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<b>Management of Viral Hepatitis</b>	<b>ACA Standards: 4-4356M, 4-4359M, 4-ACRS-4C-09</b>		
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## Management of Viral Hepatitis/Cirrhosis

### Hepatitis Panel

#### I. Hepatitis A

Hepatitis A Virus (HAV) infection is usually acquired by the fecal-oral route. Viral transmission can occur through close personal contact (cell-mates, dorm -mates, co-workers, sexual contact) and contaminated food or water. HAV infection is a self-limited disease that does not produce chronic infection or long-term liver disease.

## A. Identification

1. Acute HAV infection is usually symptomatic. Symptoms and signs can be mild to fulminant and include malaise, anorexia, fever, dark urine, pale stools, jaundice, right upper quadrant pain, and tender hepatomegaly.
2. All patients with the above symptoms should have a hepatic function panel (CPL 9175) done initially. Elevated ALT, AST, bilirubin, and INR are consistent with hepatic involvement and form a baseline for monitoring.
3. The presence of anti-HAV IgM in the serum confirms the diagnosis of acute Hepatitis A infection. Anti-HAV IgM is detectable within 5 to 10 days of the onset of symptoms and persists for up to 6 months.
4. The presence of anti-HAV IgG is indicative of previous infection with HAV and confers immunity.

## B. Isolation

Inmates with acute Hepatitis A should be considered contagious until 10 days after the onset of jaundice. Isolation in a single cell with a separate sink and toilet are recommended until clinical improvement and resolution of diarrhea occurs. The offender should be educated about strict hand washing and other practical infection control measures, and universal precautions are to be observed.

## C. Treatment

There is no specific treatment for HAV infection. The disease is self-limited, and only 0.1% of patients have a fatal, fulminant course. Supportive measures include adequate nutrition and hydration, avoidance of hepatotoxins, rest and antiemetics as needed. Avoidance of alcohol and drugs should be encouraged to prevent further hepatic damage. Parenteral nutrition is only indicated in patients that cannot eat due to persistent vomiting.

## D. Immunization

Hepatitis A vaccine is administered intramuscularly in a two-dose series, 12 months apart. Hepatitis A vaccine should not be administered to inmates with hypersensitivity to alum or other components of the vaccine.

The following are indications for Hepatitis A vaccine:

1. Inmates with chronic liver disease or cirrhosis
2. Inmates with chronic HBV or HCV infections and underlying liver disease
3. Certain at-risk offenders in the context of a contact investigation.

#### E. Follow-up

Inmates diagnosed with acute HAV infection should be seen by a health care provider for follow-up. The frequency of follow-up should be determined based on the severity of symptoms. ALT (CPL 2219) AST (CPL 2218) Bilirubin (CPL 2207), and INR (CPL 1425) should be monitored until they are normalizing.

#### F. Surveillance

Acute, anti-HAV IgM+ Hepatitis A is a reportable disease in the state of Oklahoma, and is to be reported to the OSDH by telephone (405) 271-4060 or FAX (800) 898-6734 immediately upon diagnosis, utilizing ODH form 295 "Reportable Disease Card". In addition, offenders with newly diagnosed acute HAV infection should be reported to the nurse manager (Infection Control) Fax 405.962.6147 at Medical Services, and logged on the monthly medical service report.

Contact investigations should be coordinated with the Oklahoma State Department of Health. A form for Hepatitis A Contact Investigations is included as attachment A. All food service staff and offenders should be evaluated as part of the contact investigation, with the assistance of local and state public health authorities.

Post-exposure prophylaxis should be considered for the following contacts:

1. Cellmates
2. Sexual contacts
3. Persons routinely sharing toilet facilities
4. Other food handlers if the source-case was a food handler
5. Broad-based prophylaxis in consultation with OSDH if the source-case was a food handler.
6. Persons with known immunity to HAV (positive anti-HAV IgG) do not require prophylaxis.

Post-exposure prophylaxis should be administered within 2 weeks of the exposure. It consists of pooled serum immunoglobulin (IG) 0.02 ml/kg administered intramuscularly in a single dose and/or the Hepatitis A vaccine. Healthy persons 12 months to 40 years can receive the Hepatitis A vaccine (preferred) or immunoglobulin (anti-HAV IgG). Persons over 40 years, those whom are immunocompromised, or with chronic liver disease should receive the immunoglobulin only.

II. Hepatitis B

Hepatitis B virus (HBV) is a bloodborne pathogen. It is predominantly transmitted sexually, but is also frequently transmitted by injection drug use or other percutaneous or mucosal exposures to blood or other infectious body fluids. Perinatal transmission from mother to child also occurs. Acute hepatitis B is usually self-limited. About 2-6% of adults with HBV infection progress to chronic infection. The majority of persons with chronic HBV infection are asymptomatic, and one third have no evidence of liver disease. The remainders have chronic hepatitis ranging from mild to severe that can lead to cirrhosis and hepatocellular carcinoma (HCC). Those with chronic HBV infection have a 15-20% lifetime risk of death from cirrhosis or HCC.

A. Identification

1. 30-50% of adults with acute HBV infection will have signs and symptoms: fever, jaundice, nausea, right upper quadrant pain, malaise, dark urine, pale stools, and tender hepatomegaly.
2. All patients with the above symptoms should have a hepatic function panel (CPL 9175) done initially. Elevated ALT, AST, bilirubin, and INR are consistent with hepatic involvement and form a baseline for monitoring.
3. Laboratory diagnosis of acute and chronic HBV infection involves 3 antigens:
  - a. HBsAg – Hepatitis B Surface Antigen
  - b. HBcAg – Hepatitis B Core Antigen
  - c. HBeAg – Hepatitis B e Antigen
4. In addition, these 3 antigens can result in the formation of 3 antibodies:
  - a. Anti-HBs – antibody against HBsAg
  - b. Anti-HBc – antibody against HBcAg
  - c. Anti-HBe – antibody against HBeAg
5. Finally, HBV DNA is a marker of active viral replication.

The following table summarizes the interpretation of HBV serologic markers:

HBsAg	Total anti-HBc	IgM anti-HBc	Anti-HBs	Interpretation
-	-	-	-	Susceptible, never infected
+	-	-	-	Acute infection, early incubation period
+	+	+	-	Acute infection
-	+	+	-	Acute resolving infection
-	+	-	+	Past infection, recovered and immune
+	+	-	-	Chronic infection
-	+	-	-	False positive, past infection, or low-level chronic infection
-	-	-	+	Immune from vaccination

6. Indications for HBV testing
  - a. Pregnant offenders
  - b. Inmates with a history of injection drug use or tattoos or body piercing while in prison
  - c. Inmates with HIV or HCV infection
  - d. Inmates with persistent ALT elevations of undetermined etiology
  - e. Inmates on chronic hemodialysis
  - f. Inmates with signs and symptoms of hepatitis

B. Isolation

Isolation is not required for inmates with HBV infection. Patients with acute or chronic HBV infection should be counseled on measures for preventing further transmission of HBV to others. Universal precautions should be followed.

C. Treatment

1. Acute HBV infection (HBsAg positive, IgM anti-HBc positive) – treatment of acute HBV infection is supportive. Measures include adequate nutrition and hydration, avoidance of hepatotoxins, rest and antiemetics as needed. Fulminant cases characterized by hemodynamic instability, dehydration, and delirium require hospitalization and intensive supportive care. Acute HBV infection is self-limited in 94-98% of cases.
2. Chronic HBV infection – chronic infection is diagnosed in one of two ways: 1) two positive HBsAg tests, at least 6 months apart; or 2) positive HBsAg with negative IgM anti-HBc and positive total anti-HBc.
  - a. Natural history – persons with chronic HBV infection may develop 1) chronic hepatitis, 2) asymptomatic chronic infection, or 3) spontaneous resolution of infection.
  - b. Baseline evaluation should include a targeted history and physical exam as well as the following lab studies:
    - (1) CMP (CPL 9179), INR (CPL 1425), CBC (CPL 1000),
    - (2) Evaluation of other potential causes of liver disease (iron studies, ceruloplasmin, antimitochondrial antibody CPL 2118/4213/ 4634)
    - (3) HBeAg (CPL 2735), anti-HBe (CPL 2733), HBV DNA (CPL 4286)
  - c. Normal ALT levels – In all patients with chronic HBV infection and normal ALT levels, treatment is not indicated unless the patient has evidence of cirrhosis including: Thrombocytopenia, APRI score > or = to 1.5 or FIB-4 score > or = to 3.25.

- d. Abnormal ALT levels – Patients with elevated ALT levels should have ALT repeated every three months. Over the period of one year, if 3 out of 4 ALT levels are > 2X the upper limit of normal, the patient should be worked up for HBV treatment.
- e. Liver biopsy is only indicated if other, comorbid etiologies of liver injury are suspected including: Non Alcoholic Fatty Liver Disease (NAFLD), Hemochromatosis, or Autoimmune Hepatitis.
- f. Consideration of antiviral therapy for chronic HBV should be individualized, taking into account that 25% of patients with chronic HBV will spontaneously clear the virus. The likelihood of response to treatment is increased in the following persons: 1) low HBV DNA levels, 2) high ALT levels, 3) short durations of infection, 4) acquisition of infection in adulthood.
- g. All patients with evidence of decompensated liver cirrhosis should be worked-up for HBV treatment. Clinical Decompensation includes: Hepatic Encephalopathy, jaundice, ruptured esophageal (or gastric) varices, and ascites. Further, Child Pugh class B and C are considered to be decompensated. The calculation for the Child Pugh score can be found at:  
<https://www.hepatitisc.uw.edu/page/clinical-calculators/ctp>
- h. In summary, the criteria for consideration of treatment of chronic HBV infection are below. Consultation with Hepatology, ODOC Hepatitis Clinical Coordinator, or Infectious Disease is recommended prior to treatment. Treatment of HBV is only curative in 5 % of cases, but is used to suppress the viral activity of the HBV Chronic infection. Long term treatment may be indicated for viral suppression; to prevent secondary complications which may include Cirrhosis, HCC, Glomerulonephritis, and Polyarteritis Nodosa.
  - (1) HBsAg positive for at least 6 months
  - (2) HBeAg positive or HBeAg negative/HBV DNA positive
  - (3) 3 out of 4 ALT levels > 2X upper limit of normal
  - (4) No documented drug or alcohol use in the past 6 months
  - (5) HCV co-infection, with specialty consultation.
  - (6) HBV induced glomerulonephritis, with specialty consultation.
- i. Four agents are approved for treatment of chronic HBV infection:
  - (1) Lamivudine
  - (2) Tenofovir
  - (3) Entecavir
  - (4) Combination regimens are not superior to monotherapy

The following table lists doses, contraindications, side effects, monitoring tests, and other considerations.

Medication	Pegylated IFN α2a (Pegasys)	Lamivudine	Tenofovir	Entecavir
Dose	HBeAg positive – 180 mcg every week x 12 months.	100 mg daily for at least one year. Treatment endpoint is loss of HBeAg and development of anti- HBe.	300 mg daily, indefinitely	<ul style="list-style-type: none"> <li>• 0.5 mg p.o. daily (until 6 months after eAg conversion)</li> <li>• 1 mg daily if Lamivudine resistant</li> </ul>
	HBeAg negative – 180 mcg/week x 24 months	100 mg daily for at least one year. Treatment endpoint is 6 months after disappearance of HBV DNA.	300 mg. daily, indefinitely	<ul style="list-style-type: none"> <li>• 0.5 mg daily (indefinite)</li> <li>• 1 mg daily if Lamivudine</li> </ul>
Contraindications	<ul style="list-style-type: none"> <li>•Age &gt;60 or &lt;18 years</li> <li>•WBC &lt;3000, plt &lt;100K</li> <li>•Solid organ transplant</li> <li>•Pregnancy</li> <li>•Decompensated cirrhosis</li> <li>•Current chemotherapy</li> <li>•Autoimmune disease</li> <li>•Thyroid disease</li> <li>•Uncontrolled chronic illness</li> </ul>	Renal failure	Renal failure  Dosing adjustments for patients with altered renal function	renal failure
Side Effects	Fever Fatigue Myalgia Depression Suicide Neutropenia Thrombocytopenia Thyroid	Lactic acidosis Pancreatitis Hepatic steatosis	Lactic acidosis Hepatic steatosis HIV resistance Worsening renal function Avoid NSAIDs	Lactic acidosis Hepatic steatosis HIV resistance Headache, fatigue, dizziness, nausea
Monitoring	CBC, ALT, BUN/CR, TSH Weekly visits X 1 mo. Monthly visits after 1 <sup>st</sup> mo. Mental health visits monthly	ALT, BUN/CR HBeAg, anti-HBe Weekly visits X 1mo. Monthly visits after	ALT, BUN/CR HBeAg, anti-HBe HBV DNA HBsAg, anti-HBs Weekly visits X 1mo. Monthly visits after	ALT, BUN/CR HBeAg, anti-HBe HBV DNA HBsAg, anti-HBs Weekly visits X 1mo. Monthly visits after
Other	Increases theophylline levels	***Discontinuation may cause an exacerbation of hepatitis*** Resistance may develop.	***Discontinuation may cause a severe exacerbation of hepatitis*** Resistance may develop.	***Discontinuation may cause a severe exacerbation of hepatitis***

- i. Discontinuation of antiviral treatment for chronic HBV infection should be done in consultation with a specialist. The following table indicates general guidelines for discontinuation.

Duration of Therapy

HBeAg(+)	HBeAg(-)
12 months	24 + months
6 months past conversion	Indefinite

Severe exacerbation of liver disease can occur on cessation of therapy. The effectiveness of treatment is determined by the following parameters 6 months after completion of therapy:

- (1) Absence of HbeAg
- (2) Absence of HBV DNA
- (3) Normalization of ALT

D. Follow-up

1. Acute HBV infection – patients with acute HBV infection should be monitored regularly. Frequency of visits will be determined on a case-by-case basis depending on severity of symptoms. ALT (CPL 2219) levels should be monitored until they are normalizing. HBsAg (CPL 2379) and anti-HBs (CPL 2737) should be monitored to determine if the infection resolves or becomes chronic.
2. Chronic HBV infection – monitoring in chronic HBV infection depends on ALT levels, HBeAg status, and treatment status. Monitoring during and after treatment are addressed above.
  - a. Normal ALT / HBeAg positive
    - (1) ALT every 3-6 months
    - (2) HBeAg annually to check for clearance of HbeAg
  - b. Normal ALT / HBeAg negative
    - (1) ALT every 6-12 months
    - (2) HBsAg annually to check for spontaneous resolution of chronic infection
  - c. Abnormal ALT
    - (1) ALT every 3 months – if 3 out of 4 are >2X upper limit of normal, liver biopsy indicated



## HBV 101 MEDICATIONS FOR TREATMENT OF CHRONIC HEPATITIS B

<b>DRUG</b>	<b>DOSE</b>	<b>FDA approved to treat CHB/HIV coinfection</b>	<b>Active Against HIV</b>	<b>COMMENTS</b>
Lamivudine (3TC)	300 mg daily in HIV-infected patients minimum treatment duration of 12 mo.	No	Yes	Resistance rate of 20-25%/yr among HBV isolates from HIV-infected patients. Do not include in HAART as the only HBV active agent.
Emtricitabine (FTC)	200 mg daily, optimal duration unknown	No	Yes	Similar in structure to lamivudine, so expected have same rates of resistance.
Adefovir	10 mg daily, optimal duration unknown	No	No	Concerns about HIV resistance emerging to tenofovir may limit use.
Tenofovir (TDF)	300 mg daily, optimal duration unknown	No	Yes	Recommended use as part of a HIV-replication-suppression regime.
Entecavir	0.5 mg daily In lamivudine-naïve patients. 1.0 mg in lamivudine-experienced; optimal duration unknown	Yes	No	No resistance during first 48wks in treatment naïve patients. resistance at 48 wks in HBV isolates from 7% of patients with lamivudine-resistant HBV.

Modified from: Thio CL, Sulkowski MS, Thomas D. Treatment of Chronic Hepatitis B in HIV-infected Persons: Thinking Outside of the Black Box, Clin Infect Dis. 2005;41:1035-40

\*Anti-HIV activity was noted in an HIV-Hepatitis C virus coinfection trial – Torriani FJ, Rodriguez – Torres M, Rockstroh JK, et al. Peginterferon alfa-2a plus ribavirin for chronic hepatitis C virus infection in HIV- infected patients. N. Engl J Med. 2004;351:438-50.

\*\*Marcellin P, Lau GKK, Bonino F, et al. Peg-interferon alfa-2a (40KDa) (Pegasys) monotherapy is more effective than lamivudine monotherapy in the treatment of HBeAg negative chronic hepatitis B: 72 week results from a phase III, partially double-blind study of Pegasys alone vs. Pegasys plus lamivudine vs. lamivudine [abstract 95]. Program and abstracts of the 39<sup>th</sup> Meeting of the European Assoc. of the Study of Liver Disease (Berlin). Geneva, Switzerland: Keyes International, 2004.

### E. Immunization

Hepatitis B vaccine is administered in a 3-dose series, at 0, 1, and 6 months. Spacing of the doses allows for flexibility as long as there is at least 1 month between doses #1 and #2; 2 months between doses #2 and #3; and 4 months between doses #1 and #3. The following are candidates for Hepatitis B vaccine:

1. Pregnant women (previously unvaccinated, HBsAg negative)
2. Inmates on chronic hemodialysis
3. As indicated in post-exposure prophylaxis
4. Certain inmates in the context of a contact investigation
5. Inmates with chronic HCV infection and underlying liver disease
6. Inmates with HIV
7. Inmates with cirrhosis or chronic liver disease
8. Inmates at high risk of HBV infection (injection drug use, unprotected sex with multiple partners, men who have sex with men)

F. Surveillance

Acute or chronic HBV infection (HBsAg+) is a reportable disease in the state of Oklahoma, and is to be reported to the OSDH by telephone (405) 271-4060 or FAX (800) 898-6734 within one business day from diagnosis, utilizing ODH form 295 "Reportable Disease Card". In addition, offenders with newly diagnosed acute HBV infection (IgM anti-HBc+) should be reported to the nurse manager (Infection Control) Fax 405.962.6147 at Medical Services, and logged on the monthly medical services report. Contact investigations for acute HBV infection (IgM anti-HBc+) should be coordinated with the Oklahoma State Department of Health. A form for Hepatitis B Contact Investigations is included as attachment B. Post exposure prophylaxis for HBV exposures should be managed in accordance with OP-140125 entitled, "Bloodborne Pathogen Exposure Control Program"

III. Hepatitis C

Procedures for the identification, treatment and follow-up of individuals with Hepatitis C Virus (HCV) infection will be in accordance with MSRM-140137-06 entitled, "Management of Hepatitis C"

IV. Cirrhosis – Enroll in chronic liver disease chronic clinic

Cirrhosis is a condition in which the liver does not function properly due to long term damage. Typically, the disease develops slowly over months or years.

- A. Compensated cirrhosis is cirrhosis of the liver without evidence of severe liver disease, such as ascites, encephalopathy, jaundice, marked thrombocytopenia, or bleeding esophageal varices; and with preserved hepatic synthetic function (albumin >3.5 g/dl, total bilirubin <1.5 mg/dl, INR <1.5).
- B. Decompensated cirrhosis is cirrhosis of the liver with evidence of significant liver disease, such as ascites, encephalopathy, jaundice, marked thrombocytopenia, or bleeding esophageal varices; and loss of hepatic synthetic function (albumin <3.5 g/dl, total bilirubin >1.5 mg/dl, INR >1.5)
- C. Modified Child-Pugh Score – The Modified Child-Pugh Score (attachment C) provides a tool for estimating the survival and surgical mortality for persons with cirrhosis. The score is based on the bilirubin, albumin and INR, as well as the presence or absence of ascites and encephalopathy. The following table indicates the scoring mechanism.

Parameter	1 point	2 points	3 points
<b>Bilirubin</b>	<2 mg/dl	2-3 mg/dl	>3 mg/dl
<b>Albumin</b>	>3.5 g/dl	2.8-3.5 g/dl	<2.8 g/dl
<b>INR</b>	<1.70	1.71-2.20	>2.20
<b>Ascites</b>	None	Medically controlled	Poorly controlled
<b>Encephalopathy</b>	None	Medically controlled	Poorly controlled

Class A – 5-6 points

Class B – 7-9 points

Class C – 10-15 points

The following table indicates the survival and surgical mortality by Child-Pugh class.

<b>Class</b>	<b>Survival</b>	<b>Surgical Mortality</b>
<b>A</b>	15-20 years	10%
<b>B</b>	5-15 years	30%
<b>C</b>	1-3 years	82%

- D. Goals of Therapy – Cirrhosis was once thought to be an irreversible, incurable process. However, research now suggests cirrhosis can be reversed when ALL offending agents are removed. Reversal of cirrhosis takes 5 years once all offending agents are removed. The focus of therapy is to delay progression and to manage the complications that arise. The primary complications of cirrhosis are esophageal variceal bleeding, coagulopathy, ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, jaundice, and hepatocellular carcinoma.
- E. Delaying Progression
1. Avoidance of alcohol and other hepatotoxins
  2. Avoidance of NSAIDS
  3. Acetaminophen in doses of less than 2gm/day are safe for pain control
  4. Avoid iron overload (Transferrin saturation <50%, Ferritin <200)
- F. Varices and Bleeding
1. American College of Gastroenterology recommendation – EDG every 2-3 years in patients with cirrhosis.
  2. If no varices or Grade I with normal platelets, prophylaxis is not indicated.
  3. Grade I with thrombocytopenia or Grade II, III, or IV (regardless of number of platelets) a non cardioselective Beta Blocker is indicated and should be titrated to keep HR > 55 and BP > 100.
    - a. Propranolol 10 mg TID titrated to reduce resting heart rate by 20-25%, OR Carvedilol 3.25 mg BID to reduce resting heart rate by 20-25%
    - b. Previous bleeding varices
    - c. Endoscopic band ligation AND
    - d. Propranolol or carvedilol as above

## G. Coagulopathy

1. Massive splenomegaly
  - a. Splenectomy
2. Decreased production of clotting factors
  - a. Vitamin K 10 mg IM daily X3d, then monthly
  - b. Maintain INR<2.2

## H. Ascites

1. Sodium restriction
2. Spironolactone 100-400 mg once daily
3. Add loop diuretic if needed: maintain ratio of Spironolactone 100 mg: Furosemide 40 mg with max doses of 400:160. Fluid restriction only if Na <120 mEq/L or symptomatic.
4. Indications for paracentesis
  - a. First episode of ascites (to R/O other causes)
  - b. Suspected spontaneous bacterial peritonitis
  - c. Tense ascites interfering with ventilation
  - d. Clinical deterioration not responsive to diuretics

## I. Spontaneous bacterial peritonitis

1. Ascites noted prior to clinical manifestations that includes the Triad: fever, abdominal pain, and altered mental status.
2. Hospitalization and IV antibiotics required
3. Bactrim or Norfloxacin as secondary prophylaxis may prevent recurrence

## J. Hepatic encephalopathy

1. Check for GI bleeding
2. Lactulose 15-30 ml TID-QID titrated to produce 2-3 loose stools per day
3. Neomycin 1 gm PO BID
4. Metronidazole 250 mg PO TID
5. Miralax 17 grams daily

## K. Hepatocellular Carcinoma

1. Advanced-stage – three year survival = 17%
2. <2cm tumor – five year survival = 85%

- L. Screening strategy
  1. AFP every 6 months (>20 is high risk)
  2. Hepatic ultrasound bi-annually

- M. Indications for CT of abdomen
  1. AFP >30, OR
  2. Mass identified on ultrasound

V. References

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OP -140125 entitled, “Bloodborne Pathogen Exposure Control

Program” OP –140137 entitled “Chronic Illness Management”

MSRM 140137-06 entitled, “Management of Hepatitis C”

VI. Action

The chief medical officer will be responsible for compliance with this procedure. The chief medical officer will be responsible for the annual review and revisions.

Any exceptions to this procedure will require prior written approval from the director.

This procedure will be effective as indicated.

Replaced: Medical Services Resource Manual 140125-01 entitled "Management of Viral Hepatitis" dated April 24, 2018.

Distribution: Medical Services Resource Manual

Attachments

<a href="#">Attachment A</a>	Contact Investigation-Acute Hepatitis A	Attached
<a href="#">Attachment B</a>	Contact Investigation-Acute Hepatitis B/C	Attached