Liver Cirrhosis

October 15, 2003
Case

**Patient:** 70 y.o. female from home with history of Alcoholic liver cirrhosis

**C.C:** Diarrhea

**HPI**
- 4 day history of diarrhea
- Melena stools
- GU symptoms: dysuria, polyuria and urgency
- Nausea and Vomiting
- No Fever and no Chills
- No complaints of SOB, chest pain. She has one cardiac risk factor, she was a smoker for 15 years.
- Recently admitted to the hospital for acute on chronic renal failure (creatinine 500), improved with hydration.
Past Medical History
- Liver cirrhosis
- Chronic Renal failure
- Osteoarthritis

Medications:
Pantaloc

Social History
- Drinks Alcohol- 26oz of liquor/week for the past 20 years.
- Smoker- ½ pack/day for 15 years

ROS:
- ↓ energy, anorexia and sleep disturbance

Physical Exam
- 2+ pitting edema on the ankles. JVP = 4cm
- Abdomen is distended, ++ ascites, flank dullness and fluid waves
- Splenomegaly
- Palmar erythema, spider nevi present on her thorax
Investigations:
- ↑ INR
- ↑ Crt, ↑ Bili tot
- Endoscopy and colonscopy were unremarkable

Impression:
- 70 year old woman with alcohol induced liver cirrhosis presents with symptoms and sign of UTI and melena stool.

Complications:
- Acute Renal failure- dialyses
- Thrombocytopenia and anemia
LIVER CIRRHOSIS

• CAUSES

I. TOXIN MEDIATED

• Alcohol- # 1 cause in N. America
• Aflatoxin
• Drugs – Methotrexate, Amiodarone
II. INFECTIONS

• Viral Hepatitis- Hepatitis B, C, D, EBV
• Schistomiasis
III. VASCULAR

- Budd Chiari
- Veno-occlusive Disease
- Portal Vein Thrombosis
IV. INHERITED METABOLIC DISORDERS

- Alpha 1 antitrypsin deficiency
- Wilson’s Disease
- Hereditary hemochromatosis
- Cystic Fibrosis
- Others- Glycogen storage disorders, Tyrosinemia, Porphyrias,
V. PRIMARY BILIARY CIRRHOSIS

VI. AUTOIMMUNE HEPATITIS
SPECTRUM OF ALCOHOLIC LIVER DISEASE

ALCOHOLIC LIVER DISEASE

STEATOSIS  HEPATITIS  FIBROSIS  CIRRHOSIS
Progression in Alcoholic Liver Disease

Normal liver 

90 to 100 percent 

Fatty liver 

10 to 35 percent 

Alcoholic hepatitis 

8 to 20 percent 

Cirrhosis

? 40 percent

HEPATIC METABOLISM OF ETHANOL

- Ethanol is mainly metabolised in the liver
- Oxidised by 3 enzyme systems
  - Acetaldehyde dehydrogenase (ADH)
  - Cyt P450
  - Catalase-least impt
PATHOGENESIS

1. Redox alterations

- ADH mediated Ethol oxidation leads to reduction of oxidized (NAD+) to NADH

- Inc. NADH shifts redox state of hepatocytes which affects other NAD+ dependent processes incl. Lipid & CHO metabolism leading to:
Hepatic steatosis-(Inc. NADH provoke steatosis by stimulating fatty acid synthesis & inhibiting mitochondrial beta oxidation. Fatty acids accumulate in hepatocytes & are stored as TG’s.

Mallory bodies in alcoholic hepatitis High power view of a liver biopsy in alcoholic hepatitis shows macrovesicular fat and Mallory bodies (arrows) which are eosinophilic accumulations of intracellular material. Similar changes can occur in nonalcoholic steatohepatitis. Courtesy of Robert Odze, MD.
PATHOGENESIS

2. oxidant stresses

a. Ethol oxidatn leads to formation of free radical species – hydroxyethyl, super oxide, & hydroxyl radical – which inflict oxidative damage to intracellular compounds
i. attack unsat. lipids – lipid peroxidation – tissue damage & fibrosis
ii. attack DNA – deletion & mutations – mitochondrial dysfunction

b. Dec. antioxidant defenses – Ch. Etholism leads to:
   i. dec. amts of Vits A & E – which inc. hepatic lipid peroxidation 7 causes lysosomal damage
ii. dec. glutathione
PATHOGENESIS

3. Inflammatory cell infiltration & activation

1. Kupffer cell activation & cytokine production—eg TNF, IL 1, IL 6, IL 8—causing oxidative injury

2. Immune response to altered hepatocellular proteins (caused by oxidative injury) leading to formation of ABs
PATHOGENESIS

4. Centrilobular Hypoxia

• Due to inc. O2 demand for ethanol metabolism, a zone of hypoxia around central veins, which is the farthest from oxygenated blood, develops.
MECHANISM OF FIBROSIS

• Irreversible consequence of ethanol abuse.
• Occurs in 10-15% of alcoholics
• There is activation of hepatic stellate cells which reside in Disse’s space (b/w hepatocyte & sinusoidal endothelial cell)
• In normal liver, function of stellate cell appear to be storage of Vit A
• In liver injury (as explained before), they are altered & become proliferative, myofibroblast like cells, producing collagen.
Phenotypic features of hepatic stellate cell activation during liver injury and resolution

Following liver injury, hepatic stellate cells undergo "activation," which connotes a transition from quiescent vitamin A-rich cells into proliferative, fibrogenic and contractile myofibroblasts. The major phenotypic changes after activation include proliferation, contractility, fibrogenesis, matrix degradation, chemotaxis, retinoid loss, and white blood cell chemoattraction. Key mediators underlying these effects are shown. The fate of activated stellate cells during resolution of liver injury is uncertain but may include reversion to a quiescent phenotype and/or selective clearance by apoptosis. Courtesy of Scott L Friedman, MD.
Co-factors in the development of liver disease

1. Heritable factors: rate of ethanol metabolism
2. Gender: women are more susceptible to ethanol induced liver damage - unexplained - postulated theory – gender difference in fatty acid metabolism
3. Diet & Nutrition: both under & over nutrition are risk factors for development of liver disease
4. Co-existent viral hepatitis: 18-25% of ethanolics are infected with Hep C
LIVER FUNCTIONS

Metabolic—glucose homeostasis, metabolizes ammonium to urea

Synthetic—albumin, coagulation factors complement proteins

Storage—glycogen, vitamins(ADEK and B12)

Catabolic—hormones, detoxification

Excretory—bile salts
Complications

Portal Hypertension and Hepatocellular dysfunction which may result in:

- Variceal bleeding
- Ascites
- Hepatic encephalopathy
- Hepatorenal syndrome
- Hepatocellular carcinoma
Portal Hypertension

- Abnormally ↑ pressure in the portal vein and tributaries
- Normal pressure=4-8 mmHg
- >12mmHg
- Consequence of PH:
  - Disorders in the preceding organs: Spleen (anemia and thrombocytopenia), Bowel (malabsorption).
  - Use of collateral vessels that are normally thin-walled
Portal hypertension

Portosystemic collateral routes:

1. Esophageal- submucosal veins of the esophagus which communicate with azygous vein
2. Paraumbilical- with anterior abdominal wall
3. Hemorrhoidal- iliac system
4. Retroperitoneal- vena cava
Esophageal varices

- Enlarged esophageal veins, when gradient >12mmHg can rupture
- Lead to massive bleeding: combination of thrombocytopenia and deficiency in clotting factors
- Mortality rate of 30%-60%
Ascitis

- Accumulation of excess fluid in the peritoneal cavity
- Causes:
  1. Hypoalbuminemia
  2. Portal Hypertension
  3. Vasodilation: opening of portosystemic collaterals decreases SVR, ↑ level of vasodilators (NO, glucagon, substance P, prostacyclins)

Complications: Spontaneous bacterial peritonitis (due to bacterial overgrowth and translocation of bacteria to mesenteric lymphnodes and then bacteremia then to the liver and then enters ascitic fluid.)
Hepatorenal Syndrome

• Functional renal failure
• 4% of patients, in patients with significant hepatic synthetic dysfunction and severe ascitis
• Cause: ↓ in systemic vascular resistance leads to decrease renal perfusion, decrease in GFR, decrease in Na excretion eventually lead to progressive renal ischemia
Hepatic Encephalopathy

• Spectrum of potentially reversible neuropsychiatric abnormalities due to altered brain function (personality, memory gaps to coma)
• May reflect either reversible encephalopathy, brain atrophy, brain edema, or combination

Cause:

• **Ammonia** - decreased metabolism of ammonia to urea and bacterial hydrolysis of Nit compounds in the gut, interferes with brain function by causing: brain edema, glutamate release and inhibits postsynaptic potentials
• **Toxic brain substances** (amines, phenols and short-chain fatty acids) bypass the liver in portal hypertension and interfere with synthesis of neurotransmitters
• **Low glucose** - due to impaired hepatic gluconeogenesis alters brain energy metabolism
Cirrhosis Physical Findings
Cirrhosis Physical Findings

- General Appearance
  - Cachexia
  - Proximal muscle wasting
  - Ascites
  - Jaundice
Cirrhosis Physical Findings

• Hands and Arms
  – Clubbing
  – Terry’s nails
  – Dupuytren’s contracture
  – Palmar erythema
  – Anemia
  – Asterixis
  – Eccymoses
  – Petechiae
Dupuytren’s contracture  Nodular fibrosing lesions with bands radiating distally are features of Dupuytren’s contracture. The ulnar side of the hand is affected, with the 4th and 5th fingers usually involved first. (By permission from Shear, RP, Moskowitz, RW, Goldberg, VM. Soft Tissue Rheumatic Pain: Recognition, Management, Prevention, 3rd ed, Williams & Wilkins, Baltimore 1996.)
Clubbing of the fingers: In a normal finger, the length of the perpendicular dropped from point A to point B should be greater than a similar line from C to D. In clubbing, the relationships are reversed — that is, the distance C-D is greater than the distance A-B. The other important change is the angle described by A-C-E. In the normal finger this is usually <150 degrees whereas in clubbing it is >150 degrees. Redrawn from DeRemee, RA. Facets of the algorithmic synthesis. In DeRemee, RA, (Ed), Clinical profiles of diffuse interstitial pulmonary disease. Mount Kisco, NY, Futura Publishing Company, Inc, 1950, pp. 3-44.
Cirrhosis Physical Findings

- Head and Chest
  - Jaundice (frenulum, scleral icterus)
  - Parotid hypertrophy
  - Kaysher Fleischer rings (Wilson’s)
  - Fetor Hepaticus
  - Spider angiomata
  - Gynecomastia (male)
  - Loss of chest or axillary hair (male)
Cirrhosis Physical Findings

- Abdomen and Pelvis
  - Caput medusa or prominent abdo veins
  - Ascites: bulging flanks, flank dullness, shifting dullness, fluid wave
  - Cruveilhier-Baumgarten murmur
  - Splenomegaly
  - Liver size??
  - Testicular atrophy
## Ascites

*JAMA, May 20, 1992*

<table>
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<tr>
<th>PHYSICAL SIGN</th>
<th>LR+</th>
<th>LR-</th>
<th>Sens.</th>
<th>Spec.</th>
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<tr>
<td>Bulging Flanks</td>
<td>2.0</td>
<td>0.3</td>
<td>0.81</td>
<td>0.59</td>
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<tr>
<td>Flank Dullness</td>
<td>2.0</td>
<td>0.3</td>
<td>0.84</td>
<td>0.59</td>
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<tr>
<td>Shifting Dullness</td>
<td>2.7</td>
<td>0.3</td>
<td>0.77</td>
<td>0.72</td>
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<tr>
<td>Fluid wave</td>
<td>6.0</td>
<td>0.4</td>
<td>0.62</td>
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# Liver Cirrhosis Prognosis

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<tr>
<td>Bilirubin (umol/L)</td>
<td>&lt; 34</td>
<td>35-51</td>
<td>&gt;51</td>
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<td>Albumin (g/L)</td>
<td>&gt;35</td>
<td>28-35</td>
<td>&lt;28</td>
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<td>INR</td>
<td>&lt;1.7</td>
<td>1.71-2.24</td>
<td>&gt;2.25</td>
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<td>Encephalopathy</td>
<td>none</td>
<td>slight</td>
<td>mod-severe</td>
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<tr>
<td>Ascites</td>
<td>absent</td>
<td>slight</td>
<td>moderate</td>
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<th>Survival</th>
<th>1yr (%)</th>
<th>2yr (%)</th>
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<td>A (5-6)</td>
<td>100</td>
<td>85</td>
</tr>
<tr>
<td>B (7-9)</td>
<td>80</td>
<td>60</td>
</tr>
<tr>
<td>C (10-15)</td>
<td>45</td>
<td>35</td>
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</table>
• Major goals of treatment:
  – Slowing or reversing the progression of disease
  – Preventing superimposed insults to the liver
  – Preventing and treating the complications
  – Determining the appropriateness and timing for liver transplantation
Initial therapy of Ascites

• Dietary Na restriction:
  – An 88 meq (2000 mg/day) Na diet is the most practical level of Na restriction.
  – This diet will be effective alone only in the small subset of pts whose urinary Na excretion is >78 meq/day (10 meq of non urinary loses)
  – Urine excretion of Na< dietary intake will accumulate Na and water, causing p.edema and ascites

• The common approach: Diuretics+Na restriction to all pts with cirrhosis and clinically detectable ascites.
Diuretic therapy

• The max rate of fluid removal is 300-500 ml/day, more rapid rate:
  – Causes plasma volume depletion and azotemia
  – Hypokalemia should be avoided

• Spironolactone: more effective than furosemide alone and no hypokalemia
  – Painful gynecomastia
  – Amiloride is less effective

• Best regimen:
  – Single am dose of spironolactone and furosemide 100 and 40mg respectively.
  – Dose can be doubled if no clinical response.
Diuretic Resistance Ascites

- Exclude lack of compliance with diet Na
- 24h urine Na excretion > 78 meq/day
- Random 24h Na/k ratio > 1
- On max tolerable dose of diuretics
- Diuretic related complications:
  - Lyle imbalance, azotemia, H encephalopathy
Therapeutic options

• Paracentesis:
  – Serial large volume paracentesis (LVP)
    • Pts on 88 meq/d Na and no Na excretion in urine require paracentesis of almost 8.4 liter q 2 weeks.
    • Pts with some Na excretion require less fluid removal
    • Single therapeutic para in pts with tense Ascitis and serial LVP when become diuretic resistant.
• Colloid replacement:
  – In one study, pts with ascites undergoing LVP, assigned to get Albumin (10/L of ascites removed) and no Albumin:
    • No albumin group showed more signs of HD deterioration and increase in renin plasma activity.
    • Also worsening renal function, hyponatremia
  – Albumin, dextran 70 or polygelin used
  – Postpara circulatory dysfunction less common with albumin administration.
• Trasjugular intrahepatic portosystemic shunt (TIPS)
  – A side to side portocaval shunt, through int jugular v under local anesthesia
  – Studies showed:
    • Increase in u/o, marked reduction in ascites, cessation of diuretic tx or much lower dose in about 75% of pts.
    • Higher survival without the need for transplant
    • No significant reduction in hepatic encephalopathy
    • Early thrombosis or delayed shunt stenosis
Continued..

- At present, it’s avoided in pts with child C class or high MELD score or severe spont H encephalopathy.

- Peritoneovenous shunt:
  - Drains into int jugular v, reinfuses ascites into vascular space.
  - Virtually abandoned due to excessive rate of complications:
    - DIC, shunt infection, bacteremia, SBO
• Surgical portosystemic shunt:
  – Significantly reduce HVPG and development of ascites and frequency of SBP
  – Similar mortality rate compare to TIPS
  – Lower rate of prosthesis occlusion
• Could have a role in tx of diuretic resistance ascites, in future.
Hepatic Encephalopathy

- HE, acute or chronic is reversible and a precipitating cause rather than worsening of hepatocellular function identified in majority of cases.
  - Such as GI bleed, high pr intake, hypokalemic alkallosis, infection and constipation
• Correction of hypokelemia:
  – Increases renal ammonia production
• Removing source of ammonia from GI tract
  – NG lavage in pts with UGI bleed
  – Limit pr intake
  – Treat constipation
• Lactulose and lactitol:
  – Enters the colon, catabolized by bacterial flora to FA and lowers the colonic PH to 5.0 which favors the formation of nonabsorbable NH4+ from NH3
Continued..

- Lactulose 45-90g/d
- 70-80% of pts with HE improve on this Tx
- Number of trials showed lactitol as effective as lactulose and fewer side effects

• Enemas and diet pr reduction (0.8g/kg/day)

• Oral antibiotics:
  - Neomycin: (trial) at dose of 6mg/d compare to placebo, reported no difference in outcome
  - In addition, neomycin related to oto and nephrotoxicity
  - Metronidazole, vancomycin, rifximim
    • Been found effective in some clinical trials
    • Better tolerated than neomycin
• Modification of colonic flora:
  – Increase the number of saccharolytic bacteria
    • Enterococcus faecium sf68
    • As effective as lactulose to lower blood ammonia
  – Ornithine-aspartate:
    • Ammonia removal by stimulation of glutamine synthesis in hepatocytes
  – Sodium benzoate:
    • Increase nitrogen excretion in urine
    • 5mg twice daily, similar result as lactulose in HE tx
• Recommendation:
  – Start therapy with lactulose and lactitol (70-80% respond)
  – Those with no improvement after 48h, ornithin-aspartate infusion (20g over 4 h) or oral sodium benzoate (5mg/bid)
  – Oral AB considered 2nd line in non responders to disaccharidases.
Spontaneous bacterial peritonitis

• In pts with fever, abdo pain, tenderness, or altered mental status, start tx after ascitic fluid, blood, and urine obtained for analysis and culture.

• In pts without these findings, wait until result of PMN count is available:
  – PMN>250 in a compatible clinical setting, start empiric AB therapy.
Choice of Antibiotic

• Most SBP’s due to gut bacteria, E.coli, klebsiella, however, strep and infrequently staph infections can also occur
  – So, a broad spectrum AB, cefotaxim or similar 3rd generation
  – Trial: comparing cefotaxim with AMP/GENT
    • Higher resolution rate, no nephrotoxicity and no superinfection with cefotaxim
  – Cefotaim 2g iv q8h with excellent ascitic fluid level
  – Main s.e is rash(1%)

• Emergence of resistant infection, esp when a quinolone used for SBP prophylaxis.
  • Gram positive cocci found in most of the cultures
Continued…

- Suspected bacterial peritonitis:
  - Broader coverage cefotaxim + metronidazole
- Possible oral tx:
  - Ofloxacin 400mg/bid or parenteral cefotaxim or iv cipro
    200mg iv 2 days followed by 500mg po bid 5d
- Renal failure:
  - Happens in 30-40% and is a major cause of death in SBP
  - Prevented by iv Albumin to expand plasma volume and octerotide to inhibit endogenous vasodilator release
Duration of therapy

- Studies document the efficacy of short course tx (5 day and 10 day course of cefotaxim showed similar outcomes)
  - Treat for 5 days then R/A the pt:
    - Is usual dramatic response d/c AB
    - If fever or pain persists repeat paracentesis:
      - If PMN<250 stop tx
      - PMN> pretreatment value search for surgical source
      - PMN elevated but < pretreatment value, AB continue for another 48h and repeat paracentesis.
• Intermittent antimicrobial tx to prevent infection:
  – A single weekly dose of 750 mg of cipro
  – 1 ds tab of septra 5 days/week
  – Norfloxacin only if ascitic fluid pr<1g/dl
Hepatorenal Syndrome

• Acute renal failure in patient with advanced liver disease
• First suggested association between oliguric RF and cirrhosis in 1863
• Type I more serious, 50% lowering of CRCl to <20ml/min in less than 2 week or at least 2x increase of Cr
• Type II less severe, ascites resistant to diuretics
Epidemiology


- 234 patients with cirrhosis and ascites
- probability of hepatorenal syndrome 18% at 1 year and 39% at 5 years
- 16 variables had predictive value for hepatorenal syndrome
Epidemiology (2)

- history of ascites, absence of hepatomegaly
- nutritional status
- BUN/Cr
- serum Na <133, serum K >4
- serum osmolality <279, urine osmolality >553
- urinary Na excretion <2 meq/day
- GFR <80 ml/L
- MAP <85 mmHg
- plasma renin activity >3.5
- plasma norepinephrine concentration >544 pg/ml
- esophageal varices
Epidemiology (3)

- neither etiology nor the Child-Pugh score had predictive value
- A multivariate analysis disclosed 3 independent predictors of hepatorenal syndrome:
  - low serum Na concentration, <133
  - high plasma renin activity, <3.5
  - absence of hepatomegaly
- Poor prognosis, median survival of 1.7 weeks
Pathophysiology

• Functional problem
• Splanchnic dilatation, may be due to nitric oxide
• Decline in renal perfusion, decrease effective circulating volume, decrease in GFR, altered RAAS
• Intense renal vasoconstriction
• Theory of increase in ratio of vasoconstrictor thromboxanes to vasodilator prostaglandins, playing role in progressive renal ischemia,
• Increase in endothelin promote renal ischemia


Renal Function

- Difficulty in accurate assessment
- Reduced urea and creatinine production
- Decreased muscle mass, decreased nutrition
- Liver dysfunction
- Intense Na reabsorption increases passive urea reabsorption
Clinical Presentation

- Usually no obvious precipitant
- Hypotensive
- Oliguria
- Benign urine sediment
- Low rate of Na excretion, almost no urinary Na
- Increasing creatinine
Diagnosis

- Cr >133 umol/L progressing over days to weeks in px with severe liver disease with portal hypertension
- Oliguria, <500cc/d
- Absence of other cause
- Absence of proteinuria, bland urinalysis
- Urine sodium <10meq/L
- urine osmolality > plasma osmolality
- No improvement in renal function after discontinuation of diuretics and rehydration
Ddx: Acute Tubular Necrosis

- ppt’s such as antibiotics, radiocontrast, sepsis, bleeding
- But difficult b/c prolonged renal ischemia in hepatorenal syndrome can lead to ATN
- Also difficult b/c fractional excretion of sodium may remain <2% in cirrhosis with persistent renal ischemia induced by liver disease
- Also granular and epithelial cell casts may be seen with marked hyperbilirubinemia alone
Ddx: prerenal disease

- Hepatorenal syndrome is a prerenal disease
- Other causes of prerenal dx in cirrhosis (GI loss, diuretics, NSAIDS)
- Diagnosis of hepatorenal syndrome requires lack of improvement in renal function after discontinuation of nephrotoxic agents, and rehydration
### HRS vs. Prerenal vs. ATN

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<th>HRS</th>
<th>Prerenal</th>
<th>ATN</th>
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<tr>
<td>Ur sediment</td>
<td>-</td>
<td>-</td>
<td>Heme granular casts</td>
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<tr>
<td>Ur Na</td>
<td>&lt;10-20</td>
<td>&lt;10-20</td>
<td>&gt;30</td>
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<tr>
<td>Proteinuria</td>
<td>-</td>
<td>-</td>
<td>+/-</td>
</tr>
<tr>
<td>FeNa</td>
<td>&lt;1%</td>
<td>&lt;1%</td>
<td>&gt;2%</td>
</tr>
<tr>
<td>Ur osmols</td>
<td>At least 100&gt;plasma</td>
<td>At least 100&gt;plasma</td>
<td>Same as plasma</td>
</tr>
<tr>
<td>Respond to fluid</td>
<td>-</td>
<td>Yes</td>
<td>-</td>
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Treatment

• Improve hepatic function
• Rehydrate, remove nephrotoxic agents
• Ornipressin, terlipressin (ADH analogs)
• Misoprostol (PGE analog)
• N-acetylcysteine (antioxidant)
• Midodrine (systemic vasoconstrictor)
• Octreotide (inhibitor of vasodilator release)
Unclear benefits vs. risks

- Ornipressin for splanchnic vasoconstriction increases GFR but induce renal ischemia
- Terlipressin with albumin reduced Cr but unsure about ischemic complications
- Conflicting evidence for misoprostol
- N-acetylcysteine, some evidence (uncontrolled study, rat study, models) for increasing CrCl, Ur volume, Na excretion

Octreotide and midodrine

- Controlled trial of 13 patients
- 5 px received midodrine and octreotide vs. 8 patients received dopamine
- Both received daily albumin
- Midodrine (7.5-12.5 mg tid) and octreotide (100 to 200ug sc. tid) group had significant improvements in renal function, lower Cr, increase GFR, increased urine volume
- Dopamine group had trend toward deterioration
- Minimal s/e such as tingling, goosebumps, diarrhea

Transjugular intrahepatic portosystemic shunt

- Some short term benefit
- In one study average survival following TIP was 5 months
- Increase risk of encephalopathy
- bridge to transplant?
Dialysis

- Px’s condition limited by liver failure
- Use in short term if reversible hepatic cause
- Bridge to transplant?
- Px’s morbidity not improved
Liver Transplant

- Only long term treatment
- No significant survival difference b/t HRS and non-HRS px post transplant (90 day)
- HRS px had lower GFR post transplant c/w non-HRS
- One- and 2-year actuarial survival rates in the non-HRS patients were 87.2% and 82.1%, respectively
- actuarial 1- and 2-year survival rate for the HRS patients was 76.6% (P = NS)
- 10% HRS patients developed ESRD posttransplant compared with 0.8% of non-HRS patients
Liver transplant (2)

- HRS patients can safely undergo OLTX with acceptable perioperative mortality and good long-term survival
- Most HRS patients have return of acceptable renal function
