lieve, on the basis of our studies and those of others, that self-disinfecting surfaces such as copper are an important additional tool and a significant step forward in helping to reduce the potentially infection-causing microbial bioloads that exist on clinical surfaces. Indeed, we should ask the question, why select a nonantimicrobial surface when we now know that naturally occurring metals have this intrinsic antimicrobial activity?

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Failure of HIV Postexposure Prophylaxis after a Work-Related Needlestick Injury

To the Editor—Transmission of HIV by occupational needlestick injury (NSI) is a rare event,[1,2] particularly in instances in which a healthcare provider (HCP) receives prompt postexposure prophylaxis (PEP).[3] We report a case in which PEP failed to prevent HIV transmission.

After placement of a central line in a patient with cryptococcal meningitis for whom HIV was recently diagnosed, a HCP accidentally sustained a NSI to the left thumb with the infiltration needle (25 gauge). The exposure site was cleansed thoroughly with soap and water, and the HCP was provided an antiretroviral regimen that consisted of lopinavir/ritonavir, zidovudine, and lamivudine at the time the NSI was reported (approximately 6 hours after exposure). However, the first doses of antiretroviral medication were not administered until approximately 18 hours after exposure. Serologic tests demonstrated that the source patient had negative results for hepatitis B and C, and the results of baseline hepatitis and HIV tests for the exposed HCP were also negative. The source patient had received a diagnosis of HIV 1 day before his arrival, and he had never taken antiretrovirals. He was transferred to another hospital and died less than 24 hours after the accident. No additional blood samples could be recovered for further evaluation of the source patient. The exposed HCP reported no high-risk sexual exposure, no intravenous drug use, and not having undergone HIV testing before. On the 10th day following the exposure, the HCP developed diarrhea (5–6 loose stools per day) without nausea or vomiting, which was considered an adverse effect of the PEP regimen. The regimen was changed to tenofovir (TDF), emtricitabine (FTC), and atazanavir, which the HCP continued to receive without experiencing any further adverse effects. Ultimately, the HCP received a total of 4 weeks of PEP. During the PEP period, the exposed HCP reported strict adherence to both regimens, missing none of the doses. His reports of adherence correlated with a controlled weekly pill count. On the 25th day after exposure, the HCP remained asymptomatic and the results of a second ELISA test for HIV were negative. Approximately 60 days after exposure, the HCP developed a dengue-like illness characterized by fever, thrombocytopenia, muscle pain, and fatigue; the physical examination at that time did not note either adenopathy or rash. Antibody tests for dengue had negative results; however, an ELISA for HIV had positive results (67 days after exposure). The HCP's symptoms subsequently disappeared with symptomatic treatment. On day 74 after exposure, a second ELISA test for HIV had positive results and the results of a Western blot assay were indeterminate. At that time an HIV viral load test was ordered, which detected 60,770 copies/mL with a lymphocyte TCD4 count of 672 cells/mL. The HCP reported that from the time of the NSI until the positive HIV test results, he had no sexual contact or other risk factors for HIV infection.

A blood sample for a genotype assay was collected on the 85th day after exposure (57 days after the last dose of PEP antiretrovirals was administered). Resistance mutations sequenced (ViroSeq) for protease were V3I, E35D, S37D, Q61E, L63P, I64V, C67S, H69Y, and V77I, and for reverse transcrip-

tase they were V90I, A98S, D121H, S162C, V179I, Q197K, R211Q, L214F, P272S, R277K, T286A, I293V, E297A, I329V, and Q334P. None of these mutations confer resistance to any antiretrovirals. The HCP received a triple regimen that included tenofovir, emtricitabine, and efavirenz, with adequate tolerance and adherence; 5 months later his viral load was undetectable.

This case illustrates how, despite initiating PEP during the recommended time frame,[4] transmission of HIV by NSI can still occur, and it emphasizes the importance of close follow-up for HCPs who experience occupational exposure to HIV. On the basis of the sequenced mutations, both antiretroviral regimens that were administered to the patient during the 4-week period were effective against the HIV strain with which the HCP was subsequently infected. Because we could not obtain additional samples from the source patient, we could not compare the 2 viral strains genetically. Despite the fact that the HCP denied other risk factors for HIV infection and the fact that he had no signs of exposure to other bloodborne pathogens, the lack of the genetic data is a limitation of this case report.

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