A CASE OF LIVER CIRRHOSIS & HEPATIC ENCEPHALOPATHY
• Mr N.N. 56 yr old male.
• Admitted on 22/03/02.
• 1 month Hx of abdominal distention, confusion, inability to concentrate and dyspnoea Grade 111.
• **Pmx:** Diagnosed with alcohol-induced liver cirrhosis in Jan 2000 and defaulted Rx since August 2000.
• **Social Hx:** Excessive alcohol use for approx. 20 yrs.
On Examination

- Bp 130/70  Pulse 80
- Apyrexial
- Jaundice, Clubbing, Foetor hepaticus present.
- No circulatory changes - palmer erythema, spider telangiectasia, cyanosis.
- No endocrine changes - gynecomastia, testicular atrophy.
- No bleeding tendency- bruises, purpura, epistaxis.
ABDOMEN:
• abdominal distention.
• marked ascites present.
• unable to detect visceromegaly.

CVS:
• N S1 + S2
• Not in CCF.

CHEST:
• bibasal creps.

NEURO:
• Apathetic, confused, disorientated, drowsy, slurred speech.
• Flapping tremor present.
INVESTIGATIONS

• FBC: Hb11.7L/Hct32.1L/ MCV104.9H/MCH38.1
• PLT263/WBC7.4/
• I.N.R. 2.2
• U&E: 125/4.8/97/20/8.8/257
• GLUCOSE:3.9
• LFT: ALP220/TBIL65/TP80/ALB25L/GGT137H/ALT21
• Ascitic tap: results unavailable
• Liver Bx not done.
DIAGNOSIS
1. Liver cirrhosis.

COMPLICATIONS
1. Hepatic encephalopathy (grade 3).
3. Coagulation defect.
TREATMENT

- Furosemide 80mg tds ivi.
- Aldactone 100mg bd po.
- Thiamine 100mg dly po.
- Vit B12 1000µg dly imi.
- Pregamal 1 b.d po
- Vit K 10mg dly imi.
- Lactulose 30ml bd po.
- Flagyl 400mg tds po.

Despite the above mentioned treatment the patient demised on the 25/03/02
ALCOHOLIC CIRRHOSIS

• Alcoholic cirrhosis is only one of many consequences resulting from chronic alcohol ingestion and often accompanies other forms of alcohol-induced liver injury, including alcoholic fatty liver and alcoholic hepatitis.

Etiology

• Quantity and duration of alcohol intake are the most important risk factors involved in the development of alcoholic liver disease.

• Threshold for developing severe alcoholic liver disease in men is an intake of 60-80g/d of alcohol for 10 yrs; women by consuming 20-40g/day.
• Concomitant chronic Hepatitis C virus infection accelerates development of alcoholic cirrhosis.

Pathology
• This is usually, initially micronodular often with active inflammation in fibrous septae and marked peri-cellular fibrosis, and later becomes macronodular.

Investigations
• Biochemical investigations are used to assess the severity and activity of the disease.
• Plasma transaminases reflect the activity of the disease which is usually low in established cirrhosis.
• Plasma ALP reflects the severity of cholestasis and is highest in biliary forms of cirrhosis.
• Plasma bilirubin, albumin, the Vit K corrected prothrombin reflect the severity of liver damage.
• Imaging is required for assessing the structural effects of the disease.
• Liver biopsy establishes the diagnosis of cirrhosis.

STAGING OF LIVER CIRRHOSIS
• A reliable staging system is the modified Child- Pugh classification with a scoring system of 5-15.
• Class A : 5-6
• Class B : 7-9
• Class C : 10-15
• It is used to assess prognosis in cirrhosis and provides the standard criteria for liver transplantation (class B).
# CHILD-PUGH CLASSIFICATION OF CIRRHOSIS

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum bilirubin (µmol/L)</td>
<td>&lt;34</td>
<td>34-51</td>
<td>&gt;51</td>
</tr>
<tr>
<td>Serum albumin (g/L)</td>
<td>&gt;35</td>
<td>30-35</td>
<td>&lt;30</td>
</tr>
<tr>
<td>Ascites</td>
<td>none</td>
<td>Easily controlled</td>
<td>Poorly controlled</td>
</tr>
<tr>
<td>Neurologic disorder</td>
<td>none</td>
<td>minimal</td>
<td>Advanced coma</td>
</tr>
<tr>
<td>Prothrombin time (INR)</td>
<td>0-4</td>
<td>4-6</td>
<td>&gt;6</td>
</tr>
</tbody>
</table>

<1.7 1.7-2.3 >2.3
COMPLICATIONS

• Portal Hypertension
• Ascites
• Hepatic encephalopathy
• Renal failure
• Infection
• Hepatocellular carcinoma

MANAGEMENT

• No treatment can reverse cirrhosis or prevent further progression.
• Medical therapy can promote improved general health and alleviate symptoms.
• Specific treatment is directed at particular complications.

• Ascites: Restriction of Sodium intake.
  
  Spironolactone (100-400 mg/d) is the drug of choice for long-term therapy because it is a powerful aldosterone antagonist.

  Paracentesis of 3-5L over 1-2 hrs.

• Nutrition - In the absence of hepatic encephalopathy or ascites, a high energy, protein-rich diet should be advised with strict avoidance of alcohol.
Hepatic encephalopathy is a complex neuropsychiatric syndrome characterized by disturbances in consciousness and behaviour, personality changes, fluctuating neurologic signs, asterixis and distinctive EEG changes.

- Encephalopathy may be acute and reversible or chronic and progressive.
- In severe cases, irreversible coma and death may occur.
Pathogenesis

- The most important factors in the pathogenesis are severe hepatocellular dysfunction and/or intrahepatic and extrahepatic shunting of blood into the systemic circulation so that the liver is largely bypassed.

- Various toxic substances absorbed by the intestine are not detoxified by the liver and lead to metabolic abnormalities in the CNS.

- Ammonia is the substance most often incriminated in the pathogenesis of encephalopathy. Others include mercaptans, short-chain fatty acids and phenols.
Clinical Stages of Hepatic Encephalopathy

<table>
<thead>
<tr>
<th>Stages</th>
<th>Mental Status</th>
<th>Asterixis</th>
<th>EEG</th>
</tr>
</thead>
<tbody>
<tr>
<td>i</td>
<td>Euphoria or depression, mild confusion, slurred speech, disordered sleep</td>
<td>+/-</td>
<td>Triphasic waves</td>
</tr>
<tr>
<td>ii</td>
<td>Lethargy, moderate confusion</td>
<td>+</td>
<td>Triphasic waves</td>
</tr>
<tr>
<td>iii</td>
<td>Marked Confusion, incoherent speech, sleeping but arousable</td>
<td>+</td>
<td>Triphasic waves</td>
</tr>
<tr>
<td>iv</td>
<td>Coma, initially responsive to noxious stimuli, later unresponsive</td>
<td>-</td>
<td>Delta activity</td>
</tr>
</tbody>
</table>
Factors precipitating hepatic encephalopathy

- Uraemia
- Drugs- sedatives, anti-depressants, hypnotics
- Git bleeding
- Excess dietary protein
- Constipation
- Paracentesis (>3-5l)
- Hypokalemia
- Infection
- Trauma
- Portasystemic shunts
**Treatment**

- Dietary protein reduced <20g/d.
- Lactulose diminishes ammonia absorption and production.
- Intestinal ammonia production by bacteria can be decreased by by neomycin (0.5-1g 6hrly).
- Metronidazole can also be used.
- Levodopa, bromocriptine and I.V. amino-acid formulations are of unproven benefit.
- Haemoperfusion and extracorporeal liver assist devices are also of unproven value.
Initial Evaluation:
- Exclude other causes of disordered mentation
- Identify precipitants and correct
- Determine electrolytes, Blood Urea Nitrogen, creatinine, NH3(optional), glucose

Protein Restriction

Laxative, e.g., Lactulose 30-120 mL, 1 to 4 times daily until 4 stools/day

Inadequate response?

Broad-spectrum antibiotics (eg., neomycin 500-100 mg qid, or metronidazole 250 mg tid)

Inadequate response?

Consider liver transplantation
THE END