

**MECHANISMS OF HUMAN DISEASE: LABORATORY SESSION  
HEPATOBIILIARY PATHOLOGY LAB**

**Wednesday, 1-28-09  
8:30 am – 10:00 pm**

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**GOAL:**

- 1. Describe the basic morphologic and pathophysiologic changes which occur in various conditions of the liver, gallbladder and biliary tree.**
- 2. Define (Describe) and correlate symptoms and signs of a disease with the structural changes in diseased organs alcoholic liver disease.**

**OBJECTIVE:**

- 1. Describe the morphologic changes which characterize alcoholic liver disease.**
- 2. Describe the morphologic changes which characterize cholecystitis.**
- 3. Describe the morphologic changes which characterize malignancies metastatic to the liver.**

## CASE 1

### **CHIEF CONCERN:**

Routine Physical.

### **HISTORY:**

A 24 year-old medical student with a history of “binge” drinking presents after a three-day history of excessive alcohol intake.

**PHYSICAL EXAMINATION:** The abdomen is soft and with no palpable masses or organomegaly. Mild right upper quadrant tenderness is noted.

### **LAB TESTS:**

AST 39 (ref range 8-20 U/L)

ALT 18 (ref range 8-20 U/L)

Alk Phos 30 (ref range 20-70U/L)

Bilirubin, Total 0.4 (ref range 0.1-1 mg/dL)

**AST minimally elevated; remaining hepatic enzymes within normal limits**

1. Identify the organ/describe the pathologic changes in the virtual microscopy slide.

- **Liver**
- **Mild steatosis**

2. What is your diagnosis?

**Hepatic steatosis/fatty liver (acute reversible manifestation of ethanol ingestion).**

3. Describe biochemical mechanisms responsible for this condition.

**Biochemical mechanisms responsible for fat accumulation in hepatocytes include: catabolism of fat by peripheral tissues increased and there is increased delivery of free fatty acids to the liver; metabolism of ethanol converts NAD to NADH which stimulates lipid biosynthesis; oxidation of fatty acids by mitochondria increased; acetaldehyde forms adducts with tubulin and impairs microtubule function, resulting in decreased transport of lipoproteins from the liver (Robbins page 411).**

## CASE 2

### **CHIEF COMPLAINT:**

“My eyes are yellow.”

### **HISTORY:**

53 year-old with chronic alcohol and drug abuse who has been in numerous rehabilitation facilities in the past now presents with abdominal pain after a 3-week alcoholic “binge.”

### **VITAL SIGNS:**

BP 130/70 HR 80 RR 18 T 100

### **PHYSICAL EXAMINATION:**

Patient has icteric sclerae and cutaneous “spider” angiomas. Abdominal exam significant for hepatomegaly, splenomegaly and RUQ tenderness to palpation.

### **LAB TESTS:**

AST 169 (ref range 8-20 U/L)

ALT 70 (ref range 8-20 U/L)

Alk Phos 36 (ref range 20-70U/L)

Bilirubin, Total 4.6 (ref range 0.1-1 mg/dL)

Bilirubin, Direct 3.3 (ref range 0.0-0.3mg.dL)

**AST/ALT ratio > 2; total bilirubin elevated with increased conjugated bilirubin.**

1. What is the clinical problem?

**Abdominal pain, jaundice**

2. What is your clinical differential diagnosis?

**Alcoholic or viral hepatitis, pancreatitis, cholecystitis, peritonitis**

3. Identify the organ/describe characteristic pathologic changes in the virtual microscopy slide.

**Liver; sections show focal hepatocellular necrosis with scattered inflammatory cells (including neutrophils), steatosis, and mallory bodies. Bands of fibrosis produce pseudolobules.**

4. What is your diagnosis?

**Acute alcoholic hepatitis superimposed on alcoholic cirrhosis.**

5. What are the potential complications of this disorder?

**Cirrhosis:**

- Ascites
- Esophageal varices
- Coagulopathy
- Encephalopathy

**-Hepatocellular carcinoma**

**Alcoholic Hepatitis:**

**-In severe cases liver failure or even death**

### CASE 3

#### **CHIEF COMPLAINT:**

“My stomach hurts after I eat.”

#### **HISTORY:**

An obese 40 year-old diabetic female presents with RUQ abdominal pain 30-45 minutes after eating fatty meals.

#### **PHYSICAL EXAMINATION:**

Alert and oriented female with round, protuberant abdomen who has RUQ tenderness to palpation. No masses or organomegaly are identified.

1. What is the clinical problem?

**Abdominal pain after meals**

2. What is the clinical differential diagnosis?

**Cholecystitis, cholelithiasis, gallstone pancreatitis**

3. Identify the organ/describe the characteristic pathologic changes in the virtual microscopy slide

**Gallbladder; sections show a thickened gall bladder wall with scattered chronic inflammatory cells. In some sections, Rokitansky-Aschoff (outpouching of the mucosal epithelium through the wall) sinuses are identified. Superimposition of acute inflammation on chronic inflammation would suggest an acute exacerbation in a patient with previously chronically injured gallbladder.**

4. What is your diagnosis?

**Chronic cholecystitis - most cases related to gallstones.**

**Cholesterol stones arise exclusively in gallbladder and may be pure cholesterol stones or associated with calcium carbonate; pigment stones are black or brown. Black stones found in sterile bile, brown in infected bile (see Robbins page 930).**

5. What are potential complications of this clinical problem?

**Bacterial superinfection with cholangitis or sepsis, gallbladder perforation and local abscess formation, rupture with peritonitis, biliary enteric fistula; empyema/gangrenous cholecystitis after acute cholecystitis**

## **CASE 4**

### **CHIEF COMPLAINT:**

None

### **HISTORY:**

72-year-old male with a past medical history significant for colon cancer presents with an enlarged liver.

### **PHYSICAL EXAMINATION:**

Abdomen is soft and non-tender with a well-healed scar. The liver is enlarged. No masses are palpated and no lymphadenopathy is noted.

### **LAB TESTS:**

CEA 1250 ng/ml (<5ng/ml)

AST 47 (ref range 8-20 U/L)

ALT 52 (ref range 8-20 U/L)

Alk Phos 125 (ref range 20-70U/L)

Bilirubin, Total 1.1 (ref range 0.1-1 mg/dL)

### **Elevated serum CEA, mildly elevated transaminases and alkaline phosphatase**

1. Identify the organ/describe the characteristic pathologic changes in the virtual microscopy slide.

**Liver; sections show islands of well differentiated adenocarcinoma surrounded by unremarkable liver.**

2. What is your diagnosis?

**Metastatic adenocarcinoma**

3. Review Case 4 slide for comparison with case 2 slide.

4. What is "CEA"?

**Carcinoembryonic antigen is a glycoprotein normally found in fetal gut tissue. It is increased in a variety of adenocarcinomas, particularly colorectal cancer.**

**Because it lacks both sensitivity and specificity, serum CEA is NOT a useful screening tool for colorectal cancer. However, in patients with established disease, the absolute level of the serum CEA correlates with disease burden.**