Erythema multiforme (EM) has also been described.2-6 Footskin reactions, which can be dose-limiting toxic reactions. Commonly reported eruptions include nonspecific rash and hand-foot skin reactions, which can be dose-limiting toxic reactions. Erythema multiforme (EM) has also been described.2-6

**Sorafenib-Induced Eruption Mimicking Erythema Multiforme**

Sorafenib tosylate is an orally administered small-molecule multikinase inhibitor that targets vascular endothelial growth factor receptors (VEGFR)-2 and -3, platelet-derived growth factor receptor (PDGFR), rearranged during transfection (RET), FMS-like tyrosine kinase 3 (FLT3), c-KIT, and C- and B-Raf. This drug is approved by the US Food and Drug Administration for the treatment of advanced renal cell carcinoma, unresectable hepatocellular carcinoma, and radioactive iodine-resistant, advanced, differentiated thyroid carcinoma. Cutaneous adverse effects of this drug affect up to 91% of patients.1 Commonly reported eruptions include nonspecific rash and hand-foot skin reactions, which can be dose-limiting toxic reactions. Erythema multiforme (EM) has also been described.2-6

**Report of a Case** A man in his 70s was enrolled in a clinical trial of sorafenib for patients with metastatic androgen-independent prostate cancer. He had a remote history of genital, but not orolabial, herpes simplex virus infection. His baseline dermatologic examination was unremarkable. Ten days after initiation of sorafenib therapy, 400 mg twice a day, the patient developed nontender, pruritic, erythematous papules and plaques on the trunk. He complained of fatigue but had no other systemic symptoms. Sorafenib treatment was withheld. Histologic examination of a punch biopsy specimen demonstrated a mild superficial perivascular and perianal lymphohistiocytic infiltrate with numerous eosinophils.

Two days later, the patient returned to the clinic with progression of the eruption. Numerous targetoid lesions with dusky centers, surrounding white rings, and peripheral erythematous rings were present on the trunk (Figure 1) and extremities. Several lesions had pseudovesicles or vesicles centrally. His oral mucosa showed several pinpoint white papules with an erythematous base without evidence of erosions. He denied systemic symptoms. Test results were positive for serum herpes simplex virus-2 IgG and negative for IgM.

Given the concern for EM from the clinical appearance, the patient underwent 2 additional punch biopsies from a targetoid lesion on the abdomen, and he was empirically treated with oral valacyclovir and fluocinonide, 0.05%, cream. Histopathologic analysis demonstrated spongiform dermatitis with a superficial perivascular inflammatory infiltrate consisting of lymphocytes and numerous eosinophils. No interface changes were seen (Figure 2). The histologic features seen in these specimens were most compatible with a medication-induced hypersensitivity reaction rather than EM. The eruption improved, and 8 days after its first appearance, the patient restarted a sorafenib regimen at a 50% dose reduction without recurrence of the EM-like reaction.

**Discussion** While there are numerous published reports of sorafenib-induced EM, several of these cases do not report histopathologic confirmation. One previously described patient was able to restart sorafenib therapy without recurrence of the eruption,2 but other patients have had recurrence of the eruption after beginning retreatment or have not been rechallenged.3-6 Despite the clinical appearance of our patient, histopathologic changes were not consistent with EM,
and sorafenib therapy was restarted without further sequelae. Unlike other cases reported in the literature, the patient described herein and the one described by Bilaç et al.² had clinically EM-like eruptions with targetoid lesions, nondiagnostic histopathologic findings, and successful reinitiation of sorafenib treatment. Thus, we recommend biopsy of targetoid eruptions during sorafenib therapy to differentiate between a diagnosis of EM and EM-like sorafenib reaction to minimize discontinuation of an antineoplastic agent shown to prolong progression-free survival.

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BRAF Inhibition in a Lung Transplant Recipient With Metastatic Melanoma

New treatment options like the BRAF inhibitors have been established for immunocompetent patients with metastatic melanoma, but experience in organ transplant recipients is lacking.

Report of a Case | A female double lung transplant recipient in her 60s with a standard triple immunosuppressive regimen (cyclosporine, mycophenolate mofetil, prednisolone) and chronic lung allograft dysfunction was diagnosed with metastatic melanoma and pulmonary (Figure 1A), mediastinal, hepatic, osseous, subcutaneous, and cerebral metastases (Figure 2A). A primary tumor could not be detected, and tumor cells harbored...