Chronic Septic Bursitis Caused by Dematiaceous Fungi

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eptic bursitis is a commonly encountered condition. Because of the location of the olecranon and prepatellar bursae, they are the most frequently affected sites.¹ The majority of cases of septic bursitis are due to bacterial pathogens, particularly gram-positive organisms (*Staphylococcus aureus* and streptococcal species). More recently, reports have appeared in the literature of chronic infectious bursitis caused by fungi and occurring in both immunocompromised and healthy immunocompetent individuals.

We report the first case of chronic, isolated, septic olecranon bursitis caused by *Fonsecaea pedrosoi*, review 3 other cases of bursitis caused by dematiaceous fungi, and discuss treatment.

CASE REPORT

A 65-year-old Hmong woman was seen in the rheumatology clinic for evaluation of left olecranon bursitis. She reported a history of trauma to the left elbow approximately 2 years earlier. Since then she had recurrent drainage of purulent material from the olecranon bursa. She had been treated with numerous antibiotics without relief.

She occasionally was febrile, but this was often felt to be due to other infectious causes. On examination there was evidence of a thickened left olecranon bursa with a fluctuant mass 4 cm in diameter. There was no evidence of overlying cellulitis, erythema, or lymphadenopathy. The left elbow had a full range of motion without pain. Laboratory tests included a normal white blood cell count and differential count. A plain film of the left elbow revealed soft-tissue swelling over the olecranon process, but the joint spaces were well maintained.

The left olecranon bursa was aspirated of 4 mL purulent bloody material. There were greater than 100,000 nucleated cells per cubic millimeter with 99% polymorphonuclear neutrophils. Direct smear revealed many polymorphonuclear neutrophils, as well as septate mycelium. The organism was subsequently identified as a dematiaceous fungus, *F pedrosoi*. No acid-fast bacilli or mycobacteria were found. One day later the bursa was reaspirated of

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2 mL purulent bloody material. All blood cultures were negative.

It was recommended that the patient begin treatment with oral terbinafine and undergo repeat bursal drainage as necessary. An orthopedic surgery consultation was also obtained for possible bursectomy if resolution did not occur with systemic antifungal therapy. The patient did not return to the clinic, and there was no subsequent clinical follow-up.

DISCUSSION

Septic olecranon and prepatellar bursitis is usually due to infection with gram-positive organisms and is treated with bursal aspiration and appropriate antibiotic therapy. More recent reports have described infectious bursitis caused by mycobacteria, algae, and fungi that may occur in immunocompromised or immunocompetent individuals.

The dematiaceous fungi are a large and heterogeneous group of filamentous molds that have in common the presence of brown melanin or melanin-like pigments in the cell walls of the hyphae or conidia or both. Important human pathogens include *Alternaria* species, *Exophiala* species, *F pedrosoi*, and *Phialophora* species. These organisms are found in soil, wood, and decomposing plant debris. They cause a spectrum of disease ranging from localized cutaneous or subcutaneous disease to disseminated and invasive disease with cerebral involvement associated with high

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mortality rates. Infection results from traumatic implantation and is often seen in immunocompetent hosts.²

Fonsecaea is a filamentous fungus that grows slowly and produces velvety, cottony colonies on potato dextrose agar at 25° C. It develops septate, dark brown hyphae and conidiophores that highly branch at apices.³ In humid, tropical areas, *F pedrosoi* accounts for more than 90% of cases of chromoblastomycosis, a slowly progressive infection of skin and subcutaneous tissues. The initial lesion is a small, painless papule that develops at the site of traumatic implantation. If left untreated, the papule will enlarge to form a nodule. Established infection later manifests as large, hyperkeratotic, verrucous lesions, most commonly on lower extremities. Systemic invasion is uncommon.

This appears to be the first reported case of isolated septic olecranon bursitis caused by infection with F pedrosoi. Three other cases of chronic bursitis caused by other dematiaceous fungi have been reported. A healthy 77-year-old man developed infection of the infrapatellar bursa caused by Phialophora richardsiae. He was treated with bursal irrigation, intravenously administered amphotericin B, and then oral itraconazole, with cure.⁴ An immunocompromised 62-year-old man receiving immunosuppressive therapy for Wegener's granulomatosis developed olecranon bursitis caused by Exophiala oligosperma. Weekly aspiration of bursal fluid and injection of amphotericin B achieved a cure after 10 months.⁵ Finally, a 54-year-old woman developed septic olecranon bursitis and underlying osteomyelitis secondary to infection with Phaeoacremonium species. This was treated by bursectomy and bone débridements.6

Managing Dermatiaceous Fungal Infections

The management of patients with infection caused by dematiaceous fungi is based on clinical experience without large clinical trials. Cryosurgery alone may be effective for small lesions. Itraconazole is an effective antifungal agent for chromoblastomycosis, especially when combined with liquid nitrogen. However, not all cases of *F pedrosoi* respond to this treatment. Oral treatment with terbinafine led to mycological cure in a large percentage of cases of infection with *F pedrosoi*.⁷ In addition, 4 patients with long-standing chromoblastomycosis caused by *F pedrosoi* that failed antifungal treatment achieved a response with combination treatment of itraconazole and terbinafine.⁸

CONCLUSIONS

Clinicians need to be aware of the expanding spectrum of infectious agents that have been reported to cause septic bursitis. Although the majority of cases of septic bursitis are still due to gram-positive organisms, unusual presentations (including a prolonged history of symptoms) and lack of response to usual therapeutic agents should prompt evaluation for other etiological agents.

AUTHOR'S DISCLOSURE STATEMENT AND ACKNOWLEDGMENTS

The author reports no actual or potential conflict of interest in relation to this article.

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