Zeroing in on the Appropriate Management of Occupational Exposures to HIV-1

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As an arrow shot From a well-experienc'd archer hits the mark His eye doth level at. William Shakespeare, Pericles, Act 1, Scene 1

Transmission of human immunodeficiency virus type 1 (HIV-1) in the healthcare setting continues to be a concern for the healthcare community. Although in the tenth year of the HIV epidemic in the United States we have learned a great deal about some aspects of the occupational risks associated with the provision of care for HIV-l-infected individuals, we still lack several important pieces of information regarding these risks.

While several prospective clinical studies have provided information regarding the magnitude of risk for HIV-l infection associated with percutaneous or mucous membrane exposures to body fluids containing HIV-l,' we have yet to gain much insight into factors associated with occupational injuries that influence risks for infection. For example, most of the instances of documented occupational/nosocomia1 transmission of HIV-l described in the literature have resulted from inadvertent occupational exposures to what might be called "hot" needles (i.e., healthcare worker infections resulting from parenteral exposures to needles [or other sharp objects] that had been removed from HIV-l-infected patients or from specimens containing blood from such patients only moments prior to the exposure).¹ Such exposures are also likely to

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produce the most obvious concern among healthcare providers and are, therefore, perhaps the most likely to be reported and carefully documented. Thus, the precise role of the immediacy of the exposure in producing occupational infection remains unclear.

Management of occupational exposures to blood in the healthcare setting would seem, on cursory examination, to be relatively straightforward. Nonetheless, even in this setting, there are significant gaps in the available scientific information base, and, for this reason, the target (i.e., the optimal management of employees sustaining such exposures) remains elusive. In this issue of *Infection Control and Hospital Epidemiology*, Shirazian and colleagues have attempted to provide an important piece of missing information.²

In their study, Shirazian, et al. describe the successful and reproducible detection of both HIV-1 antigens and antibodies directed against HIV-1 from reconstituted dried blood from the surfaces of needles and broken glass. These techniques are of particular interest to *Infection Control and Hospital Epidemiology* readers because responsibility for the development of policies and procedures for managing occupational/nosocomial exposures has often landed directly "in the laps" of hospital epidemiologists, hospital infection control committees and employee health staff.

Since the early days of the HIV-l epidemic, infection control and employee health staff have been concerned about the appropriate management of so-called "source unknown" or "high-risk location" exposures. Elaborate schemes have been developed to attempt a rational (if not scientifically-grounded) approach to this issue.' Data from the article by Shirazian and his colleagues provide another approach to this difficult problem. A second important finding described by Shirazian and colleagues is that dry heat decreases the detectability of both anti-HIV-l antibodies and HIV-l antigen (as measured by enzyme-linked immunosorbent assay [ELISA]).

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Whereas the data from the article by Shirazian and coworkers provide additional arrows for the employee health physician's quiver, the article falls somewhat short of its target. Their study fails to provide clear answers to some of the most relevant questions regarding "source unknown" exposures. Although Shirazian and colleagues demonstrated that anti-HIV-l antibodies and HIV-1 antigens are detectable in the experimental conditions outlined, their data do not address the more relevant issue of the infectivity of the material from which they detected antigen or anti-HIV-l antibodies. Shirazian and colleagues did not attempt to culture HIV-l from the needles and glass used to evaluate their technique. The authors acknowledge that detection of viability of HIV-l would have provided a more reliable estimate of risk.

In the absence of such information, the relevance of their findings remains unclear. HIV-l antigen and/or antibody detection may or may not be sensitive, and, most importantly, specific indirect measures of infectivity. The applicability of this technique will ultimately rest on its sensitivity and specificity in clinical use. Shirazian, et al. have apparently not yet attempted clinical application of the technique. Often, both sensitivity and specificity suffer considerably when a technique is moved from the laboratory to the clinical setting.

Similar studies performed to assess the role of environmental contamination in the occupational/ nosocomial transmission of hepatitis B virus (HBV) may provide a valuable comparison. Several studies have demonstrated persistence of hepatitis B surface antigen (HBsAg) in the healthcare workplace environment³⁻⁵; others have demonstrated the stability of HBsAg when exposed to standard disinfecting agents.⁶ Based on these in vitro antigen detection experiments, many investigators concluded that HBV was extraordinarily refractory to most commonly-used disinfectants.⁶ Nonetheless, because of the lack of an in vitro system to culture HBV, these early studies could not directly address the infectivity of HBV in these samples. In an elegant primate study, Bond and coworkers demonstrated that exposure of HBV to most disinfectants resulted in a significant decrease in infectivity.⁶ In this study the treated HBV samples did not produce infection when injected into primates, despite the persistence of measurable titers of radioimmunoassay-detectable HBsAg in many of the samples.⁶

Because an in vitro system is available to culture HIV-l, data from such cultures would provide important confirmatory evidence for the antigen/ antibody detection methods described by Shirazian, et al. In the absence of such confirmatory data, I would emphasize that detection of HIV-l antigen and/or antibody, either individually or in concert, should not be equated with infectivity. Despite these shortcomings, I would equally emphasize that refinement and additional evaluation of the techniques described by Shirazian, et al. may yield a new approach to, as well as additional flexibility in, the evaluation of "source unidentifiable" exposures.

A more difficult issue raised by Shirazian, et al. is how and when to use the HIV-l antigen/antibody detection technique. As the authors note, the procedure is ideal for use in the setting in which a healthcare worker has sustained an injury with a sharp object contaminated with blood from an unknown source. Shirazian, et al. also advocate use of this procedure in situations in which the source patient for the sample is known but refuses voluntary HIV-l serologic testing. However, the authors also note that the use of the technique in this setting is controversial.

Some states (i.e., Virginia) have enacted statutes that allow HIV-l antibody testing of samples obtained from "source" patients of occupational exposures, even if the source patient refuses to have the HIV-l serology performed voluntarily. In states with similar statutes, the standard ELISA test (performed on serum) will probably be a more reliable measure of infection than the techniques described by Shirazian and colleagues. Conversely, if serum from such a patient cannot be obtained, antigen or antibody detection procedures may provide reasonable alternatives.

In states where such legislation has not been implemented, involuntary testing remains problematic. As advocates for both patients and healthcare workers, the hospital epidemiology community finds itself in an uncomfortable position. As of yet, we do not have an effective therapeutic intervention for healthcare workers sustaining occupational HIV-l exposures. Alternatively, the physician involved in the management of a healthcare worker who has sustained such an exposure could provide much more relevant information to an exposed employee if the "donor" serologic status were known. The Centers for Disease Control (CDC) has stressed the advantages of determining (with the source patient's consent) the "donor's" HIV-l serostatus.7 Counseling strategies emphasizing techniques to minimize the risk for secondary transmission are more likely to be heeded if the donor is known to be seropositive.

Conversely, if the sensitivity and specificity of the new technique are ultimately found to be limited in clinical application, use of the test may actually do more harm than good. For example, false-negative antigen and/or antibody tests may offer a false sense of security, and perhaps even result in secondary HIV-l transmission in settings in which transmission would not have occurred had the current CDC recommendations7 been followed.

Decisions are most difficult when important pieces of data are missing, and this situation is an excellent example. One has to carefully balance the not-insignificant 0.4% risk for transmission associated with each percutaneous HIV-l exposure event¹ with the risk to the patient of having the result of an involuntary test inadvertently made public. The risk to the patient is frequently not given serious consideration by advocates of involuntary testing. Nonetheless, this risk is both real and, unfortunately, somewhat unique to HIV-l infection. Although I do not advocate basing major decisions on anecdotal reports, I also believe that, in addition to the widely-disseminated newspaper reports of houses being burned down and HIV-1infected children prevented from entering public schools, most individuals working in HIV-l-related fields over the past nine years have acquired enough personal experiences to substantiate the reality of this risk for patients.

Several other complex issues are raised by proposing any type of "involuntary" HIV-l serologic testing. For example, if the test is found to be positive, will the patient (i.e., the "donor" who is being involuntarily tested) be notified of the result? If not, is such a policy ethically and legally sound? If so, how will notification be accomplished, and who will be responsible for both notification and the requisite counseling to minimize the risk for subsequent spread? If a "donor" is involuntarily tested and found to be HIV-l-infected, is the testing physician also responsible for making certain that the "donor" notifies his or her sexual or needlesharing partners of his or her infection status? Some authorities suggest that an institution choosing to perform any "involuntary" testing may be obligated to inform patients of this practice at the time of admission and that this notification should also discuss the management of patient and partner notification.

Finally, I would underline the recommendations of Admiral James Watkins and the Presidential Commission on the Human Immunodeficiency Virus Epidemic. The Commission's final report emphasizes that HIV-l-infected individuals must be guaranteed their civil rights and that such individuals must be protected against discrimination by the legal system.⁸ In the absence of effective legislation guaranteeing HIV-l-infected patients' civil rights, any involuntary testing program may, unfortunately, result in discrimination against the "donor." Should such legislation be passed on the national level, "involuntary" testing, particularly in the setting outlined by Shirazian, et al., may be less problematic.

One of the most difficult aspects of trying to focus on any of the complex problems related to HIV-l infection in society is that one is always "shooting at a moving target." What seems to be the correct position today (or perhaps more appropriately, the most nearly correct position) becomes untenable as the issues change. In addition, perhaps more frequently than for any other medical issue, scientific discussions about HIV-l-related issues seem to become increasingly clouded by moral, political, religious or legal biases. As we gain more experience with HIV-l infection in society and as HIV-1infection (hopefully) becomes less stigmatizing, these issues will continue to change subtly.

Based on my own assessment of the current scientific/social milieu, I would be uncomfortable with the use of this technique for involuntary testing, especially in the absence of both legal precedent and of clear guarantees of patient (i.e., "donor") protection. Nonetheless, experience has taught the hospital epidemiology community that HIV-l-related target issues are fluid and are subject to scientific and, unfortunately, political influences. Nonetheless, we should pattern our behaviors after Shakespeare's experienced archer, making certain that are our eyes are steadily leveled on the appropriate target before loosing our arrows.

REFERENCES

- Henderson DK. HIV-1 in the health-care setting. In: Mandell GF, Douglas RG Jr., Bennett JE, eds. Principles and Practice of Infectious Diseases. 3rd ed. New York, NY: John Wiley;1990:2221-2236.
 Shirazian D, Herzlich BC, Mokhtarian F, Grob D. Detection of human
- Shirazian D, Herzlich BC, Mokhtarian F, Grob D. Detection of human immunodeficiency virus antibody and antigen (p24) in residual blood on needles and glass. *Infect Control Hosp Epidemiol*. 1990;11:180-184.
 Favero MS, Maynard JE, Petersen NJ, et al. Hepatitis B-antigen on
- Favero MS, Maynard JE, Petersen NJ, et al. Hepatitis B-antigen on environmental surfaces. *Lancet*.1973;2:1455.
- Dankert J. Ultentius J, Houwen B, Tegzess AM, van der Hem GK. Hepatitis B surface antigen in environmental samples from hemodialysis units. J Infect Dis. 1976;134:123-127.
- Lauer JL, Van Drunen NA, Washburn JW, Balfour HH. Transmission of hepatitis B virus in clinical laboratory areas. J Infect Dis. 1979;140:513-516.
- Bond WW. Favero MS, Petersen NJ, Ebert JW. Inactivation of hepatitis B virus by intermediate-to-high-level disinfectant chemicals. J Clin Microbiol. 1983;18:535-538.
- Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. MMWR.1987;36(suppl.2S):1S-19s.
- Report of the Presidential Commission on the Human Immunodeficiency Virus Epidemic. Washington, DC: U.S. Government Printing Office; June; 1988.