gynecomastia, whereas indinavir has already been described as a cause of breast enlargement in women. One of the patients we described and the two previously reported women with indinavir-associated breast enlargement developed redistribution of body fat, a feature of the syndrome of peripheral fat wasting (lipodystrophy), hyperlipidemia, and insulin resistance that has been recently described in patients who received HIV-1 protease inhibitors [1]. One of our patients had associated lipid abnormalities; repeated fasting blood glucose levels have remained within normal limits.

Gynecomastia is a benign glandular enlargement of the male breast. This entity has been associated with the use of various drugs including anabolic steroids; antimicrobials (isoniazid, ketoconazole, metronidazole); cardiovascular, antineuropathy, and psychoactive medications; and certain chemotherapeutic agents [6]. Breast enlargement, although uncommon, should be included among the adverse effects associated with use of protease inhibitors in both men and women. The mechanism for this side effect is unknown, but does not appear to be associated with any obvious endocrine abnormalities. Whether this effect is exclusively due to indinavir is a matter of speculation, and it remains to be determined if gynecomastia is another feature of the syndrome of HIV-1 protease inhibitor–associated peripheral lipodystrophy.

References


In April 1997, diarrhea and fever recurred. He was admitted to the hospital with severe dehydration. Testing for sepsis was negative except for a positive result for C. difficile toxin in his stool. At this point, tacrolimus was discontinued. In addition, he received oral vancomycin solution, with complete resolution of symptoms in the next 2 weeks. A repeat stool test was negative for C. difficile toxin. He continues to do well while receiving hemodialysis.

Antibiotic-associated pseudomembranous colitis became a major clinical problem in the 1960s and 1970s, particularly with the use of broad-spectrum agents such as lincomycin and clindamycin, which caused diarrhea in ~10% of patients and pseudomembranous colitis in 1% [1].

In 1978, C. difficile was identified as the source of cytotoxin responsible for antibiotic-associated pseudomembranous colitis [2, 3]. It is now established as the most common nosocomial enteric pathogen causing pseudomembranous colitis, antibiotic-associated colitis, and antibiotic-associated diarrhea [4]. Antibiotic treatment, older age, and underlying illness are the major risk factors for the development of symptomatic disease [4]. In the last 3 years, there have been reports of pseudomembranous colitis following treatment with clarithromycin, a newer macrolide antibiotic indicated for eradication of Helicobacter pylori in peptic ulcers [5], and with third-generation cephalosporins [6].

Tacrolimus is a newer macrolide used for immunosuppression, and there have been no previous published reports of C. difficile diarrhea associated with its short-term use. The manufacturer of Prograf has received isolated reports of C. difficile–induced diarrhea in association with Prograf use (personal communication, P.C. Blahunka, Medical Information Department, Fujisawa Healthcare, 1998). This case serves as a warning of the need for attentiveness to the side effects of macrolide molecular structure.
Pacemaker-Induced Endocarditis Due to Propionibacterium acnes

Propionibacterium acnes and other Propionibacterium species are branching, gram-positive, anaerobic bacilli that are part of the normal human microflora of the skin, conjunctiva, external ear, sebaceous follicles, and mouth and upper respiratory tract [1–5]. Although P. acnes is of low virulence, it has been identified as the etiologic agent in a variety of infections including CNS shunt infections, brain abscesses, endophthalmitis, neurosurgical wound infections, and rarely, pulmonary infections [6–10]. Invasive disease usually involves a foreign body [5]. When identified in blood cultures, P. acnes is generally considered a skin contaminant. To our knowledge, we describe herein the first case of infectious endocarditis due to P. acnes associated with a pacemaker.

A 78-year-old man was admitted to Winthrop-University Hospital (Mineola, New York) for evaluation of syncope and intermittent fever with chills, night sweats, fatigue, and malaise of 6 months’ duration. The patient’s medical history included hypothyroidism, hypertension, and implantation of a permanent pacemaker in 1982. After cardiac surgery, the patient was sent home with iv ampicillin/gentamicin to complete a 6-week course of therapy. He had an uneventful recovery.

Admission laboratory studies included the following values: WBCs, 11.4/mm³; hemoglobin, 151 g/L; hematocrit, 45%; platelets, 50,000/mm³; and creatinine, 0.8 mg/dL. Results of a urinalysis were negative. A chest radiograph showed no infiltrate or effusion.

On physical examination, the patient was alert, oriented, and in no acute distress. A pulse generator was palpable at the left upper anterior chest wall, but there was no inflammation or tenderness. The heart rate was normal, the rhythm was regular, and first and second heart sounds (S1/S2) were normal; no murmur was noted. The remainder of the physical examination findings were noncontributory. There were no ocular hemorrhages, Janeway lesions, splinter hemorrhages, or Osler nodes, and there was no splenomegaly.

References

Figure 1. Pacemaker leads surrounded by organized, laminated clot containing Propionibacterium acnes, from a patient with endocarditis.