specific gravity of $<1010$ [3]. Failure to respond to vasopressin confirms the diagnosis of nephrogenic diabetes insipidus. A variety of drugs have been reported to cause nephrogenic diabetes insipidus, including amphotericin B [4]. The clinical course in our patient, with prompt resolution of symptoms after cessation of the drug proved that amphotericin B was the underlying cause. The exact mechanism by which amphotericin B causes nephrogenic diabetes insipidus is unclear. Successful use of indomethacin in a similar case suggests that renal prostaglandins and/or modulation of phosphodiesterase activity are involved [5]. Considering the potential adverse effects of indomethacin in a patient with chemotherapy-induced mucosal damage and thrombocytopenia, we favored other treatment options. First, we tried fluconazole, which proved to be a safe and effective substitute in leukemia patients with chronic disseminated candidiasis and serious amphotericin B–related toxicities [6]. Because neutropenic fever recurred during fluconazole therapy we used liposomal amphotericin B, according to a recent report [7], as a second option with success. Our case underscores the value of liposomal amphotericin B as an alternative therapy in amphotericin B–induced nephrogenic diabetes insipidus.

### Septic Shock Caused by *Staphylococcus lugdunensis*

Coagulase-negative staphylococci are commensal organisms of the human skin. They have recently been recognized as major nosocomial pathogens. This has prompted more interest in their detailed characterization. We describe a patient who developed septic shock caused by *Staphylococcus lugdunensis* acquired through platelet transfusion.

A 71-year-old man with a history of pancytopenia secondary to myelodysplasia presented to the emergency room complaining of shortness of breath and rectal bleeding of several hours’ duration. He denied cough, fever, or malaise. He also had a history of prostate cancer that had been treated with radiation. During the past year he had had multiple admissions to the hospital because of severe anemia caused by rectal bleeding. He had refused further work-up for the bleeding, which was believed to be related to radiation colitis.

Physical examination on admission was positive for pallor, tachycardia, and rectal bleeding. He did not have an iv catheter at the time of admission. Chest radiography was within normal limits. Laboratory evaluation revealed the following values: WBCs, 900/mm³ with 17% neutrophils; hematocrit, 16%; and platelet count, 8,000/mm³. The patient was admitted to the intensive care unit, and he received platelets and two units of packed red blood cells. While receiving the second unit of red blood cells, he became febrile (temperature, 104°F). Later he become hypotensive and developed respiratory failure. Blood for cultures was obtained; he was intubated, connected to mechanical ventilation, treated with broad-spectrum antibiotics, and supported with vasopressors. The transfused blood was retyped and cultured. The platelets were also cultured. Two days later, the blood cultures and the platelet culture yielded *S. lugdunensis* that was susceptible to penicillin. A few days later the patient had recovered, after completion of therapy with vancomycin (due to concern of a penicillin allergy). There was neither evidence of allergic reaction to the blood products nor of any other source of infection. The transfused blood was sterile. There were no reports of any other infected blood products in the blood bank.

Platelet components contaminated with bacteria are an important source of transfusion-associated bacterial sepsis, but the rates are low (0.08%) [1]. Although coagulase-negative staphylococci are usually considered avirulent or usual contaminants of blood cultures, their roles as causes of disease are well established in certain clinical situations, particularly when prosthetic material is present or in profoundly immunocompromised hosts. Wade et al. [2] reported *Staphylococcus epidermidis* as a frequent pathogen in patients with severe granulocytopenia. Winston et al. [3] reported coagulase-negative staphylococci as the most common cause of bacteremia in patients receiving immunosuppressive therapy.

*S. lugdunensis*, first described in 1988, is a coagulase-negative species of the genus *Staphylococcus*, and it may represent up to 10% of the coagulase-negative staphylococci [4]. However, its role as a pathogen has been well established, and, although the majority of clinical isolates represent colonization [4], it can cause serious infections [5]. Indeed, it has been suggested that *S. lugdunensis* may be more pathogenic than other species of coagulase-negative staphylococci [6]. However, this is the first report of *S. lugdunensis* as a cause of septic shock.

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Mycobacterium neoaurum—An Unusual Cause of Infection of Vascular Catheters: Case Report and Review

*Mycobacterium neoaurum* was initially isolated from soil by Tsukamura in 1972 [1]. *M. neoaurum* has seldom been encountered in human disease; only two cases of infection have been reported, both from Australia [2, 3]. We report the first case of *M. neoaurum* infection in the United States. In all three cases (2, 3 and the current case), the presentation consisted of a febrile syndrome in a patient with a long-term indwelling Hickman catheter with mild or absent inflammation and no drainage at the catheter insertion site. *M. neoaurum* was isolated from multiple cultures of blood obtained through the Hickman catheter in each case.

A 46-year-old man with a history of primary pulmonary hypertension presented with a 2-month history of recurring fevers (temperature, to 103°F), diaphoresis, and rigors occurring every other night. A Hickman catheter had been placed 10 months previously for continuous infusion of prostacycline. In between febrile episodes the patient felt well. Physical examination revealed a well-appearing man with a hyperdynamic precordium, palpable pulmonic second sound (P2), fixed split second heart sound (S2), and a loud holosystolic murmur at the left lower sternal border, unchanged from previous examinations. The Hickman catheter insertion site was mildly indurated with 2 cm of surrounding erythema and mild tenderness; no drainage could be expressed. Physical examination findings were otherwise unremarkable. The WBC count was 4,700/mm³ with a normal differential.

The patient had undergone two previous evaluations for his fevers; these had included four sets of peripheral blood cultures that had demonstrated no growth. He had also received several courses of empiric antibiotics: two courses of 3 days each of intravenous vancomycin and ceftriaxone administered through the Hickman catheter, and a 10-day course of oral amoxicillin/clavulanate. With each treatment he would defervesce, but the fevers would recur within 1 week after antimicrobials had been discontinued. Careful questioning on his third hospital admission revealed that the patient’s wife always flushed his Hickman catheter every other night, and his fever syndrome always occurred 3 to 4 hours thereafter. He had had no fevers on the nights that his Hickman catheter was not flushed. Peripheral blood was again obtained for cultures which remained without growth, but two cultures of blood obtained from the Hickman catheter yielded a rapidly growing scotochromogenic mycobacterial species after 48 hours, which was subsequently identified as *M. neoaurum* by use of biochemical testing and chromatography (University of Iowa Clinical Microbiology and Hygienic Laboratories) (figure 1). Identification of the mycobacterium as *M. neoaurum* was confirmed by the Centers for Disease Control and Prevention Mycobacterial Identification Laboratory. The Hickman catheter was removed and no antibiotics were administered. Surveillance cultures of blood obtained after Hickman catheter removal remained negative. The patient was afebrile without further symptoms at 4 months’ follow-up.

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