



NURSING CARE *of the* HIV-Infected INMATE

HIV & HEPATITIS
COINFECTION
Module 4 of 5

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Albany
Medical
Center

Department of HIV Medicine and
The Albany Medical Center
Hospital Nursing Continuing
Education Provider Unit

MISSION:

This learning series is designed to equip correctional nurses with the basic knowledge needed to provide safe, comprehensive care to inmates infected with HIV. Each learning module will provide an overview of a pertinent topic, and will serve as a foundation for the succeeding module. At the completion of the 5-module series, each participating facility will have a valuable resource readily available for issues regarding nursing care for HIV-infected inmates. To obtain copies of Modules 1 – 3, please visit Albany Medical College's website at:

www.amc.edu/Patient/hiv/index.htm

(go to *correctional education*).

LEARNING OBJECTIVES:

After reading this monograph, the corrections nurse should be able to:

- 1) Discuss the impact of hepatitis B & C in the correctional population including the clinical effects on HIV coinfection.
- 2) Outline the diagnosis, manifestations, and treatment of hepatitis B & C.
- 3) Compare and contrast hepatitis B, hepatitis C and HIV.

DISCLOSURE STATEMENT:

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Albany Medical College

Albany, NY

Production Assistant

Jim Ybarra

Education and Outreach Assistant

Division of HIV Medicine

Albany Medical College

Albany, NY

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NURSING CARE *of the* HIV-Infected INMATE

LEARNING MODULE 4: HIV AND HEPATITIS COINFECTION

MODULE DIRECTIONS

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There is no fee for the nursing continuing education credit for this monograph.

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1. Time yourself throughout all portions of this activity.
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NURSING CARE *of the* HIV-Infected INMATE

LEARNING MODULE 4: HIV AND HEPATITIS COINFECTION

Effective treatment for HIV disease has resulted in significant reductions in AIDS-related morbidity and mortality. Now that people are living longer, one result is an increase in significant co-morbidities as other illnesses have the opportunity to develop. Due to shared modes of transmission, coinfection with HIV and hepatitis B and C are more common. However, we do not have a true picture of historical hepatitis seroprevalence rates as hepatitis B and C did not become reportable illnesses until January 2003. Nonetheless, we are clearly seeing more people presenting to health care with coinfection. This learning module will focus on current issues in hepatitis infection and treatment, particularly among HIV+ individuals.

HEPATITIS C

Epidemiology:

It is estimated that there are 5 million Americans infected with the hepatitis C virus (HCV), making it the most common blood-borne disease in the United States with 35,000 new infections reported annually. It is expected that this number will quadruple by 2015 due to increased testing and public awareness. These cases will reflect people infected before 1989. Cases of new infection have decreased dramatically since routine screening of blood for transfusions was implemented (CDC, 2003).

Higher seroprevalence rates are found among African Americans and people aged 40-59. Intravenous drug use (IVDU) is the most common mode of transmission of HCV, so it is no surprise that seroprevalence among inmates is quite high. 1.3-1.4 million inmates are infected, 9-10 times higher than in the non-incarcerated population. Although seroprevalence rates are not available yet from most regions, states that have begun to track HCV in their correctional facilities are finding rates ranging from 12% to 35%. New York State also reported high rates of coinfection, with 40-50% of those testing positive for HIV also infected with HCV (NYSDOH, 2002). Seroprevalence rates among incarcerated women are higher than men. In 1997, it was estimated that 30% of people with chronic hepatitis C infection had been

incarcerated at some point that year (NCCHC, 2002). Clearly, the correctional facility is a key setting for the identification and assessment of HCV patients.

Risk Factors and Transmission:

Transmission of HCV occurs through exposure to infected blood. Although IVDU accounts for the majority of transmissions (60%), other modes include the following:

- High risk sexual practices (15%)
 - Blood transfusions prior to 1992 (10%)
 - Perinatal transmission and percutaneous exposure through body piercing, tattooing and occupational exposures (5%)
 - Infections from an unknown cause (10%)
- (CDC, 2003)

In the New York study previously cited, it is notable that many inmates who tested HCV+ had not reported any risk factors. However, given the high prevalence of risk behaviors among incarcerated individuals, all inmates should be considered high risk and routine screening for hepatitis B and C is appropriate. Risk factors that warrant testing for HCV and HBV are listed in Table 1.

Natural History:

Hepatitis C is an RNA virus that replicates 10 trillion times a day. Similar to HIV in this regard, rapid replication results in frequent mutations of the virus. Unlike HIV, however, HCV is not systemic but is confined to the liver. Therefore, it is possible for HCV to be cleared from the body (unfortunately not true of HIV).

Approximately 15% of people exposed to HCV will clear the virus from the body on their own. These people will have a positive HCV antibody but no detectable virus or clinical sequelae. African American males and patients coinfecting with HIV are less likely to spontaneously clear the virus. The majority of people exposed (85%) will develop chronic infection, verifiable by detectable HCV virus in their blood and abnormalities of liver function tests. Of this group, 80% will remain stable over time. However, 20% will progress in their liver disease, developing cirrhosis. This group is at high risk for developing end stage liver disease, hepatocellular carcinoma and death. It generally takes 20-30 years from the time of infection to progress to end stage disease. However, coinfection with HIV is frequently associated with more rapid disease progression.

Diagnosis and Monitoring:

During the acute stage of HCV infection most patients are unfortunately asymptomatic, and therefore, may be unaware that they have been exposed to HCV. When acute HCV is symptomatic, malaise, jaundice, fatigue and weakness typically arise 4-12 weeks post-exposure and a positive HCV antibody may be detected at this time. As with HIV, the antibody to HCV serves only as a marker of disease exposure. It does not infer any immunoprotection. False negative antibodies occur more commonly in HIV+ and other immunosuppressed patients. False positive antibodies can also occur, primarily in patients with autoimmune disease.

TABLE 1 RISK FACTORS FOR HCV INFECTION

Intravenous Drug Use (IVDU)
Blood transfusions before 1992
HIV+
Clotting factors before 1987
History of sexually transmitted diseases
Occupational exposure due to percutaneous injury
Unexplained abnormal liver function tests
Long term hemodialysis

TABLE 2 TESTS USED IN THE DIAGNOSIS AND EVALUATION OF HCV

TEST	PURPOSE	COMMENTS
Enzyme Immunoassay (EIA)	Confirm presence of HCV antibody	Recommended as initial screening
Qualitative RNA assay (PCR)	Detect active virus/confirm chronic infection	Approved for diagnosis of HCV
Quantitative RNA assay (PCR)	Measure level of virus in the serum	Necessary for monitoring response to treatment
Genotype assay	Determine strain of virus	Predictive of response to treatment
Alanine aminotransferase (ALT)	Monitor hepatic inflammation	Varies during course of disease

Also similar to HIV, there is a window period following exposure before the positive HCV antibody develops. It is detectable in 90% of patients by 3-6 months. Alanine aminotransferase (ALT) levels become elevated in the first 4-12 weeks indicating hepatic injury. However, the ALT will fluctuate over the disease course and at times may be normal. Therefore, it is not considered an accurate indicator of liver pathology.

There are 6 genotypic variants of hepatitis C (1 – 6). Genotype 1 is most common in the United States but is also least amenable to current treatment. Consequently, identifying the HCV genotype prior to therapy is helpful as it is a major predictor of a patient's clinical response to treatment.

Liver biopsy is the gold standard for staging liver disease. It gives valuable information about the amount of inflammation and presence or absence of fibrosis and/or cirrhosis. It is very helpful in determining how quickly the disease is progressing and whether immediate treatment is needed. Fibrosis is staged from zero to four (zero= absence of fibrosis, four= cirrhosis). Patients with significant fibrosis and inflammation should be considered for treatment, whereas those with only mild fibrosis may elect to defer treatment.

The assessment of the patient with risk factors, elevated LFTs, and/or symptoms suggestive of HCV is as follows:

- Check for HCV antibody (full hepatic panel including hepatitis A and B serologies are recommended so that the patient can be immunized if needed)
- If HCV antibody positive (or false negative suspected), check qualitative HCV RNA PCR (viral load)
- If HCV RNA PCR positive, check HCV genotype and consider liver biopsy
- If treatment indicated, check quantitative HCV RNA PCR which will also be monitored during the course of treatment to determine response.

In the presence of advanced hepatic disease, other abnormal laboratory findings may include: hypoalbuminemia, anemia, thrombocytopenia and prolonged prothrombin time (PT).

Effects of HIV on HCV:

Patients who are coinfecting with HIV and hepatitis have been the subject of recent research to determine how the diseases and their treatments impact each other. How does HIV affect the course of HCV disease?

Coinfected patients are more likely to develop chronic HCV infection than those with HCV alone. Studies have also shown more rapid progression to cirrhosis. Higher HIV viral load appears to be a contributing factor, suggesting a direct role of HIV in hepatic damage (Nelson, 2003). Lower CD4 counts

(< 250 cells/mm) also correlate with more rapid disease progression: 5 years from acute infection to cirrhosis in the setting of uncontrolled HIV.

When a patient is coinfecting, the issue of which disease to treat first arises. In the setting of more advanced disease, (lower CD4 count, higher HIV viral load), it is important to start highly active antiretroviral treatment (HAART) as soon as possible to control HIV viremia, reconstitute the immune system and possibly slow the accelerated course of liver pathology. Since most HIV medications are metabolized in the liver, these patients need to be monitored very closely, especially during the early weeks on treatment. Baseline liver function tests are standard for all patients starting HAART but may need to be followed more closely in the coinfecting patient.

HAART-related toxicities can be worsened in the setting of coinfection and are more common in patients with lower CD4 counts and higher baseline ALTs. All classes of antiretrovirals (discussed in detail in Module #2) have been implicated in hepatic complications. However, within each class there is considerable variation among drugs in this regard.

- **Nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs):** in this class, the 'd' drugs (d4T/Zerit, ddI/Videx and ddC/Hivid) are most likely to cause liver problems by causing hepatic steatosis. This condition may occur in association with lactic acidosis, a rare but potentially fatal complication if not diagnosed promptly. Symptoms of lactic acidosis are usually non-specific and include fatigue, nausea, vomiting, diarrhea, abdominal distention, or dyspnea.
- **Non-nucleoside reverse transcriptase inhibitors (NNRTIs):** among the drugs in this class, nevirapine (Viramune) has the greatest potential for hepatotoxicity. It is most likely to occur in the first several weeks on treatment. LFTs should be monitored every 2 weeks for the first month, followed by every 4 weeks for 12 weeks and then every 1-3 months.
- **Protease inhibitors (PIs):** patients can develop hepatotoxicity at any time during the course of taking a PI-containing regimen. Nelfinavir (Viracept) and atazanavir (Reyataz) are the least likely to cause problems whereas ritonavir (Norvir) is most likely to result in hepatotoxicity. LFTs should be closely monitored. Patients on PIs who are coinfecting are at an increased risk for hyperglycemia. Therefore, blood glucose levels should be monitored every 3 months.

Many coinfecting patients tolerate HAART well; transient elevations of LFTs early in the course of treatment often normalize within the first few months. However, close

monitoring is a must. Any complaints of nausea, vomiting, anorexia, malaise, lethargy or abdominal pain require prompt evaluation. Be sure to make note of any other potentially hepatotoxic medications the patient is taking and eliminate those that are unnecessary.

Effects of HCV on HIV:

More studies are needed to assess exactly how HCV affects the natural disease process of HIV by monitoring these patients over time. One phenomena that has been observed is that coinfecting patients often have a blunted immune response to HAART, indicated by a diminished increase in CD4 cell count when compared with HIV+ patients without hepatitis (Greub, 2000). This would suggest that there is benefit to treating HCV before initiating HAART so that the patient will have a better tolerance and response to treatment.

Treatment Issues:

Careful consideration must be given to determine which illness to treat first. It is definitely **not** advisable to begin treatment for HIV and HCV at the same time due to tolerability issues. Both treatments cause adverse events and adherence is critical to the success of both HIV and HCV therapy. Although each case must be considered individually, common scenarios include:

- If the patient has either well controlled HIV disease on HAART, or early disease as evidenced by low HIV viral load and a CD4 count > 350 cells/cmm without HAART, HCV should be addressed and treated if indicated. Patients tolerate HCV treatment better if their HIV disease is well controlled or asymptomatic. HCV treatment is time-limited and, once completed, may afford the patient the best possible response to HAART (greater immune response and less drug-induced hepatotoxicity). Notably, patients receiving HCV therapy may have an initial drop in their absolute CD4 counts but the CD4 percentages remains stable. PCP prophylaxis should be initiated if the CD4 count decreases to < 200 cells/cmm. If the patient is already on HAART, he/she may need some revision of the HAART regimen. Specifically, ddI use during HCV treatment has been associated with hepatic decompensation, lactic acidosis and pancreatitis. This drug must be replaced with another medication in its class prior to initiating anti-HCV treatment. Additionally, HCV treatment results in anemia as does Retrovir (AZT). The clinician may opt to replace AZT with another NRTI for the duration of HCV treatment.
- If the patient has more advanced HIV disease indicated by opportunistic infection, symptomatic disease, low CD4 count or high viral load, treatment with HAART should take precedence to control and prevent HIV complications, reconstitute the immune system and possibly slow the progression of hepatic damage. Although HAART has no direct effect on HCV replication, by controlling HIV viral replication it is hoped that the accelerated HCV disease course can be slowed. When patients are started on HAART they may experience immune reconstitution syndrome. As the immune system begins to recuperate with successful viral suppression, there is an increased inflammatory response. Hepatitis can flare and result in a transient increase in ALTs. This underscores the necessity of closely

monitoring liver status in coinfecting patients starting HAART.

Who should be considered a candidate for HCV treatment? "Treatment is recommended for patients who are at increased risk for progression to cirrhosis: measurable HCV RNA, a liver biopsy with portal or bridging fibrosis, and at least moderate inflammation and necrosis." (NIH, 2002). HCV treatment is costly and has considerable toxicities. Patients with early/mild disease may do better to defer treatment if they are stable. Chronic illnesses such as diabetes, thyroid, pulmonary, cardiac, renal and psychiatric disease are not contraindications to treatment, but these conditions should be stabilized prior to treatment initiation.

Contraindications for treatment are:

- Known sensitivity to Interferon and/or ribavirin
- Chronic active hepatitis B
- Pregnancy
- Decompensated liver disease (Childs-Pugh class B and C)
- Hemoglobinopathies (sickle cell disease, Thalassemia major)
- Unstable or significant cardiac or cerebrovascular disease
- Severe depression/history of suicide attempts (Brown, 2003)

The **primary treatment goal** for HCV is viral eradication. Patients who experience an undetectable or a 2-log (100 fold) decrease in HCV viral load at 12 weeks of therapy are considered to have an early viral response (EVR). The EVR is considered highly predictive of achieving a sustained virologic response (SVR) defined as undetectable HCV viral load 24 weeks after treatment is completed. Coinfecting patients are less likely to achieve EVR or SVR.

Secondary goals of treatment are to slow disease progression, improve liver histology, decrease hepatocellular carcinoma and improve quality of life.

What are the current treatment modalities for HCV?

Initially, treatment consisted of interferon-alfa 2b (IFN) 3 million units subcutaneously (SC) 3 times a week for 24-48 weeks. Approximately 50% of those treated, (non HIV-coinfecting), showed improved liver histology and normalization of their ALTs. However, for more than half of that group the response was not durable. Relapse of chronic infection occurred within 6 months of completing treatment. More aggressive dosing regimens resulted in improved response rates, but significant drug toxicities. When IFN was combined with the nucleoside analogue ribavirin (Rebetol/Copegus), response rates improved. Recently IFN has received FDA approval in a sustained release form (pegylated or PEG/IFN) given by weekly subcutaneous (SC) injection. Comparative studies demonstrate superior efficacy of PEG/IFN/ribavirin over either standard IFN/RBV or IFN alone. 76% of genotype 2 and 3 patients achieved an SVR, whereas only 46% of genotype 1 patients did so. Among coinfecting patients, response rates are lower (47.6% for genotypes 2 and 3, 19.5% for genotypes 1 and 4 in one study). Results may vary depending upon patient characteristics (Voight, 2003). Additional studies in coinfecting patients are underway with results expected later this year.

Dosing of PEG-IFN is dependent upon which of the recently approved formulations are used. PEG-Intron (pegylated interferon alfa 2b) has weight-based dosing

at 1.5 µg/kg weekly by SC injection. PEGASYS (pegylated interferon alfa 2a) has standardized dosing at 180 µg weekly by SC injection. Either formulation is given along with ribavirin, twice daily by mouth with food. Ribavirin is available as Rebetol or Copegus. When using PEG-Intron, ribavirin is dosed 400 mg PO BID regardless of genotype. Ribavirin is dosed differently when used with Pegasys and this is based on the patient's genotype:

- Genotypes 1 and 4** Patients receive ribavirin 400 mg/600 mg BID if < 75 kg or 600 mg BID if > 75 kg
- Genotypes 2 and 3** Patients receive ribavirin dosed 400 mg BID

Duration of treatment for patients with HCV mono-infection with genotype 2 or 3 HCV infection is 24 weeks, whereas it is 48 weeks for the more difficult to treat genotype 1 or 4. However, all coinfecting patients should be treated for 48 weeks regardless of genotype. Patients who do not experience an EVR are highly unlikely to achieve the goal of viral eradication and consequently, treatment should be stopped at 12 weeks. This relationship has been best defined for mono-infected patients. Later time points, such as 24 weeks, may be

more appropriate for the assessment of EVR in the coinfecting patient. These patients should be assured that future treatment options are in development.

Response rates to HCV treatment are dose-dependent. If the patient is able to tolerate full dose treatment, he/she has a significantly better chance of achieving an EVR/SVR (Manns, 2003). Maintaining full dose therapy includes adequate adherence to at least 80% of doses. Therefore, successful therapy is highly contingent upon patient education and aggressive management of side effects.

Patient education should include information on:

- Disease transmission and course
- Importance of ETOH abstinence (> 50 gm/day is linked to increased mortality)
- Avoidance of recreational drugs
- Importance of vaccinations against hepatitis A virus and hepatitis B virus if the patient is a candidate
- Need for double barrier contraception during treatment and 6 months after completion of treatment for both male and female patients due to the teratogenicity of ribavirin. This is especially critical if the inmate will be paroled within 6 months of completion of treatment (see Table 3)

TABLE 3 SIDE EFFECTS OF HCV TREATMENT	
INTERFERON (IFN) SIDE EFFECTS	RECOMMENDATIONS
<p><u>Hematologic:</u></p> <ul style="list-style-type: none"> - Neutropenia (ANC > 500 cells/cmm) - Anemia (Hgb < 12 gm/dl) - Thrombocytopenia 	<ul style="list-style-type: none"> - Check CBC at 2 and 4 weeks, then monthly - Treat with neupogen 300 mcg SC 3 times a week - Treat with erythropoietin 40,000 units SC q week - Dose reduction of IFN or ribavirin may be needed if above treatments are not successful; discontinue treatment if hemoglobin < 8.5 and/or platelets < 50,000
<p><u>Neuropsychiatric:</u></p> <ul style="list-style-type: none"> - Depression; anxiety - Irritability; nervousness - Insomnia - Impaired concentration - Suicidal ideation 	<ul style="list-style-type: none"> - Treat with antidepressant; consider monthly preemptive therapy if patient has history of depression - Monitor closely; refer to psychiatry prn
<p><u>Constitutional:</u></p> <ul style="list-style-type: none"> - Flu-like symptoms - Low grade fever - Myalgias/arthralgias - Malaise - Alopecia - Rash - Dyspnea/cough <p><i>Assure patient that symptoms often occur in first few weeks of treatment but then diminish in severity and frequency.</i></p>	<ul style="list-style-type: none"> - Pre-medicate with Benadryl, Tylenol, or NSAIDs - Dose at bedtime
RIBAVIRIN SIDE EFFECTS	RECOMMENDATIONS
<p><u>Hematologic:</u></p> <ul style="list-style-type: none"> - Anemia - Leukopenia 	<ul style="list-style-type: none"> - Same monitoring and intervention as with IFN
<p><u>Teratogenicity</u></p>	<ul style="list-style-type: none"> - Monthly pregnancy tests; teaching about need for birth control during and 6 months post treatment

- Referral for alcohol and substance abuse treatment should be made when indicated to minimize disease transmission and progression

In the correctional setting, concern exists about initiating HCV treatment due to uncertainty over whether the inmate will remain incarcerated for the 12 month treatment period. One study showed that 64.3% of inmates diagnosed with HCV at their intake facility had sentences over 2 years duration (Maryland DOH, 2003). This strongly suggests that if routine screening for HCV is done at intake, there is ample time to identify and treat inmates for HCV.

Future Treatment Options:

Interferon remains the most potent agent available for HCV treatment. Its antifibrotic effect yields great benefit in eradicating virus as well as decreasing liver damage, even in those who are not able to clear the virus completely (Imazeki, 2003). However, it must be used in combination therapy and research is looking at using it together with different types of medications that will be more tolerable. Alternatives to ribavirin that are safer, as well as new classes of HCV drugs such as helicase and protease inhibitors, appear promising.

HEPATITIS B

Epidemiology:

Worldwide, 400 million people are infected with hepatitis B virus (HBV) which is a major cause of liver-related morbidity and mortality. The disease is endemic to Southeast Asia, China and Africa and immigrants from these areas have a 1 in 7 chance of being infected (CDC, 2003). In the United States, it is estimated that 1.25 million people have chronic HBV. The disease is 4 times more prevalent among African Americans than Caucasians. Despite an overall decline in HBV rates in the United States due to immunization efforts, 78,000 new infections were reported in 2001 (CDC, 2003). Incarcerated individuals have 2-6 times the rate of HBV infection compared with the non-incarcerated population. Two percent of inmates and recent releasees have current or chronic HBV. These

individuals comprise 12% of those infected in the United States (NCCCHC, 2002).

Risk Factors and Transmission:

HBV is blood-borne and is transmitted by exposure to blood or body fluids. Highest concentrations are found in serum, blood and wound exudates. Moderate amounts of virus are found in saliva, semen and vaginal secretions. The virus is also present in small amounts of urine, feces, sweat, tears and breast milk (Valenti, 2003). It is 100 times more infectious than HIV and 10 times more infectious than hepatitis C. In the developing world, the majority of HBV transmission is perinatal which results in a high rate of chronic infection. In the United States, more than 50% of new infectious are acquired sexually through either hetero or homosexual contact. Parenteral exposure via IVDU, piercing, tattooing and other percutaneous injuries account for 15% of reported cases. A large percentage of cases have no identified cause (CDC, 2003).

Natural History:

HBV is a DNA virus whose genetic material remains in the cells. For this reason it cannot be eradicated from the chronic carrier, but can become dormant in the setting of immune compromise. Chronic HBV infection progresses to cirrhosis or hepatocellular carcinoma in approximately 25% of chronic infections; as with HCV, the damage occurs over many years. In coinfecting patients, the disease course is more rapid and severe. If a patient has HIV, he/she has 3-6 times the risk of becoming a chronic carrier of hepatitis B after acute HBV infection (Piliero, 2002).

Diagnosis and Monitoring:

Acute HBV infection may be asymptomatic. When symptoms occur they include nausea, vomiting, anorexia, malaise, fatigue, myalgias and arthralgias. Clinical signs include jaundice and dark urine. The incubation period is 45-180 days. Serologic testing during acute infection will show increased liver enzymes and a positive HBeAg which is

TABLE 4 SEROLOGIC TESTING FOR HBV

HBsAg	Hepatitis B surface antigen. This marker is acutely seen by week 2-4 of HBV infection and it only persists in the setting of chronic HBV infection.
Total anti-HBc	Total antibody to hepatitis B core antigen. This marker for exposure to HBV is positive by 6-12 weeks.
IgM anti-HBc	Immunoglobulin M antibody to hepatitis B core antigen. This marker is positive during acute HBV.
IgG anti-HBc	Immunoglobulin G antibody to hepatitis B core antigen. This marker usually indicates resolved infection.
Anti-HBs	Antibody to hepatitis B surface antigen. This marker usually indicates resolved infection or protection from vaccination if seen without anti-HBcIgG.
HBeAg	Hepatitis B e antigen. This is a marker of active replication and when persistent in chronic HBV infection, indicates high infectivity.
Anti-HBe	Antibody to hepatitis B e antigen; develops as eAg wanes and indicates resolving infection.

The above tests need to be interpreted in total, not in isolation. Interpretation can be tricky, especially in the coinfecting patient. Infectious disease consultation may be advisable.

a marker of viral replication. Treatment is rest, hydration and supportive measures. Table 4 reviews the serologic testing for HBV.

Interaction of HBV and HIV:

Chronic HBV infection is estimated to exist in 10-15% of HIV+ patients, but greater than 90% of all HIV+ patients have evidence of prior exposure to HBV. HBsAg and anti-HBs, used to screen for chronic or past infection, may not always be present in the coinfecting patient. A high percentage of patients with HIV/HCV and HBV will have an isolated IgG anti-HBc with a negative HBsAg (Gandhi, 2003). This usually suggests past resolved infection, but if there is history of risk exposure and unexplained elevated LFTs, an HBV RNA PCR (viral load) should be checked.

Although HIV accelerates liver pathology in HBV coinfection, there is no evidence at present that HBV influences the progression of HIV disease. There is also no evidence to show an impaired response to HAART in coinfecting patients as is seen in HCV.

Since liver damage in chronic HBV is immune-mediated, patients may experience a flare of the disease when they begin HAART therapy and undergo immune reconstitution. Fortunately, some of the drugs used to treat HIV also have activity against HBV. All coinfecting patients should be receiving at least one of these medications. It should be clearly documented in the patient's medical record that he/she is receiving these medications for both HIV and HBV. If there is a discontinuation or change in therapy, the abrupt removal of these drugs can cause a severe flare of hepatitis that can result in fulminant liver failure. Even if the HAART regimen is switched due to HIV resistance, the drug(s) being used to concurrently treat HBV must remain on board.

Much remains to be learned about how HIV and HAART affect HBV. Hepatotoxicity can develop due to immune reconstitution, direct HAART toxicities or resistance to/withdrawal from anti-HBV therapy. How HIV alters serologic markers of HBV also requires more study.

Treatment:

The goals of treatment are to suppress viral replication and thereby stop progression of liver injury (Malik, 2000). The patient should have an HBV viral load assay prior to beginning treatment for HBV. Some clinicians will also do a liver biopsy. These tests will help in monitoring response to treatment. Currently available medications to treat HBV are:

- **Interferon alfa-2b (Intron):** given at 10 million units SC 3 times a week for 4 months, this treatment is generally not used in the coinfecting patient due to poor sustained response rates and intolerance. Currently, Peg-IFN is being studied and appears to be twice as effective in patients with HBV alone. Peg-IFN is also being studied in combination with epivir and adefovir.
- **Adefovir (Hepsera):** given 10 mg PO QD this medication appears to be well tolerated and effective in even advanced disease. Dose reduction is required in renally-impaired patients. Generally this medication is not used in coinfecting patients as it does not have activity against HIV.
- **Lamivudine (Epivir-HBV):** given 100 mg PO QD in the patient with HBV mono-infection. If the patient is coinfecting, standard lamivudine dosing is 150 mg PO BID or 300 mg PO QD in order to adequately treat both infections. This medication is well tolerated but

development of HBV and HIV viral resistance is common over time.

- **Tenofovir (Viread):** this drug is not FDA approved for the treatment of HBV but is approved for HIV treatment. It is very active against 3TC-resistant HBV as well as wild-type virus (Cooper, 2003). Tenofovir is given in one 300 mg dose daily with food and is well-tolerated.
- **Emtricitabine (FTC):** this drug is not FDA approved for treatment of HBV but was recently approved for HIV treatment. It is a similar compound to epivir and is potent in vitro against HBV. Studies are evaluating its role in HBV treatment.

In treating HBV, a quantitative PCR should be checked at baseline and at 12 weeks on therapy. A minimum 2 log reduction in viral load is indicative of treatment response. Some experts recommend that patients with chronic HBV have an annual alpha fetoprotein and right upper quadrant ultrasound to monitor for hepatocellular carcinoma.

Vaccination:

All HIV+ patients and HCV patients should be assessed for immunity to hepatitis A and B and vaccinated if non-immune. All HIV/HBV coinfecting patients should receive an hepatitis A vaccine if non-immune. Hepatitis A, ordinarily a self-limited illness with no sequelae, can lead to fulminant liver failure in patients with HCV. Hepatitis B, as discussed, is a preventable illness that has the capacity to lead to chronic liver disease.

Hepatitis A vaccine is given in 2 doses: 0.5 mg SC at baseline and again at 6 months. Hepatitis B vaccine is given in 3 doses: 0.5 mg SC at baseline, 1 month and 6 months. A booster dose or repetition of the series can be given if there is no subsequent HBsAb detected. In patients with AIDS (CD4 < 200 cells/cmm) there is a lower likelihood of achieving a successful immune response to vaccines. If the patient is going to be started on HAART, some experts delay vaccinations for a period of 6-9 months pending immune reconstitution.

Conclusion:

Hepatitis coinfection has created new challenges in the era of HAART. As patients live longer with HIV, identifying and treating liver disease is critical to safely treat HIV, limit hepatic damage and stem the tide of rising liver-related deaths in HIV patients. The correctional facility is an ideal location to identify those already infected, and prevent infection among those at highest risk for these diseases. By intervening while people are incarcerated, the public health should be protected as the vast majority of inmates will return to their communities.

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ADDITIONAL INFORMATION AND RESOURCES

HELPFUL WEBSITES:

www.hcvadvocate.org www.hepb.org
www.hepnet.com www.hivandhepatitis.com
www.hcvinprison.org www.liverfoundation.org

SAVE THE DATE!

***Management of HIV/AIDS in the Correctional Setting:
A Live Satellite Videoconference Series
Antiretroviral Update 2004***

Tuesday, March 16, 2004
12:30-3:30 PM Eastern Time

Visit Albany Medical Center's website in January of 2004 register as a downlink site: www.amc.edu/Patient/hiv/hivconf/index.htm. For additional information, please contact Jim Ybarra at (518) 262-4674 or ybarraj@mail.amc.edu.

Continuing Nursing Education credits offered.

COMMUNICABLE DISEASES: ARE YOU AT RISK?

This 30-minute videotape encourages inmates to get tested for HIV, hepatitis B & C, and sexually transmitted diseases by discussing risk factors. This resource will be available in late Spring of 2004 and was developed in collaboration with Albany Medical College, the New York State Department of Correctional Services and the pharmaceutical industry. To be notified when this videotape is available, call (518) 262-4674 or e-mail ybarraj@mail.amc.edu. The cost is \$15.00.

COMING WINTER 2004:

Module 5: Health Maintenance of the HIV-infected Inmate

Please share this monograph with your nursing colleagues making photocopies of the Continuing Nursing Education documents if needed. Additional copies of this monograph can also be downloaded from Albany Medical College's website at: www.amc.edu/Patient/hiv/index.htm (go to correctional education).

PLEASE GO TO PAGE 9

SELF ASSESSMENT TEST

MODULE 4: HIV AND HEPATITIS COINFECTION

DIRECTIONS: Please select the BEST answer and circle your response directly on the self assessment test. To obtain Continuing Nursing Education credit, a minimum of 80% of the questions must be answered correctly. To assure your receipt of Continuing Nursing Education credit, please complete the self assessment test, program evaluation and reader information form.

This activity is eligible for nursing credit through **April 30, 2004**. Individuals who mail the required documentation noted above after this date will be ineligible for credit. The estimated time for completion of this activity is 1-hour.

There is no fee for the nursing continuing education credit for this monograph.

Albany Medical College's mailing information is on the reverse side of this document.

- 1) **The following inmates should be screened for hepatitis B and C:**
 - A. Only inmates reporting recent risk activities such as IVDU or high risk sexual activity
 - B. All inmates
 - C. Any inmate with unexplained, abnormally elevated liver function tests
 - D. Inmates recently diagnosed with gonorrhea
- 2) **Which of the following statements is true?**
 - A. Both HIV and HCV can be eliminated from the body with treatment
 - B. HIV and HCV have a slow rate of viral replication
 - C. Modes of transmission for HIV and HCV are similar
 - D. Antibodies for both HIV and HCV indicate immunoprotection against chronic illness
- 3) **Modes of transmission for hepatitis B and C include all except:**
 - A. Transfusions before 1992
 - B. Oral-fecal contamination
 - C. Perinatal (mother to child)
 - D. Tattoos/piercing
- 4) **All of the following statements are true regarding the patient coinfecting with HIV and HCV except:**
 - A. False negative HCV antibody tests may occur
 - B. Progression to cirrhosis is more rapid
 - C. Treating the patient's HCV will not offer any benefit when HAART is initiated
 - D. Close monitoring of liver functions and clinical status is crucial
- 5) **The best clinical outcomes of hepatitis C treatment are seen in patients who:**
 - A. Are coinfecting with HIV
 - B. Are infected with HIV with non-genotype 1 HCV
 - C. Have a .5 log reduction in HCV viral load at 12 weeks on treatment
 - D. Have decompensated liver disease
- 6) **Which of the following statements about hepatitis B is true?**
 - A. It is less infectious than HIV
 - B. It has a short disease course
 - C. It can be prevented by vaccine
 - D. It can be eradicated from the body
- 7) **Ramon F. is being treated for HIV with Combivir (AZT+Epivir) and Sustiva. He also has chronic hepatitis B. The correctional nurse is aware that:**
 - A. This treatment will likely be adequate to control Ramon's hepatitis B for a long time
 - B. If Ramon requires a change in HIV medications, he needs to continue his Epivir
 - C. The addition of interferon alfa will likely benefit Ramon
 - D. The goal of this treatment is to eradicate both viruses from Ramon's body
- 8) **Sheila M. is a 45 year old African American inmate coinfecting with HIV and HCV. Her baseline testing indicates HCV genotype 1a, CD4 count 125 cells/cmm, HIV viral load 250,000 by PCR. She has a history of PCP. The best plan for Sheila is:**
 - A. Initiate HCV treatment before beginning HAART
 - B. Treat neither HCV nor HIV but immunize against HAV and HBV
 - C. Initiate treatment for both HCV and HIV
 - D. Initiate HAART to get HIV under control, and plan to further assess HCV and liver status at a later time
- 9) **Paul J., who is in his 3rd week of HAART and is infected with HCV, presents at sick call with nausea and vomiting, fatigue and abdominal pain. Appropriate action would be:**
 - A. Stop his HAART
 - B. Check his LFTs and lactic acid
 - C. Check his HCV viral load
 - D. Treat his side effects and reschedule him for one month follow-up
- 10) **George M. is HIV/HCV coinfecting with a CD4 count of 125. Vaccination for hepatitis A and B should be considered:**
 - A. At intake or initial contact
 - B. Never
 - C. When his CD4 count increases to > 350 but no later than 9 months
 - D. When he has been on HAART for at least 12 months

OVER

PROGRAM EVALUATION & READER INFORMATION FORM

MODULE 4: HIV AND HEPATITIS COINFECTION

To assure your receipt of Continuing Nursing Education credit, please mail your completed self assessment test, program evaluation, and reader information form to: **Jim Ybarra, Albany Medical College, 47 New Scotland Avenue, Mail Code 158, Albany, NY 12208.** Please allow 6-8 weeks for education credit processing. An attendance certificate and self assessment test answer key will be mailed to you at that time. If you have any questions, please contact Jim Ybarra at (518) 262-4674 or ybarraj@mail.amc.edu.

PLEASE COMPLETE THIS FORM BY COMPLETELY FILLING IN THE CIRCLES WITH BLACK PEN OR PENCIL.

	STRONGLY DISAGREE	DISAGREE	AGREE	STRONGLY AGREE
1. As a result of completing the program, I am able to meet the following program goal: to provide an overview of the emerging problem of hepatitis B & C in HIV coinfection.	①	②	③	④
2. As a result of attending this program, I am able to achieve the following objectives:				
a. Discuss the impact of hepatitis B & C in the correctional population including the clinical effects on HIV coinfection.	①	②	③	④
b. Outline the diagnosis, manifestations, and treatment of hepatitis B & C.	①	②	③	④
c. Compare and contrast hepatitis B, hepatitis C and HIV.	①	②	③	④
3. The objectives of this program were relevant to the overall goals of the program.	①	②	③	④
4. The monograph was an effective learning tool for me.	①	②	③	④

Time required to complete this learning activity: _____ minutes

Comments: _____

READER INFORMATION FORM

(Please print legibly as all information is needed for education credit processing.)

Name (first and last): _____

Degree: _____ Title: (NP, RN, LPN) _____

Facility Name: _____

Facility Address: _____
STREET

CITY STATE ZIP CODE

E-mail Address (if applicable): _____