Cutaneous aspergillosis complicating pyoderma gangrenosum

Christopher B. Harmon, MD, W. P. Daniel Su, MD, and Margot S. Peters, MD
Rochester, Minnesota

Ninety percent of all leg ulcers are a consequence of chronic venous insufficiency, and approximately 5% are a result of arteriosclerosis obliterans. The remaining 5% are associated with hematopoietic disorders (such as cryoglobulinemia, sickle cell anemia, and thrombocytopenic purpura), collagen vascular diseases (such as systemic lupus erythematosus, scleroderma, rheumatoid arthritis, and dermatomyositis), cutaneous neoplasms, traumatic insults (such as burns, decubiti, radiation, and factitial causes), infectious diseases (such as deep fungal infection), and systemic diseases (such as diabetic microangiopathy).

Since its initial description by Brunsting et al. in 1930, pyoderma gangrenosum has been included in the differential diagnosis of nonhealing leg ulcers. Although inflammatory bowel diseases (ulcerative colitis and Crohn's disease) and rheumatoid arthritis are the most commonly associated systemic diseases, pyoderma gangrenosum has also been associated with collagen vascular diseases, viral infections, leukemia, and myeloma. Approximately 25% of cases, however, are not associated with any underlying disease. Treatment of pyoderma gangrenosum often requires systemic corticosteroids or nonsteroidal immunosuppressive agents, such as azathioprine and cyclosporine. We describe a patient who illustrates that treatment-resistant pyoderma gangrenosum requires evaluation for the possibility of secondary infection.

CASE REPORT

A 42-year-old man with a 5-year history of recurrent pyoderma gangrenosum was previously responsive to combination therapy with dapsone, prednisone, and cyclosporine. After a 12-month remission, posttraumatic lower extremity ulcers developed (Fig. 1). They were resistant to further therapy, including high-dose prednisone with cyclophosphamide. Because of the progression of lesions, tissue specimens were obtained for histologic examination and fungal cultures. Hematoxylin-eosin and methenamine silver stains demonstrated acutely branching septate hyphae and fruiting bodies consistent with aspergillosis (Fig. 2). Culture confirmed infection with Aspergillus flavus. The patient was treated with surgical debridement and grafting, intravenous amphotericin B, and reduction in immunosuppressive agents. The grafted sites healed successfully, and despite surgical manipulation, there has been no reactivation of the pyoderma gangrenosum.

DISCUSSION

Species of Aspergillus are ubiquitous, can be isolated from most organic materials, and are known to
cause pulmonary infections in pigeon and poultry handlers. Only five of the species cause infections in humans: *A. fumigatus*, *A. flavus*, *A. niger*, *A. terreus*, and *A. nidulans*. *A. fumigatus* is the most common cause of disseminated aspergillosis, and *A. flavus* is more often associated with cutaneous disease.\(^5\)\(^6\)

Cutaneous aspergillosis most often occurs as a secondary manifestation of disseminated disease in an immunocompromised patient; however, primary cutaneous aspergillosis does occur rarely. Since 1970, 15 patients with primary cutaneous aspergillosis have been reported; the skin served as the presumed portal of entry. Most of these patients had an underlying hematologic or lymphoreticular malignancy.\(^5\) To our knowledge, there have been no previous case reports of pyoderma gangrenosum associated with primary cutaneous aspergillosis.

Our patient had no signs or symptoms to suggest disseminated aspergillosis; two chest radiographs

---

*Fig. 2. A, Widespread proliferation of hyphae invading ulcerated tissue. (Hematoxylin-eosin stain; ×210.) B, Acutely branching septate hyphae and fruiting bodies of *Aspergillus flavus*. (Methenamine silver stain; ×340.)*
were normal and four blood cultures were negative. The diagnosis of primary cutaneous aspergillosis was based on the histologic features and fungal cultures. Because of the frequent contamination of clinical specimens, it is best to base the diagnosis of cutaneous aspergillosis on both microscopic findings and culture.5

This report emphasizes the importance of obtaining tissue for histologic examination and culture in patients with a previous diagnosis of pyoderma gangrenosum who exhibit a poor response to immunosuppressive treatment. The case also illustrates that patients with pyoderma gangrenosum who are receiving immunosuppressive therapy are susceptible to infection with opportunistic organisms.

REFERENCES

Sun between 3 PM and 4 PM can burn!

Bryan C. Schultz, MD,a and James S. Sweltzer, PhDb Maywood and Chicago, Illinois

The dermatologic community and such organizations as the American Academy of Dermatology (AAD) and the American Cancer Society have advised patients to avoid the burning rays (UVB 290 to 320 nm) of the sun from 10 AM-3 PM. The warning time is meant to encompass 2 hours before or after local solar noon on standard (12 PM) or daylight savings (1 PM) time. Peak sunlight (local solar noon) is about 1 PM for those on daylight savings time and near the center of their time zone (e.g., Chicago). Data collected from a Robertson-Berger (R-B) UVB meter1 since 1983 in a western suburb of Chicago confirms that maximum UVB exposure on a clear sunny summer day usually occurs at 1 PM.

Conversion of the data for UVB intensity to a minimal erythema dose (MED) gives an approximate time of 19 minutes (704 units) for June 21, 1992 (a clear, sunny day). This number decreased only 15% to 595 units by 3 PM and 40% to 423 units by 4 PM (Central Daylight Time). Put another way, between 3:00 and 3:30 PM the UVB dose is still 85% of maximum and 60% of maximum from 3:30 to 4 PM; data were collected as 30-minute totals.

After 4 PM UVB decreased dramatically because of the longer path taken by the sun's rays through the UV-absorbing atmospheric air mass as the sun's angle of elevation above the horizon decreases. UVB is negligible when the solar elevation angle drops below 30 degrees. Between 3 PM and 4 PM on June