

Health Care–Associated Transmission of Hepatitis B & C Viruses in Dental Care (Dentistry)

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KEYWORDS

- Dentistry • Transmission • Hepatitis B
- Hepatitis C • Prevention

Of the nine million health care providers practicing in the United States, 608,000 are dental health care personnel (DHCP), including 176,000 dentists, 217,000 dental hygienists, and 362,00 dental assistants.¹ It is projected that by 2016, this number will grow to approximately 755,000 DHCP. Dental patients and DHCP are at risk for infection with microorganisms that either colonize or infect the oral cavity and the respiratory tract or may be present in the oral tissues from the circulating blood.² Among these, there are several blood-borne viral diseases, including infections caused by hepatitis B virus (HBV) and hepatitis C virus (HCV).³ Dentistry is considered as one of the health professions with the highest risk of HBV exposure, with infection rates among dentists that are 3 to 10 times higher than the general population.⁴⁻⁹ Historically and prior to the institution of mandatory HBV vaccination for health care workers, the prevalence rate for HBV markers among dentists was shown to be 16% to 28%,^{10,11} and several clusters of HBV transmission from infected dentists and oral surgeons to patients were reported.¹²⁻²¹ In terms of hepatitis C, one longitudinal cohort study showed a slightly higher prevalence rate for carrier state among DHCP compared to the other health care workers in the study (1.7% vs 1.4%).¹¹ In dental care settings, microorganisms can be transmitted through direct contact with contaminated instruments or surfaces, splash or spray of infectious fluids or materials in the mucosa of the eyes or mouth, and by inhalation of airborne infectious agents.^{2,22}

HEPATITIS B EPIDEMIOLOGY

An estimated 400 million people have chronic HBV infection worldwide.¹³ Every year, an estimated 900,000 people die of acute hepatitis, liver cirrhosis, or hepatocellular

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carcinoma caused by chronic HBV infection.²³ In the United States, it is estimated that between 800,000 to 1.4 million people have chronic HBV infection, and each year an estimated 50,000 new infections occur.²⁴ Community HBV transmission involves exposure to infectious body fluids such as blood, semen, and saliva, mostly through vertical, sexual, and parenteral modes. The current HBV incidence rate in the United States reflects a significant drop compared to mid-1980s, primarily because of aggressive vaccination of infants and high-risk populations.²⁴ Currently, heterosexual transmission accounts for approximately 39% of new HBV infections among adults; transmission among men who have sex with men comprises approximately 24% of cases, and injection drug use (IDU) accounts for approximately 16% of new HBV infections.²⁵ Other adult cases of chronic HBV infection are seen among household contacts of other chronically infected individuals, patients on hemodialysis, travelers to areas with high endemic rates for hepatitis B, and occupational exposures.²⁵ In health care settings, needle sticks or other sharps injuries are responsible for most occupational HBV transmissions. In addition, HBV has been shown to be able to survive for as much as 1 week on environmental surfaces.²⁶ Therefore, lapses in infection control practices and poor barrier techniques account for the remainder of transmission cases among health care workers and also may be responsible for many cases of nosocomial or patient-to-patient transmissions.²⁷⁻²⁹

CLINICAL FEATURES AND NATURAL HISTORY OF HBV INFECTION

HBV, a DNA virus from the *Hepadnaviridae* family, is capable of causing hepatic inflammation and the clinical syndrome of acute jaundice.³⁰ After an exposure event involving a susceptible host, the virus reaches the liver through the bloodstream and successfully establishes an infection in hepatocytes. HBV infection occurs with an average incubation period of 90 days (range: 60 to 150 days) from exposure to the onset of jaundice and 60 days (range: 40 to 90 days) from exposure to the onset of abnormal serum alanine aminotransferase (ALT) levels.^{31,32} Newly acquired HBV infection may be symptomatic. For instance, in infants, children aged younger than 5 years, and immunosuppressed adults, acute infection is typically asymptomatic, whereas 30% to 50% of children aged over 5 years and adults have initial clinical signs or symptoms.³³ These may include signs of hepatic disease such as clinical jaundice, anorexia, malaise, nausea, vomiting, abdominal pain, and extrahepatic signs like skin rashes, arthralgias, and arthritis.³⁰ A small proportion of patients may develop severe acute hepatitis B. The risk for severe acute hepatitis B may be increased in persons who are coinfecting with hepatitis C or D.³³ The fatality rate among persons with reported cases of acute hepatitis B is 0.5% to 1.0%, with the highest rates in adults aged over 60 years.²⁵

The earliest marker of infection, appearing within 4 to 10 weeks after the exposure, is hepatitis B surface antigen (HBsAg), a glycoprotein associated with the surface of the viral envelope.²³ The hepatitis B virion contains two other major antigens in its core, along with a partially double-stranded DNA and a DNA polymerase enzyme. One of the core antigens, hepatitis B core antigen (HBcAg), is not readily detectable in blood. The other, hepatitis B e antigen (HBeAg) appears in blood shortly after the HBsAg and is associated with significant liver inflammation manifested by a marked increase in serum transaminases and bilirubin.³⁴ The presence of HBeAg generally indicates high levels of HBV DNA in the blood.²⁵

The first humoral response to HBV infection is the development of immunoglobulin (Ig) M antibody to HBcAg (anti-HBc), which is detectable in blood shortly after the

appearance of HBsAg. Shortly thereafter, this antibody is replaced with IgG anti-HBc, remaining in blood for years after the infection. The next events in individuals who clear the infection involves development of anti-HBe, marking the end of active liver disease and ultimately anti-HBs, indicating recovery and immunity.³⁴ Approximately 95% of primary infections in adults with normal immune status follow a self-limited course and involve the elimination of the HBV from blood and subsequent lasting immunity to reinfection (development of anti-HBs).²³ A small proportion of individuals who clear the infection will have intermittent low levels of HBV DNA in serum referred to as latent hepatitis B. In these infected persons, progression to liver disease is unlikely, but viral reactivation may occur with severe immunosuppression.²³

Chronic infection occurs in about 5% of infected individuals over the age 5, in approximately 30% of infected children aged less than 5 years, and in almost all infected infants, with continuing viral replication in the liver and persistent viremia.^{25,33} Primary infections become chronic more frequently in immunosuppressed persons (eg, hemodialysis patients and persons with human immunodeficiency virus [HIV] infection)^{25,35,36} and persons with diabetes.^{25,37} Persons who were infected as adults or adolescents and have developed chronic HBV infection eventually enter the inactive carrier phase, whereas in those infected at birth or in early childhood, the disease continues to progress. The inactive carrier state is associated with clearing the HBeAg and developing anti-HBe (HBeAg seroconversion) with undetectable or low levels of HBV DNA, normalization of serum ALT levels, and reduced liver inflammation.²³ It is noteworthy that HBV DNA remains present in the blood during the inactive carrier phase, but at lower levels than during the active phase.²³

Overall, approximately 25% of persons who become chronically infected during childhood and 15% of those who become chronically infected after childhood die prematurely from cirrhosis or liver cancer; most remain asymptomatic until the onset of cirrhosis or end-stage liver disease.^{25,38} In chronically HBV-infected individuals, medical evaluation and regular monitoring are critical to ensuring sustained suppression of HBV replication and remission of liver disease. Currently, the therapeutic agents approved by the US Food and Drug Administration (FDA) for treating chronic hepatitis B include interferons (interferon- α 2b and peginterferon- α 2a) and nucleoside or nucleotide analogues (lamivudine, adefovir, entecavir, tenofovir, and telbivudine).¹³ The major goals of anti-HBV therapy are to prevent progressive liver disease, cirrhosis, liver failure, and subsequent development of hepatocellular carcinoma and death. It is not yet clear if the short-term positive results reported by anti-HBV therapies will, in the long term, provide protection against liver failure and carcinoma development. Periodic screening with ultrasonography and alfa-fetoprotein has been demonstrated to enhance early detection of hepatocellular carcinoma.³⁹

HEPATITIS B AND THE DENTAL CARE SETTING

In dental care settings, microorganisms can be transmitted through

- Direct contact with blood, oral fluids, or other patient materials
- Indirect contact with contaminated objects (eg, instruments, equipment, or environmental surfaces)
- Contact of conjunctival, nasal, or oral mucosa with droplets (eg, spatter) containing microorganisms generated from an infected person and propelled a short distance (eg, by coughing, sneezing, or talking)

Inhalation of airborne microorganisms that can remain suspended in the air for long periods.²²

For many of these exposures, transmission of HBV is very plausible because of contamination with blood and saliva. It has been shown that both infectious viruses and HBsAg particles are present in saliva; however, the number of infectious viruses is very low even in HBsAg-positive blood.⁴⁰ Generally, percutaneous injuries with sharp instruments such as cutting instruments and anesthetic needles are the most common source of occupational exposures in dentistry.^{10,41-45} A survey of dental practitioners by the American Dental Association showed that private practitioners experience an average of 3.2 injuries per year.⁴⁴ This rate has been reported to be much higher for dental education institutions⁴⁶ and with an approximate 0.3% to 0.5% of the US population being chronically infected with hepatitis B, the great potential for transmission of HBV to the DHCP is quite evident. In fact, some of the earlier prevalence studies showed the prevalence of HBV serologic markers among dentists to range from 16% to 28%.^{10,11} Since the early 1980s, the transmission of HBV to DHCP has declined dramatically (prevalence of serologic markers dropped to 9% in 1992), mostly as a result of better compliance with HBV vaccination and improved infection control practices.⁴⁷

Several reports, published between 1970 to 1987, described nine clusters of HBV transmission from three infected general dentists and six oral surgeons to their patients.¹²⁻²¹ The number of patients varied in each cluster and was as high as 55¹⁶ and 37,¹⁴ both involving oral surgeons, to 3¹⁹ and 4,²¹ from an oral surgeon and a general practitioner. No such transmission has been reported since 1987 in dentistry, most likely because of more widespread HBV vaccination of DHCP, universal glove use, and implementation of the 1991 Occupational Safety and Health Administration (OSHA) Bloodborne Pathogens Standard.⁴⁸ One case of patient-to-patient transmission has been documented where the exact mechanism of transmission was unproven.⁴⁹

HEPATITIS C EPIDEMIOLOGY

HCV, first identified in 1989,⁵⁰ is now considered responsible for chronic infection in 3% of the world population, approximately 170 million individuals.^{50,51} Globally 3 to 4 million new infections occur every year.⁵¹ In the United States, an estimated 3.2 million people are living with chronic HCV infection, with roughly 19,000 new infections reported in 2006 alone.²⁴ The most efficient mode of HCV transmission is through blood exposure. Therefore, most cases (60%) of infections in the United States are seen among injection drug users. Of persons injecting drugs for at least 5 years, 60% to 80% are infected with HCV, compared with about 30% infected with HIV.⁵² Donor deferrals and screening of blood and blood products for HCV antibodies and surrogate markers (ALT), which began in 1990, have been effective in significantly reducing the number of new cases of post-transfusion infections.⁵³ As of 2001, the risk of HCV infection from a unit of transfused blood is less than one per million transfused units.⁵² Sexual transmission is responsible for 15% of the reported cases in the United States.⁵² A total of 5% of exposures are caused by hemodialysis, employment in the health care field, and birth to an HCV-infected mother; for 10% of cases, no recognized source for infection has been identified.⁵² It has been shown that HCV in plasma can survive drying and environmental exposure to room temperature for at least 16 hours,⁵⁴ suggesting a potential for transmission through blood contamination of environmental surfaces and inanimate objects.

CLINICAL FEATURES AND NATURAL HISTORY OF HCV INFECTION

HCV, an RNA virus of the *Flaviviridae* family, has 6 genotypes and more than 50 subtypes.⁵⁵ Genotype 1 accounts for 70% to 75% of all HCV infections in the United States and is associated with a lower rate of response to treatment.^{56,57} HCV replicates preferentially in hepatocytes but is not directly cytopathic, leading to persistent infection.⁵⁵

The incubation period for acute HCV infection is 1 to 3 weeks. Within an average of 4 to 12 weeks, HCV RNA can be detected in blood and corresponds to the onset of symptoms and elevated serum ALT levels. Acute infection can be severe but rarely is fulminant.⁵⁵ Symptoms are uncommon but can include malaise, weakness, anorexia, and jaundice.²⁴ Symptoms usually subside after several weeks as ALT levels decline. An average of 60% to 70% of infections lead to a chronic carrier state; 10% to 20% of chronic carriers develop liver cirrhosis, and 1% to 5% develop hepatocellular carcinoma.⁵⁸ Older age at time of infection, male gender, diabetes, alcohol use, and co-infection with HIV or HBV appear to increase the risk of progressive liver disease.^{23,52,55,59} Patients with chronic hepatitis C can present with extrahepatic manifestations such as rheumatoid arthritis, keratoconjunctivitis sicca, lichen planus, glomerulonephritis, lymphoma, and essential mixed cryoglobulinemia.⁵⁵ HCV-associated chronic liver disease is the most frequent indication for liver transplantation among adults.⁵²

During chronic infection, HCV RNA reaches high levels, generally ranging from 10^5 to 10^7 IU/mL. Ultrasensitive enzyme immunoassays (EIAs) that can detect HCV antibodies are used for screening at-risk populations and are recommended as the initial test for patients with clinical liver disease. A negative EIA test rules out chronic HCV infection in immune-competent patients, but patients on hemodialysis and patients with immune deficiencies may have false-negative EIAs.⁵⁵ For these patients, an assay for HCV RNA (target amplification, polymerase chain reaction [PCR], or signal amplification techniques like branched DNA) is necessary for diagnosis of chronic infection.

The goal of HCV treatment is to prevent complications of chronic infection. This is principally achieved by eradication of infection. Current treatment standards involve the use of pegylated interferon (alfa-2a and alfa-2b) in combination with ribavirin.^{60–62} HCV genotype 1 requires a longer course of treatment with pegylated interferon and higher dose of ribavirin than other genotypes.⁶⁰ Early viral response demonstrated by a rapid drop in the HCV viral load is predictive of sustained viral response (at 6 months).⁶⁰ Treatment is recommended for patients with an increased risk of developing cirrhosis. These patients include those with detectable HCV RNA levels (higher than 50 IU/mL), a liver biopsy indicative of portal or bridging fibrosis, or moderate inflammation and necrosis. Most also have persistently elevated ALT values.^{60,61}

HEPATITIS C AND DENTAL CARE SETTING

Approximately, 50% of HCV-infected individuals have HCV-RNA in their saliva.⁶³ In addition, there is a direct relationship between the presence of HCV in saliva and the plasma HCV load.^{63–65} Studies also have shown HCV particles to be present in oral epithelial cells.^{66,67} These findings may suggest that HCV would be transmissible through household contacts, explaining the roughly 10% of new cases where no specific mode of transmission can be identified.⁵² It also may imply that the dental environment may be at especially high risk for occupational HCV transmission. Evidence, however, suggests that the infectivity of HCV in saliva may be very low. As reviewed by Ferreiro and colleagues,⁶⁸ studies have not been able to demonstrate a high capacity for infectivity by HCV particles found in different oral compartments.

Also, there is no epidemiologic evidence for HCV transmission through orogenital exposure, kissing, or household contact. Moreover, the prevalence rates of DHCP have been shown to be only slightly higher than the general population.¹¹ One study of general dentists and oral surgeons showed anti-HCV antibodies in 2% of oral surgeons and 0.7% of general practitioners.⁶⁹ To date, no case of transmission from an HCV-infected dentist to his or her patients has been reported.⁷⁰

OCCUPATIONAL EXPOSURES TO HBV AND HCV IN DENTISTRY

In June 2001, the Centers for Disease Control and Prevention (CDC) published its last updated recommendation for the management of occupational HIV, HBV, and HCV exposures, and recommendations for postexposure prophylaxis.⁴⁰ Since then, other updates have been published to include new antiretroviral drugs for postexposure prophylaxis against HIV, but the recommendations for HBV and HCV have remained the same.⁷¹ This section provides a summary of the US Public Health Service (PHS) guidelines and also includes specific recommendations for infection control in dental settings.⁷²

HBV infection is a well recognized occupational risk for DHCP.⁴⁻⁹ For each exposure incident involving an HBV-infected source patient, the risk of HBV infection is related directly to the degree of contact with blood or other infected body fluids and the HBeAg status of the source person.⁷² The risk of developing clinical hepatitis after exposure to blood that is both HBsAg- and HBeAg-positive has been shown to range from 22% to 31%; the risk of developing serologic evidence of HBV infection is 37% to 62%. In contrast, the risk of developing clinical hepatitis from a needle contaminated with HBsAg-positive, HBeAg-negative blood is about 1% to 6%, and the risk of developing serologic evidence of HBV infection is 23% to 37%.⁷³ Recent data indicate fluctuating levels of HBV DNA among hepatitis B carriers.⁷⁴ Therefore an assessment of the viral DNA load, regardless of the HBeAg status, may be necessary to assess a person's degree of infectivity.⁷⁵

Percutaneous injuries with sharp dental instruments and hollow bore anesthetic needles are among the most efficient modes of HBV transmission. However, direct or indirect blood or body fluid exposures that can inoculate HBV into cutaneous scratches, abrasions, burns, other lesions, or on mucosal surfaces have been documented.²⁷⁻²⁹ This type of exposure may not only be immediate, as HBV has been demonstrated to survive in dried blood at room temperature on environmental surfaces for at least 1 week.²⁶ The potential for HBV transmission through contact with environmental surfaces has been demonstrated in investigations of HBV outbreaks among patients and staff of hemodialysis units.⁷⁶⁻⁷⁸

HCV is not transmitted efficiently through occupational exposures to blood. The average incidence of anti-HCV seroconversion after accidental percutaneous exposure from an HCV-positive source is 1.8% (range: 0% to 7%).⁷⁹⁻⁸² Transmission rarely occurs from mucous membrane exposures to blood, and no transmission has been documented from intact or nonintact skin exposures to blood.^{83,84} Although it has been shown that HCV can survive on environmental surfaces for a few hours,⁵⁴ epidemiologic data do not support a significant risk for HCV transmission in the health care setting through environmental contamination with blood.^{43,85} The risk for transmission from exposure to fluids or tissues other than HCV-infected blood also has not been quantified but is expected to be low.

POSTEXPOSURE PROPHYLAXIS

For percutaneous or mucosal exposures to blood, factors to be considered are the HBsAg status of the source and the hepatitis B vaccination and vaccine-response

status of the exposed person. Such exposures usually involve persons for whom hepatitis B vaccination is recommended. Any blood or body fluid exposure to an unvaccinated person should lead to initiation of the hepatitis B vaccine series.

If a DHCP has not been vaccinated against HBV, or if he or she has started the vaccination series but has not completed it, or is known not to have responded to the initial vaccination series, then a single dose of hepatitis B immunoglobulin (HBIG) should be administered as soon as possible after exposure (preferably within 24 hours). The effectiveness of HBIG when administered more than 7 days after exposure is unknown. For all these individuals hepatitis B vaccine is indicated:

- To initiate the series for the nonvaccinated
- To complete the series for those who have already received one or two doses
- To repeat the vaccination series for the nonresponders.

The vaccine also should be administered as soon as possible (preferably within 24 hours), and it can be administered simultaneously with HBIG at a separate site (vaccine always should be administered in the deltoid muscle).

DHCP exposed to an HCV-positive source should receive baseline testing for anti-HCV and ALT activity and also follow-up testing (eg, at 4 to 6 months) for anti-HCV and ALT activity (if earlier diagnosis of HCV infection is desired, testing for HCV RNA may be performed at 4 to 6 weeks). Confirm all anti-HCV results reported positive by EIA using supplemental anti-HCV testing by recombinant immunoblot assay (RIBA II; Ortho Diagnostics, Raritan, NJ, USA). The exposed DHCP should be counseled on the risk for HCV infection and appropriate medical follow-up. They should be advised to refrain from donating blood, plasma, organs, tissue, or semen. The exposed person does not need to modify sexual practices or refrain from becoming pregnant.

Immunoglobulin and antiviral agents are not recommended for postexposure prophylaxis after exposure to HCV-positive blood. In addition, no guidelines exist for administration of therapy during the acute phase of HCV infection. Limited data, however, indicate that antiviral therapy might be beneficial when started early in the course of HCV infection. When HCV infection is identified early, the person should be referred for medical management to a specialist knowledgeable in this area.

No modifications to an exposed person's patient care responsibilities are necessary to prevent transmission to patients based solely on exposure to HBV- or HCV-positive blood. If an exposed person becomes acutely infected with HBV, the person should be evaluated for his or her medical status. No recommendations exist regarding restricting the professional activities of HCP with HCV infection. As recommended for all health care providers, DHCP who are chronically infected with HBV or HCV should follow all recommended infection control practices, including standard precautions and appropriate use of hand washing, protective barriers, and care in the use and disposal of needles and other sharp instruments.⁸⁶

SUMMARY

The dental environment is associated with significant risk for HBV transmission and to a lesser degree for HCV exposure and infection. DCHP are required to receive hepatitis B vaccination and must follow infection control strategies that are consistent with standard precautions to reduce the risk for HCV transmission. For occupational exposures in dentistry, the latest PHS guidelines are used to properly guide postexposure evaluation and management of DHCP.

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Human Immunodeficiency Virus (HIV) Transmission in Dentistry

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ABSTRACT

HIV transmission in the health-care setting is of concern. To assess the current position in dentistry, we have reviewed the evidence to November 1, 2005. Transmission is evidently rare in the industrialized nations and can be significantly reduced or prevented by the use of standard infection control measures, appropriate clinical and instrument-handling procedures, and the use of safety equipment and safety needles. We hope that breaches in standard infection control will become vanishingly small. When occupational exposure to HIV is suspected, the application of post-exposure protocols for investigating the incident and protecting those involved from possible HIV infection further reduces the likelihood of HIV disease, and also stress and anxiety.

KEY WORDS: HIV, AIDS, mouth, dental, transmission, occupational.

INTRODUCTION

Viruses can be transmitted in health-care settings including dentistry, albeit rarely, notably where standard infection control measures are not implemented. The epidemic of Acquired Immune Deficiency Syndrome (AIDS) has been recognized for about 25 years, and concern about the transmission of human immunodeficiency viruses (HIV) is therefore not new. In the case of HIV, transmission is evident from cases where health-care professionals (HCPs) have seroconverted because of occupational exposure to HIV (Marcus, 1988; Tokars *et al.*, 1993; Centers for Disease Control and Prevention, 1995), but the risk of transmission is low, with a seroconversion rate of 0.1% after percutaneous exposure and 0.63% after mucous-membrane contamination (Ippolito *et al.*, 1993). Review of data reported to December, 2001, in the HIV/AIDS Reporting System and the National Surveillance for Occupationally Acquired HIV Infection revealed 57 HCPs with documented occupationally acquired HIV infection; most (86%) had been exposed to blood, and most (88%) had percutaneous injuries (Do *et al.*, 2003). However, to assess the current position in dentistry, we have reviewed the evidence to November 1, 2005. We have focused on HIV and do not discuss other blood-borne pathogens, such as hepatitis viruses, herpesviruses, prions, bacteria, fungi, or parasites. Definitions relevant to this paper are outlined in Table 1.

HEALTH-CARE WORKERS WITH POSSIBLE OCCUPATIONALLY CONTRACTED HIV INFECTION

In the USA until December, 2001, the Centers for Disease Control and Prevention (CDC) reported that there had been 57 occupational HIV infections in HCPs (Centers for Disease Control, 2002), mainly from percutaneous (sharps; needlestick) injuries. Of these, none was reported to be in dental HCPs (Table 2). In addition, 139 other cases of HIV infection or AIDS have been recorded among HCPs who have not reported other risk factors for HIV infection, but who report a history of occupational exposure to HIV-infected blood, body fluids, or laboratory material, but where seroconversion after exposure was not documented. Six have been dental HCPs; each had a history of percutaneous or mucous membrane exposure to HIV-infected body fluids, but seroconversion could not be linked to specific occupational exposure (Centers for Disease Control, 2001). The occupations of these HCPs are presented in Table 2.

In the UK, until May, 2005, the Health Protection Agency (HPA) reported that there had been 5 documented HIV seroconversions through occupational exposure in the health-care setting, and 12 possible/probable occupational seroconversions—but none was in dental HCPs. A further 14 probable cases of occupational acquisition of HIV in HCPs have been diagnosed in the UK. The majority of these HCPs had worked in countries of high HIV prevalence, and are presumed to have been infected outside of the UK (Heptonstall *et al.*, 1993).

According to McCarthy, there are, worldwide, > 300 reports

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Table 1. Terminology Relevant to HIV Transmission

Term	Definition
Infection control	Infection control refers to policies and procedures used to minimize the risk of spreading infections, especially in hospitals and health-care facilities.
Standard infection control precautions	Preventive practices used to reduce blood exposures, particularly percutaneous exposures, include: careful handling of sharp instruments; the use of rubber dams to minimize blood spattering; handwashing; and the use of protective barriers (e.g., gloves, masks, protective eyewear, and gowns), based on the premise that all blood and body fluids, excretions, and secretions should be treated as infectious. http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5217a1.htm
Sterilization	Sterilization is defined as the complete destruction of all organisms, including a large number of highly resistant bacterial endospores.
Disinfection	Disinfection is defined as the removal of all vegetative bacteria and nearly all recognized pathogenic organisms except bacterial endospores.
Decontamination	Decontamination is any process or treatment that makes a machine, component, instrument, medical device, or environmental surface incapable of transmitting infectious particles.
Occupational exposure	Reasonably anticipated skin, eye, mucous membrane, or parenteral contact with blood or other potentially infectious materials that can result from the performance of an employee's duties. Exposure is not synonymous with transmission of an infectious agent.
Possible occupational transmission	The term implies that a HCP has been found to be HIV-infected, and that subsequent investigations have revealed no other identified risk for infection other than occupational exposure. There is variation between and among countries on what constitutes a "possible occupational transmission".
Documented occupational transmission	An exposure for which there is documented evidence of HIV seroconversion (a recorded negative result of a test for anti-HIV, followed by a subsequent positive result) associated in time with a specific occupational exposure to a source of HIV. There is little variation between and among countries on what constitutes a "documented occupational transmission".
Exposure-prone procedure	Procedures during which there is a recognized risk for percutaneous injury to the health-care professional (HCP), and if such an injury occurs, the HCP's blood is likely to come into contact with the patient's body cavity, subcutaneous tissues, and/or mucous membranes. Exposure-prone procedures are those invasive procedures where there is a risk that injury to the HCP may result in the exposure of the patient's open tissues to the blood of the HCP. These include procedures where the HCP's gloved hands may be in contact with sharp instruments, needle tips, or sharp tissues (e.g., spicules of bone or teeth) inside a patient's open body cavity, wound, or confined anatomical space, where the hands or fingertips may not be completely visible at all times. However, other situations, such as pre-hospital trauma care and care of patients where the risk of biting is regular and predictable, should be avoided by health-care workers restricted from performing exposure-prone procedures.

(102 confirmed) of occupational transmission to HCPs, including up to nine dental HCPs (unconfirmed) (McCarthy *et al.*, 2002). Exposure to HIV has been reported by 0.5% dentists/year (McCarthy *et al.*, 2002). There are few data from resource-poor countries or regions where the prevalence of HIV is, and risk of infection must be, higher.

HEALTH-CARE PROFESSIONALS WITH HIV INFECTION OF UNKNOWN ORIGIN

Many HCPs do have HIV infection or AIDS, but the infection has often been contracted non-occupationally. Of about 23,000 HCPs with AIDS reported to the CDC, fewer than 500 are dental HCPs, but there is no reported evidence of any of them having acquired HIV occupationally in the dental health-care setting (ADA, 2003) (Table 3).

DENTAL STAFF WITH POSSIBLE OCCUPATIONALLY CONTRACTED HIV INFECTION

Reports of those dental HCPs who do appear to have contracted HIV infection occupationally are as follows:

Dentist 1

The first case of a dental HCP reported with apparently occupationally contracted HIV was a male dentist in the USA (Klein *et al.*, 1988). He lived among and treated New York City "Greenwich Village" patients—a high HIV/AIDS risk population—and he used protective equipment only intermittently, denied other high-risk behavior, and tested HIV-positive in a survey of 1309 dental HCPs (Klein *et al.*, 1988). His HIV exposure could not be documented, and the CDC concluded that if the dentist did contract HIV occupationally, then standard infection control precautions would have prevented transmission to his patients.

Dentists 2 & 3

There is a reference to two HIV-seroconverted dental HCPs, among a group of 69 HCPs with no identifiable risk for infection (Neiburger, 2004). These dentists evidently worked in a correctional facility (treating high-risk patients), experienced needlesticks from equipment used on unidentified patients, and died before HIV-DNA studies and in-depth interviews could be done (Centers for Disease Control, 1992a).

Table 2. Documented and Possible Occupational Transmissions of HIV to Health-care Professionals

Health-care Occupations	Documented Occupational Transmissions	Possible Occupational Transmissions
Dental health-care worker	0	6
Embalmer/morgue technician	1	2
Emergency medical technician/paramedic	0	12
Health aide/attendant	1	15
Housekeeper/maintenance worker	2	13
Laboratory, Worker, clinical	16	17
Technician, non-clinical	3	0
Nurse	24	35
Physician, Non-surgical	6	12
Surgical	0	6
Respiratory therapist	1	2
Technician, Dialysis	1	3
Surgical	2	2
Other	0	9
Other	0	5
Total	57	139

Adapted from the Centers for Disease Control and Prevention (CDC), 1999 and 2001.

There was little other information on these two dentists or any potential high-risk behavior.

Dentists 4, 5, & 6

The CDC, in several years of HIV/AIDS Surveillance Reports, indicated that there were seven dental HCPs who were possible cases of occupational HIV transmission, but this was later revised to six possible cases (Centers for Disease Control, 1993, 1997, 1999). Of these seven (or six) dental HCPs, three were general dental practitioners, two were dental students, one was a pedodontist, and one a periodontist. Dental HCPs in the UK have not been reported to have contracted HIV from HIV-infected patients (Public Health Laboratory Service, 2005).

HIV TRANSMISSION FROM HCP TO PATIENT

Available information indicates that the risk of HIV transmission in the dental office is very low (Centers for Disease Control, 1990). There is general agreement that there can be some risk of HIV transmission from an HIV-infected HCP to a patient, but it is small, and may be minimized by the use of standard infection-control measures.

In attempting to assess the risk, one must consider not only statistical data, but also the nature of the procedure being performed. Should the HIV-infected HCP incur a surgical accident or percutaneous injury in an exposure-prone procedure (EPP), there may be the potential for exchange of blood or other potentially infected fluid, such as saliva, but the susceptibility of oropharyngeal and other mucous membranes to transmission of HIV is unknown.

In only three reported instances (discussed below)—the Florida dentist (Ciesielski *et al.*, 1992), the French orthopedic surgeon (Lot *et al.*, 1999), and the nurse (Goujon *et al.*, 2000)—have there been possible transmissions from an HIV-infected HCP to patients, but although genetic relatedness was

Table 3. Adults Reported with AIDS and a History of Employment in US Healthcare, where Job is Known, by Occupation, as of December, 2002

Occupation	Number with AIDS
Nurses	5,378
Health aides	5,638
Technicians	3,182
Physicians	1,792
Therapists	1,082
Dental workers	492
Paramedics	476
Surgeons	122
Other	5,050
Total	23,212

From CDC, 2001.

demonstrated, only in the orthopedic case was the route of transmission clear.

Worldwide, all other retrospective studies of patients exposed to the potential risk of transmission of HIV during EPP have failed to identify any patients who have become infected by this route. Analysis of the data available from patient notification exercises also supports the conclusion that the overall risk of transmission of HIV from infected HCPs to patients is very low. Between 1988 and 2001 in the UK, there were 22 patient notification exercises, but no detectable transmission of HIV from an infected HCP to a patient, despite about 7000 patients having been tested (Public Health Laboratory Service, 2005).

THE FLORIDA DENTIST CASE

Although AIDS has been recognized in the USA since 1981, the cases described related to the Florida dentist remain the only ones in which HIV transmission has been convincingly documented in any way in dental practice (Centers for Disease Control, 1991), and even this is controversial. Possible transmission of HIV infection during an invasive dental procedure was first reported in a young woman (patient A) with AIDS in Florida, USA. She had no identified risk factors for HIV infection and was infected with a strain of HIV apparently closely related to that of her male dentist, as determined by viral DNA sequencing. Because the dentist had known behavioral risk factors for HIV, his infection was probably not occupationally acquired.

The dentist then wrote to his former patients, which prompted 591 persons to be tested for HIV, when two patients (B and C) were found to be HIV-seropositive. Another patient (patient D) was identified as HIV-infected when the list of available names of the dentist's former patients was matched with Florida's state AIDS surveillance records, and one more patient (E) contacted the CDC to report that she was HIV-infected and had been a former patient of the dentist. Of these four additional HIV-infected patients of the dentist, only two were infected with HIV strains closely related to those of the dentist and patient A, but not to strains from other persons residing in the same geographic area as the dental practice. Another 1100 persons who may have been patients of the dentist were contacted for counseling and HIV-antibody testing; of

these persons, 141 were tested, but all were HIV-seronegative.

This investigation strongly suggests that at least three (possibly six) patients of the Florida dentist with AIDS were infected with HIV during their dental care, since they had no other confirmed exposures to HIV, all had had invasive procedures performed by the HIV-infected dentist, and DNA sequence analyses of the HIV strains indicated a high degree of similarity of these strains to each other and to the strain that had infected the dentist. These HIV strains were also distinct from strains from patient D (who had known behavioral risks for HIV infection), from strains of the eight HIV-infected patients residing in the same geographical area, and from the 21 other North American HIV isolates. The precise mode of HIV transmission to patients A, B, and C remains uncertain, though all three had invasive dental procedures at times when the dentist was known to be HIV-infected and would have had high blood viral titers, and patients B and C had multiple invasive procedures.

Although barrier precautions were reportedly used in the Florida dental office, they were neither consistent nor in compliance with recommendations. Transmission might also have occurred by the use of instruments or other dental equipment that had been previously contaminated with blood from either the dentist or an infected patient.

There have been continued controversy and speculation over this case, and the truth will probably never be established, since the dentist has died.

OTHER REPORTS FROM THE USA ON PATIENTS TREATED BY HIV-POSITIVE HCPs

The CDC have reported HIV test results for 15,795 patients who were treated by 32 HIV-infected HCPs, including some dental HCPs (Centers for Disease Control, 1992b). The total number of patients treated by these HCPs and the number of patients who underwent invasive procedures are unknown. However, 23 of these HCPs (11 were dentists/dental students) had 10,270 of their patients tested, and no seropositive persons were reported. For the remaining nine HCPs (five were dentists), 5525 of their patients were tested, and 84 HIV-infected patients were identified. Follow-up was completed for 47 of these 84 HIV-seropositive patients: Seven patients had established HIV risk factors identified (*e.g.*, male-to-male sexual contact, injecting-drug use, receipt of a blood transfusion from a retrospectively identified HIV-infected donor); five were documented to be infected before receiving care from the HIV-infected HCP; and the remaining 35 were male inmates in a state correctional facility. These 35 inmates were among a total of 962 male inmates who received treatment from two HIV-infected dentists, and for whom HIV-antibody test results were known. The prevalence of HIV infection for male inmates tested (3.6%) was less than that documented among male inmates upon entrance into the state correctional system (8.6%). Established risk factors were identified for 33 of the 35 HIV-infected inmates. Because both dentists died, specimens for HIV genetic sequence analysis were not available.

The 37 HIV-infected persons in the same study (Centers for Disease Control, 1992a), for whom investigations were in progress, were patients treated by three HCPs, two of whom were dentists. Dentist 1 practiced in an area with high background prevalence of HIV infection, and, of 1162 patients tested, 29 were HIV-infected. Established risk factors could not

be identified for 17 of these 29 patients, but epidemiologic investigations determined that many may have had opportunities for exposure to HIV (*e.g.*, multiple sex partners and/or exchange of sex for drugs or money). HIV genetic sequence analysis results do not appear to have been published. More than 800 patients of Dentist 2 were tested, and five proved to be HIV-positive. Three of these patients had established risk factors identified. Eighteen months after the last visit to the dentist, a fourth patient was documented to be seronegative but was seropositive when re-tested 2 years later. No risk factors were identified for the remaining patient, who had visited the dentist only once, for an examination.

As of 1 January 1995, information about investigations of 64 HCPs infected with HIV had been reported to the CDC, with HIV test results available for 22,171 patients of 51 of these HIV-infected HCPs (Robert *et al.*, 1995). For 37 of the 51 HCPs, no HIV-seropositive patients were reported among 13,063 patients tested. For the remaining 14 HIV-infected HCPs, 113 seropositive patients were reported among 9108 patients. However, epidemiologic and laboratory follow-up did not show any HCPs to have been a source of HIV for any of the patients tested (Robert *et al.*, 1995).

Data from the above investigations, as well as risk estimates from modeling techniques, continue to indicate that the risk for HIV transmission from an HIV-infected HCP, whether dental or other, to a patient during an invasive procedure is very small.

THE FRENCH ORTHOPEDIC SURGEON

This HIV-infected orthopedic surgeon practiced in Paris, France, for 12 years after his HIV diagnosis was known, and 983 of 3004 of his patients treated during that period were HIV-tested. Only one HIV-positive woman, negative before hip prosthesis and without other risk factors, was identified as HIV-positive, and the strain of HIV from both surgeon and patient was similar (Lot *et al.*, 1999).

This seems to confirm transmission of HIV from the HCP.

THE FRENCH NURSE

The first known case of HIV transmission from a nurse practicing near Paris, France, to a 61-year-old female surgical patient has been reported, without evidence of blood exposure (Goujon *et al.*, 2000). Phylogenetic analyses strongly suggested that the HIV-seropositive 51-year-old female night nurse, who was also infected with hepatitis C virus (HCV), appears to have infected the patient with HIV but not HCV, despite not having performed invasive procedures (Goujon *et al.*, 2000). Interestingly, another HIV-infected nurse attending the patient appears not to have been involved in the transmission.

TRANSMISSION OF OTHER VIRAL INFECTIONS IN DENTAL PRACTICE

There is no doubt that blood-borne viral infections such as hepatitis B and other pathogens have been transmitted from dental HCPs to patients and *vice versa*, especially when the dental HCPs were those practicing surgical procedures, and in the era before standard infection control measures were widely adopted.

The current level of risk of transmission, however, is debatable. Dental HCPs do not now seem to be particularly at risk for occupational acquisition of blood-borne hepatitis

viruses transmissible by percutaneous injuries or blood products, such as either hepatitis C virus or transfusion-transmitted virus.

UNIVERSAL AND STANDARD INFECTION CONTROL PROCEDURES

Universal infection control precautions were based on the concept that all blood and body fluids might be contaminated with blood and should be treated as infectious, because patients with blood-borne infections can be asymptomatic or unaware that they are infected. Standard infection control precautions integrate and expand the elements of these universal precautions into a standard of care designed to protect both HCPs and patients from pathogens that can be spread by blood or any other body fluid, excretion, or secretion. The latest detailed guidelines are available elsewhere (Kohn *et al.*, 2003) and will not be described here.

COMPLIANCE WITH INFECTION CONTROL PROCEDURES IN DENTAL PRACTICE

Despite improvements in infection control over the period of the HIV pandemic (Scully *et al.*, 1992), there have been substantial improvements with compliance in some areas of infection control in dentistry—for example, glove-wearing. However, other aspects, such as the effective management of needlestick injuries, remain problematic (Gordon *et al.*, 2001), and there remain widespread shortcomings in facilities, equipment, operational procedures, management, and staff training in some health services (Glennie Report, 2004), and the available evidence suggests that compliance in dental practice is sometimes lacking, even in developed areas such as North America and Europe (McCarthy *et al.*, 1999a; Bagg *et al.*, 2001).

PERCUTANEOUS INJURIES IN DENTAL HEALTH-CARE PROFESSIONALS

The circumstances varied among 51 percutaneous injuries in one US study of HCPs, with the largest proportion (41%) occurring after a procedure, 35% occurring during a procedure, and 20% occurring during disposal of sharp objects (Do *et al.*, 2003). Factors that increase the risk of contracting HIV infection from a percutaneous injury in a HCP include the volume of blood involved and, probably, a higher HIV titer in the source patient's blood (Cardo and Bell, 1997; Cardo *et al.*, 1997). Other factors include:

- terminal HIV-related illness in the source patient (Saag *et al.*, 1991),
- a deep injury,
- visible blood on the device that caused the injury, and
- injury with a needle that had been placed in a source patient's artery or vein.

Blood is effectively removed from many hollow needles or suture needles when the needle passes through one or more layers of latex or vinyl gloves before coming into contact with the skin (Mast *et al.*, 1993).

Dental HCPs are also at risk, but tend to under-report percutaneous injuries, particularly when there is potential HIV contamination (Ramos-Gomez *et al.*, 1997). The CDC, from June, 1995, through August, 2001, reported 208 exposures—199 percutaneous injuries, six mucous membrane exposures, and three skin exposures—in dental HCPs (Cleveland *et al.*, 2002). One-third of these injuries were caused by small-bore

hollow syringe needles, and most were moderately deep. Nearly half the devices involved were visibly bloody at the time of injury. Twenty-four (13%) of the known source patients were HIV-positive; 14 had symptomatic HIV infection or a high viral load. In this study, three of four dental HCPs exposed to an HIV-positive source warranted a three-drug post-exposure protocol (PEP) regimen. Twenty-nine (24%) dental HCPs exposed to a source patient, who subsequently was found to be HIV-negative, took PEP; six took PEP for 5 to 29 days. No exposures resulted in HIV infection (Cleveland *et al.*, 2002).

Most dental HCPs appear to be careful to try to avoid injury during intra-oral procedures, but it is during extra-oral procedures—such as laboratory work, operatory clean-up, and instrument preparation for sterilization—that most percutaneous injuries occur (Porter *et al.*, 1990; Cleveland *et al.*, 1995; Gooch *et al.*, 1995; McCarthy *et al.*, 1999b).

Fortunately, the rate of occupational injuries among dental HCPs appears to have decreased over the last decade (Bednarsh and Klein, 2003). Post-exposure prophylaxis after percutaneous injuries reduced transmission by over 80% (Cardo *et al.*, 1997), but prevention of injuries is much more important.

PREVENTION OF OCCUPATIONAL TRANSMISSION OF PATHOGENS

Strategies for preventing occupational HIV transmission to HCPs have been summarized by the CDC (Centers for Disease Control, 2002). In the USA, in 1991, the US Department of Labor's Occupational Safety & Health Administration (OSHA) issued the Bloodborne Pathogens Standard to protect workers from the risk of exposure to blood-borne pathogens such as Hepatitis B, Hepatitis C, and HIV/AIDS. In 2002, in response to the Needlestick Safety and Prevention Act, OSHA revised the Bloodborne Pathogens Standard 29 CFR 1910.1030. The revised standard clarifies the need for employers to select safer needle devices and to involve employees in identifying and choosing these devices. The updated standard also requires employers to maintain a log of injuries from contaminated sharps (OSHA, 2002).

Engineering controls to eliminate or isolate the hazard (*e.g.*, puncture-resistant sharps containers or needle-retraction devices) are the primary strategies for protecting dental HCPs and patients. Where these are not appropriate or available, work-practice controls that result in safer behaviors, coupled with the use of personal protective equipment (PPE) (*e.g.*, protective eyewear, gloves, and masks), can prevent or minimize exposure.

An effective sharps injury prevention program is also required. This includes two main components: organizational steps for developing and implementing a sharps injury program, and operational processes. A culture of safety, reporting injuries, analyzing data, and selecting and evaluating devices must be engendered. Instruments, rather than fingers, should be used to grasp needles, retract tissue, and load/unload needles and scalpels. Safer local anesthetic syringes and retractable scalpels are available. It is important that HCPs not pass any needles unsheathed, or recap needles using two hands. Use of a mechanical recapping device or a scoop technique is recommended. Sharps disposal containers and needles and other sharps devices with an integrated engineered sharps injury prevention feature are essential (Centers for Disease Control, 2004).

PROTOCOLS FOR DEALING WITH PERCUTANEOUS INJURIES AND OTHER POSSIBLE OCCUPATIONAL EXPOSURES TO HIV INFECTION

Occupational exposures should be considered urgent to ensure timely post-exposure management and administration of hepatitis B immune globulin, hepatitis B vaccine, and/or HIV post-exposure prophylaxis (PEP) (Smith *et al.*, 2001). Post-exposure prophylaxis with zidovudine appears to be protective against HIV infection (Cardo *et al.*, 1997). Current protocols employ nucleoside reverse-transcriptase inhibitors (NRTIs) and/or nucleotide reverse-transcriptase inhibitors (NtRTIs).

CDC recommendations for PEP (Centers for Disease Control, 2005) for most HIV exposures include beginning, within hours, a basic four-week regimen of 2 anti-retroviral drugs, using 2 NRTIs, or one NRTI and one NtRTI. Regimens include zidovudine [ZDV] and lamivudine [3TC] or emtricitabine [FTC]; d4T and 3TC or FTC; and tenofovir [TDF] and 3TC or FTC. Where there is an increased risk for HIV transmission, an expanded drug regimen is recommended, which includes the addition of a third protease inhibitor [PI]-based drug, usually lopinavir/ritonavir [LPV/RTV]. When the source person's HIV is known or suspected to be resistant to one or more of the PEP drugs, then drugs to which the source person's virus is unlikely to be resistant are recommended. In addition, the CDC outlines several special circumstances (*e.g.*, delayed exposure report, unknown source person, pregnancy in the exposed person, resistance of the source virus to antiretroviral agents, or toxicity of the PEP regimen) when consultation with local experts and/or the National Clinicians' Post-Exposure Prophylaxis Hotline ([PEPline] 1-888-448-4911) is advised (Centers for Disease Control, 2005).

European guidelines suggest that PEP should be started as soon as possible with any triple combination of antiretroviral drugs approved for the treatment of HIV-infected patients; initiation of PEP is discouraged after 72 hours. Rapid HIV testing of the source could reduce inappropriate PEP. HIV testing should be performed at baseline, 4, 12, and 24 weeks, with additional clinical and laboratory monitoring of adverse reactions and potential toxicity at weeks 1 and 2. HIV resistance tests in the source and direct virus assays in the exposed HCP are not recommended routinely (Puro *et al.*, 2004). Specific UK recommendations are also available (Department of Health, 2004, http://www.dh.gov.uk/PublicationsAndStatistics/Publications/PublicationsPolicyAndGuidance/PublicationsPolicyAndGuidanceArticle/fs/en?CONTENT_ID=4083638&chk=qtPweH). The UK Department of Health recommends zidovudine as first choice, with lamivudine and nelfinavir, and recommends that PEP be considered whenever there is significant exposure to high-risk body fluids. In an ideal situation, PEP should be commenced immediately, preferably within 1 hour, but starting PEP up to 2 weeks after exposure may still be beneficial.

CONCLUSIONS

HIV transmission in the dental care setting continues to be of concern, but is rare in the industrialized nations and can be significantly reduced or prevented by the use of standard infection control measures, appropriate and careful clinical and instrument-handling procedures, and the use of safety equipment and safety needles. We hope that breaches in standard infection control will become vanishingly small and that percutaneous injuries will reduce even further.

When percutaneous exposure to HIV is suspected, the application of post-exposure protocols for investigating the incident and protecting those involved from possible HIV infection further reduces the likelihood of HIV disease, as well as the associated stress and anxiety.

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Clinical Communications

AN UNUSUAL CAUSE OF 'PTOSIS'

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□ **Abstract**—A 29-year-old man with a history of dental restoration procedure was referred for a left Bell's palsy. At the emergency department, he complained instead of deteriorating unilateral ptosis and dysphagia. Incidentally, trismus was also noted. He was diagnosed with cephalic tetanus, which rapidly progressed to generalized tetanus. Ptosis is an unusual presenting complaint of tetanus. In this case, we attempt to explain how facial weakness, ptosis, and cephalic tetanus are all related. We also highlight the key aspects of tetanus in relation to the emergency physician. © 2007 Elsevier Inc.

□ **Keywords**—tetanus; ptosis; Bell's palsy; cephalic; differentials

INTRODUCTION

Tetanus is caused by a ubiquitous bacillus with resilient spores. Although it is a disease that plagues developing countries, it is still seen in developed countries, with decreasing incidence (1,2). Emergency physicians play a significant role, as vaccination and judicious wound care remains the cornerstone of prevention. In addition, early recognition of this clinical disease entity would circumvent subsequent unnecessary investigations, delays, and irrelevant treatment. Unfortunately, mortality and morbidity remain high despite intensive care, as few advances in treatment have been made (1).

CASE REPORT

A 29-year-old man, an immigrant maintenance worker on an offshore island, was referred by a primary health

care clinic to the emergency department (ED) of a tertiary hospital. The referral noted left-sided facial weakness of 1 week's duration. He was initially diagnosed with left-sided Bell's palsy and treated with oral prednisolone. However, his symptoms persisted.

On presentation at the ED, his complaint, instead, was that of left-sided ptosis of 1 week's duration, and dysphagia for 1 day. His swallowing was worse with liquids than with solids. His past medical history included a tooth restoration procedure in Myanmar 3 months ago, which was uncomplicated, and he denied any other form of injury since. He was particularly concerned if the dental procedure was related to his symptoms.

Vital signs were normal: temperature 37.1°C, pulse rate 95 beats/min, respiratory rate 16 breaths/min, and blood pressure 137/87 mm Hg. Physical examination revealed the following: left-sided partial ptosis with normal pupils and no fatigability; inconsistent diplopia on left gaze with full extraocular movements noted by the examining physician. Fundoscopy was unremarkable. All other cranial nerves were intact and jaw opening was noted to be reduced to two finger breadths.

He had normal power of all limbs, hypertonic and hyper-reflexic lower limbs with flexor plantar reflexes and absent clonus. Examination of the lumbosacral spine and digital rectal examination were both unremarkable. There were no cerebellar signs and the gait was normal. There was no nuchal or abdominal rigidity. Neither was there any abnormal posturing or muscular tenderness.

Full blood count, renal panel, and electrolytes were normal.

In view of his ptosis, trismus, dysphagia, lower limb hypertonia and possible hyper-reflexia, he was admitted for further evaluation. Creatine kinase, sent from the ward, was noted to be elevated at 508 U/L. It was later that he recollected a trivial finger laceration by a metal plate while at work 2 weeks before presentation. He sought no medical attention then; neither had he received any tetanus vaccination.

Tetanus immunoglobulin, anti-tetanus toxoid, intravenous penicillin, and metronidazole were commenced. Prophylactic tracheostomy was done early the next day in view of the trismus and anticipated intubation difficulties. Computed tomography (CT) scan of the mandible was normal and the dental surgeon's opinion was that of secondary caries under the tooth restoration. The affected teeth were promptly extracted.

He was transferred to the intensive care unit 48 h later when he developed respiratory difficulties. Ventilation was instituted via tracheostomy mask. Creatine kinase escalated to 1239 U/L by the end of the second day, and he required sedation and intubation. He underwent a stormy course complicated by progression to generalized tetanus, transient apneic episodes, autonomic instability, nosocomial *K. pneumoniae*, and *S. aureus* pneumonia. Creatine kinase peaked at 3124 U/L on the 11th day.

After 15 days of intensive care, he was successfully weaned off the ventilator and transferred to the general ward for rehabilitation. He was reviewed by the ophthalmologist, who could not find any objective diplopia, and noted that a recent passport photograph taken 2 months earlier already showed a left partial ptosis. Exactly 1 month after admission, he was discharged and subsequently returned to Myanmar for recuperation.

DISCUSSION

Based on the clinical features of trismus, dysphagia, ptosis, hypertonia, and possible hyper-reflexia, our differential diagnoses in the ED included: tetanus, mandibular/perioral infections, demyelinating disease, and myasthenia gravis. Mechanical causes related to temporomandibular joint dysfunction were not considered due to the lack of pain and significant history of trauma or dislocation.

In the context of his dental operation, mandibular/perioral infections could possibly cause dysphagia and a certain degree of trismus. However, features against these included the presence of ptosis, a lack of local symptoms suggesting infection such as pain, and systemic features such as fever and leukocytosis. Hypertonia and hyper-reflexia with flexor plantar reflexes are occasionally seen in the anxious patient, who fails to relax and cooperate with the examining physician, and therefore are possibly unreliable signs.

Demyelinating diseases such as multiple sclerosis or Guillain Barré syndrome are other possibilities. These diagnoses are suggested by disseminated neurological features, which cannot be explained by a focal neurological cause. Multiple sclerosis (MS) could have accounted for the ptosis, hyper-reflexia, and hypertonia. Trismus could be attributed by muscle spasticity. He also had some inconsistent diplopia. Blood tests are frequently normal in MS as well. However, it is a rare disease, with a predominance in females (3,4).

Guillain Barré syndrome may cause neuromuscular weakness, which may present with dysphagia, trismus due to weakness of the muscles of mastication, ptosis, diplopia, and a normal sensation. However, it is usually ascending in nature from the peripheries and associated with hyporeflexia/areflexia instead of hypertonia and brisk reflexes. Of note, a Miller Fisher variant may present with cranial nerve palsies early in the illness (5,6).

Myasthenia gravis may present with ptosis, dysphagia, weakness of the muscles of mastication, and subjective diplopia (7). It does not cause hypertonia or hyper-reflexia. The admitting emergency physician did screen the patient for fatigability of upgaze and repetitive movements, but there was none elicited.

Other causes of polyneuropathies/cranial nerve palsies related to infectious diseases such as Lyme disease were unlikely due to their rarity locally, and the fact that he was well in the preceding months. A dystonic reaction was unlikely as well due to the absence of any drug history.

Tetanus is caused by an anaerobic Gram-positive spore-forming bacillus, *Clostridium tetani* (*C. tetani*) (1). It is diagnosed clinically and defined by the acute onset of hypertonia or by painful muscular contractions (usually of the muscles of the jaw and neck) and generalized muscle spasms without other apparent medical cause (8). Unfortunately, there are no characteristic laboratory tests. *C. tetani* is recovered in only 30% of wounds and can be present in patients without tetanus as well. A low level of anti-tetanus antibody (<0.1 IU) may suggest susceptibility. However, patients with a normal level may also succumb to the disease (9). Muscular rigidity, trismus, and dysphagia are the three most common complaints (2,10,11). Our patient presented with the latter two symptoms. Dysphagia in tetanus occurs, especially to liquids, as illustrated in our case, as opposed to dysphagia to solids, when there is a mechanical obstruction. Ptosis, however, was an unusual complaint.

Tetanus may manifest as one of four clinical entities: generalized tetanus, localized tetanus, cephalic tetanus, and tetanus neonatorum. In cephalic tetanus, cranial nerve palsies are known to occur, commonly with the VII cranial nerve (12). Our patient's ptosis was not associ-

ated with the impairment of extraocular movements or pupillary responses, therefore, a III cranial nerve palsy or Horner's Syndrome was unlikely. Interestingly, based on the ophthalmologist's review, it was concluded that his ptosis was likely to be chronic. This was validated by a passport photograph taken 2 months before admission. We recalled that he was initially misdiagnosed with a Bell's palsy. This is likely due to partial weakness of the frontalis muscle and *orbicularis oculi*. Because patients with congenital ptosis frequently compensate lid drop with hyperactivity of the frontalis muscle, we postulate that involvement of the VII cranial nerve from cephalic tetanus, with consequent weakness of the frontalis muscle, would have diminished his ability to compensate, therefore presenting with deteriorating ptosis.

It is not surprising that in the ED initially our patient could not recall any injury. In up to 25% of the cases, there is no identifiable source or history of injury. Puncture wounds (stepping on nails, barbed wire, and wooden splinters) have been cited as the most common cause of acute injury (50%). A laceration that our patient sustained was the second most common cause (33%) followed by abrasions (9%) (1,13). In addition, a higher proportion of cases are actually associated with lower limb injuries (51%) compared to upper limb injuries (36%) (13).

It was only after admission to the ward that the patient recalled a finger laceration. This was the likely site of inoculation. *C. tetani* spores can be found in soil, and animal and human excreta. The spores are highly resistant to extremes of temperature and moisture and commonly enter the body through breaches in the skin, germinating under conditions of hypoxia (14). The dental procedure 3 months ago was unlikely to be the source, due to the prolonged duration. However, in a summary of 89 tetanus cases, the incubation period for tetanus varied from 0 days to as long as 112 days (over 3 months), with a mean of 7 days (15). Generally, a shorter incubation period was observed to be associated with increasing disease severity (16).

Tetanus is an uncommon disease in developed countries since the introduction of tetanus toxoid vaccination. In the United States, the incidence has declined from 3.9 cases/million population in 1947 to 0.16 cases/million in the period 1998–2000 (13). In Singapore, tetanus vaccination is a mandatory component of our childhood immunization schedule. Our patient, however, was from Myanmar. He was uncertain of his tetanus vaccination status and it was plausible that he had not been vaccinated before.

In view of his signs and symptoms, unknown vaccination history, and recent injury, tetanus was the most likely diagnosis. This was further suggested by the elevated levels of creatine kinase, and development of nuchal rigid-

ity the next day. Of note, strychnine poisoning, a constituent of rodenticide, may produce a similar clinical picture as tetanus (17).

He was empirically commenced on antibiotics and human tetanus immunoglobulin. Immunoglobulin serves to neutralize tetanospasmin, a potent neurotoxin that is produced by the vegetative organisms. It should be administered at the earliest opportunity, as tetanospasmin undergoes retrograde axonal transport to the central nervous system, where it irreversibly blocks presynaptic inhibitory (GABA-ergic and glycinergic) neurons, giving rise to muscular rigidity (14). Although intrathecal administration of immunoglobulin has been shown to decrease morbidity and duration of illness, it is given intramuscularly (18). This is because immunoglobulin contains thimerosal, a neurotoxic component. An alternative to human immunoglobulin is equine immunoglobulin, which is available at a lower cost in certain centers, but with the added risks of allergy and anaphylaxis (1).

Our patient was administered both penicillin and metronidazole. Antibiotics are necessary to treat the wound infection and to eliminate production of tetanospasmin. Penicillin is the traditional antimicrobial of choice. However, one randomized control trial (RTC) in 175 patients showed that metronidazole was superior in mortality reduction (19), whereas another RTC of 1059 patients showed no significant difference (20). Penicillin has the added theoretical disadvantage of resembling GABA structurally. Hence, there are concerns that penicillin may aggravate neuronal disinhibition.

Surgical debridement of tetanus-prone wounds is an important facet of management. It improves survival (21). In our patient, the finger laceration had already healed, thus, there was no surgical role for debridement. However, because it was impossible to entirely exclude his dental procedure as a cause, the secondary caries that had developed under his dental restoration were treated with tooth extraction.

Despite early aggressive intervention, cephalic tetanus frequently progresses to a generalized form, as in our case. Airway management constitutes a vital component of supportive therapy (12). Muscular spasms and periods of apnea may require ventilation. Intubation may be potentially difficult due to the abnormal extensor posturing and rigidity. Our patient underwent tracheostomy in view of his early nuchal rigidity. Subsequent episodes of apnea were thus easily managed via the tracheostomy. In addition, a tracheostomy facilitates convenient suctioning of tracheal secretions.

Our patient was fortunate to have survived tetanus. This is given the fact that inadequately vaccinated patients have a higher morbidity and mortality compared to those who have received a complete course of tetanus immunization (13,22). To this day, the case fatality ratio of

tetanus remains high. In a study of 113 tetanus patients, the mortality rate was 18%, of which three-quarters were aged 60 years and above (13). Tetanus, once described as an 'inexcusable disease', is ultimately best prevented, rather than treated (23).

In developed countries where standard immunization programs and improved wound care facilities have drastically reduced the incidence, lack of awareness by both doctors and the public still remains a major pitfall (2). Contaminated tetanus-prone injuries are often inappropriately dismissed by patients without adequate antibody levels. A population-based seroprevalence survey conducted from 1988 to 1994 in the United States showed that despite immunization, 20% of adolescents aged 12–19 years had inadequate levels of antibodies (24). In the United States 1998–2000 surveillance of 129 tetanus cases, only 37% sought medical treatment and only 63% of them received tetanus toxoid (13). Compliance with established vaccination guidelines is vital. Patients with tetanus-prone wounds and an unknown vaccination history are occasionally inadequately managed with only tetanus toxoid, but not the immunoglobulin. Therefore, emergency physicians, in their daily practice, play a pivotal role, via patient education, vaccination, and fastidious wound management, in the prevention and recognition of tetanus.

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Cephalic tetanus following tooth extraction in a Nigerian woman

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Sir,

Tetanus is an acute, often fatal, disease caused by an exotoxin produced by the bacterium *Clostridium tetani*. Out of the four major clinical types of tetanus (generalized, localized, neonatal and cephalic), cephalic type is the rarest.[1] We encountered a case of cephalic tetanus in a young Nigerian woman six months after she had tooth extraction.

The patient is a 22 year old Nigerian lady, who four days before admission, developed difficulty in opening her mouth as a result of pain, which was associated with inability to swallow, excessive salivation and neck pain. At the time of presentation, patient was noticed to be having intermittent spasm of the muscles of the face and neck. Angle of her mouth was also deviated to the right; however, patient was conscious with ability to appropriately respond to communication in between spasm. There was no discharge from the ear or the nostrils. No relevant history of cough or head trauma was given. Six months before the onset of her symptoms, patient had a mouth infection for which her tooth was extracted from a tertiary health centre. There was no tetanus toxoid given to the patient at the time, though the patient claimed she completed her infancy tetanus toxoid schedule according to the National Programme on Immunization. The patient had no history of genital instrumentation.

At the time of physical examination, the patient had a body temperature of 37.2°C, pulse of 102 beats per minute which was regular, a respiratory rate of 24 cycles per minute and a blood pressure of 100/80 mmHg. Significant physical findings were seen in the neurological examination. Though patient was conscious, she was tensed up and had trismus. She was able to communicate with difficulty amidst intermittent spasm of the muscles of the face and neck. She had neck stiffness but negative Kernig's and Brudzinski's sign. There was left incomplete ptosis and lower motor neuron type left facial nerve palsy. Power in the four limbs was normal and deep tendon reflexes were also normal. Sensory functions were apparently within normal limits though examination was limited because of the patient's condition.

Chest radiograph and lumbar puncture results were within normal limits. Electrocardiogram revealed a sinus tachycardia with a ventricular rate of 103 and a non specific ST-T wave changes. There was leucocytosis with a total white blood cell count of 16,000/mm³. The patient was managed in the general ward with antitetanus sera, tetanus toxoid, intravenous antibiotic, diazepam and ophanedrine. Other supportive treatments were given as well.

Patient did well and was discharged home after 8 days on admission. She had since been seen twice in the medical outpatient with sustained improvement.

Cephalic tetanus presents with cranial nerve palsy, particularly of the facial nerve, and is always scored as severe or very severe, since approximately two thirds of cases progress to generalized tetanus.[2] However, our patient did not progress to develop generalized tetanus. We could not send the patient for

neuroimaging such as Computerized tomography scan or magnetic resonance imaging scan because they were not available in our centre and the nearest facility where there was a functional one is more than 200 kilometer far from our centre.

In conclusion, it is important to highlight a few aspects of this case.

First, this patient's incubation period was long and she never progressed to develop generalized tetanus which probably might have influenced the good clinical outcome.[3] Second, the management of this patient has once again exposed the poor facilities prevailing in our health sector. Finally, tetanus is still a major health burden in Nigeria. There is need to promote health education to reduce the prevalence of this disease in this part of the world.

References

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