agar in an atmosphere of 5% CO2 and incubation for 5 days on chocolate culture remained negative for year before hospital admission. Urine ally associated with UTI during the could not recall any symptoms usu-
no underlying urologic condition and patients in previous reports. He had reported here differed from those of
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Mycobacterium chelonae Wound Infection after Liposuction

To the Editor: We recently investigated a case of Mycobacterium chelonae abdominal wound infection after liposuction performed under local anesthesia at an outpatient medical office. Our aim was to determine whether other cases of atypical mycobacterial infections had previously occurred after liposuction. M. chelonae is widely distributed in soil and water, including tap water. Atypical mycobacterial infections have been associated with skin and soft tissue infections, including infections after cosmetic surgeries, and outbreaks have been documented (1–4). Previously reported potential sources of liposuction equipment contamination have been inadequate disinfection or sterilization after rinsing of liposuction equipment with tap water, tap water used in cleaning liposuction cannulae, or the quaternary ammonium solution used to disinfect liposuction equipment (2,4). Increased numbers of procedures performed in freestanding medical centers (not connected with hospitals) that are not routinely monitored by infection control committees or equivalent oversight bodies may contribute to atypical mycobacterial infection (1).

Our investigation showed that proper cleaning, disinfection, and sterilization of liposuction equipment and other infection control issues at this medical office were concerns.

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Except for the physician, only unlicensed medical assistants worked at this office. This staff had been trained to clean and sterilize liposuction equipment, but no written procedures existed for processing reusable liposuction equipment, no logs were kept of autoclave use for sterilization, and preventive maintenance checks and verification of sterility on the autoclave by using biological indicators as recommended by the manufacturer were not performed. The office did not have any general written infection control policies. Office staff mixed leftover solutions from open small bottles of povidone iodine and placed this mixture into larger containers. Staff stored wet alcohol-soaked cotton balls in multisuse containers for wiping tops of multidose vials instead of using individual alcohol prep pads; and 70% isopropyl alcohol solution from an open nonsterile bottle was used instead of sterile irrigation solutions to dislodge tissue from the ports during the procedure.

Case finding and surveillance of acid-fast bacilli results routinely reported to our public health tuberculosis program did not indicate any other cases of postliposuction wound infections caused by atypical mycobacteria associated with this office. Laboratory testing of environmental samples, including tap water and faucet aerator samples, also did not indicate a source for M. chelonae in this outpatient office. This case was likely an isolated occurrence in which the case-patient acquired infection through an environmental source unrelated to this office. However, because of the infection control concerns observed in this office and because the incubation period for M. chelonae can be as long as 5 months (2), the physician was advised to develop infection control policies and procedures; develop protocols for cleaning, disinfecting, and sterilizing liposuction equipment in accordance with the manufacturer’s recommendations; ensure autoclave sterility by using biological indicators; educate office staff about basic infection control practices and use of aseptic techniques; and notify public health officials of any further infections post-liposuction.

Risk factors that cause or contribute to infectious disease outbreaks in outpatient settings include inadequate cleaning, disinfection, sterilization, and storage of instruments and equipment; inappropriate use of barrier equipment, such as gloves, by healthcare personnel; inadequate handwashing practices by healthcare workers; failure to use aseptic techniques; and lack of familiarity with established infection control practices by ambulatory care personnel (5). Also, in the outpatient setting, responsibility for implementing an infection control program usually is not assigned to a specific person (5), and outpatient medical offices are not routinely monitored by oversight bodies or infection control committees as are hospitals and outpatient surgical centers (6).

The California Business and Professions Code requires that outpatient surgery settings using anesthesia, other than local anesthesia or peripheral nerve blocks, be accredited by an oversight body. However, because this facility used only local anesthesia, it did not fall under this code of regulations for facility accreditation, oversight, certification standards, and quality assurance for general public health safety and welfare.

Lack of adherence to basic infection control principles, specifically in outpatient settings, has resulted in outbreaks (1,7–10). Our findings at this medical office further highlight the unaddressed infection control problems in outpatient settings. Because of insufficient oversight for the outpatient setting, professional organizations, state medical boards, and federal and state authorities should consider the need to systematically address infection control standards and monitoring tailored for this setting. As more healthcare procedures move to the outpatient setting, ensuring appropriate infection control practices can prevent outbreaks.

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Influenza A Pandemic (H1N1) 2009 Virus and HIV

To the Editor: The effects of influenza A pandemic (H1N1) 2009 virus infection in HIV-infected patients are unknown. We describe an HIV-infected patient with severe pandemic (H1N1) 2009 virus infection.

The patient was a 37-year-old, HIV-positive, former intravenous drug user in a methadone-substitution program. She had a history of smoking, hepatitis C, and mild chronic obstructive pulmonary disease not requiring treatment. Since 2007 her viral load had been <50 HIV RNA copies/mL, for which she received tenofovir, emtricitabine, and lopinavir/ritonavir. Her CD4 count in March 2009 was 542 cells/μL (25%).

On June 24, 2009, the patient entered the Hospital Universitario La Paz after 3 days of dyspnea and fever, without cough or sputum. Temperature was 39°C, blood pressure 118/74 mm Hg, pulse rate 110 beats per minute, respiratory rate 30 breaths per minute, and oxygen saturation 85% on room air (fraction of inspired oxygen [FiO2] 21%). Chest radiograph findings were consistent with bacterial pneumonia (Figure, panel A). Empirical treatment with clarithromycin and ceftriaxone was started. After full clinical recovery, the patient was discharged on June 30 and prescribed oral clarithromycin and cefixime.

At a routine follow-up visit on July 2, the woman was asymptomatic and had fewer leukocytes and neutrophils, creatine kinase 800 U/L (reference <145), and lactate dehydrogenase 373 U/L (reference <247). On July 3, she returned to the hospital because of dyspnea and high fever (38.5°C). Oxygen saturation was 75%, pO2 33.8 mm Hg (FiO2 21%), and blood pressure 90/54 mm Hg. Chest radiographs showed alveolar infiltrates in the right lower lobe (Figure, panel B). Because her deterioration was attributed to nosocomial infection, she was given meropenem, linezolid, and levofloxacin and was hospitalized. Within the next 96 hours, her condition deteriorated further to drowsiness, hypotension, oxygen saturation 92%, pO2 60.5 mm Hg (FiO2 50%), and new radiographic bilateral alveolo-interstitial infiltrates appeared (Figure, panel C).

On July 7, real-time reverse transcription–PCR of a nasopharyngeal swab confirmed influenza A pandemic (H1N1) 2009 virus (1). Other infectious causes for bacterial and viral pneumonia were excluded. The patient received mechanical ventilation for 7 days and oseltamivir. She was discharged after 21 days. She still had dyspnea after exertion and radiologic sequelae on chest radiograph (Figure, panel D).

No patients or healthcare workers who had had contact with the patient had confirmed pandemic (H1N1) 2009 virus infection. Address for correspondence: Moon J. Kim, Acute Communicable Disease Control Program—County of Los Angeles, Department of Public Health, 313 N Figueroa St, Rm 222, Los Angeles, CA 90012, USA; email: mokim@ph.lacounty.gov


Figure. Chest radiographs of 37-year-old, HIV-positive woman with severe pandemic (H1N1) 2009 virus infection, 2009. A) June 24, alveolar infiltrate in the right lower lobe. B) July 3, minimal pleural effusion, alveolar infiltrate on right lower lobe, and possibly left lower lobe infiltrate. C) July 6, bilateral alveolo-interstitial infiltrates. D) July 29, bilateral peribroncovichacular thickening with fibro-cicatricial changes; conserved lung volumes.