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Expert consultation is recommended when managing occupational HIV exposures

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Risk of HIV exposure in health care personnel is associated with percutaneous injury (i.e., needle stick or cut with a contaminated needle/instrument) or contact of mucous membranes or non-intact skin with blood, tissue or other potentially infectious body fluids. Blood and all visibly bloody body fluids are potentially infectious. Other body fluids considered potentially infectious include semen and vaginal secretions (though neither of these have been associated with known occupational HIV transmission), CSF, synovial, pleural, peritoneal, pericardial and amniotic fluids. The transmission risk associated with these latter fluids is not known. Other body fluids not considered potentially infectious unless visibly bloody are feces, urine, nasal secretions, saliva, sputum, tears and vomitus.

The estimated risk of transmission from percutaneous exposure to HIV-infected blood is 3 in 1000 (0.3%). For mucous membrane exposure, the risk is approximately 1 in 1000 (0.09%). The risk of transmission from non-intact skin exposures is not known but thought to be less. The risk after exposure to other potentially infectious body fluids and tissues is thought to be even lower. The risk of HIV transmission increases with exposure to a larger volume of blood which is in turn associated with visible patient blood on the sharp, hollow-bore needles, procedures involving a needle placed directly into the patient's vein or artery and deep injuries. Advanced HIV disease in the source patient also confers higher risk for transmission.

Postexposure prophylaxis (PEP) should be initiated as soon as possible

See *Occupational PEP*, next page

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Pharmacy

Update: Antiretroviral agents in expanded access

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The expanded access process is designed to make promising antiretroviral products available as early in the drug evaluation process as possible to patients without therapeutic options either because they have exhausted or are intolerable of approved therapies. This process includes treatment

IND (investigational new drug) protocols and parallel track protocols usually designed for sicker, more advanced patients who are unable to participate in controlled studies.

Currently estimated rates of resistance among HIV-infected patients

An estimated 40,000 HIV-infected patients have developed resistance to avail-

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Medicine

Some drugs should be avoided in PEP due to toxicities

Occupational PEP, from previous page

after a significant exposure has occurred, within hours not days, and should be continued for four weeks. The guidelines for selection of a PEP regimen are summarized in Tables 1 and 2. The revised guidelines published in 2005 changed and expanded the drugs that are recommended for use in PEP regimens. Additional information on toxicities associated with some antiretroviral medications has led to the recommendation to avoid specific drugs, both in general and in pregnancy (see Tables 3 and 4).

The preferred expanded PEP regimen (three or more drugs) is now protease inhibitor based. In general, expanded PEP is recommended for higher risk exposures and basic or two-drug PEP for lower risk exposures. If the HIV status of the source patient is unknown, rapid HIV testing should be performed if possible to guide the decision regarding PEP initiation. If not possible, PEP should be offered if the source patient has risk factors for HIV infection. For unknown source exposures (e.g., discarded needle or sharp), PEP should be offered if the risk of contamination is high, for example, an exposure involving a sharp from an area in a healthcare facility where HIV-positive patients are treated.

If the source patient's virus has documented or suspected antiretroviral resistance, the choice of PEP drugs should be modified to include drugs to which the virus is less likely to

be resistant. Resistance should be suspected in patients who are taking antiretrovirals and failing. Expert consultation should be sought whenever possible. Information about viral resistance mutations and antiretroviral history can assist in the selection of a PEP regimen. PEP initiation should, however, not be delayed if either consultation or information regarding the source patient's virus or medications is not readily available. PEP modifications can be made in the first few days if additional

**Healthcare worker
HIV exposures
should be treated
as urgent
medical events.**

information becomes available. Resistance testing at the time of exposure is not recommended since the results will not be available to guide initial PEP selection. PEP changes made after resistance test results are known (one to two weeks later or more) have not been shown to alter the outcome of PEP.

HIV-antibody testing should be done at baseline, six weeks, twelve weeks and six months after exposure. Extended HIV testing is only recommended for health care workers who acquire hepatitis C after exposure to an

HIV/HCV co-infected source patient. Symptoms consistent with HIV seroconversion syndrome in exposed health care workers should prompt HIV testing regardless of the amount of time that has elapsed since the exposure.

Monitoring for toxicity related to PEP should include a baseline CBC and renal and hepatic function tests. Repeat testing should be done after two weeks. Additional testing should be based on the exposed health care worker's underlying medical conditions, if any, and the specific toxicities associated with the PEP drugs used. Blood glucose monitoring should be included for protease inhibitor-containing PEP regimens and monitoring for kidney stones, hemolytic anemia and hepatitis should be added for indinavir-containing regimens. If toxicity occurs, modification of PEP should be considered, as well as the need for further diagnostic testing.

Prior to the initiation of PEP, the health care worker should be counseled about the importance of completing the PEP regimen. He/she should also be evaluated for drug interactions with preexisting medications and given information about side effects, side effect management, toxicity and monitoring, and potential drug interactions. Reevaluation 72 hours after initiating PEP should be strongly considered for assessment and treatment of side effects, especially nausea, vomiting and diarrhea as these occur frequently in health care workers taking PEP. Potential changes in



the PEP regimen based on additional information about the exposure or the source patient may also be instituted at this time.

The current HIV PEP guidelines emphasize that health care worker HIV exposures should be treated as urgent medical events. When indicated, PEP should be initiated promptly within hours. The selection of PEP drugs should avoid unwarranted toxicities. Close follow-up care is recommended to manage side effects, maximize adherence and monitor for adverse events. Expert consultation in all aspects of HIV occupational exposures and management is stressed. ❖

REFERENCE

CDC. Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. MMWR 2005;54(No.RR-09):1-17.

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Table 1

	Basic PEP	Expanded PEP (add to basic)
Preferred	AZT +3TC or FTC tenofovir +3TC or FTC	lopinavir/ritonavir
Alternatives	stavudine +3TC or FTC didanosine +3TC or FTC	atazanavir + ritonavir fosamprenavir +/- ritonavir indinavir + ritonavir saquinavir + ritonavir nefinavir efavirenz enfuvirtide *

*Use in PEP only with expert consultation

Table 2

PEP RECOMMENDATIONS BY EXPOSURE TYPE/SOURCE STATUS

Exposure type	HIV + asymptomatic or VL < 1500	HIV + symptomatic or VL > 1500	HIV status of source unknown	Unknown source
Percutaneous, high volume	Expanded PEP (>= 3 drugs)	Expanded PEP	No PEP / consider basic 2 drug PEP if HIV risk factors	No PEP / consider basic 2 drug PEP if HIV exposure likely
Percutaneous, low volume	Basic PEP (2 drugs)	Expanded PEP	No PEP / consider basic 2 drug PEP if HIV risk factors	No PEP / consider basic 2 drug PEP if HIV exposure likely
Mucous membrane/non-intact skin, large volume	Basic PEP	Expanded PEP	No PEP / consider basic 2 drug PEP if HIV risk factors	No PEP / consider basic 2 drug PEP if HIV exposure likely
Mucous membrane/non-intact skin, small volume	Consider basic PEP	Basic PEP	No PEP	No PEP

Table 3

ANTIRETROVIRALS TO GENERALLY AVOID IN PEP

Drug	Rationale
Abacavir	Hypersensitivity reaction
ddC	Neuropathy, poor potency
Delavirdine	Allergic reaction
ddI/d4T combination	Neuropathy, pancreatitis
Nevirapine	Hepatotoxicity, hypersensitivity syndrome

Table 4

ANTIRETROVIRALS TO AVOID IN PREGNANCY

Drug	Rationale
Efavirenz	Teratogenic potential
ddI/d4T combination	Lactic acidosis, fatal and non-fatal
Indinavir	Hyperbilirubinemia (avoid near delivery)



Pharmacy

Patients without options can qualify for early access to new drugs

Expanded Access, from page 1

able antiretroviral agents and so rely on a complex array of available drugs to treat their infection. Drugs currently in expanded or early access include entry inhibitors such as the CCR5 inhibitors, an intravenous monoclonal antibody entry inhibitor, an oral integrase inhibitor and a second generation non-nucleoside reverse transcriptase inhibitor (NNRTI).

This article will review the four main antiretroviral agents currently available by expanded access: 1) the CCR5 entry inhibitor Maraviroc, 2) the monoclonal antibody entry inhibitor TNX-355, 3) the oral integrase inhibitor MK-0518, also called raltegravir, and 4) the second generation NNRTI Etravirine.

Maraviroc (UK-427,857)

This is a CCR5 antagonist designed to prevent HIV infection of CD4 cells by blocking the CCR5 receptor. CCR5 blockade means that "CCR5-tropic" HIV can no longer engage in a T-cell to infect the cell. The CCR5 variant predominates in early stages of HIV infection while, later in the disease, viruses adapted to the use of the CXCR4 receptor are known to dominate.

This agent, which is the first in its class, is being developed by Pfizer Inc. and was offered in early expanded access by 2006 to HIV-infected patients with little or limited options for continued therapy. An FDA advisory panel will meet to examine safety and efficacy data for this drug by April 24, 2007.

Maraviroc:

Results of early Phase I studies

Preliminary results of early Phase I studies were presented in major conferences by 2003. In one of the major studies, 25 HIV-infected patients with CCR5-tropic viruses were randomized to Maraviroc 25 mg daily or 100 mg two times daily or placebo for 14 days as monotherapy. Steady state (SS) drug levels were reached in seven days with better drug levels in the fasted state. By day 14, the 100 mg twice daily group

had experienced a viral load drop of 1.4 logs versus 0.4 log drop for the 25 mg daily group. The drug was well tolerated and no rebound viral load was reported upon cessation, indicating that a population of receptors remained blocked for sometime. A second placebo-controlled study confirmed the above. Dosages recommended from these early studies are 100 mg twice daily or higher. Other dose-ranging studies also showed similar decreases in viral load of greater than one log at doses above 100 mg daily or twice daily with no effect of food on the antiretroviral efficacy of the agent.

Effects of Maraviroc on lipids, hematological parameters and blood chemistry

A placebo-controlled study of Maraviroc 100 mg and 300 mg twice daily in 54 HIV-negative adults showed no effects on lipids, blood chemistry or hematological parameters. No ECG or changes in heart rate were noted in this study.

Further studies on Maraviroc

In Pfizer's two main studies on Maraviroc, a total of 1,049 HIV-positive patients were studied. Eight hundred and forty received Maraviroc with their regular drug regimens with an optimized background regimen (OBR), following both genotypic and phenotypic testing. The rest received a placebo with their regular antiretroviral regimens. All patients had developed resistance to all three classes of available antiretroviral agents.

After 24 weeks of a 48-week study, more than 40% of the patients who received Maraviroc had undetectable viral load; 60% of patients on Maraviroc also experienced declines in their viral loads to manageable levels.

Toxicity of Maraviroc

The major side effects reported in the key studies were headache, dizziness, flatulence, asthenia, postural hypotension that was dose-related and reported to be rare at doses less than 300 mg twice daily, abnormal vision, arthralgias, flu syndromes, myalgias and increased liver function tests.

Recent studies have shown that resistance to Maraviroc probably emerges slowly but is not always associated with a loss of use of the CCR5 co-receptor.

Metabolism and key drug-drug interactions of Maraviroc

Maraviroc is metabolized by the CYP3A isoenzyme and is also a P-glycoprotein substrate. However it does not inhibit activity of expressed enzymes such as CYP3a4, CYP2C9, CYP2C19 or CYP1A2. It is a weak inhibitor of CYP2D6. It is 5-15% excreted unchanged in urine and so is not likely to need adjustment in patients with renal impairment. CYP3A4/P-glycoprotein inhibitors such as ketoconazole, saquinavir, lopinavir/ritonavir, atazanavir and ritonavir caused significant increases in systemic exposure of Maraviroc ranging from 2-5 fold mean increase in C_{max} and 3-10 fold increase in AUC. CYP3A4/P-glycoprotein inducers such as efavirenz and rifampicin resulted in significant reduction in systemic exposure of Maraviroc ranging from 56-70% mean decrease in C_{max} and AUC.

Alpraviroc (GSK-873, 140)

This is an investigational entry inhibitor that is being studied in clinical trials for the prevention and treatment of HIV infection. All studies have been halted because of severe side effects to the liver which occurred in both treatment-naïve and treatment-experienced patients. No further studies are planned for this agent at this time.

TNX-355

This is an experimental entry inhibitor being developed by Tanox Inc. It contains genetically-engineered antibodies known as monoclonal antibodies that bind to the CD4 receptor on T-cells, thus preventing the connection of the HIV virus with the surface of T-cells and so preventing viral infection of healthy T-cells. The dosages are not yet quite established; the drug is effective upon intravenous use, once every two weeks and the dose depends upon body weight.



Efficacy, proof of concept and Phase II studies

Eighty-two HIV-infected, highly treatment-experienced patients with failures to other agents were studied. They received TNX-355 with an optimized background regimen based on genotypic and phenotypic studies. Two out of three patients received one or two doses of TNX-355 (10 mg or 15 mg per kg BW). One out of three patients received placebo through an intravenous central line once a week for the first nine weeks, then once every other week for the remainder of the study period of 48 weeks. After 48 weeks, treatment with the 10 mg/kg dose resulted in a viral load decline of 0.96 logs while treatment with the 15 mg/kg dose resulted in a viral load decline of 0.71 log. Viral load decline in the placebo group was 0.14 log. Patients given the two different doses of TNX-355 reported a greater increase in their T-cells compared to the placebo group (an average increase of 51 T-cells in the 15 mg/kg group and an increase of 48 T-cells in the 10 mg/kg group). Both doses of TNX-355 were well tolerated. In order to move into Phase III clinical trials, FDA has requested additional early-stage Phase II studies in order to determine the correct dosage of the drug.

Oral integrase inhibitors in expanded access by 2007

MK-0518, now raltegravir, represents a new class of antiretroviral called integrase inhibitors, which work by preventing the insertion of the HIV viral DNA into the human DNA genome and by so doing block the ability of the virus to replicate and infect other cells. It is being developed by Merck Inc.

Review of major Phase II/III studies

Two main studies of about 700 HIV-infected patients were funded by Merck. All participants had highly resistant viruses failing multiple therapies in all three drug classes. In 16 weeks of standard drugs using optimized background regimens (OBRs) versus OBRs plus MK-0518. After 16 weeks, viral loads fell to below levels of detection in 62% of patients on MK-0518 versus 32% of those who received the standard drugs alone. Eighty percent of patients receiving raltegravir had a decrease in viral loads to a level that was considered manageable. The

drugs were well tolerated and side effects were minimal and manageable.

Etravirine (TMC-125)

Etravirine is a second-generation NNRTI that works by blocking the reverse transcriptase enzyme. Expanded access program on this agent started by Tibotec by September of 2006. It comes as capsules and tablets for peroral dosing and is being currently studied with food and with no food.

Several small dosing studies of etravirine have been presented at conferences. Results of these studies show significant reductions in viral load after just one week of treatment. A placebo-controlled Phase IIA study was designed to test the antiretroviral activity of 900 mg dosed twice daily. After one week of treatment in 12 persons, average viral load fell by two logs and ten out of twelve patients achieved viral loads below 400 copies/ml. No resistance was seen nor were any serious adverse effects reported.

Efficacy of etravirine (TMC-125)

It appears to be effective in persons resistant to efavirenz and other NNRTIs. In a study of 16 persons failing an EFV-based regimen, seven days after substituting etravirine for the failing NNRTI viral load fell by an average of 0.9 logs despite decreased susceptibility to efavirenz and nevirapine. No relationship was seen between response and genotypic and phenotypic resistance nor between drug concentrations and treatment response. Etravirine alone was just as potent as a combination of AZT, 3TC and abacavir with indinavir and nevirapine. Viral load declined by 1.2 logs in the etravirine group in the first seven days of treatment compared to 1.55 logs in the 5-drug group.

Resistance profile of etravirine

Etravirine showed a slower emergence of resistance compared to virus exposed to efavirenz or nevirapine. Etravirine shows specific flexibility in its attachment to the reverse transcriptase enzyme that continues to allow inhibition when older NNRTIs have failed. Mutations V179F, Y181C, L214F and M230L are associated with resistance to etravirine. There is clinical evidence that virus which has high level resistance to efavirenz and nevirapine is susceptible to etravirine.

Side effects and drug interactions with etravirine

Side effects that were most frequently reported for etravirine were dizziness, headache, blurred vision, mild diarrhea, flatulence and mild rash. All of these resolved in a few days; rash appeared to be the related to the maximum concentrations (C_{max}) of etravirine. The maximum tolerated doses have not yet been established since no grades 3-4 adverse events have so far been reported in these studies.

Drug interactions with protease inhibitors make combinations with etravirine rather complicated. This is especially important for a second-generation NNRTI that may be used in salvage therapy with PIs. For example, etravirine blood levels are decreased by 45% with ritonavir and increased by 45% and 17% when co-administered with indinavir and tipranavir respectively. Etravirine reduces levels of saquinavir by 40% (both hard and soft-gel versions). Phase II studies are being repeated to further investigate the best dose, which is likely to be between 400 and 1600 mg twice daily.

Several new and exciting agents are currently available for early expanded access to heavily-treatment-experienced HIV-infected patients with limited options. Many of these patients are currently achieving good virological and immunological outcomes using optimized background regimens along with enfuvirtide in salvage regimens that are continuing to make their HIV infection a manageable disease. FDA review of one of the agents is scheduled for April 2007 and approval of at least one and possibly more of these agents is anticipated later this year if all studies go as planned. ♦

References available upon request.

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Nursing

Understanding and compassion are key to transgender HIV care

Carole Pindaro, RN, MSN, MPH

Have you ever had to wear clothes that did not fit correctly, or were just not your style? You may have felt self-conscious or out of place wearing them. This analogy can be used to describe transgendered persons who “do not feel right in their own skin.” The intent of this article is to define some of the unique issues faced by male-to-female transgendered persons: nomenclature, specific health concerns, and medication therapy. Much of what is noted here, except medication therapy, applies to female-to-male transgenders as well. Because male-to-female transgenderism is predominant in the area where I work, it will be the focus of this article.

Having worked in an HIV outpatient clinic for 20 years, I have had the opportunity to care for many HIV-infected transgenders. They report past health care experiences in which they were misunderstood, not taken seriously, experienced judgmental comments, unnecessary questioning, outright rejection or denial of care. Consequently, many have had no primary, much less HIV-specific, care in years. For this reason, some come into HIV care late, with a low CD4 count.

As clinicians, it is our duty to learn about the care needs of the transgendered population. They deserve to be treated with the same dignity and respect as any other patients. If after self examination we cannot put aside our biases and care for

transgendered individuals in an effective, non-judgmental manner, then we should refer them to a clinician who will.

The Diagnostic and Statistical Manual (DSM-IV-TR) uses the term gender identity disorder (GID) to refer to those “with strong and persistent cross-gender identification (and not merely a desire for any perceived cultural advantages of being the other sex), and a persistent discomfort with their sex, or a sense of inappropriateness in the gender role of that sex.” These individuals may truly believe that they were born the wrong sex. They suffer significant distress or impairment in social or work settings and may spend much time working on the appearance of being female.

The term “transgender” has come into popular usage. It is not a formal diagnosis, but is used to refer to a person with any type of gender identity issue, in a value free manner. There is no connotation of psychopathology.

Those with GID are to be differentiated from transvestites, who wear clothes of the opposite sex in order to experience temporary membership in the opposite sex. They do not dress in clothing of the opposite sex full time, nor do they desire a permanent change to the opposite sex.

Because transgenderism is delineated in the DSM, it may lead one to believe that it is a psychiatric disorder. This is not necessarily true, unless the person exhibits severely maladapt-

tive behaviors and experiences great mental suffering. Recall that the purpose of the DSM is to guide diagnosis for proper treatment and research. For the transgendered individual, a formal diagnosis is often a requisite step in getting a hormone prescription or a referral for gender reassignment surgery. Designating a person as having GID is not a license to stigmatize or discriminate.

Imagine that you are in your busy clinic and you hurriedly pull the chart of the next person to be seen. After glancing at the chart cover, you proceed to the waiting room and call the patient using only first name and last initial, as per clinic confidentiality policy. When you call “Donald M” a very tall woman with flawless hair and nails stands. Once in the exam room you begin talking and you notice that her voice is a bit deep, her hands and feet seem large, and she has an Adam’s apple. Thoughts racing through your mind might relate to whether this is really a woman, how you will ask her without being offensive, and how her care needs differ because of her situation. Although you may feel overwhelmed, angry or betrayed, you try to keep a neutral facial expression and an open body posture.

When faced with this type of situation, you should take a deep breath and realize that the patient before you is likely having a more difficult time than you are because of past negative experiences with the health



care system. This is the time to call on your active listening and interviewing skills.

It has been my experience that most transgendered patients will honestly tell you their story if you inquire politely, listen carefully and treat them with the courtesy and respect that you would give to any other patient. Explain that your reason for asking is so that you can provide the best care possible to meet the individual's specific needs.

The medication history is often revealing, as when the patient reports a female hormone as one of her current medications. If the patient is not currently taking one, it may be

**A respectful approach
can mean the
client is more likely
to return for
follow-up HIV care.**

that she has not had a means of getting a prescription. It is important to ask about lifetime hormone use. Earlier age of initiation of female hormones is associated with more pronounced female characteristics. Past use of injectible hormones with shared needles, and a history of trading sex for medication, may be the source of her HIV infection, and is an area of focus for education about reducing transmission of the virus.

The surgical history may reveal any number of feminizing

surgeries or none at all.

It would not be out of place to ask if the patient is satisfied with the results of her hormone and/or surgical therapy, and what her ultimate goals are.

The psychosocial history should address who the patient's support system includes and whether they are accepting of her current lifestyle. Ask about the patient's means of financial support. Substance use history is important, as alcohol and drugs may be used more frequently in persons who are marginalized and not accepted by society as "normal."

With an interested and respectful approach, you have the beginnings of a good rapport and the transgendered client is likely to return for follow-up care which is essential to effective HIV treatment.

The origins of GID are unknown and there are few scientific studies to guide treatment. Despite limitations of knowledge in this area, there are international standards of care to guide treatment (The Harry Benjamin International Gender Dysphoria Association's Standards of Care for Gender Identity Disorders, Sixth Version, February, 2001). The goal of care should always include helping transgendered clients to feel comfortable with the gender they are trying to become, so they can achieve long lasting psychological well-being and self-fulfillment.

Nearly all of the transgendered clients presenting to my clinic for an initial visit have identified as transgendered for at least a year prior. Many have been taking female hormones since their teens or early 20s. Frequently these medications have been obtained from illegal

sources and used in an injectible form, possibly with contaminated needles. Often they have had silicone injections to their breasts, hips or face to soften or feminize their features. Some have had breast implants and fewer have had castration and vaginoplasty as most cannot afford these costly surgical procedures which are not covered by insurance. Some wish only to take female hormones and forego any type of surgery.

The client presenting with a history of female hormone use is approached differently from the client who has never taken female hormones or had feminizing procedures. Because initiating hormones can be associated with irreversible body changes such as breast development, it would be prudent to involve a mental health clinician experienced in working with transgendered clients prior to instituting any therapy associated with permanent changes.

Once it is determined that a client has been living as a female and it is apparent from the female body characteristics present, it is generally safe to prescribe female hormones unless there are absolute contraindications. Precautions and contraindications to the use of female hormones in this population are the same as for genetic females. Prescription of female hormones should be done in the context of primary and HIV care, and can be an incentive to get patients into care and to stay in care. HIV and treatment for HIV alone are not contraindications for female hormone use. While drug-drug interactions can occur, there are no known serious interactions or causes of antiretroviral failure.

See *Transgender*, next page



Nursing

Hormone therapy can be incentive for some patients to stay in care

[Transgender](#), from [previous page](#)

The client should be informed of the risks associated with female hormone use and an informed consent obtained. Safer sex practices should be encouraged. A thorough history, physical and laboratory testing should be done to rule out any contraindications to hormone therapy. Clients should be made aware that while hormones are being prescribed, they also are expected to attend to their general and HIV-specific health care needs. Ongoing prescription may be contingent upon regular follow-up, but care should be taken to be non-punitive. Some clinicians may prefer a harm reduction approach where prescriptions for female hormones are more liberally given so that clients will not obtain them illegally with potential use of used needles.

A guiding principle for prescribing female hormones is: "more is *not* better." This can be a difficult myth to overcome as patients erroneously believe that they will have better results faster with larger doses. In truth, heredity limits tissue response to hormones and so a larger dose cannot overcome genetics. Patients can be told to expect at least a year and possibly two before reaching their maximum breast size, and it is not likely they will have a larger breast size than their first degree female relatives. It is always best to use the lowest effective dose. (See Table 1 for starting, usual and maximum dosages*.) Note that estrogen doses can be re-

duced to a minimum after gender reassignment surgery or after maximum feminization, which is usually after two years of higher doses. Patients often wrongly believe that they have to continue the dose they started with or escalate the dose even though they may have been taking estrogens for many years.

As part of HIV care, we provide primary and health maintenance care according to national standards. As a matter of routine, we give vaccines when they are due, diligently apply PPDs and counsel about smoking cessation. We must not forget that our male-to-female transgender client needs a mammogram and clinical breast exam, *and* PSA and digital rectal exam as indicated by age. Keep in mind that the parts of the exam pertaining to unwanted gender are the most difficult and embarrassing for the client. Drape the client appropriately and reassure her that you are providing essential components of comprehensive, preventive health care.

This article provides some basics for working with transgendered clients. You may contact the editor (tnewto@lsuhsc.edu) for clinical consults, reference materials, and consent for care forms. With a little extra care and understanding, you can add quality of life for those who may have been disenfranchised in the health care system. You will undoubtedly contribute to their commitment to stay in care for HIV which is a noble goal in itself. ♦

**from the Protocols for the Reassignment*

of Gender, Tom Waddell Health Center Transgender Team, San Francisco Department of Public Health, San Francisco, CA.

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The Harry Benjamin International Gender Dysphoria Association's Standards of Care for Gender Identity Disorders, Sixth Version, February 2001. www.wpath.org (The World Professional Association for Transgender Health, formerly known as the Harry Benjamin International Gender Dysphoria Association)

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Table 1 APPROXIMATE ESTROGEN BIO-EQUIVALENCES	
Medication	Gender Reassignment Dose
conjugated estrogens (Premarin)	Starting: 1.25-2.5mg/d Average: 5mg/d Maximum: 10mg/d
estradiol	Starting: 2-3mg/d Average: 4mg/d Maximum: 8mg/d
estradiol valerate for injection (Delestrogen)	Starting: 20-40mg IM q2wks Average: 40mg IM q2wks Maximum: 40-80mg IM q2wks
estradiol patch	Starting: 0.1-0.2mg/d Average: 0.2-0.3mg/d Maximum: 0.3mg/d

Source: Tom Waddell Health Center Protocols for Hormonal Reassignment of Gender (2006)

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Bacterial endocarditis and antibiotic prophylaxis in HIV dentistry

Kishore Shetty, DDS

Infective endocarditis (IE) is a rare infection of the endocardial surface, with an incidence of 0.6 to 11.6 cases per 100,000 person-years.¹ A number of other names have been used for IE, such as bacterial endocarditis, acute bacterial endocarditis, subacute endocarditis, and chronic endocarditis. Though they demonstrate the variable presentations of the clinical disease, they are all essentially the same process: a microbiological infection of the endothelium of the heart.²

The relationship between immunosuppression and the development of infective endocarditis (IE) in humans is not clear. A number of studies have demonstrated higher rates of IE among HIV-infected intravenous drug users (IDUs) than among HIV-negative IDUs and an inverse relationship between IE occurrence and CD4 lymphocyte count. The introduction of highly active antiretroviral therapy (HAART) has reduced morbidity, mortality, and hospitalizations associated with HIV infection. In addition, the epidemiology of HIV has changed, with more women, minorities, and IDUs becoming infected in the past decade. Studies in the pre-HAART and early HAART eras indicated an increased risk of IE in HIV-infected patients and higher mortality rates from IE among severely immunosuppressed HIV-infected patients.

There are cardiac conditions that require antibiotic prophylaxis for certain kinds of dental treatment in order to reduce the risk of IE. Medical and dental practitioners are all familiar with this, and there is a clear consensus that for a certain group of high-risk cardiac and high-risk dental procedures, antibiotic prophylaxis is mandatory. However, a consistent regimen is not always used when it comes to other similar circumstances, such as for the intermediate group of cardiac and dental procedures which calls for individual evaluation and determination of whether prophylaxis is taken. Some even feel that the efficacy of antibiotic prophylaxis against infective endocarditis has not been demonstrated and that there is not much evidence to support specific recommendations made by professional organizations.¹ This report will discuss bacterial endocarditis,

protocols for antibiotic prophylaxis, patient management, and treatment considerations in the HIV-positive patient population

Bacterial Endocarditis/ Infective Endocarditis

Endocarditis occurs when bacteria enter the bloodstream and infect damaged endocardium or endothelial tissues. The process of pathogenesis can be divided into four basic steps: 1) endocardial damage, 2) establishment and persistence of bacteria within the endocardium, 3) bacterial growth with local tissue damage, and 4) clinical IE with cardiac and non-cardiac manifestations.² Normal endothelium is a single cell layer that lines the inside of the heart and all blood vessels and is resistant to colonization by circulating bacteria. The most common sites of damage to endothelial cells occur in the mitral and aortic valves, ventricular septal defects, and complex congenital abnormalities, which are regions with the most mechanical stress.^{2, 3, 18} Damage also leads to metabolic and synthetic events as well as morphological changes, and inflammatory activation of these cells cause thrombotic vegetations to develop on the surfaces of valves. These events further change the haemodynamic properties, creating an ideal situation for bacteria to adhere and colonize, as well as being a significant source of emboli. For patients who have already had previous valve damage, such as scarring from rheumatic fever, or who have previously had bacterial endocarditis, such changes are the greatest. In addition, artificial materials, such as those used in prosthetic heart valves, are especially thrombogenic. Once the bacteria have colonized, they must survive, mature, and become fully enveloped, all while evading or resisting host defenses. Clinical manifestations of IE are quite variable with fever being the most common symptom. Most of those with IE also have a heart murmur, which may have been pre-existing, splenomegaly, or petechiae of the skin, conjunctiva or oral mucosa. Less common symptoms are anorexia, weight loss, malaise, and night sweats. Non-cardiac manifestations include arterial emboli and are apparent in about 50% of patients with neurologic symptoms being

secondary to embolic strokes and occurring in 40% of patients. IE is diagnosed from clinical, laboratory, and echocardiographic data. Treatment is complex and requires close collaboration among specialists in infectious diseases, cardiology, cardiac surgery, and microbiology.^{3, 13} Penicillin G is the drug of choice for most cases of endocarditis caused by *S. viridans* with a starting dose of four million units i.v. every six hours for about four weeks. In the case of penicillin-resistant streptococci, high doses of penicillin G (18 to 30 million units per day) and gentamicin, ampicillin plus gentamicin, or vancomycin plus gentamicin are used, with treatment often extending to six weeks.³ Often times cardiac surgery is required as well. The mortality rate, with good treatment and care, is generally 20-25%.

Of particular significance to the dentist is a patient with a past history of rheumatic fever. Rheumatic fever is an autoimmune disease triggered following a streptococcal sore throat. It occurs due to a cross reactivity of streptococcal antigen and antibody produced against it within the immune system. These antigen-antibody complexes may localize within tissues such as the heart, kidney, and joints, activate the complement system and lead to tissue damage resulting in rheumatic heart disease, arthritis, nephritis, or a combination. Also, a patient may have had rheumatic fever without any such complications. Though a common disease until the development of antibiotics, rheumatic fever has virtually disappeared in developed countries.⁴

Antibiotic Prophylaxis

It is known that dental treatment produces a greater bacteremia than normal physiologic function. Though it is still low grade and of short duration, there is rationale for the use of antibiotic prophylaxis for certain dental procedures, especially for certain patients, i.e. high risk cardiac patients. Although the primary mechanism by which antibiotics might prevent endocarditis has not been established, there are important general principles involved in ideal antibiotic prophylaxis. They include these: 1) the specific organism should be known, 2) an antibiotic effective against that organ-

See *Endocarditis in HIV*, next page



Dentistry

Rates of IE are higher among HIV+ intravenous drug users

[Endocarditis, from previous page](#)

ism should be selected, 3) the proper dosage of the antibiotic should be used, 4) the antibiotic should be given just before the procedure to provide maximum blood levels at time of injury, and 5) the antibiotic should be continued as long as bacteria can be released (usually short duration).³ Ultimately the prudent dentist must make the decision after assessing all factors related to the patient, the procedure, and whether the risk of a potential problem outweighs those of using antibiotics. The American Heart Association guidelines for the prevention of bacterial endocarditis meet most of these principles for effective prophylaxis. It is designed to manage alpha-hemolytic streptococci, most commonly found in transient bacteremias, with amoxicillin being the most effective. The United States, Britain, Australia, and several other countries have their own guidelines that have many similarities but subtle differences between them in regards to dental prophylaxis. In the U.S., the guidelines put forth by the American Heart Association are followed as the standard guideline. Another recommendation from the AHA is the use of local irrigation with chlorhexidine prior to any invasive dental procedure that can result in bacteremia. Though it is yet to be determined whether this is sufficient to prevent IE in high or moderate risk patients, a possible disadvantage is the selection of resistant streptococci such as *Strep sanguis* from regular use of chlorhexidine. Endocarditis from resistant organisms would have a higher mortality rate than one caused by viridans streptococci.^{5,11}

Simply providing prophylaxis "when in doubt" or to "cover oneself" is not a prudent practice by the dental practitioner. As do all drugs, the antibiotics recommended for prophylaxis will cause harm for some patients, greater harm than any protection that prophylaxis may provide. The allergy rate to penicillin type antibiotics is about 3% for uticular type reactions. Anaphylaxis occurs in one of 2500-5000, with death from anaphylaxis occurring in 10%. Though a patient may deny having an allergy to penicillin, there is still a chance that the subsequent intake will cause a

reaction.⁴ In addition, the development of resistant organisms has increased through the years due to liberal use of antibiotics. The prevalence of penicillin resistant *Streptococcus viridans* in blood cultures has increased through the years. It is imperative that the dentist analyze the risks versus the benefits in many situations when the need for antibiotic prophylaxis is not a definite necessity. To put it into one perspective, it is estimated that 1.36 people per million population are likely to die from penicillin anaphylaxis to prevent IE and 0.26 deaths per million population are due to dental procedure-induced endocarditis. In other words, patients receiving penicillin (amoxicillin) prophylaxis to prevent IE are five times more likely to die from an anaphylactic reaction to the drug than to die from getting endocarditis.^{5, 10}

In many instances of dentoalveolar surgery, some have provided antibiotic prophylaxis to prevent infection at the surgical site. However, several studies and reviews from third molar surgery and implant placement conclude that there is little or no evidence of benefit from antibiotic prophylaxis in fit patients. Antibiotic prophylaxis during implant placement is still debated. Preoperative antibiotics appear to decrease the rate of implant failure, but studies have emphasized prevention of implant failure rather than prevention of infection.¹⁸ Consequently, antibiotic prophylaxis is indicated when the risk of infection is low but the consequences are quite serious. For example, the incidence of post-operative infection is 0.39% for orthopedic hip replacement. But if it does become infected, 4% need amputation at the level of the hip with the mortality rate being 5%. According to the 2003 Advisory Statement put forth by the ADA and AAOS, antibiotic prophylaxis is not routinely indicated for most dental patients with total joint replacements, pins, or plates and screws. However, those patients who are immunocompromised and/or have comorbidities such as previous prosthetic joint infections, HIV, type I diabetes, or malignancy may be at higher risk for infection and should be considered for the AHA suggested antibiotic prophylaxis regimen.¹⁷ In addition, the patient must be assessed individually since increasing

age, medical comorbidities, immunosuppression, increased length of the procedure and hospitalization will all increase the risk of wound infection.⁶

In one study of IE in an elderly population, *Streptococcus viridans* was the most common oral pathogen in the IE of oral etiology regardless of age group with majority of cases detected in women. Though the prevalence of IE of oral origin in patients older than 60 is low, the high frequency of heart disease, poor oral health, and number of dental procedures in the elderly population makes this group at risk for IE of oral origin.⁷ For the pediatric population, it is important to be fully aware of dosing when antibiotic prophylaxis is used. Children require treatment more frequently than do adults, and no matter what the calculation, the maximum pediatric dose should never exceed the adult dose (2 g amoxicillin, 600 mg clindamycin). Also, fixed acrylic appliances, which often harbor streptococci, should be avoided in children with cardiac defects.⁸ Patients with congenital heart disease that need antibiotic prophylaxis must establish a lifelong practice of doing so while maintaining excellent dental health. In a study of IE in patients growing up with congenital heart disease, it was found that 13.5% of the patients received dental work without antibiotic prophylaxis and this was focused as the possible reason for transient bacteremia.⁹

Conducting a thorough medical history cannot be overemphasized. Another study of 273 patients with endocarditis found that 38% knew of cardiac conditions and 6% of the control patients were aware. Also, patients with endocarditis had a history of mitral valve prolapse, congenital heart disease, valve surgery, rheumatic fever or heart murmur more frequently than did control patients.¹² Some even feel that patients with valvular heart disease and artificial valves have inadequate understanding of endocarditis and prophylaxis of it. One study reported that though 69% of adolescents with congenital heart disease could name their cardiac condition, only 4% could define endocarditis and 40% knew an antibiotic was necessary before dental procedures.¹⁶

There is no hard evidence either supporting antibiotic prophylaxis preventing infective endocarditis nor that it does



not protect those at increased risk. One study in Spain reported that between 34% and 57% of IE cases developed in patients who did not have previously known at risk cardiac conditions as defined by the American Heart Association.¹⁴ In a recent study conducted in France, researchers concluded that antibiotic prophylaxis reduces the risk of IE after a dental procedure, but that due to the limited risk of spontaneous IE after unprotected procedures in adults with known predisposing cardiac conditions, a huge number of doses of prophylaxis must be prescribed to prevent a very low number of IE cases. Considering this and the adverse reactions to antibiotics, prophylaxis was viewed as being best to target the procedures and populations

Studies have shown an inverse relationship between IE occurrence and CD4 lymphocyte count.

with the highest risk and that energy should focus on such populations to improve adherence to prophylaxis recommendations.¹⁵ The dentist must take into account the advantages and risks of antibiotic prophylaxis, all while keeping the individual patient situation in mind before making the final decision. ❖

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