Invasive Streptococcus pyogenes infection in a surgeon after an occupational exposure

prophylaxis; the authors concluded that prophylaxis was not warranted for patients undergoing TEE. Görge et al. [4], however, did recommend its systematic use based on an observed incidence of bacteremia of 17% after TEE. Other authors have documented rates of post-TEE bacteremia lower than 5%, leading to varying recommendations [5–11]. The lack of studies of risk factors for developing post-TEE endocarditis and our recent observation, lead us to strongly recommend the use of antibiotic prophylaxis in all patients at high risk of endocarditis who undergo a TEE, i.e. patients with previous endocarditis or those with a prosthetic valve.

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Invasive *Streptococcus pyogenes* infection in a surgeon after an occupational exposure

Surgeons and other healthcare workers are at risk of developing an occupational infection caused by viral blood-borne pathogens such as hepatitis B (HBV) and C (HCV) viruses, and human immunodeficiency virus (HIV), and universal precautions for preventing these troublesome professional diseases have been properly provided [1]. Much less attention is paid to bacterial pathogens such as *Streptococcus pyogenes*, which can cause severe infections as well. We report the first—to our knowledge—documented invasive streptococcal infection occurring in a surgeon following accidental injury by a medical instrument.

A febrile 50-year-old woman with a voluminous uterine leiomyoma was admitted to our hospital for a suspected acute abdominopelvic pathology; she underwent hysterectomy and bilateral adnexectomy. The peritoneal cavity appeared to be filled with purulent material. During surgery, a 47-year-old obstetric–gynecologic surgeon accidentally pricked the inner side of his right middle finger with a pointed lancet. Unfortunately, a plan of action for the control of bacterial infection following occupational exposure did not exist in our hospital at that time, so the surgeon was included in a protocol for prevention of HBV, HCV and HIV transmission alone. Starting the following day, he noted the sequence of: (1) a small bullous lesion on his injured finger; (2) a few hours later, an erythematous edge all around the lesion, followed by serosanguinous discharge and evidence of a necrotic base; (3) within 36 h, fever of up to 39 °C with shivers, and a red line of an ascending lymphangitis on his right arm, for which reason he began self-treatment with oral amoxycillin; (4) within 48 h, a right axillary lymphadenopathy. At this time, our colleague was admitted to the infectious disease unit with the diagnosis of bullous cellulitis. Physical and laboratory evaluation revealed: temperature, 38.8 °C; blood pressure, 150/85 mmHg; erythrocyte sedimentation rate, 56 mm/h; leukocyte count, 5040/mm^3. As the culture of peritoneal liquid intraoperatively obtained from the index patient grew a *S. pyogenes* strain susceptible to all the antibiotics tested (ampicillin, erythromycin, imipenem, penicillin G, piperacillin, teicoplanin, and vancomycin), we initiated intravenous therapy with penicillin G 4 mU every 4 h plus clindamycin 900 mg every 8 h. Such an aggressive therapeutic approach, recommended for severe streptococcal skin infections [2], was chosen because of the rapid progression of the disease. Defervescence was achieved within the second day, and the other physical signs progressively improved by the sixth day, when the surgeon was discharged with oral clindamycin 900 mg

every 8 h plus intramuscular ceftriaxone 1 g/day for 5 more days.

Blood cultures obtained on admission were negative, but a culture from the finger lesion yielded a *S. pyogenes* strain which showed the same antimicrobial susceptibility pattern as the woman's isolate. Antistreptolysin O and streptozyme test titers were 255 Todd units and 1 : 200, respectively, on admission, and, 4 weeks later, 310 and 1 : 400. The woman's and surgeon's isolates were sent to the National Health Institute for typing. Both strains were found to be T-and M-non-typable, producers of serum opacity factor and protease, and negative for the genes encoding erythrogenic toxin (*speA* and *speC*). The macrorestriction profile, studied by pulsed-field gel electrophoresis (PFGE) after digestion of the DNA by *Sma*I according to Stanley et al [3], was identical in both strains (data not shown). DNA macrorestriction endonuclease analysis using PFGE to confirm the spread of *S. pyogenes* among close contacts of infected patients has already been used by other researchers [4].

S. pyogenes is primarily a common agent of pharyngotonsillitis, but an increasing frequency of severe invasive infections due to this pathogen, such as toxic shock syndrome (TSS), necrotizing fasciitis, and other skin and soft tissue infections, has been observed [5]. Healthcare workers are aware of the occupational risk of developing HBV, HCV or HIV infection, but bacterial pathogens such as *S. pyogenes* are a well-known serious hazard in medical practice as well. In 1847, Kolletschka, a professor of medical jurisprudence in Vienna, died of presumptive streptococcal septicemia after pricking a finger during a necropsy on a victim of puerperal fever. In the first decades of this century, similar accidents have been described in surgeons, nurses and pathologists after finger pricks or scratches during attendance on a septic patient [6]. Much more recently, a TSS has been observed in a fire-fighter exposed during prehospital resuscitation to the secretions of an *S. pyogenes*-infected child [7], and invasive infections (bullous cellulitis, ascending lymphangitis, necrotizing fasciitis) have been developed by physicians who came in contact with body fluids or secretions of patients with streptococcal TSS [8], or who scratched a finger with the needle used in a patient with group A streptococcal sepsis [9].

To our knowledge, we have documented the first invasive streptococcal infection in a surgeon following professional injury by a medical instrument. The case we describe demonstrates that: (1) surgeons and other health workers must adhere to the well-known isolation precautions for avoiding transmission of viral and bacterial pathogens when caring for all patients [10]; and (2) the availability of a plan of action to use in the case of occupational exposure would be important for prompt diagnosis and early aggressive therapy in order to minimize the clinical consequences of an invasive streptococcal infection.

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Comparison of the Bactec 460TB system and the Bactec MGIT 960 system in recovery of mycobacteria from clinical specimens

The recent increase in tuberculosis and other mycobacterioses all over the world [1] has led to a search for faster and more accurate detection and identification procedures. Despite the great advances in direct detection with molecular biology methods [2], culture is still fundamental for mycobacterial detection, species identification/confirmation and drug susceptibility testing. Solid media such as Löwenstein–Jensen or Middlebrook agar have been used for such purposes, but may take several weeks to become positive. Recently introduced media such as the Middlebrook 7H12 (12B medium) and the modified Middlebrook 7H9 used in MGIT (MGIT medium)