

beats per minute. She had marked jugular venous distension, a diffusely enlarged thyroid gland, and no lid lag or exophthalmos. Lung bases were dull to percussion with decreased breath sounds. A 2/6 holosystolic murmur was heard at the apex with no third heart sounds. The abdomen was distended with massive ascites that yielded a transudative fluid. Laboratory studies revealed thyrotoxicosis (serum thyrotropin <0.01  $\mu$ IU/mL).

Electrocardiography showed atrial fibrillation. Chest radiographs showed cardiomegaly and bilateral moderate pleural effusions. Ventilation-perfusion lung scanning was normal. Chest computed tomography and echocardiography showed normal pericardium and severe pulmonary hypertension (60 mm Hg), with marked dilatation of the right atrium and ventricle, and moderate mitral and tricuspid regurgitation. There was paradoxical motion of the septum and normal LV function. Cardiac catheterization showed normal coronary arteries and confirmed the echocardiographic findings (wedge pressure, 23.2 mm Hg; right ventricular pressure, 56/18 mm Hg). Rapid volume expansion by normal saline infusion ruled out constriction. After treatment (furosemide, propranolol, propylthiouracil), she had a rapid diuresis and the anasarca resolved. She remains euthyroid and well 4 years after presentation.

Because of the positive effects of triiodothyronine on cardiac function (2), overt heart failure in thyrotoxicosis is unexpected (1). Occasionally, patients develop primarily systolic heart failure because of the toxic effects of thyroid hormones on the myocardium or chronic tachycardia (1). In contrast, predominantly right-sided heart failure in thyrotoxic patients with preserved left ventricular function is rare (Table). It can be due to myocardial dysfunction of the right ventricle or to increased afterload. Review of the literature supports the second alternative.

Pulmonary artery pressure in these patients (including our patients) decreased following resolution of the hyperthyroid state (Table). The normalization of pulmonary artery pressure and resolution of all signs of heart failure with treatment of the thyrotoxicosis support a causal association. Hyperthyroid rats have been observed to develop ventricular hypertrophy affecting predominantly the right ventricle (8). A study of 4 thyrotoxic patients revealed that all patients had clinically "silent" pulmonary hypertension that decreased about 35% when they were euthyroid (6). Another study reported mild pulmonary hypertension ( $29 \pm 6$  mm Hg) in 35% of patients with recently diagnosed hyperthyroidism (9). Pulmonary artery pressure in these patients correlated with serum thyrotropin and free thyroxine levels ( $r = 0.8$ ;  $P < 0.001$ ), but was normal in the euthyroid control group. The mechanism, however, remains unclear (10). Nonetheless, hyperthyroidism should be considered in the differential diagnosis of right-sided heart failure with pulmonary hypertension.

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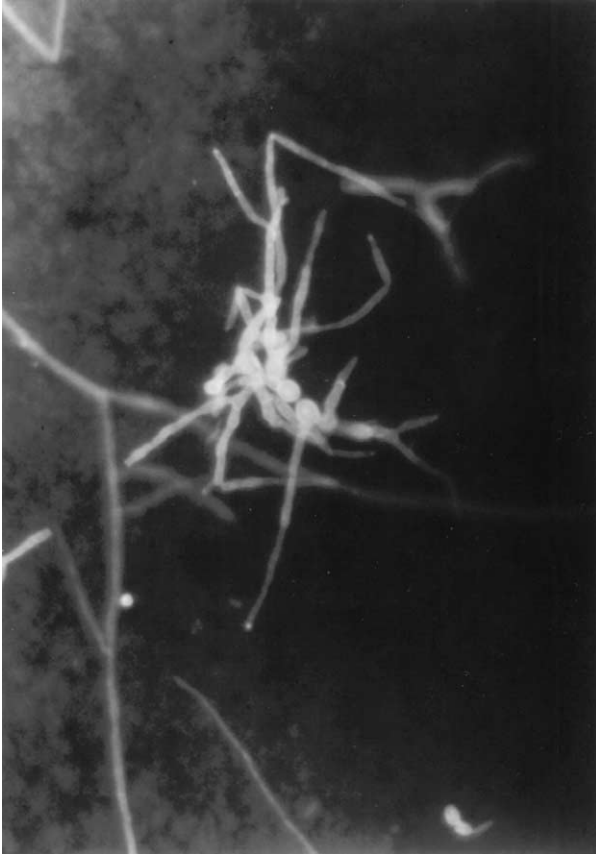
## PHIALOPHORA RICHARDSIAE BURSTITIS TREATED MEDICALLY

### To the Editor:

Although *Staphylococcus aureus* is responsible for most cases of septic bursitis, fungi are rarely isolated (1). *Phialophora richardsiae*, a dematiaceous fungus, was first reported as a cause of human infection in 1968 (2). There has been one case of septic bursitis caused by *P. richardsiae* (3). The dearth of information on antifungal susceptibility for *P. richardsiae*, and the favorable response to excision, have made surgery the treatment of choice (4). We report a case of septic bursitis with contiguous cellulitis caused by *P. richardsiae* that was treated successfully with systemic antifungal agents and irrigation of the bursa.

A healthy 77-year-old man presented with chronic swelling below the right knee. On examination, the infrapatellar bursa was markedly enlarged without warmth or erythema. Microscopic examination of fluid aspirated from the bursa revealed abundant neutrophils. Gram's stain and bacterial culture were negative. A presumptive diagnosis of "Carpenter's knee" was made. Methylprednisolone was instilled into the bursa to reduce inflammation.

The infrapatellar bursa subsequently diminished in size; however, 2 months later the patient presented



**Figure 1.** Fungifluor stain of infrapatellar bursa fluid (original magnification  $\times 40$ ).

with redness, pain, and swelling of the bursa. The infrapatellar bursa was enlarged with overlying erythema and warmth. There was contiguous extension to the lateral calf with edema and skin pustules. A bursal aspirate yielded 15 mL of purulent material. Microscopy revealed abundant neutrophils; however, septate hyphae were noted on Gram's stain and subsequently confirmed by Fungifluor stain (Figure 1). The patient was hospitalized for treatment of a locally invasive fungal infection.

The bursal aspirate, an aspirate of a superficial pustule, and a punch biopsy specimen grew *P. richardsiae* (Figure 2). Microscopic examination of the skin specimen showed dermal neutrophilic infiltrate and perivascular lymphohistiocytic infiltration. Fungal blood cultures were negative.

Treatment with intravenous amphotericin B was instituted. Erythema and edema decreased rapidly. The bursa was irrigated with normal saline through a draining tract. Neither incision and drainage nor debridement was undertaken given the clinical improvement. Following 2 weeks of amphotericin, oral itraconazole was administered for 3 months, which led to resolution of the infection. Minimum inhibitory concentrations (performed in the laboratory of Dr. Michael Rinaldi, Department of Veterans Affairs Mycology Reference Laboratory, South Texas Veteran's Health Care System) were as follows: amphotericin B, 0.25  $\mu\text{g}/\text{mL}$  at 24 hours and 0.5  $\mu\text{g}/\text{mL}$  at 48 hours; itraconazole, 0.25  $\mu\text{g}/\text{mL}$  at 24 hours and 0.5  $\mu\text{g}/\text{mL}$  at 48 hours; voriconazole, 0.25  $\mu\text{g}/\text{mL}$  at 24 hours and 0.5

$\mu\text{g}/\text{mL}$  at 48 hours; and posaconazole, 0.06  $\mu\text{g}/\text{mL}$  at 24 hours and 0.13  $\mu\text{g}/\text{mL}$  at 48 hours.

*Phialophora richardsiae* rarely causes infection in humans. Subcutaneous granuloma, the most common clinical presentation, is characterized by a solitary, painless, well-encapsulated, subcutaneous lesion on the extremities without overlying skin abnormalities or regional adenopathy (4). Histologic examination typically demonstrates granulomas with central necrosis.

The extension of the infection from the bursal space into adjacent soft tissues, confirmed by culture of both a skin biopsy specimen and an aspirate from a superficial pustule, make our patient atypical. Although it is plausible that this soil organism was introduced into the bursa during the patient's daily activities, it is notable that he had recently received a corticosteroid injection. There has been one report of *P. richardsiae* infection following corticosteroid injection (5). In our patient, it is unknown whether the fungus was in the bursa before the injection, was inoculated into the bursa after the injection, or was introduced into the bursa by the injection. Contamination of the steroid preparation, as proposed previously (5), seems less likely. Regardless, the effects of the local steroid almost certainly contributed to the contiguous spread of infection.

The most salient feature of this case is that surgical excision was not required. Most previous cases of subcutaneous granulomas achieved cure through surgical excision alone. Antifungal therapy has been used in one case of subcutaneous granuloma as an adjunct to surgical excision (6). That patient died of unrelated causes. Two reports of invasive infection (endocarditis and osteomyelitis) involved treatment with systemic antifungal agents but yielded indeterminate results (7,8). Although we considered surgical intervention in this patient, we determined that empiric antifungal therapy with close



**Figure 2.** Lactophenol cotton blue stain of organisms obtained from culture specimen (original magnification  $\times 40$ ).

clinical monitoring was a reasonable approach given his intact immune system. He showed rapid clinical improvement and has not had recur-

rence of infection 1 year later. In select immunocompetent patients with *P. richardsiae* infection, systemic antifungal therapy and percutaneous

drainage may obviate the need for surgery.

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