Introduction:

Identification of abnormal liver biochemical tests is one of the most common discoveries during health examinations (8.9%). Physicians of all specialties need a working knowledge of the meaning of these abnormalities, and a general sense of when urgency for evaluation is necessary. The following is my suggested guideline for the evaluation of ↑ LFT's.

A focused evaluation of abn LFT's includes 4 areas of importance:

1. Categorization of Severity, Symptoms and Duration:
   a. Mild vs. Moderate vs. Severe
   b. Asymptomatic vs. Symptomatic
   c. Acute vs. Chronic (≥ 6 mo)

2. Complete Medical History
   a. Exposures: “bad” water (fresh & salt), blood, needles, partners, toxins, ETOH,...
   b. Travel: O-con-US
   c. Family history: liver disease, alcohol abuse, origins
   d. Medications, OTC’s, Herbals, Foods (seafood, non-potable irrigation, ..)
   e. A history of arthralgias and myalgias predating jaundice, suggests viral or drug-related hepatitis, while jaundice associated with the sudden onset of severe right upper quadrant pain and shaking chills suggests choledocholithiasis and ascending cholangitis.
   f. Occupational exposure to chemicals, solvents, fumes and vapors.

3. Complete Physical Examination
   a. Stigmata of Chronic Liver Disease
      ✔ Stigmata of chronic liver disease include spider nevi, palmar erythema, gynecomastia, caput medusae
Dupuytren's contractures, parotid gland enlargement, and testicular atrophy are commonly seen in advanced Laennec's cirrhosis and occasionally in other types of cirrhosis.

Temporal and proximal muscle wasting suggest longstanding diseases.

An enlarged left supraclavicular node (Virchow's node) or peri-umbilical nodule (Sister Mary Joseph's nodule) suggest an abdominal malignancy.

Jugular venous distension, a sign of right sided heart failure, suggests hepatic congestion.

A right pleural effusion, in the absence of clinically apparent ascites, may be seen in advanced cirrhosis.

b. Stigmata of other systemic disorders

- Systemic disorders involving both skin and liver: SLE, Sjogren's syndrome, Scleroderma, Psoriasis, Dermatitis Herpetiformis, Mastocytosis, Melanoma.
- Rheumatologic Disorders or medicines used to treat these disorders: SLE, RA, Felty Syndrome, Sjogren's Scleroderma, AS & Meds: NSAIDs, Penicillamine, Sulfasalizine, Leflunomide, MTX, anti-TNF α agents.
- Pregnancy related: Hyperemesis gravidarum (HG), Intrahepatic Cholestatis of Pregnancy (ICP), Hemolysis, Elevated liver Enzymes, and Low Platelets (HEELEP syndrome), Acute Fatty Liver of Pregnancy (AFLP).
- Cardiovascular disease: Ischemic Hepatopathy, Congestive Hepatopathy,
- Pulmonary Disorders: α-1-Antitrypsin Deficiency, Cystic Fibrosis, Sarcoidosis, TB.
- Endocrine Disorders: Metabolic Syndrome, Thyroid, Adrenal Insufficiency, Cushing's Syndrome, Hyperestrogen states,
- Hematologic & Oncologic Diseases: Hemolytic anemia's, Sickle cell anemia, Malaria, DIC, Porphurias, Leukemia, Lymphoma, Metastasis.
- Infectious Diseases: Viruses (EBV, CMV, HSV, Yellow fever, Dengue), Bacteria (Salmonella, Mycobacterium TB, Brucella, Coxiella bumerii, Leptospira, spirochetes), Parasites (Schistosomasis, Plasmodium) and Fungi (Candida and Histoplasma capsulatum).
4. Stratification of Severity

a. Hepatic Dysfunction: Synthetic (Albumin, Protein, Coagulation factors), Detoxification (Bilirubin, PSE, Ammonia, Protein, Drugs, Toxins), Portal HTN (Ascites, Varices, Thrombocytopenia, Organomegaly, Edema).

b. Degree of abn LFT's
   ✓ Mild (2-4x ULN)
   ✓ Moderate (5-10x ULN)
   ✓ Severe (≥ 10x ULN)

c. Rapidity of Δ LFT abnormality and associated clinical deterioration
   ✓ Hours to Days
   ✓ Weeks
   ✓ Months

Definitions:

The term "Liver Function Tests" (LFT's) is misnomer, since the measurement of liver associated enzymes (LAE's = AST, ALT, Alk Phos, Bilirubin, δ GGT, 5' nucleotidase) does NOT measure liver FUNCTION! Elevation of LAE's is more accurately a measure of the release of cytosolic enzymes from inflamed or injured hepatocytes and/or bile duct epithelium. Indirect measurements of liver function would include (synthetic: albumin, protein, prothrombin time; detoxification: ammonia and excretory: bilirubin), while direct measurement of liver function would include caffeine or lidocaine clearance tests that are only performed at research centers.

Laboratory Testing

The pattern of the abnormal LFT's can be divided into three categories, that become branch points in the evaluation of hepatitis:

- Patterns predominantly reflecting hepatocellular injury (↑ ALT/AST)
- Patterns predominantly reflecting cholestasis (↑ Alk Phos +/- ↑ Bili)
- Mixed hepatocellular and cholestatic (↑ Both ALT/AST & Alk Phos)

Note: Increases in conjugated bilirubin can be seen in each category.

- ↓ Albumin suggest severe hepatitis, cirrhosis or malnutrition
- ↑ PT suggests significant cholestasis, hepatitis or vitamin K deficiency
- Bilirubin in the urine reflects direct or conjugated hyperbilirubinemia
- Unconjugated bilirubin is bound to albumin (only in urine with renal disease)
Step one

Stratify the severity of hepatitis!

If any of the following are evident, then the urgency for complete evaluation becomes heightened.

- Moderate or Severe hepatitis = ↑ LFT's > 5-10x ULN
- Clinically symptomatic hepatitis
- Evidence of Cirrhosis or Hepatic Decompensation by History & Physical or Ancillary Tests:

If hepatitis is "mild" without warnings, then determine the slope of hepatic injury = \( \Delta \text{LFT}/\Delta \text{time} \).

### Extent and Pace of Work Up

**Moderate to Severe ↑LFT's**
- Intermediate/Rapid slope
- Signs Chronic Liver Disease

**Mild ↑ LFT's, Asymptomatic**
- Flat Slope
- No Signs of Chronic Liver Disease

**Work up in "launch" mode**
- Early GI/Hepatology Input
- Many tests take wks to return

**Focused work up**
- GI/Hepatology Input at 4-6 mo.
- Liver biopsy for ↑ LFT ≥ 6 mo.
Moderate to Severe Elevation LFTs Intermediate to Rapidly Rising Slope

This type of pattern and trend for LFT’s elevation should be evaluated more expeditiously, especially if symptoms of hepatic decompensation are present.

Mild Chronic Elevation in LFTs

The laboratory evaluation of patients with chronic (≥ 6 mo), mild elevation (<4x ULN). the selection of additional tests should be guided by the history and physical examination, but remember Rule #1: "Common Diseases are Common!"

Rule 1: "Common diseases are common!"
- Fatty Liver
- Medications, OTC’s, Herbals, Supplements
- Alcohol
- Viral Hepatitis (HBV, HCV)
- Hemochromatosis

Fatty Liver

The presence of hepocellular injury from hepatitis due to obesity is a downstream impact of the obesity(BMI > 30) epidemic in America, 70% of obese persons have fatty liver disease ==> 20-30% of American adults have fatty liver disease. The projected impact of obesity on liver disease is most riveted by the projection that the leading indicator for liver transplant in 2020 with be fatty liver. The difficulty is determining which patients have Non-Alcoholic Fatty Liver Disease (NAFLD) vs. Non-Alcoholic Steatohepatitis (NASH), which is a potentially progressive liver disease leading to cirrhosis, Figure 1.

Natural History for NAFLD

![Natural History for NAFLD](#)
NASH is more common in women and associated with Syndrome X. In contrast to alcohol related liver disease, the ratio of AST:ALT is usually < 1; yet when AST:ALT is > 2, NASH progression to cirrhosis is likely. Ultrasound is the most economical test to evaluate for hepatic steatosis, however radiologic imaging is not specific for NAFLD and cannot identify inflammation or distinguish simple hepatic steatosis from NASH. Nevertheless, because of the absence of effective medical therapy for NASH, many experts do not advocate a liver biopsy unless one of the following is present:

- Peripheral stigmata of chronic liver disease
- Splenomegaly
- Cytopenia
- Abnormal iron studies
- Diabetes and/or significant obesity in an individual over the age of 45

**Medications**

Over 300 drugs in current use have been implicated to cause liver injury. Drug Induced Liver Injury (DILI) causes ~5% of community cases of acute hepatitis and 10-40% of hepatitis cases admitted to the hospital. Hepatic injury patterns defined by LFT profiles and known risk factors for incidence and severity can be helpful in identifying DILI.

<table>
<thead>
<tr>
<th>Hepatocellular</th>
<th>Cholestatic</th>
<th>&quot;Mixed&quot; Cholestatic Hepatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ ALT &gt; 2-3x</td>
<td>↑ Alk Phos &gt; 2-3x</td>
<td>↑ ALT &gt; 2-3x and</td>
</tr>
<tr>
<td></td>
<td>Alk Phos ratio ALT &lt; 2</td>
<td>↑ Alk Phos &gt; 2x</td>
</tr>
<tr>
<td>INH, Sulfonamides, Phenytoin,</td>
<td>Estrogen, Anabolic</td>
<td>Chlorpromazine, Macrolide</td>
</tr>
<tr>
<td>Disulfiram, Ketoconazole, Nitrofurantoin, Minocycline, Nicotinic acid</td>
<td>Steroids, Tamoxifen,</td>
<td>antibiotics, TCA's, Augmentin,</td>
</tr>
<tr>
<td></td>
<td>Azathioprine</td>
<td>Ketoconazole, NSAID's, Captopril, Enapril</td>
</tr>
</tbody>
</table>

Chronologic association between initiation of drug and the onset of liver injury is often helpful. The latent period can be hours to days (acetaminophen), immunoallegic DILI reactions usually delayed 2 to 10 weeks, amoxicillin-clavulanate associated DILI may arise up to 6 wk after discontinuation.

Consumer use of herbal remedies is common place in the US, with 1 in 5 adults taking at least one herbal agent. This industry is largely unregulated, persons with preexisting liver disease should be cautious and consult their doctor and reputable websites, [http://nccam.nih.gov/](http://nccam.nih.gov/) Potentially Hepatotoxic Herbs: Autoimmune hepatitis: Syo-saiko-to, Ma-huang, Germander; Cirrhosis: Syo-saiko-to, Chaparral, Greater celandine, Jin Bu Huan; Cholestasis Hepatitis: Cascara sagrada, chaparral, Greater celandine, Kava, Syo-saiko-to; Fulminant Hepatic Failure: Atractylis gummifera, Chaparral, Cocaine, Germander, Kava; and Veno-occlusive disease: pyrollizidine alkaloids (Teas), Skullcap.

**Alcohol abuse**

CAGE questionnaire.
AST: ALT ratio of 2:1
Elevation GGT and AST
Elevation MCV
**Hepatitis B**

The pretest probability for hepatitis B is increased among the unvaccinated with a history of parenteral exposure or birth in or travel to areas of high endemcity (Southeast Asia, China, and sub-Saharan Africa). Pockets of high prevalence in US (African immigrant workers; San Francisco, Seattle, New York, Miami,...)

**Hepatitis C**

Chronic hepatitis C is very common in the United States and other parts of the world. The risk is highest in individuals with a history of parenteral exposure (blood transfusions, intravenous drug use, occupational), cocaine use, tattoos, body piercing, and high risk sexual behavior.

**Hereditary hemochromatosis**

Hereditary hemochromatosis (HHC) is a common genetic disorder, frequency of heterozygotes is about 10 percent in Caucasian populations in the United States and western Europe, with a frequency of about 1 per 200 (0.5 percent) for the homozygous state.

Screening Tests:

Serum iron/TIBC = % iron saturation (value > 45 suggests, need to screen for HHC with serum Ferritin).

Serum ferritin: 400 ng/mL ♂, suggests HHC -- should do Liver biopsy and gene tests for HFE mutation

300 ng/mL ♀, suggests HHC -- " "

Liver Biopsy: A hepatic iron/age index (hepatic iron concentration in micromoles per gram dry weight divided by the patient's age) greater than 1.9 is consistent with homozygous HHC. A liver biopsy is not necessary for patients less than 40 years of age with genotypically defined hemochromatosis (C282Y homozygous or C282Y/H63D mutations, so-called compound heterozygotes) with normal liver function tests.

**Steps 2 & 3**

**Step 2:** Tests should look for non-hepatic causes of elevated aminotransferases: muscle disorders, thyroid disease, celiac disease and adrenal insufficiency.

**Step 3:** Tests aimed at identifying rarer liver conditions: Autoimmune hepatitis, Wilson's disease, Alpha-1Antitrypsin Deficiency, and Adult bile ductopenia.

**Autoimmune hepatitis**

Autoimmune hepatitis (AIH) is a condition found primarily in young to middle-aged women (♀ to ♂ ratio of 8:1). The diagnosis is based upon the presence of elevated serum aminotransferases, the absence of other causes of chronic hepatitis, and features (serological and pathological) suggestive of AIH.
Screening tests:

Serum Protein Electrophoresis (SPEP) > 80% AIH have hypergammaglobulinemia (↑ γ-glob ≥ 4)

Antinuclear Antibodies (ANA)

Anti-smooth Muscle Antibodies (ASMA)

Liver-Kidney Microsomal Antibodies (LKMA)

Height of LFT elevation is usually 5-10 x ULN

**Wilson's disease**

Wilson disease, a genetic disorder of biliary copper excretion, may cause elevated aminotransferases in asymptomatic patients. While the prevalence of Wilson disease is very low, it is a treatable liver disease and needs to be identified. Patients usually present between ages 5 to 25, but the diagnosis should be considered in patients up to the age of 40.

Screening Tests:

Serum ceruloplasmin: ↓ in ~85 percent of patients.

Ophthalmologist for Kayser-Fleischer rings

24-hour urine collection for quantitative copper excretion

Liver biopsy for quantitative copper > 250 mcg/gm of dry weight is seen with Wilson's Disease.

**Alpha-1 antitrypsin deficiency**

Alpha-1 antitrypsin deficiency is an uncommon cause of chronic liver disease in adults.

Screening Test:

SPEP: ↓ α-1-antitrypsin or on direct measure (note levels may increase in response to inflammation and conceal the diagnosis)

Alpha-1 antitrypsin phenotype is probably the most cost-effective test

Liver biopsies should be stained with PAS (PAS (+) diastase resistant globules indicate A1AT def)

**Adult bile ductopenia**

Adult bile ductopenia is a rare inherited condition that presents with elevated aminotransferases. In mild forms, patients are asymptomatic, while in more serious forms, patients have pruritus and elevations of plasma alkaline phosphatase. The diagnosis is based upon liver biopsy findings. In the healthy liver, there are approximately 1.5 to 2 bile ducts cut in cross section per portal triad. In adult bile ductopenia, there are typically fewer than 1.2.
Some patients respond to ursodeoxycholic acid (12 to 15 mg/kg body weight per day). Such patients have normalization of the plasma aminotransferases and generally do not progress to cirrhosis. By contrast, in the severe form, the disease progresses despite treatment, and patients may eventually require liver transplantation.

**Step four**

A liver biopsy is often considered in patients in whom all of the above testing has been unyielding or staging severity of liver injury is necessary.

**Whom to observe and not perform a liver biopsy?**

Some recommend observation only in patients in whom the ALT and AST are less than twofold elevated and no chronic liver condition has been identified by the above noninvasive testing. This approach was supported by a preliminary study in which expectant clinical follow-up was found to be the most cost-effective strategy for managing asymptomatic patients with negative viral, metabolic, and autoimmune markers and chronically elevated aminotransferases. Another study included 36 patients with a chronic elevation of the serum ALT, AST, or alkaline phosphatase (50 percent or greater above normal).

**Whom to biopsy**

All patients with persistent ↑ ALT &/or AST 2x ULN for ≥ 6 months should undergo liver biopsy for diagnosis and staging of the degree of liver injury.

**Isolated Hyperbilirubinemia**

Isolated hyperbilirubinemia occurs principally in two settings:

- Overproduction of bilirubin
- Impaired uptake, conjugation, or excretion of bilirubin

The initial step in evaluating a patient with an isolated elevated hyperbilirubinemia is to fractionate the bilirubin to determine whether the hyperbilirubinemia is predominantly conjugated or unconjugated. Recall that in the absence of renal disease only conjugated bilirubin will appear in the urine.

**Unconjugated hyperbilirubinemia**

**Over Production of Bilirubin = Hemolysis** (serum bilirubin rarely exceeds 5 mg/dL)

Test: Peripheral smear, ↑ reticulocyte count, ↓ haptoglobin, and ↑ LDH

Inherited hemolytic disorders: spherocytosis, sickle cell anemia, G6PD deficiency. Acquired hemolytic disorders: microangiopathic hemolytic anemia (eg, hemolytic-uremic syndrome), paroxysmal nocturnal
hemoglobinuria, and immune hemolysis. Ineffective erythropoiesis occurs in cobalamin, folate, and iron deficiencies.

**Impaired hepatic uptake or conjugation**

Impaired hepatic uptake or conjugation of bilirubin should be considered in the absence of hemolysis. This is most commonly caused by certain drugs (rifampicin and probenecid) which diminish hepatic uptake of bilirubin, or Gilbert's syndrome. Gilbert's syndrome affects ~5% of the US population. Impaired conjugation of bilirubin is due to ↓ UDP glucuronosyl transferase activity. Unconjugated hyperbilirubinemia levels are always less than 6 mg/dL. The serum levels may fluctuate and jaundice is often identified only during periods of illness or fasting. In an otherwise healthy adult with mildly elevated unconjugated hyperbilirubinemia and no evidence of hemolysis, the presumptive diagnosis of Gilbert's syndrome can be made without further testing.

**Conjugated hyperbilirubinemia**

Elevated conjugated hyperbilirubinemia is found in two rare inherited conditions: Dubin-Johnson syndrome and Rotor syndrome. Patients with both conditions present with asymptomatic jaundice typically in the second decade of life. Normal levels of Alk Phos and GGT help to distinguish these conditions from disorders associated with biliary obstruction.

**Isolated Elevation of Alk Phos &/or GGT**

Serum alkaline phosphatase is derived predominantly from the liver and bones.

Other sources of Alk Phos:

- Placenta
- Individuals with blood types O and B can have elevated serum alkaline phosphatase after eating a fatty meal due to an influx of intestinal alkaline phosphatase.
- Infants and toddlers occasionally display transient marked elevations of alkaline phosphatase in the absence of detectable bone or liver disease.
- There are also reports of a benign familial occurrence of elevated serum alkaline phosphatase due to intestinal alkaline phosphatase.

**Determining the source of the alkaline phosphatase**

The first step in the evaluation of an elevated alkaline phosphatase is to identify its source.

**Serum Tests:**

↑ 5'-nucleotidase or GGT suggest biliary origin for increased Alk Phos

Normal 5'-nucleotidase or GGT should suggest a bone disorder as cause for increased alk Phos.

**Initial testing for ↑ Alk Phos of hepatic origin** (↑ 5'nucleotidase or GGT)
Chronic cholestatic or infiltrative liver diseases should be considered in patients in whom the alkaline phosphatase is determined to be of liver origin and persists over time.

Differential Diagnosis:

- Neoplasm
- PBC (AMA)
- PSC (ANCA) 75% of PSC associated with IBD
- Adult bile ductopenia
- Drugs: phenytoin and androgenic steroids
- Granulomatous Diseases: sarcoid, drugs,

Testing:

Ultrasound or CT scan to evaluate for bile duct obstruction, mass, infiltration or "ductopenia". If negative blood tests with AMA and ANCA should be obtained. If these studies are negative, and no signs of IBD, then liver biopsy and either ERCP or MRCP should be performed.

**Gamma glutamyl transpeptidase**

Gamma glutamyl transpeptidase (GGT) is found in hepatocytes and biliary epithelial cells. In normal full-term neonates, serum GGT activity is six to seven times the upper limit of the adult reference range; levels decline and reach adult levels by 5 to 7 months of age. GGT is sensitive for detecting hepatobiliary disease, but its usefulness is limited by its lack of specificity. Elevated levels of serum GGT have been reported in a wide variety of clinical conditions, including pancreatic disease, myocardial infarction, renal failure, chronic obstructive pulmonary disease, diabetes, and alcoholism. High serum GGT values are also found in patients taking medications such as phenytoin and barbiturates.

Some authorities have advocated using the GGT to identify patients with occult alcohol use. The reported sensitivity of an elevated GGT for detecting alcohol ingestion has ranged from 52 to 94 percent. Its lack of specificity makes its use for this purpose questionable. A population-based study found that men with increased GGT levels who also had a hyperechogenic liver by ultrasound (suggesting the presence of steatosis) has increased all-cause mortality rates but more data are needed.

We suggest GGT be used to evaluate elevations of other serum enzyme tests (eg, to confirm the liver origin of an elevated alkaline phosphatase or to support a suspicion of alcohol abuse in a patient with an elevated AST and an AST:ALT ratio of greater than 2:1). An elevated GGT with otherwise normal liver tests should not lead to an exhaustive work-up for liver disease.
References


ALT/AST < 5 x ULN

H&P + Medication List

LFT, PT, Albumin, CBC, PLT, Hepatitis A, B, C, Fe, TIBC, Ferritin

Consider US, ANA, ASMA, Ceruloplasim, A1AT

Liver Bx

(-) Serology, Asx without Liver Decompensation

Lifestyle Modification
Stop ETOH
Stop Toxic Rx
Wt Loss
DM control

Observe
Repeat LFT

US & Serologies: ANA, ASMA, ceruloplasim, A1AT, Gliadin-AB, TTG/AEmAB

Liver Bx

(+1 Serology

(+ HAA IgM > Observe

(+ HBV or HCV

Follow Clinically, Serial LFT

Improve, NL LFT < 6 mo
No better ↑ LFT > 6 mo

**Observe
Liver Bx
### Case 1

<table>
<thead>
<tr>
<th>51 yo ♀ seeking life insurance</th>
<th>PE: 5'4” 175lbs = BMI 30 (obese)</th>
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<tbody>
<tr>
<td>PMSHx: HTN, BTL, T&amp;A, TVH</td>
<td>Normal except liver 11 cm</td>
</tr>
<tr>
<td>Rx: Lisinopril</td>
<td>Labs:</td>
</tr>
<tr>
<td>OTC &amp; Herbals: -0-</td>
<td>CBC – nl</td>
</tr>
<tr>
<td>ETOH: 1-2/wk</td>
<td>ALT  50 IU</td>
</tr>
<tr>
<td>Social: married, 2 kids, secretary</td>
<td>AST  37 IU</td>
</tr>
<tr>
<td>ROS: (-) “hepatic”, p-menopausal</td>
<td>Radiology: 0</td>
</tr>
</tbody>
</table>

**Notes:**
### Case 2.

**50 yo ♂ with increased LFT’s**

- PMSHx: HTN, DJD - debilitating T2DM, BPH, ED, L4-5 fusion,
- Rx: Lisinopril, NSAID, Metformin, Cialis
- OTC & Herbals: -0- ETOH: -0- yrs
- Social: Unemployed, married, 2 kids
- ROS: (-) “hepatic”, depressed

**PE: 5’10” 200 lbs**

- Over weight, depressed, walks slow with a limp, liver 12 cm, OA changes to hands, venous stasis and 1+ PTE

**Labs:**

- CBC – PLT 100K
- ALT  50 IU
- AST  37 IU

**Radiology: US & Hand X-rays**

### Notes:
### Case 3:

<table>
<thead>
<tr>
<th>30 yo ♀ seeking life insurance</th>
<th>PE: 5'4&quot; 160lbs</th>
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<tbody>
<tr>
<td>PMSHx: HTN, ↑ Lipids, T&amp;A, TVH</td>
<td>Normal except liver 11 cm</td>
</tr>
<tr>
<td>Rx: BCP, Lisinopril, Zocor</td>
<td>Labs:</td>
</tr>
<tr>
<td>OTC &amp; Herbals: Tylenol &amp; -0-</td>
<td>CBC – nl</td>
</tr>
<tr>
<td>ETOH: 1-2/wk</td>
<td>ALT  50 IU</td>
</tr>
<tr>
<td>Social: married, Vanguard $ Mgm</td>
<td>AST  37 IU</td>
</tr>
<tr>
<td>ROS: (-) “hepatic”</td>
<td>Radiology: 0</td>
</tr>
</tbody>
</table>

**Notes:**
### Case 4:

<table>
<thead>
<tr>
<th>34 yo ♀ 5 yr history RA, visiting from Phoenix</th>
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<tbody>
<tr>
<td>New onset jaundice, myalgia, low grade fever, dark urine</td>
</tr>
<tr>
<td>PMSHx: RA, GERD, UGIB – nasaid ulcer 2000, BTL, T&amp;A</td>
</tr>
<tr>
<td>Rx: MTX 15mg/wk 4 yrs, Naprosyn, tylenol, ADA 1 mo</td>
</tr>
<tr>
<td>OTC &amp; Herbals: -0- ETOH: -0- Social: married, 2 kids, ROS: (-) “hepatic”</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PE: 5’4” 175lbs = BMI 30 (obese)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal except liver 11 cm</td>
</tr>
<tr>
<td>Labs:</td>
</tr>
<tr>
<td>CBC 13.5WBC HCT 34 PLT 160</td>
</tr>
<tr>
<td>ALT 500 IU</td>
</tr>
<tr>
<td>AST 430 IU</td>
</tr>
<tr>
<td>APO 200 IU</td>
</tr>
<tr>
<td>TB 4.1</td>
</tr>
<tr>
<td>Radiology: 0</td>
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**Notes:**
Case 5:

18 yo ♂ with Strep tonsillitis and maxillary sinusitis 6 wk ago.
New onset jaundice, myalgia, low grade fever, dark urine

PMSHx: Appy at 10 yrs

Rx: o

OTC & Herbals: -0- ETOH: -0-
Social: scholar athlete DU -2011
ROS: (-) “hepatic”

PE: 6’3” 190 lbs

ENT adenonapthy
Liver 14 cm ? Spleen tip

Labs:

CBC 13.5WBC HCT 48 PLT 260
ALT 440 IU
AST 210 IU
APO 400 IU
TB 6.1

Radiology: 0

Notes:
Case 7:

54 yo ♀ presented with 6 wks of recurrent RUQ pain, intermittent sweating
New onset jaundice, nausea, myalgia, low grade fever, dark urine
PMSHx: DM, ↑ Lipids, HTN, OA, ROAD, GERD
Rx: Glyburide, Vytorin, Lisinopril, Naprosyn, PPI
OTC, Herbals & ETOH: -0-
Smoker 1 ppd x 20 yrs
Social: welfare
ROS: (-) “hepatic”

PE: 5’4” 195lbs
VS: 80/40 HR 118 R 20 T 103 O2 84% RA
Very ill, confused, jaundiced, (+) Murphy’s sign, râles bilateral lung bases
Labs:
CBC 19.5WBC HCT 57 PLT 160
ALT 200 IU
AST 180 IU
APO 230 IU
TB 4.1
Radiology: 0

Notes: