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## Liver cirrhosis with homoeopathic perspective

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### Abstract

Liver cirrhosis is an emerging enigma of today's world. Even youth are not spared from this evil disease. Cirrhosis is the resultant or the last stage change in structural unit of liver and it's surrounding which eventually disrupt the harmonious processings of liver functions.

In this article an accustomed idea regarding liver and the chronicity of liver disease is mentioned along with the factual concept of homoeopathy in the all three pillar aspects which is organon, repertory as well as materia medica. The homoeopathic literature is being tried to put under the same head covering liver and it's homoeopathic approach.

**Keywords:** Liver cirrhosis, homoeopathy, miasmatic analysis, synthesis repertory, lycopodium, fatty liver, liver cell dysfunction, ALT, AST, Liver function, chelidonium

### Introduction

Fibrosis and nodule formation of the liver secondary to long-term provocation advancing into changes in it's normal lobular textural organization of the liver is known as Cirrhosis<sup>[1]</sup>. Based on the absence or presence (or previous history) of variceal bleeding, ascites, jaundice or encephalopathy chronic liver disease aka cirrhosis differentiated as compensated or decompensated type<sup>[2, 3, 4, 5]</sup>.

### General consideration

#### Hepato-cellular injury that leads to Cirrhosis via

- Diffuse liver involvement
- Bridging necrosis and fibrosis
- Nodular regrowth,

It's an irrevocable state and leading cause of mortality.

### About Our Liver

Right from blood detoxification from unwanted entities such as pathogens, chemicals like alcohol and storing iron, vitamins and glucose to destroying old RBCs, and bile secretion hence contributing in digestive mechanism, our Liver which is body's largest gland and organ, is a multitasker! Wired to carry out such critical responsibility while supporting nearly every organ of the body in various crucial aspects. Preserving the health of Liver is foremost for it to maintain the metabolism and to execute over hundreds of the functions vital for surviving a normal life.

Liver aids in production of blood clotting proteins and it absorbs fats present in our diet, vitamins A, D, E and processing everything which is in took, inhaled or rubbed on skin: liver metabolizes dynamically fulfilling the need of energy to effectively perform other functions of the body, such as regulating hormones and much more. Lifestyle habits like long-term alcohol abuse, smoking and complications like chronic or acute hepatitis infections, fatty liver disease, etc. directly attack healthy liver tissues replacing them slowly with fibrous scar tissues, thereby failing liver functioning. Liver cells can replicate and grow back from as little as 25% available liver mass, after a chemical injury or a surgical removal hence liver has wonderful capability to regenerate,. However, regeneration cannot take place when affected person is habitual of alcohol intake or have an untreated liver infection; and then takes place an excessive deposition of components of extracellular matrix, for e.g. collagens and glycoproteins etc. which leads to fibrotic scar and abnormal nodular regeneration. These abnormal nodules block the flow of blood and gradually hamper the production of all the substances including proteins.

The metabolism of toxins and drugs hormones, nutrients, etc. Liver gets severely affected as well. The slow metabolism diseased tissues continuously damage the tissues and causes Liver Cirrhosis- a medical illness, in which into that structurally abnormal nodules and fibrous scar tissues is made by conversion of a normal liver architecture.

#### Clinical features due to

1. Liver cell dysfunction
2. Porto-systemic shunting

#### Scarred Liver: How common is it?

##### 1. Fatty livers

Sedentary lifestyle, high fatty foods intake, increased tendency for smoking and consumption of alcohol are hugely prevalent in ongoing generation who are care-less about the health overall, leading India to become the "world capital of liver diseases". Individuals with uncontrolled blood sugar, obesity, etc. will develop fatty livers with excessive fat infiltration in hepatocytes (liver cells) which is usually harmless at start, but if unattended and ignored, leads to the development of silent liver disease nonalcoholic steatohepatitis (NASH). It has also been observed that NASH over time is responsible for acute liver damage, and also occurs in those individuals without any apparent risk factor, diabetes and obesity, but with normal blood cholesterol and lipid levels.

##### 2. Hepatitis wreaking havoc!

Approximately 1 million Indians are at a risk of Hepatitis B infection (HBV) annually, whereas around 100,000 deaths result from HBV infection. Hepatitis D infection is uncommon in India but is observed in around 10-20% of HBV positive patients.

Another major causal factor of liver damage spread by blood to blood contamination is Hep.C infection with a population prevalence of around 1%. Infected needles and razors, vertical transmission of viruses from infected mother to their newborns, blood transfusion and sexual transmission are the major modes of acquiring these life threatening viral hepatitis infections that damage the liver causing inflammation and persistent infections over time.

##### 3. Genetic diseases

Many inherited diseases cause interference with liver's processing, production and storage of enzymes. An autosomal recessive (codominant) disease, Alpha-1 antitrypsin deficiency is caused upon mutations in SERPINA1 gene which encodes for serine protease inhibitor AAT which is synthesized by liver cells is essential to protect lungs from proteolytic damage. Cystic fibrosis resulting upon an altered activity of cystic fibrosis transmembrane Conductance regulator CFTR chloride channel due to mutations in CFTR gene, leads to altered bile flow followed by inflammation and proliferation of liver cells leading to biliary cirrhosis.

##### 4. Other Causes

Certain diseases damaging or destroying bile ducts like primary biliary cirrhosis lead to accumulation of bile in the liver that causes inflammation and scarring of bile ducts, to an extent that they disappear, resulting in cirrhosis.

Repeated instances of heart failure with liver congestion, parasitic infections, prolonged exposure to toxic chemicals and drug reactions may also lead to scarring of the liver which worsens to cirrhosis.

#### Recognise the signs that your liver is suffering!

Fibrotic tissue buildup causing cirrhosis is an insidious process. As soon as cirrhosis progresses, fibrotic tissue slowly replace the healthy liver tissues, the liver first expands followed by shrinking upon worsening of liver cirrhosis.

Initial stages of cirrhosis are asymptomatic, at this stage the liver continues to function, but with the progression of disease and shrinking of liver occurs. Following are the manifestations:-

- Fatigue
- Itching
- Nausea and weakness
- Easy bruising and bleeding
- Loss of appetite and weight loss
- Jaundice (Yellowing of skin and eyes)
- Edema (Swelling due to fluid buildup in feet, ankles etc.)
- Bloating Or fluid buildup in abdomen
- Gallstones
- Confusion and slurred speech
- Spiderangiomas (Spider Like blood vessels on skin)

#### Classification based on histology

Divides cirrhosis:-into micronodular, macro nodular, and mixed forms.

*Micro-nodular cirrhosis*: exclusive of alcoholic liver disease - the nodules are less than 1 mm in diameter.

*Macronodular cirrhosis* larger nodules, measure several centimeters in diameter.

#### Causes of cirrhosis

- Infections- Viral Hepatitis B And C, C. Schistosomiasis
- Alcoholic & NASH
- Cryptogenic
- Metabolic- A 1- AT Deficiency, Wilson's Disease, Hemosiderosis
- Biliary Obstruction - Primary Biliary Cirrhosis, Sclerosing Cholangitis
- Venous Out Flow Obstruction- Budd Chiari Syndrome, Venous Occlusive Disease
- Drugs- A Methotrexate, «Meadron, OCA,
- Cardiac causes- CHF, constrictive Pericarditis

#### Pathophysiology <sup>[6]</sup>.

Mechanisms of CLD is underlying fibrosis of the liver. primarily responsible for hepatic fibrosis and its progression to cirrhosis is myofibroblast-like cells that encircle the sinusoids k/a hepatic stellate cells. In response to liver injury, stellate cells undergo alteration; characterized by contraction, fibro-genesis, chemotaxis and extracellular matrix degradation. Under conditions of persistent factor causing the cell injury or sinusoidal insults the behavioral reaction of stellate cells act in order to create fibrosis in the liver texture.

#### Clinical features

##### Symptoms

The onset of symptoms either Insidious or may be abrupt.

Oftenly its asymptomatic at beginning.

- Weakness
- Fatigability
- Disturbed sleep muscle cramps are common.
- Anorexia with associated nausea and vomiting sometimes.
- Abdominal pain may precipitate to hepatic impotence and loss of libido causing sterility and gynaecomastia in men.
- Presentation with complications like -
- Bloating of abdomen due to ascitis or
- Confusion, altered sleep pattern and behaviour due to encephalopathy.
- Hematemesis due to varices.

### Sign

- Weight Loss, Anaemia, Jaundice
- Temperature- Fever In About 35% Cases.
- Alopecia, Loss Of Axillary & Pubic Hair, Facial Hair
- Glossitis & Chelitis D/T Vitamin Deficiency.
- Parotid Gland Swelling
- Ecchymotic Changes.
- *Spider Naevi*: Dilated Capillaries Seen In The Body at Upper Part.
- *Palmer Erythma*: Mottled Redness on the Hypothenar & Thenar Eminences.
- Dupuytren's Contracture: Contracture on Palmer Fascia.
- Nail Changes: White Nails.
- Clubbing Of Fingers,
- Peripheral Edema
- Gynecomastia

### Investigations

#### Abdominal examination

Hepatomegaly in 70% cases firm, nodular with sharp edges. Left lobe may be prominent.

Splenomegaly: in 30% cases - 50% cases.

Caput medusa: dilated superficial veins of anterior abdominal wall around umbilicus.

Ascites: in most of the cases

Chest examination:- pleural effusion m/b present.

CNS examination:- evidence of encephalopathy eg. Confusion, asterixis.

#### Laboratory findings

1. Anemia: mostly folate deficiency due to alcoholism, hemolysis due to hypersplenism. Occult or overt blood logs.
2. WBC decreased. Platelet decreased.

#### 3. Liver function test

- Serum bilirubin increased, serum albumin decreased.
- Increased ALT, AST
- Increased alkaline phosphatase,
- Prolonged prothrombin time.

usually mild to moderate elevation of Aminotransferases. Alanine aminotransferase (ALT) less than Aspartate aminotransferase (AST)<sup>[7]</sup>.

Elevation of Immunoglobulins, especially the gamma fraction<sup>[8]</sup>.

#### 4. Liver biopsy--fibrosis with regenerative nodules

##### Imaging

1. Endoscopy may show esophageal varices
2. USG for liver size, ascites, splenomegaly. USG with Doppler study can detect patency of portal, hepatic

vein.

3. CT/MRI with contrast for characterization of hepatic nodule.

#### Complication of cirrhosis

1. Portal HTN, Spontaneous bacterial peritonitis.
2. Hepatocellular carcinoma
3. Hepatic encephalopathy b/o shunting
4. Hepatorenal syndrome.
5. Increase systemic infection b/o increase susceptible to infection b/o decreased detoxification.

#### Miasmatic Representation of Liver Disease with stages & advancement<sup>[9]</sup>.

a. Sycotic

- Chronic Hepatitis
- Hepatomegaly

b. Tubercular

- Oesophageal varices

c. Syphilitic

- Atrophy of liver
- Metabolic failure

#### Repertorial Literature available-<sup>10,11</sup>

##### 1. Liver, enlarged

3 marks: Chin, Lyc, Mag-m, Nat-s, Nux-v

2 marks: Ars, Aur-m, Bry, Calc, Calc-ars, Carb-v, Card-m, Chel, Chion, Cocc, Con, Dig, Ferr, Fl-ac, Hep, Hippoz, Iod, Kali-c, Laur, Merc, Merc-d, Nat-m, Nit-ac, Nux-m, Phos, Podo, Stel, Sulph, Sul-ac, Sul-I, Tarax, Tarn, Tub, Tephrosia-pur, Vip, Zinc, Zinc-p

1. 1 mark: acon, aesc, am-m arg-n, aur-a, aur-I, aur-s, baj, bell, boerh, calc-sil, can-s, chin-s, corn-c, cupr, cur, doli, eel's serum, form, gins, glyc, grap, hell-f, Ins, kali-i, lac-d, lept, mang, myr, nat-p, nyct, ol-i, pin-s, pul, pyro, querc, senna, sep, staph.
2. Emaciation, body, liver affection: 2 marks: Hydr, mag-m
3. Emaciation body, liver hypertrophied: 2 marks: Chen-v
4. Enlarged spleen: 3 marks: Cean, chin
5. Abdominal veins, distended portal system:

##### 2 Marks

Abs, agar,.. anthr, apis,.. arn, ars, ars-I, ars-s-f, asaf, aur-m, bar-c, bell, bels, brom, bry, calc-I, carb-ac, card, cedr, caps, chin-s, chion, cit-v, cocc, con, corn-c, corn-f, ferr, ferr-m, helia, hell, hippoz, ign, kali-I, kali-m, ..lach, linar, luffa-a, mez, nat-m, nat-s, nit-ac, nux-v, ph-ac, phos, pin-s, pod, poly m, polyp, puls, querc, ran-s, sec, sulph, sul-ac, sul-I, thuj, urt-u, xanthor-ap

6. White, nails: 1 mark: Cupr, nit-ac

Totality: Chin: 6/2, Lyc: 5/3, Mag-m: 6/3, Nux-v: 7/3, Sulph: 6/3

##### Cirrhosis of liver

abies-c. am-be. apoc. ars. arS-i. aur. aur-m. Aur-m-n. berb. boerh-d. calc-ar. Card-m. cas-s. chel. chin. chlorpr. crot-h. Cupr. cur. diosm. dulc. euon. f-ac. hed. Hep. Hydr. hydr. iod. kali-bi. kali-i. lact. lyc. mag-m.merc. merc-d. Mur-ac. nast-o. nit-m-ac. nux-v. perh-mal. Phos. plb. quas. senec. Sulph. urea vip.

##### Abdomen - atrophy - liver

Accompanied by

##### Ascites

Apoc. Mur-ac.

**Abdomen - dropsy - ascites-  
Accompanied by - liver cirrhosis of  
Vomiting bile  
Cur.**

#### Treatment

1. Treatment of underlying cause like abstinence from alcohol.
2. Adequate diet giving 25-35kcal/kg/day with protein intake of approximately 1- 1.5gm/kg/day.
3. There is fluid retention restriction of salt. Fluid restriction approximately 1L/day.
4. Diuretics in case of fluid retention

#### Cirrhosis diet

- No alcohol. Restrict salt 2gm/day.
- Protein 15%, CHO 55%, Fat 30%
- SOURCE:

CHO: grain-whole grain bread/brown rice

Protein: Milk, egg daily boiled egg, avoid meat (decrease protein & iron load), sprouts (gives vitamin B also).

FAT: medium chain triglyceride (fractionated coconut oil)

VEG.: bitter gourd/egg plant

#### Miraculous liver remedies of homoeopathy <sup>[12]</sup>.

##### **Lycopodium clavatum**

Lycopodium is adapted more especially to ailments gradually developing, functional power weakening, with failures of the digestive powers, where the function of the liver is seriously disturbed. Liver sensitive. Brown spots on abdomen. Dropsy, due to hepatic disease. Hepatitis, atrophic from of nutmeg liver. Pain shooting across lower abdomen from right to left.

##### **Carduus Marianus**

The action of this drug is centered in the liver, and portal system, causing soreness, pain, jaundice. Has specific relation to the vascular system. Abuse of alcoholic beverages, especially beer. Varicose veins and ulcers. Diseases of miners, associated with asthma. Dropsical conditions depending on liver disease, and when due to pelvic congestion and hepatic disease. Disturbs sugar metabolism. Influenza when liver is affected. Debility. Haemorrhages, especially connected with hepatic disease. Pain in region of liver. Left lobe very sensitive. Fullness and soreness, with moist skin. Constipation; stools hard, difficult, knotty; alternates with diarrhoea. Stools bright yellow. Swelling of gall bladder with painful tenderness. Hyperaemia of liver, with jaundice. Cirrhosis, with dropsy.

##### **Chelidonium Majus**

A prominent liver remedy, covering many of the direct reflex symptoms of diseased conditions of that organ. The jaundiced skin, and especially the constant pain under inferior angle of right scapula, are certain indications. Jaundice due to hepatic and gall-bladder obstruction. Gall-colic. Distention. Fermentation and sluggish bowels. Constriction across, as by a string. Liver enlarged.

##### **Podophyllinum**

Distended; heat and emptiness. Sensation of weakness or sinking. Can lie comfortably only on stomach. Liver region painful, better rubbing part.

Torpidity of the liver; portal engorgement with a tendency to haemorrhoids, hypogastric pain, fullness of superficial

veins, jaundice.

#### Conflict of Interest

Not available

#### Financial Support

Not available

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