest that the lesions are irreversible; in other cases, the lesions disappear after implant removal or spontaneously. Spontaneous lesion resolution may be one factor contributing to the underdiagnosis of RTE. A case has been reported in which RTE appeared prior to implant extrusion. In our opinion, although this circumstance may be incidental, it does seem advisable to follow-up with patients with RTE to assess possible implant extrusion or infection.

The origin of RTE has been associated with the heat and electric or magnetic fields generated by cardiac devices. In our opinion, the most plausible explanation is that RTE is attributable to local microcirculatory changes secondary to healing or to mechanical obstruction of blood flow caused by the device or by the anatomic characteristics of the implant site.

Conclusion
We present a case with typical clinical features of RTE associated with an implantable cardioverter defibrillator. The clinical course and complementary evaluations allow us to discard other disorders. Cardiologists and dermatologists should know this entity to avoid unnecessary diagnostic or therapeutic procedures.

REFERENCES