
بسم الله الرحمن الرحيم

Basic Metabolic Panel
Charting Shorthand

BMP	Na	Cl	BUN	glucose
	K+	CO2	Creatinine	

Clinical Chemistry Panels: Comprehensive Metabolic Panel Includes

- BMP except CO2
- Albumin
- Serum enzymes (alkaline phosphatase, AST [SGOT], ALT [SGPT]
- Total bilirubin
- Total calcium Phosphorus, total cholesterol and triglycerides often ordered with the CMP

Liver Function Tests

- Clinical symptoms in liver disease often lag behind the progression of disease
- Evaluation of liver function can often be achieved by determination of serum analytes in a test profile known as **liver function tests**
- Many of these components are not unique to liver

Routine LFT

- Total protein
- Albumin
- Globulins
- Total bilirubin
- Direct bilirubin
- AST (Aspartate aminotransferase)
- ALT (Alanine aminotransferase)
- ALP (Alkaline phosphatase)
- GGT (Gamma Glutamyl transferase)*

Liver Function Tests

- **Metabolic function**
  - Bilirubin
  - Ammonia
  - Lipids
- **Synthetic function**
  - Protein synthesis (Albumin, alpha-1-antitrypsin, ceruloplasmin, clotting factors)
- **Tests of liver injury**
  - Aminotransfrases (AST,ALT)
  - Lactate dehydrogenase (LDH)
  - Alkaline phosphatase, Gamma Glutamyl transferase (GGT)

- **Viral Hepatitis tests**
- **Autoimmune markers**
  - Antimitochondrial Ab, ANCA, ANA

تست‌های کبدی

- برای انجام این تست‌ها نیازی به ناشتا بودن بیمار نیست،ولی برای انجام هر چه بهتر تست بهتر است که بیمار حداقل چند ساعت ناشتا باشد.
- مصرف الکل سبب آسیب به کبد شده و در نتیجه بسیاری از آنزیم‌های کبدی از جمله آلکالین فسفاتاز، SGPT,SGOT و GGT افزایش می‌یابد.
- مصرف فنوباربیتال و فنی توئین که جزءداروهای ضدصرع هستند سبب افزایش سطح آنزیم‌های کبدی می‌شود.
- حداقل ۳ روز قبل از آزمایش هیچ گونه تزریق عضلانی انجام ندهید.

آنزیمهای کبدی

✓ **آلانین آمینوترانسفراز (ALT):**

- آنزیم درون–یاخته ای
- عمدتاً در کبد است ولی ایزوفرمهای آن در کلیه، قلب، ماهیچهٔ اسکلتی و لوزالمعده نیز یافت می شود.
- مردان < ۴۵ U/L
- زنان < ۳۴ U/L
- افزایش: التهاب و آسیب کبدی
- کاهش: کمبود پیریدوکسین

✓ **آسپارتات آمینوترانسفراز (AST):**

- آنزیم درون–یاخته ای
- عمدتاً در قلب و سپس در کبد، ماهیچهٔ اسکلتی، لوزالمعده، گویچه های سرخ و طحال
- مردان< ۳۵ U/L
- زنان < ۳۱ U/L
- افزایش: MI، میوکاردیت، آسیب کبدی، بیماریهای ماهیچه ای، همولیز، کمخونی وخیم، انفارکتوس کلیوی، پانکراتیت حاد، بدخیمی
- کاهش: کمبود پیریدوکسین

ALP

- دریافت مواد غذایی در ساعات نزدیک به ازمایش سطح ان را بالا می برد
- نیازی به ناشتایی برای این ازمایش نیست
- دارو هایی که سطح ALP را بالا می برند: متوتروکسات، نیکوتینیک اسید، انتی بیوتیک ها، فلوراید ها
- دارو هایی که سطح ALP را پایین می برند: مشتقات ارسنیک، سیانید ها، نیتروفورانتوئین، اگزالات ها و نمک های روی

URIC ACID

2-8 M

2-7.5 F

<u>علل افزایش:</u>

- نقص ژنتیکی یا متابولیکی(نقرس)
- در اثر کمبود روی
- در اثر کمبود مس
- در اثر افزایش فریتین
- بالا بودن پورین رژیم
- فروکتوز بالا

معرفی بیمار:

آقای ۶۰ ساله BUN و کراتینین نرمال.اسید اوریک بالا

- ✓ اقدام اول: یادآمد برای مصرف روی .مس . آهن
- ✓ اقدام دوم:تست فریتین
- ✓ اقدام سوم: رژیم کم پورین

اگه خیلی بالا بود باید دارو بگیره

کاهش اسید اوریک علت پاتولوژیک خاصی ندارد.

- Copper(70-155 mcg/dl)

-Ceruloplasmin (23-24 mg/dl)

-Zinc (0.85-1.25 mcg/ml)

- آزمایش روی منبع درستی از تخمین ذخایر نیست.
- در بچه های کم اشتها و کم وزن روی می دهیم(۱-۲ ماه) اگر طولانی مدت باشد با جذب cuو fe تداخل می کند
- خاک ایران فقیر از روی است.
- مس کم باشد سرولوپلاسمین کم و اگر سرولوپلاسمین کم باشد مس هم کم است
- در ویلسون سرولوپلاسمین کم است مس در بدن رسوب میکنه و مس ادرار بالا ست.

- درمان: مکمل روی (زینک سولفات ۲۵۰ mg/day) و شلاته کننده های مس(دیپنسین آمین) در نهایت اگه به دارو جواب نداد رژیم فاقد مس میدیم

BUN= ازت اوره خون

علل افزایش:

- مشکل کلیه (شایعترین علت)
- خونریزی دستگاه گوارش
- رژیم با پروتئین بالا
- داروهای استروئیدی
- شوک
- آدیسون
- سوختگی- بدخیمی
- دهیدراته شدن (وقتی میگی ناشتا بعضی ها آبم نمیخورند.باید بهشون گفت آب میشه خورد.البته نه زیاد)
- نارسایی قلبی
- اسهال
- سوختگی
- نکروز بافتی
- فشار خون بالا
- سنگ کلیه

کراتینین سرم

- فراورده متابولیسم فسفوکراتین در ماهیچه
- تولید: ۱۰-۲۵ mmol/d (۲/۱-۶/۰ mg/dL)
- عوامل مؤثر بر سطوح سرمی:
- میزان تولید: LBM
- میزان دفع: GFR
- افزایش: نارسایی کلیوی، انسداد مجاری ادراری، داروهای ن
- کاهش: کاهش LBM

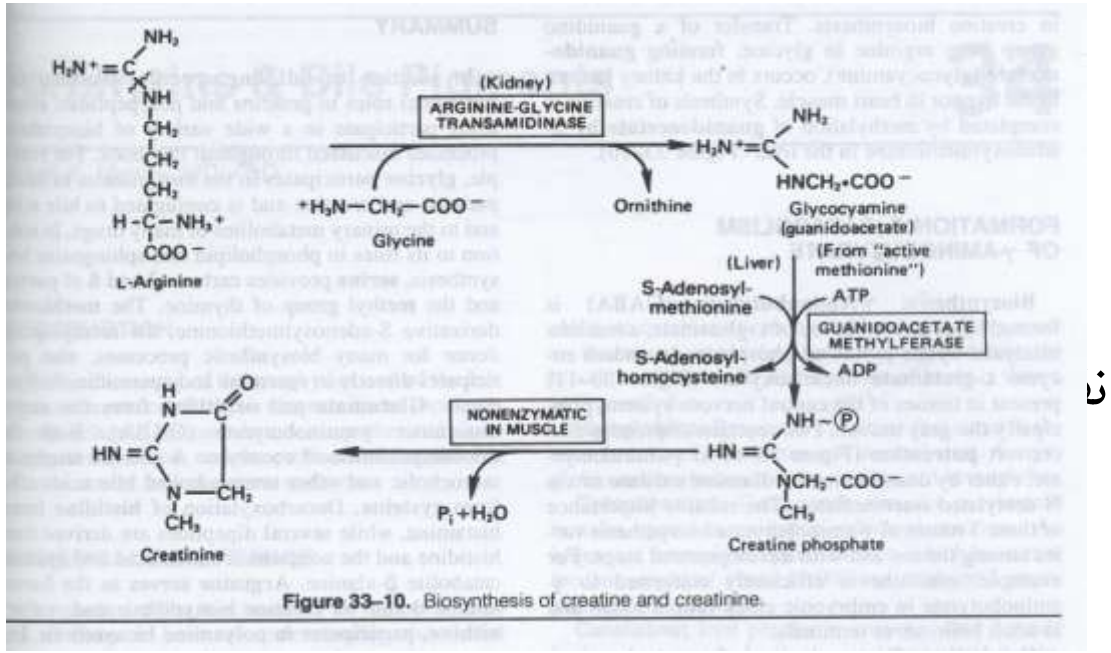
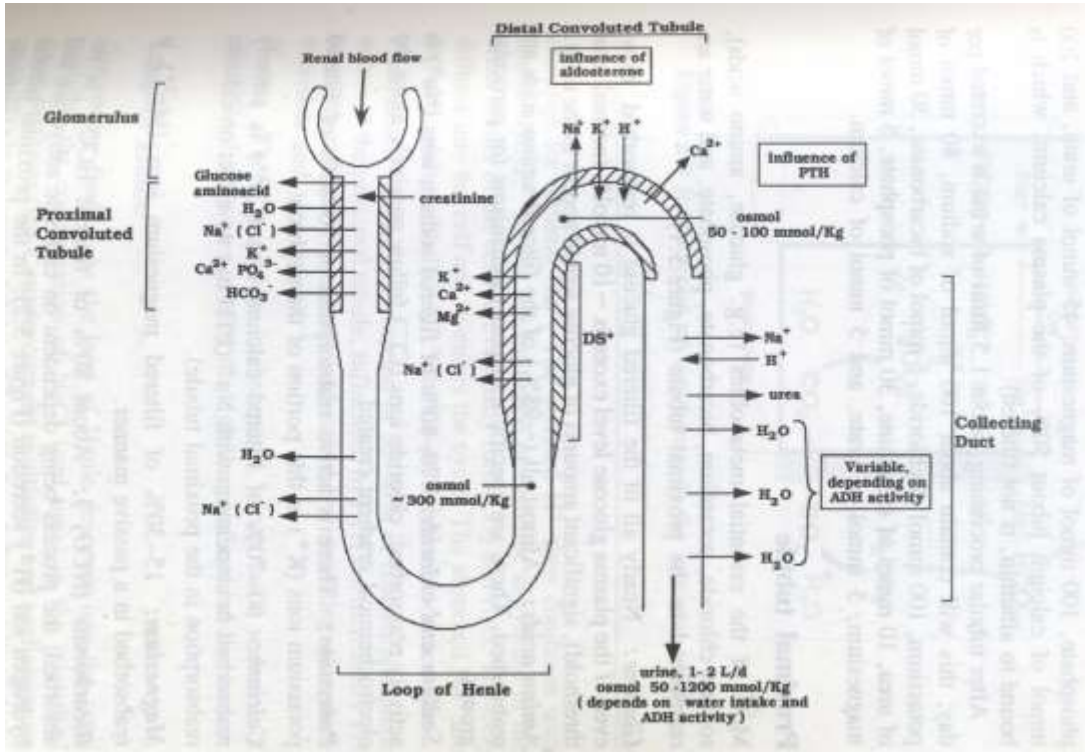


Figure 33–10. Biosynthesis of creatine and creatinine.

- پس از مصرف غذا خصوصا غذا هایی که حاوی گوشت زیادی باشند کراتینین به میزان مختصری افزایش می یابد.



تست BUN/Cr

- به طور طبیعی ۱۲:۱ تا ۲۰:۱
- افزایش: کم آبی، خونریزی گوارشی و افزایش کاتابولیسم
- کاهش: ATN، بیماری کبدی پیشرفته، دریافت پروتئین پایین، به دنبال همودیالیز

CPK

- به بیمار گفته شود جهت انجام ازمایش نیاز به محدودیت آب و غذا نیست.
- ورزش سنگین و جراحی های اخیر باعث افزایش سطح ان میشود.
- مصرف الکل، لیتیوم، پروپرانولول و مورفین سبب افزایش سطح ان میشود.

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- فیدانزا ف. ارزیابی وضع تغذیه در پزشکی بالینی و بهداشت همگانی: روش شناسی تن سنجی، بالینی و آزمایشگاهی. ترجمهٔ: نیستانی ت، ستوده گ، مداح م. دانشگاه علوم پزشکی تهران، ۱۳۸۳.

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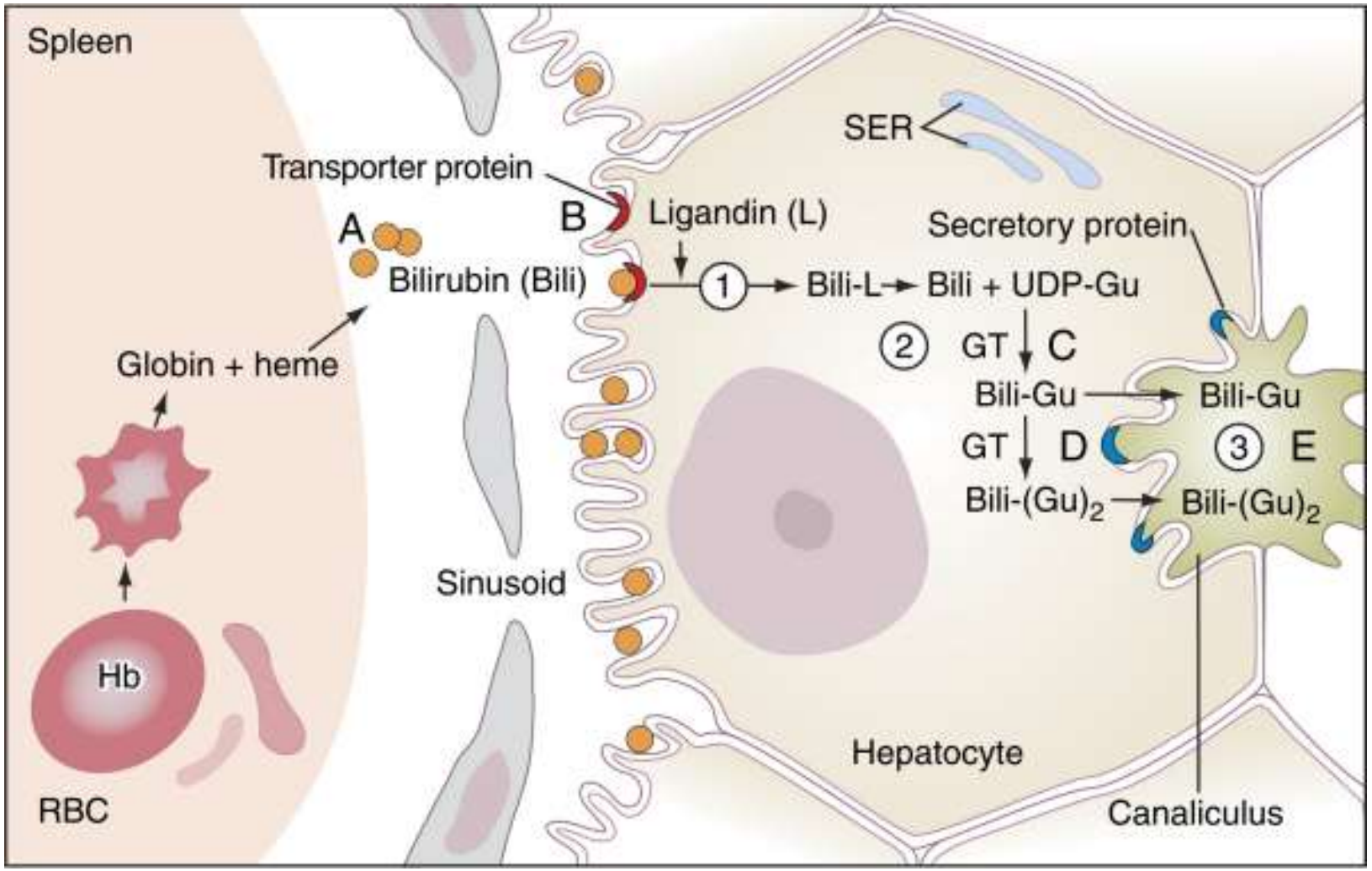
Bilirubin

- Product of heme breakdown
- Metabolized in liver to water soluble Glucuronate conjugate
- In body: Conjugated and Unconjugated
- IN lab:
  - Total
  - Direct: roughly equal to conjugated form
  - Total – direct = indirect bili


Spleen
SER
Transporter protein
B
Ligandin (L)
A
Secretory protein
Bilirubin (Bili)
1
Bili-L
Bili + UDP-Gu
2
GT
C
Globin + heme
Bili-Gu
Bili-Gu
GT
D
3
E
Bili-(Gu)2
Bili-(Gu)2
Sinusoid
Hb
Hepatocyte
RBC
Canaliculus

Bilirubin

- Method: diazotized sulfanilic acid method
- **Reference values:**
  - Total bili: 0.2-1.2 mg/dL
  - Direct bili: 0-0.1 (0.2) mg/dL
- Preanalytic point: Prolonged exposure to light causes photoisomerization → Increasing direct-reacting bilirubin

Elevated unconjugated Bili.

- Hemolysis
- Gilbert Sx
- Criggler-Najjar Sx
- Sepsis
- Hepatitis

Elevated Conjugated Bili.

- Dubin-Johnson Sx
- Rotor Sx
- Biliary obstruction
- Sepsis
- Hepatitis

Ammonia

- Toxic product of amino acids & nucleic acid metabolism that metabolized in liver via Krebs–Henseleit or urea cycle to urea
- In severe injury (>80%) or fulminant hepatitis these cylces don't work → increase ammonia → hepatic encephalopathy

Ammonia

- Method: enzymatic method using glutamate dehydrogenase
- Points:
  - Arterial blood is preferred
  - Tourniquet: Minimal use
  - Fist clenching and relaxing avoided during collection
  - Specimens should be kept in ice water until separation of cells from plasma

Proteins

- All proteins except **von Willebrand factor and immunoglobulins** synthetized in liver
- Total protein
- Albumin

Proteins

- Methods:
  - Total protein: Biuret method, Coomassie blue
  - Albumin: Bromcresol green & bromcresol purple
- Reference range:
- Total protein: 6-7.8 g/dL
- Albumin: 3.5-5 g/dL
- At least 60% total protein must be albumin

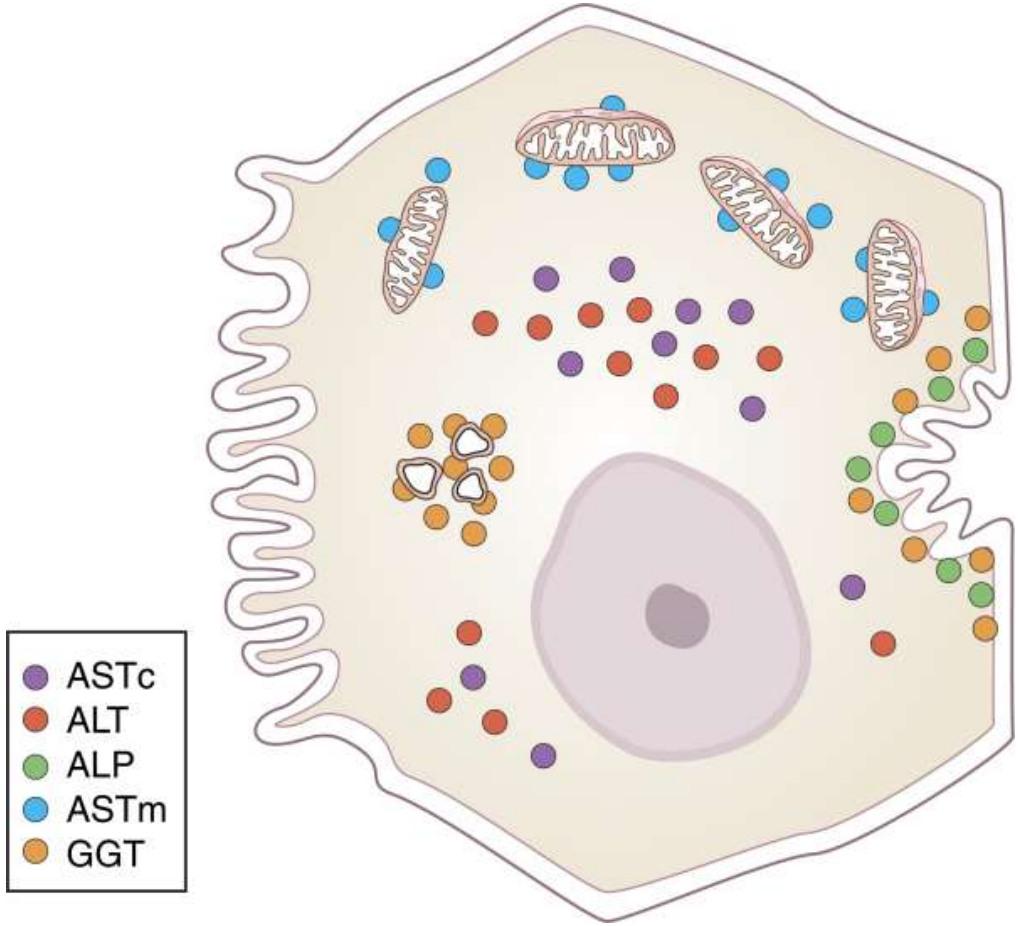
Proteins

- Decrease in total protein (albumn):
  - Liver disease, cirrhosis,
  - Renal disease, malnutrition, protein-losing enteropathies, chronic inflammatory diseases
- Increase in total protein
  - Hemoconcentration
  - Increase Ig level like Multiple myeloma
- **A decrease in albumin is one of the major prognostic features in patients with cirrhosis**

Clotting factors

- All coagulation proteins (except vWf) and their inhibitory proteins synthetized in liver
- **Prothrombin time and INR**
  - Efficacy of extrinsic coagulation pathway
  - Factors: II, **VII**, IX, X

Plasma enzyme levels



Aminotransferases

- Aspartate aminotransferase (AST), SGOT
- Alanine aminotransferase (ALT), SGPT
- AST: Liver, Heart, Muscle
- ALT: Liver, Kidney
- Reference value: <40 IU/L

Aminotransferases

- **Increase in hepatocyte damage**
- Acute hepatocyte injury:
  - AST>ALT
  - After 24-48 hrs ALT>AST
- Acute alcohol induced hepatitis
  - AST>ALT due to release mitochondrial AST
- Chronic hepatocyte injury (cirrhosis)
  - ALT>AST
  - With progression of fibrosis AST>ALT
  - In end stage cirrhosis: AST & ALT not elevated

Aminotransferases

- ALT activity is more specific for detecting liver disease in nonalcoholic, asymptomatic patients
- AST is used for monitoring therapy with potentially hepatotoxic drugs
- Chronic elevation of aminotransferase activities in asymptomatic patients:
  - Alcohol or medication use
  - Chronic viral hepatitis
  - Nonalcoholic fatty liver disease

LDH

- Liver, skeletal muscle, myocardium, RBC, kidney
- **Space occupying lesions of liver** (metastatic ca., hepatocallular ca. , hemangioma)
  - **LDH (>500 IU/L)**
  - **Alkaline phosphatase (>250 IU/L)**
  - **Near normal AST & ALT**

Enzymes for canalicular injury

- **Alkaline phosphatase**
- **Gamma Glutamyl transferase**
- **5' nulcleotidase**

- Canalicular enzyme activities within hepatocytes are typically quite low.
- Focal hepatocyte injury seldom causes significant increases in canalicular enzyme levels

Alkaline Phosphatase (ALP)

- Liver, bone, kidney, placenta, intestine
- Marker of biliary dysfunction
- Reference value
  - 30-120 IU/L
  - In children up to 1200 IU/L

Alkaline Phosphatase (ALP)

- Rise in hepatic ALP (>10x normal)
  - In obstruction of the biliary tract from stones in the ducts or ductules
  - Infectious processes resulting in ascending cholangitis
  - From space-occupying lesions
- Other causes of increase
  - Passive congestion of liver
  - Jaundice from hepatic injury
- Increase in intestinal isoenzyme
  - cirrhosis

Gamma-Glutamyl Transferase (GGT)

- Discriminate the source of elevated ALP
- Reference value:
  - 5-40 IU/L
- Increase:
  - Chronic cholestasis due to primary biliary cirrhosis or sclerosing cholangitis
  - Chronic alcohol abuse (rough correlation between amount of alcohol intake and GGT activity)

Condition	AST	ALT	LD	ALP	TP	Albumin	Bilirubin	Ammonia
1. Hepatitis	H	H	H	H	N	N	H	N
2. Cirrhosis	N	N	N	N–sl H	L	L	H	H
3. Biliary obstruction	N	N	N	H	N	N	H	N
4. Space-occupying lesion	N or H	N or H	H	H	N	N	N–H	N
5. Passive congestion	Sl H	sl H	sl H	N–sl H	N	N	N–sl H	N
6. Fulminant failure	Very H	H	H	H	L	L	H	H

Patterns of cholestasis

Type	Location of obstruction	ALT	ALP	BILI	Examples
"bilirubinostasis"	Hepatocyte	Nl	Nl	Inc	Sepsis TPN
Cholestatic hepatitis	Canalicular	Inc	Inc	Inc	Drug
Ductular injury	Intrahepatic bile ducts	Nl to mild Inc	Inc	Nl to mild Inc	PBC, PSC
Complete obstruction	Extra hepatic bile ducts	Inc	Inc	Inc	Gall stone, head of pancreas cancer

Feature	Hepatocellular injury	cholestasis
ALT	>10x, persist for weeks	Transient >10x, falls quickly
ALP	<3x	>3x, may be nl in early
GGT	< 5X	>5x
Bili	50-80% direct	50-80% direct
PT	Nl to mild increase Vit K Nonresponsive	Nl to mild increase Vit K responsive
Imaging	Nl ducts	Abnl ducts with obstruction

Hepatology Pearls

- Hepatitis: ⇑AST and ALT
- Cholestasis: ⇑ TB and ALP
- ALT more specific than AST
- Measures of function: ALB, Coags, Bili
- Alcoholic hepatitis AST>ALT 2-3:1 (NASH with cirrhosis also)

Abnormal LFT's

- Asymptomatic elevation of ALT is most common problem
- If isolated and less than 3-fold elevation then stop alcohol or drug and recheck in 2-3 months
- If persistent then further workup is needed

Abnormal LFT's

- ALT >10 fold (>400)
  - Acute viral
  - Drug/toxin
  - Ischemic/Budd Chiari
  - Autoimmune hepatitis
  - Wilson's disease

Abnormal LFT's

- Modest ALT (<300) has a wide differential
- Usually EtOH or chronic viral hepatitis
- Remember AST:ALT > 2:1 highly suggestive of EtOH
- AIH, NASH/NAFLD, Wilson's, Hemochromatosis, infiltrative/granulomatous dz

Abnormal LFT's

- Mildly high ALP or TB without evidence of biliary dz, think infiltrative (TB, sarcoid, fungal) or metastatic disease
- Workup mainly by history and risk factors
- Image or biopsy for diagnostic purposes is not always needed

Abnormal ALP

- Hepatic
  - PBC (middle aged women)
  - PSC (IBD history)
  - Gallbladder/stone disease
  - Meds (tetracyclines, OCP's, ceftriaxone)
  - Infiltrative liver dz (sarcoid, TB, CA)
- Pregnancy
- Bone (Mets or Paget's disease)

- Albumin → Half life = 20 days
  Low in malnutrition, also in infection, burns, fluid overload, hepatic failure, cancer, nephrotic syndrome.
- Transferrin → Half-life = 10 days
  Low in protein energy malnutrition, but also affected by iron status
- Prealbumin → Half-life = 2-3 days
  Low in malnutrition, also in infections, liver failure and increased in renal failure
- CRP — Positive acute phase reactant. Helps determine whether above proteins are reduced because of inflammatory process or due to inadequate substrate, as in malnutrition.

Lab Indicators of Malnutrition in Adults

- Serum Prealbumin <15 mg/dl. Best marker for Malnutrition. See Prealbumin for interpretation and monitoring.
- Serum Albumin <3.4 mg/dl.
- Serum Transferrin <200 mg/dl.
- Total Lymphocyte Count <1500/mm3.
- Total Cholesterol <160 mg/dl.

Creatinine-height index (CHI) is a ratio of a patient's 24-hour creatinine excretion and the expected normal creatinine excretion. CHI correlates with the degree of muscle depletion.

CHI: 24 hour creatinine excretion of subject 24 hour creatinine excretion of normal child of same height
has been devised for estimating the relative muscle mass of children. Age is not considered because children of developing areas are very often retarded in height. The normal CHI is close to 1.0, both in well nourished children and in fully recovered malnourished ones, with heights ranging from 64.8 to 135 cm.

A normal urea level in the urine is 12 to 20 grams over 24 hours. Individual labs may have reference ranges that vary slightly and can be different based on sex or age. Low levels of urea in the urine may suggest: malnutrition. too little protein in the diet. kidney disease.

Table 3. Pros and cons of serum nutritional markers

Nutritional marker	Pros	Cons
Albumin	• Ease of measurement • Low cost • Reproducibility • Excellent predictor of surgical outcomes • Consistent response to interventions	• Long half-life • Decreased levels in • infection, burns, fluid overload, hepatic failure, cancer and nephrotic syndrome
Transferrin	• Shorter half-life (8–10 days) • Responds more rapidly to changes in protein status	• Influenced by several factors including liver disease, fluid status, stress and illness • Unreliable in the assessment of mild malnutrition and its response to nutritional intervention • Expensive
Prealbumin	• Half-life of prealbumin (2–3 days) is much shorter	• Levels may be increased in the setting of renal dys-

Nataloni et al. investigated the role of PAB in 45 consecutive head-injury patients admitted to the intensive care unit (ICU) and found that PAB(prealbumin) was the most sensitive serum marker for the early diagnosis of malnutrition and for assessing the appropriateness of the nutritional therapy for malnourished

TLC(Total lymphocyte count) is another popular serum marker with proposed usefulness for determining nutritional status. Levels of TLC have been shown to vary with the degree of malnutrition. Levels < 1500/ mm3 correlate well with malnutrition, and those < 900/mm3 reflect severe malnutrition [33,34]. However, a study of 161 elderly subjects reported that TLC was not a good marker of malnutrition in the elderly population. They reported that TLC was more “reflective of age rather than nutritional status”

Types of Liver Tests

- grouped by the liver function they assess
- measures of hepatobiliary cell injury
- measures of transport efficiency of organic compounds
- measures of hepatic synthetic function

Tests Reflecting Cell Injury

- Aminotransferases (ALT & AST)
- Alkaline Phosphatases
- Transpeptidases
- 5’-Nucleotidase

Tests Reflecting Cell Injury

Aminotransferases

- Aspartate aminotransferase (AST)

in cytosol and mitochondria
liver > heart > skeletal muscle > kidneys > brain > pancreas > lungs > WBCs > RBCs

- Alanine aminotransferase (ALT)

in cytosol
predominantly liver
more sensitive and specific than AST

Tests Reflecting Cell Injury

Aminotransferases

- Elevated in nearly all liver diseases (ALT > AST)
- marked □ is usually hepatocellular disease

- Levels may/may not reflect extent of damage

- Do not correlate with eventual outcome

- Usually <500 in obstructive jaundice

- Usually parallel each other

AST > ALT with EtOH, fulminant, and pregnancy

Tests Reflecting Cell Injury

Alkaline Phosphatase

- Elevation may be due to induction of enzyme synthesis rather than inability of liver to secrete it into the bile

- Increases seen with cell injury or obstruction

slight to moderate (1-2x) – usually hepatocellular

large increases (3-10x) – obstruction or cholestasis

Tests Reflecting Cell Injury

- Alkaline Phosphatase
  - isolated elevations
    - infiltrative disease – tumor, abscess, granuloma, amyloid
  - Non-liver causes of elevations:
    - bone disease » diabetes
    - chronic renal failure » intestinal disease
    - renal cancer » genetic (pseudoelevation)
    - pregnancy » osteitis deformans
    - sepsis (esp. GNRs) » multiple bone fractures
    - Hodgkin's disease » intraabdominal infections
    - hypothyroidism » pernicious anemia
    - congenital hypophosphatasia » zinc deficiency

Tests Measuring Transport Efficiency

Types of Bilirubin

Direct Bilirubin	Indirect Bilirubin
conjugated	unconjugated
water soluble	lipid soluble
polar non-polar	
seen in urine	not in urine

Tests Measuring Synthetic Function

- Prothrombin Time (PT)
- Albumin

- The liver is the only source of albumin and the prothrombin group of clotting factors

Tests Measuring Synthetic Function

Prothrombin Time (PT)
sick liver can't make clotting factors
factors 2, 5, 7, 9, 10 (made only in the liver)

prolonged PT reflects failure of liver synthesis

Other causes of prolongation:
congenital deficiencies
consumptive coagulopathies (i.e., DIC)
drugs (i.e., warfarin)
vitamin K deficiency (i.e., dietary, ⍰ bile output)

The Approach

- <u>Hepatocellular Injury</u>
  - mainly ↑ AST & ALT +/- ↑ AP, GGT, bilirubin
  - ≥ 2 enzyme elevations → high likelihood of liver dz
  - guides:
    - *Mild* (<3 x normal)
      - fatty liver, EtOH, chronic hepatitis
    - *Moderate* (2-10 x normal)
      - EtOH, chronic hepatitis, cirrhosis, neoplasm, gallstones
    - *Severe* (>10x normal; usually >1,000)
      - ischemic, viral, toxic (e.g., acetaminophen, herbs)

The liver is your body's 'factory' carrying out hundreds of jobs that are essential to life.

- **Produces bile to help break down food in the gut**
- **Processes food once it has been digested**
- **Stores carboyhdrates, fat, vitamins and minerals, including iron**
- **Controls energy balance l Filters and removes chemicals, toxins and drugs from the blood**
- **Produces proteins such as albumin and clotting factors**
- **Activates a number of other important processes such as water balance and hormones**

Basic Metabolic Panel
Charting Shorthand

BMP	Na	Cl	BUN	glucose
	K+	CO2	Creatinine	

- Serum albumin is synthesized in the liver, has the longest half-life at 18 to 20 days.
- Indicator of dietary intake during the preceding three weeks.
- Low serum albumin (<2.2 g/dL) is a marker of a negative catabolic state, and a predictor of poor outcome.
- Serum albumin is not a good nutritional marker in the setting of disorders causing large protein losses from the circulation, such as ascites, protein losing enteropathy, proteinuria, liver disease, or extensive burns and inflammation. Serum albumin concentration gradually returns to normal after initiation of nutritional therapy, but this may take up to three weeks.

Laboratory Values and Hydration: BUN

Lab Test	Hypo-volemia	Hyper-volemia	Other factors influencing result
BUN Normal: 10-20 mg/dl	Increases	Decreases	Low: inadequate dietary protein, severe liver failure High: prerenal failure; excessive protein intake, GI bleeding, catabolic state; glucocorticoid therapy Creatinine will also rise in severe hypovolemia

Laboratory Values and Hydration Status: BUN:Creatinine Ratio

Lab Test	Hypo-volemia	Hyper-volemia	Other factors influencing result
BUN: creatinine ratio Normal: 10-15:1	Increases	Decreases	Low: inadequate dietary protein, severe liver failure High: prerenal failure; excessive protein intake, GI bleeding, catabolic state; glucocorticoid therapy

aboratory Values and Hydration: Alb, a+

b Test	Hypo-volemia	Hyper-volemia	Other factors influencing result
rum oumin	↑	↓	Low: malnutrition; acute phase response, liver failure High: rare except in hemoconcentration
rum dium	Typical-ly ↑ can be normal or ↓	↓, normal or ↑	Serum sodium generally reflects fluid status and not sodium balance

Visceral Proteins: Serum Albumin

- Reference range: 3.5-5.2 g/dl
- Abundant in serum, stable (half-life 3 weeks)
- Preserved in the presence of starvation (marasmus)
- Negative acute phase reactant (declines with the inflammatory process)
- Large extravascular pool (leaves and returns to the circulation, making levels difficult to interpret)
- Therefore, albumin is a mediocre indicator of nutritional status, but a very good predictor of morbidity and mortality

Urinary Creatinine

- Formed from creatine, produced in muscle tissue
- The body's muscle protein pool is directly proportional to creatinine excretion
- Skeletal muscle mass (kg) = 4.1 = 18.9 x 24-hour creatinine excretion (g/day)
- Confounded by meat in diet
- Requires 24-hour urine collection, which is difficult