symptoms arise within 6 months of the initiation of therapy with the drug thought to be responsible. The causative role of the drug use is confirmed by complete symptom resolution within 3 months of its discontinuation and by the reduction or normalization of antibody titers in the subsequent months. The medications most frequently associated with this entity are antibiotics (eg, rifampicin and penicillamines), antimycotics (eg, griseofulvin and terbinafine), nonsteroidal anti-inflammatory drugs (eg, naproxen), antihypertensive agents (eg, diuretics, angiotensin-converting enzyme inhibitors, and calcium antagonists), interferons (alfa and beta), and statins (eg, simvastatin and pravastatin sodium).1

Report of a Case. A 41-year-old man presented with erythematous lesions on his face and erythematous plaques on the back of his hands that had developed after intense and acute solar exposure (Figure 1). His general condition was good, and routine laboratory and examination findings were normal. For several years, he had been receiving prophylactic antiepileptic therapy with phenobarbital and barbexaclone for a brain lesion that had been caused by peripartum hypoxemia. The sole element of note in his medical history was that 6 weeks earlier carbamazepine had been added to his drug regimen. He exhibited strong positivity for anti–extractable nuclear antigen antibodies, including anti-Ro/SSA (30 U/mL) and anti-La/SSB (47 U/mL); antinuclear antibodies (fine speckled pattern); lupus anticoagulant; and LE phenomenon. He also demonstrated a reduction in serum C4 levels (<645 µg/mL); anti-DNA antibody levels were at the higher limit of the range (20 allergenic units per milliliter). He was negative for anti-Sm, antiribonucleoprotein, and antihistone antibodies. On histologic examination, a plaque from the back of his hand revealed a dermatitis with focal aspects of vacuolization of the epidermal basal layer and a perivascular dermal lymphocytic infiltrate (Figure 2). Direct immunofluorescence of lesional skin showed uniform granular IgM, IgG, and C3 deposition along the dermoepidermal junction; the lupus band test yielded negative results in samples of healthy deltoid and buttock skin.

Comment. The patient’s history together with the clinical, histologic, and immunopathologic data and the absence of systemic symptoms led to a suspected diagnosis of Ro/SSA CLE induced by carbamazepine therapy. This hypothesis was confirmed by the complete resolution of symptoms 2 months after discontinuation of the therapy and by normalization of immunopathologic findings at 5 months. Carbamazepine use had previously been associated with the systemic form of drug-induced LE, which is characterized by symptoms of systemic involvement, with sporadic cutaneous manifestations and presence of antihistone antibodies. To the best of our knowledge, it has never been implicated in drug-induced Ro/SSA-positive CLE. This is the first report of drug-induced Ro/SSA-positive CLE related to the use of carbamazepine.

Subcutaneous Myeloid Sarcoma

Myeloid sarcomas are extramedullary tumors composed of immature myeloid precursors. We describe a patient with myeloproliferative disorder (MPD) who developed lesions that clinically resembled...
an inflammatory panniculitis but on histologic analysis were found to be subcutaneous myeloid sarcomas.

Report of a Case. A 75-year-old morbidly obese woman presented for evaluation of progressive lethargy, weakness, bone pain, and an 18-kg weight loss. Her white blood cell count was $139 \times 10^9/L$, with 16% band cells and 8% blast cells on peripheral smear. A bone marrow biopsy showed hypercellular marrow, an increased myeloid-erythroid ratio, and 4% blast cells. A Philadelphia chromosomal rearrangement was not detected. She was diagnosed as having unclassifiable MPD.

Our department was consulted to evaluate 3 subcutaneous nodules present on the patient's abdomen and right arm for 2 weeks. They were firm, smooth, minimally tender, and each measured approximately 2 cm. The overlying epidermis was mildly erythematous. On histopathologic examination, an infiltrate of atypical cells was found to be concentrated in the subcutis and deep dermis (Figure 1). Fibrous thickening of the subcutaneous adipose septa was apparent. On higher magnification, immature myeloid cells with large vesicular nuclei and nucleoli were seen stranding in the septa of the subcutis and infiltrating the fat lobules (Figure 2). Results of myeloperoxidase and CD43 immunohistochemical staining were strongly positive throughout the infiltrate; results of CD3, CD20, CD34, and CD56 immunostaining were negative. A diagnosis of subcutaneous myeloid sarcoma was made. The patient was treated with hydroxyurea for 6 months, and her skin lesions resolved, and her white blood cell count returned to normal.

Comment. Myeloid sarcomas are rare tumors composed of immature cells of granulocytic lineage. Synonyms include chloroma, granulocytic sarcoma, and extramedullary myeloid cell tumor. They can occur at any site, including subperiosteal bone, visceras, lymph nodes, and skin. Myeloid sarcomas most commonly arise in the setting of an underlying leukemia. In the setting of MPD or myelodysplastic syndrome the detection of a myeloid sarcoma signifies blastic transformation. Most patients with myeloid sarcoma and no peripheral blood involvement will go on to develop overt leukemia within several months.

The diagnosis of myeloid sarcoma is based on identifying myeloblastic and granulocytic elements in tissue by positive immunohistochemical staining with antibodies to myeloperoxidase, lysozyme, or naphthol-ASD chloroacetate esterase. Most myeloid sarcomas have a CD43-positive phenotype.

In our patient, detection of a dense infiltrate of neoplastic myeloperoxidase/CD43-positive granulocytes in the adipose tissue, with relative sparing of the overlying dermis, led to a diagnosis of subcutaneous myeloid sarcoma. The lesion was panniculitislike rather than a true panniculitis in that clear histologic features of inflammation were absent. Although lymphomatous and leukemic panniculitis are included in the clinical differential diagnosis of panniculitis, they are best considered pseudopanniculitides. Subcutaneous panniculitislike T-cell lymphoma and extranodal natural killer–cell and/or T-cell lymphoma are the most commonly reported panniculitisslike lymphomas. In contrast, panniculitislike leukemias and panniculitic myeloid sarcomas have been reported only rarely.

In summary, our patient had multiple subcutaneous myeloid sarcomas arising in the setting of MPD. Furthermore, we suggest that the differential diagnosis of tender subcutaneous nodules in the setting of MPD or myelodysplastic syndrome should include both a true
Telangiectatic Reticular Erythema Unrelated to Cardiac Devices

Reticular telangiectatic erythema (RTE) is a new and recently described skin complication that is associated with the use of pacemakers or implantable cardioverter defibrillators. To date, RTE has not been associated with the use of other devices or subcutaneously implanted metal devices; therefore, it has been suggested that RTE is exclusively associated with the use of cardiac devices. The present report describes a case of RTE that was unrelated to the use of either pacemakers or implantable cardioverter defibrillators.

Report of a Case. In 1973, a 22-year-old man was involved in a traffic accident that required implantation of a hip prosthesis. In 1995, he was referred to the pain treatment unit of our hospital for the management of pain in the region of the hip prosthesis. In February 2000, a subarachnoid morphine reservoir was implanted (Celsite ST204; Braun Inc, Syracuse, NY). After undergoing the implantation, the patient experienced dizziness, requiring removal of the reservoir. In February 2001, an intrathecal drug delivery system was implanted in the left flank region for the administration of tramadol hydrochloride. The system was composed of 2 implantable components: a programmable continuous infusion pump (Synchromed model 8626L; Medtronic Inc, Minneapolis, Minn) and an intraspinal catheter. Fifteen days later, an erythematous and pruriginous plaque developed over the pump site. Because there were no signs of infection, topical corticotherapy was started, without improvement. In October 2001, the patient was referred to our dermatology department for evaluation.

A nonelevated, reticulate, macular, erythematous, slightly itchy lesion with poorly defined margins and telangiectasias that were more apparent at the periphery (Figure 1) was noted on the skin over the implant site. A skin biopsy specimen revealed telangiectatic vessels in the reticular and papillary dermis, associated with a perivascular lymphohistiocytic infiltrate (Figure 2). To rule out possible contact dermatitis, patch testing was performed with the standard battery of the Spanish Contact Dermatitis Research Group and with antigens to additional metals, epoxy resins, glues, and plastics, as well as the pump component materials, as specified by the manufacturer. The results of all patch tests were negative, and RTE was diagnosed.

The lesions persisted without significant changes, until an erosion of the skin over the implant site developed in April 2003, requiring removal of the device. At follow-up 2 months later, all skin lesions had disappeared.

Comment. The origin of RTE has been associated with heat and electric or magnetic fields generated by cardiac devices. However, the infusion pump does not generate heat or electromagnetic fields of sufficient intensity to support this hypothesis. In our opinion, the most plausible explanation is that RTE is attributable to local microcirculatory changes due to healing or to mechanical obstruction of blood flow caused by the device or the anatomical characteristics of the implant site. Nevertheless, it should be taken into account that to date no such lesions have been reported in surgical patients with...