Management of Viral Hepatitis/Cirrhosis/Fatty Liver

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 CBC (CPL 1000), CMP (CPL 9179), PT/INR (CPL 1425) and acute hepatitis panel (CPL 9325) drawn. 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 -

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. Liver enzymes, bilirubin and INR 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) 426-8710 or FAX (405) 900-7591 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 405-881-6965 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)) 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 remainder 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 CBC (CPL 1000), CMP (CPL 9179), PT/INR (CPL 1425) and acute hepatitis panel (CPL 9325) drawn 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 envelope 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), Hepatitis Panel (CPL 162), HBV PCR DNA, (CPL 4286) HIV antibody (CPL 3540), HBeAg (CPL 2735), anti-HBe (CPL 2733), Urine Drug Screen (CPL 3311)

      (2) Evaluation of other potential causes of liver disease (iron studies, ceruloplasmin, antimitochondrial antibody CPL 2118/ 4213/ 4634) may be indicated

   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 1.7 or FIB-4 score > or = to 4 or Fibrosure of F3 or F4.

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. The American Association for the Study of Liver Diseases (AASLD) recommends using an ALT >35 U/L for men and >25 U/L for women as the upper limit of normal (ULN) rather than local laboratory values.

e. Liver biopsy may be indicated if other, comorbid etiologies of liver injury are suspected including: 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 receive 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. Expert Consultation 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 (ALT >35 U/L for men and >25 U/L for women as the upper limit of normal (ULN)

(4) No documented drug or alcohol use in the past 6 months

(5) Negative HIV status

(6) HCV co-infection, with specialty consultation.

(7) HBV induced glomerulonephritis, with specialty consultation.

3. Tenofovir (300 mg and 25 mg) is approved for treatment of chronic HBV infection.

There is extensive experience demonstrating the virologic efficacy of Tenofovir for the treatment of chronic HBV. It is effective regardless of the patient's hepatitis B e antigen (HBeAg) status, the presence or absence of cirrhosis, or prior use of nucleos(t)ide analogues.

Tenofovir has also been used in patients with severe spontaneous exacerbations of chronic HBV. A positive response to treatment is characterized by suppression of HBV DNA and loss of HBeAg (in patients who were initially HBeAg positive). This is followed by loss of hepatitis B surface antigen (HBsAg) in a small percentage of patients during the course of follow-up. Additionally, no signature mutation for tenofovir resistance has been identified, even after eight years of treatment.

4. Patients with normal renal function and Creatine Clearance ≥ 50 mL/minute: Tenofovir disoproxil fumarate (Viread) 300 mg once daily administered without regards to meals.

   Treatment monitoring parameters to include:

   a. HIV status prior to initiation of therapy

   b. Serum creatinine, urine glucose and urine protein (prior to initiation and as clinically indicated during therapy)

   c. Serum phosphorus (in patients at risk for renal impairment)

   d. Bone density (in patients with a history of bone fracture or have risk factors for bone loss)

   e. CMP every 3 months during therapy X 4 followed by every 6 months along with Creatine Clearance calculations and dose adjustments as follows:

      (1) CrCl ≥50 mL/minute: No dosage adjustment necessary

      (2) CrCl 30 to 49 mL/minute: 300 mg every 48 hours

   f. CBC every 6 months

   g. HBV PCR DNA every 3-6 months

   h. Assess for seroconversion with HBsAg and anti-HBs every 6 months

   i. HBeAg and anti-HBe (in patients who are HBeAg-positive to monitor for seroconversion)

5. Tenofovir disoproxil fumarate (Viread) Warnings/Precautions: Decreased bone mineral density, Immune reconstitution syndrome, Lactic acidosis/hepatomegaly, renal toxicity.

6. Patients with abnormal renal function and Creatine Clearance ≤ 30 mL/minute: Tenofovir alafenamide (Vemlidy) 25 mg once daily administered without regards to meals.

   Treatment monitoring parameters to include:

   a. HIV status prior to initiation of therapy

b. Serum creatinine, urine glucose and urine protein (prior to initiation and as clinically indicated during therapy)

c. Serum phosphorus

d. Bone density (in patients with a history of bone fracture or have risk factors for bone loss)

e. CMP every 3 months during therapy X 4 followed by every 6 months along with Creatine Clearance calculations and dose adjustments as follows:

(1) CrCl ≥15 mL/minute: No dosage adjustment necessary

(2) CrCl < 15 mL/minute: Use is not recommended

f. CBC every 6 months

g. HBV PCR DNA every 3-6 months

h. Assess for seroconversion with HBsAg and anti-HBs every 6 months

i. HBeAg and anti-HBe (in patients who are HBeAg-positive to monitor for seroconversion)

7. Tenofovir alafenamide (Vemlidy) Warnings/Precautions: Lactic acidosis/hepatomegaly, renal toxicity

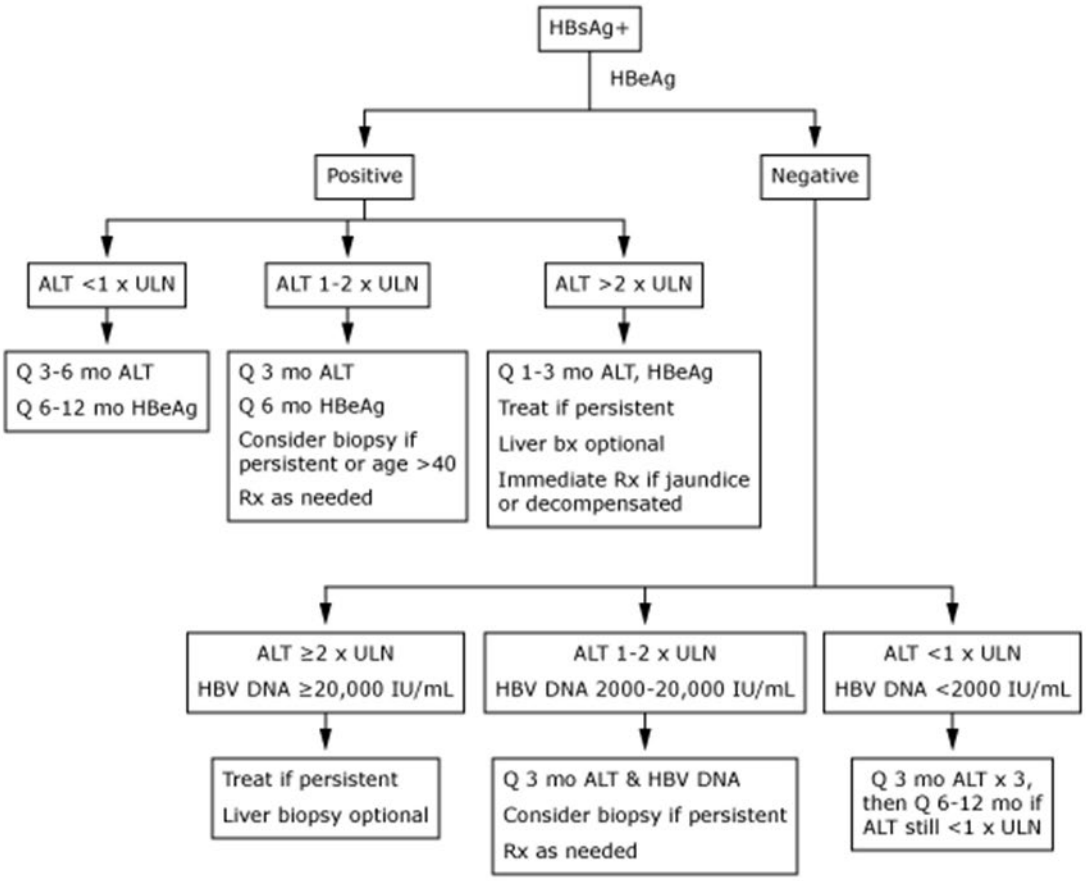
8. The optimal duration of therapy for the oral drugs is not well established. Most patients receiving nucleos(t)ide analog therapy will require at least four to five years of treatment, and some may require indefinite treatment. As severe exacerbation of liver disease can occur on cessation of therapy; Discontinuation of antiviral treatment for chronic HBV infection should be done in consultation with a specialist. For patients with cirrhosis, lifelong therapy with oral agents is typically administered to reduce the risk of clinical decompensation if a relapse occurs.

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. Liver enzymes, Bilirubin, and INR should be monitored until they are normalizing. HBsAg (CPL 2379) and anti-HBs (CPL 2737) should be monitored every 6 months – 1 year to determine if the infection resolves or becomes chronic.

2. Chronic HBV infection – monitoring in chronic HBV infection depends on ALT levels, HBeAg status, HBV PCR DNA levels and treatment status. Monitoring during and after treatment are addressed above. All patients with chronic HBV infection should be enrolled in Chronic Clinic (ICD-9 system favorite code: 070.32).

Management of chronic HBV infection*



Algorithm for follow-up of HBV carriers who are HBeAg-positive or HBeAg-negative.

ALT: alanine aminotransferase; ULN: upper limit of normal; Rx: treat; HCC: hepatocellular carcinoma.
* HCC surveillance if indicated.

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a. Normal ALT / HBeAg positive

(1) ALT every 3-6 months

(2) HBeAg and anti-HBe, HBsAg and anti-HBs annually to check for seroconversion

b. Normal ALT / HBeAg negative / HBV PCR DNA < 2000 IU/mL

(1) ALT every 6-12 months

(2) HBsAg and anti-HBs annually to check for seroconversion

c. Abnormal ALT / HBeAg positive

(1) ALT every 3 months – work up for treatment if ALT is persistently elevated, especially if HBV PCR DNA > 20,000 IU/mLd. Abnormal ALT / HBeAg Negative / HBV PCR DNA ≥ 20,000 IU/mL

(2) Work up for treatment

(3) Abnormal ALT / HBeAg Negative / HBV PCR DNA < 20,000 IU/mL

(4) ALT and HBV PCR DNA every 3 months. Work up for treatment if ALT is persistently elevated.

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) 426-8710 or FAX (405) 900-7591 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 405-881-6965 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 cirrhosis chronic clinic (ICD- 9 571.5)

Cirrhosis is a condition in which the liver does not function properly due to long term damage. Typically, the disease develops slowly over years. Thrombocytopenia (< 140 K/UL) is the most common hematologic abnormality seen in cirrhosis and is often the first clinical sign of cirrhosis.

A. Compensated cirrhosis is cirrhosis of the liver without evidence of stigmata of liver disease, such as ascites, encephalopathy, jaundice, 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 stigmata of liver disease, such as ascites, encephalopathy, jaundice, 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
Bilirubin	<2 mg/dl	2-3 mg/dl	>3 mg/dl
Albumin	>3.5 g/dl	2.8-3.5 g/dl	<2.8 g/dl
INR	<1.70	1.71-2.20	>2.20
Ascites	None	Medically controlled	Poorly controlled
Encephalopathy	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.

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

D. Goals of Therapy – Cirrhosis was once thought to be an irreversible, incurable process. However, research now suggests compensated cirrhosis can be reversed when ALL offending agents are removed and generally takes 5 years. 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 (illicit drugs including marijuana)

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. The rationale for esophageal varices screening is to identify patients at risk for and to prevent bleeding, improving survival in such patients. Screening for esophageal varices with EGD every 1-3 years is indicated in all with decompensated cirrhosis (annually) and those with compensated cirrhosis and platelets < 150,000 K/UL (every 2-3 years). For patients with cirrhosis who are already taking a non-cardio-selective beta blocker (NCSBB), a screening upper endoscopy is not indicated because the presence of varices will not modify management.

2. Esophageal varices are classified by size (small, medium, or large) and by the presence of red wale marks. High-risk Esophageal Varices (medium or large EV, or small EV with red wale marks) should receive prophylactic treatment with a NCSBB titrated to keep HR > 55 and BP > 100.

   a. Propranolol 10 mg TID titrated to reduce resting heart rate by 20-25%, OR Carvelidol 3.25 mg BID to reduce resting heart rate by 20-25%

3. Variceal bleeding is a decompensating event with a high risk of rebleeding following initial recovery. All patients who recover from esophageal variceal bleeding and do not have a transjugular intrahepatic portosystemic shunt (TIPS) are at risk for rebleeding and should receive prophylactic intervention.

   a. The preferred strategy is endoscopic variceal ligation (EVL) combined with a beta blocker for patients without contraindications to beta blockers.

   b. Propranolol 10 mg TID titrated to reduce resting heart rate by 20-25%

4. Contraindications to NCSBB include: Hyponatremia, Acute Kidney Injury, Spontaneous Bacterial Peritonitis, Diuretic-resistant ascites, adverse effects with BBs (bronchoconstriction, heart failure), Systolic Blood Pressure < 90 mmHg.

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 rule out 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 3-4loose stools per day
3. Xifaxin 550 mg BID
4. 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 NG/mL is high risk)

2. RUQ/splenic ultrasound every 6 months

M. Indications for triple phase CT or MRI of liver

1. AFP >20 NG/mL OR

2. Mass or lesion ≥ 1 cm identified on ultrasound

V. Steatotic (fatty) Liver

Steatotic (fatty) liver disease refers to liver steatosis of any etiology that is detected by liver imaging (Ultrasound) or biopsy. Fatty liver is generally a benign condition that is now the most common chronic liver disorder in the United States as obesity rates have increased.

A. Steatotic liver disease may be further classified as:

1. Metabolic dysfunction-associated steatotic liver disease (MASLD; previously termed nonalcoholic fatty liver disease [NAFLD]).

2. Alcohol-associated liver disease (ALD) – Patients with liver steatosis and heavy alcohol intake (ie, >50 g daily [350 g per week] for females and >60 g daily [420 g per week] for males) have predominantly alcohol-associated liver disease.

3. Toxin or Drug-induced liver disease – Illicit drug use including Marijuana and Medications (amiodarone, methotrexate, tamoxifen, glucocorticoids) are associated with liver steatosis.

4. Chronic hepatitis C virus (HCV) infection – Liver steatosis related to hepatitis C (predominantly genotype 3).

5. Wilson disease – Patients with symptomatic Wilson disease may have elevated liver enzymes in addition to signs of copper overload (eg., Kayser-Fleischer rings, neurocognitive involvement) and family history of Wilson disease.

6. Parenteral nutrition – Use of parenteral nutrition has been linked to elevated liver enzymes and liver steatosis.

B. There is no cure for Fatty Liver. Treatment aims to control the underlying conditions such as obesity, diabetes, hyperlipidemia. Strict avoidance of drugs and alcohol is recommended along with healthy weight loss in those with obesity. Insulin resistance is the key mechanism leading to hepatic steatosis; therefore, optimization of blood glucose control is a key component of treatment. Vaccinations for hepatitis A virus and hepatitis B virus should be given to patients without serologic evidence of immunity.

VI. References

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40)

OP -140125 entitled, “Bloodborne Pathogen Exposure Control

Program” OP –140137 entitled “Chronic Illness Management”

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

VII. 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 March 11, 20204.

Distribution: Medical Services Resource Manual

Attachments

Attachment A	Contact Investigation-Acute Hepatitis A	Attached
Attachment B	Contact Investigation-Acute Hepatitis B/C	Attached