IRREVOCABILITY CAN BE REVERSIBLE – CIRRHOSIS OF LIVER WITH HOPE IN AYURVEDA – A CASE STUDY.

1Dr. Kamal Kishore, 2Dr. Prerana Singhal, 3Dr. Satender Tanwar and 4Dr. Kiran M. Goud.

1JR in Department of PG Studies in Kayachikitsa, SKAMCH & RC, Bangalore.
2JR in Department of PG Studies in Panchakarma, SKAMCH & RC, Bangalore.
3JR in Department of PG Studies in Shalyatantra, SKAMCH & RC, Bangalore.
4Head of Institution, Department of PG Studies in Panchakarma, SKAMCH & RC, Bangalore.

ABSTRACT
Miracles do happen in Ayurveda. The same is the live example of the present case scenario. Liver is the largest organ and gland of the body. The highest level of regeneration happens in the liver. The decompensation stage starts when the 95% of the liver damage took places. The liver is the organ which gets the maximum insult in the body. There are various stages in the damages in the liver such inflammation, degeneration, necrosis, fibrosis and cirrhosis. Till the necrosis there is chances of the reversibility but when it enters into the fibrosis and cirrhosis the dark and black zone starts called as the zone of irreversibility. The present case is the live example of the treatment protocol mentioned for the management of the Parenchymal liver disease such as cirrhosis (udararoga) in Ayurveda. The treatment planned in the present case is Gomutra Haritaki, Antra Basti and Dashanga Lepa with oral medicatoin. The result were significant both on laboratory parameter and the clinical observations.

KEYWORDS: The decompensation, cirrhosis, Gomutra Haritaki, Antra Basti and Dashanga Lepa, mediciatoin.

INTRODUCTION
Cirrhosis is a condition in which the liver does not function properly due to long-term damage. Typically, the disease comes on slowly over months or years. Early on, there are often no symptoms. As the disease worsens, a person may become tired, weak, itchy, have swelling in the lower legs, develop yellow skin, bruise easily, have fluid build up in the abdomen, or develop spider-like blood vessels on the skin. The fluid build-up in the abdomen may become spontaneously infected. Other complications include hepatic encephalopathy, bleeding from dilated veins in the esophagus or dilated stomach veins, and liver cancer. Hepatic encephalopathy results in confusion and possibly unconsciousness.

Cirrhosis is characterized by the replacement of normal liver tissue by scar tissue. Some causes of cirrhosis, such as hepatitis B, can be prevented by vaccination. Treatment partly depends on the underlying cause. The goal is often to prevent worsening and complications. Avoiding alcohol is recommended.

Cirrhosis resulted in 1.2 million deaths in 2013, up from 0.8 million deaths in 1990. Of these, alcohol caused 384,000, hepatitis C caused 358,000, and hepatitis B caused 317,000. In the United States, more men die of cirrhosis than women.

Cirrhosis has many possible manifestations. These signs and symptoms may be either as a direct result of the failure of liver cells or secondary to the resultant portal hypertension. There are also some manifestations whose causes are nonspecific, but may occur in cirrhosis. Likewise, the absence of any does not rule out the possibility of cirrhosis. Cirrhosis of the liver is slow and gradual in its development. It is usually well advanced before its symptoms are noticeable enough to cause alarm. Weakness and loss of weight may be early symptoms. Ayurveda gives a wide range of pathologies related to the liver such as Pandu (anemia), kamala

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(icterus), kumbhakkamala (Jaundice with ascites), halimaka (chlorosis). Madya having usha and teekshnaguna is responsible for the movement of doshas from koshtha to shakha making the dhatushithil. Due to excessive indulgence in pitta karahara, vihara the morbid pitta dosha further makes an individual an ashtsaranisarapurusha affecting rasa, rakta, mamsadhatus and eventually affecting him with pandu which when not controlled causes kamala leading to yakrutudara which does the dushti of either prana (cardiac origin), apana (renal origin) or agni/hepatic origin. Also the management in Ayurveda is also explained in a wise manner and the step ladder pattern.

Liver dysfunction
- The following features are as a direct consequence of liver cells not functioning.
- Spider angiomata or spider nevi are vascular lesions consisting of a central arteriole surrounded by many smaller vessels (hence the name "spider") and occur due to an increase in estradiol. One study found that spider angiomata occur in about 1/3 of cases.
- Palmar erythema is a reddening of palms at the thenar and hypothenar eminences also as a result of increased estrogen.[9]
- Gynecomastia, or increase in breast gland size in men that is not cancerous, is caused by increased estradiol and can occur in up to 2/3 of patients. This is different from increase in breast fat in overweight people.
- Hypogonadism, a decrease in sex hormones manifest as impotence, infertility, loss of sexual drive, and testicular atrophy, can result from primary gonadal injury or suppression of hypothalamic/pituitary function. Hypogonadism is associated with cirrhosis due to alcoholism and hemochromatosis.
- Liver size can be enlarged, normal, or shrunken in people with cirrhosis.
- Ascites, accumulation of fluid in the peritoneal cavity (space in the abdomen), gives rise to flank dullness (needs about 1500 ml to detect flank dullness). This may be visible as increase in abdominal girth.
- Feter hepaticus is a musty breath odor resulting from increased dimethyl sulfide.
- Jaundice is yellow discoloration of the skin and mucous membranes (with the eye being especially noticeable) due to increased bilirubin (at least 2–3 mg/dL or 30 µmol/L). Urine may also appear dark.

Portal hypertension
- Cruevillier-Baumgarten murmur is a venous hum heard in the epigastric region (on examination by stethoscope) due to collateral connections forming between portal system and the periumbilical veins as a result of portal hypertension.

Advanced disease
Pathophysiology
The liver plays a vital role in synthesis of proteins (for example, albumin, clotting factors and complement), detoxification, and storage (for example, vitamin A). In addition, it participates in the metabolism of lipids and carbohydrates.

Cirrhosis is often preceded by hepatitis and fatty liver (steatosis), independent of the cause. If the cause is removed at this stage, the changes are still fully reversible. The pathological hallmark of cirrhosis is the development of scar tissue that replaces normal parenchyma. This scar tissue blocks the portal flow of blood through the organ therefore disturbing normal function. Recent research shows the pivotal role of the stellate cell, a cell type that normally stores vitamin A, in the development of cirrhosis. Damage to the hepatic parenchyma (due to inflammation) leads to activation of the stellate cell, which increases fibrosis (through production of myofibroblasts) and obstructs blood flow in the circulation. In addition, it secretes TGF-β1, which leads to a fibrotic response and proliferation of connective tissue. Furthermore, it secretes TIMP 1 and 2, naturally occurring inhibitors of matrix metalloproteinases, which prevents them from breaking down fibrotic material in the extracellular matrix.

The fibrous tissue bands (septa) separate hepatocyte nodules, which eventually replace the entire liver architecture, leading to decreased blood flow throughout. The spleen becomes congested, which leads to hypersplenism and increased sequestration of platelets. Portal hypertension is responsible for most severe complications of cirrhosis.

Pathology
Cirrhosis leading to hepatocellular carcinoma (autopsy specimen). Macroscopically, the liver is initially enlarged, but with progression of the disease, it becomes smaller. Its surface is irregular, the consistency is firm, and the color is often yellow (if associated steatosis). Depending on the size of the nodules there are three macroscopic types: micronodular, macronodular, and mixed cirrhosis. In micronodular form (Laennec's cirrhosis or portal cirrhosis) regenerating nodules are under 3 mm. In macronodular cirrhosis (post-necrotic cirrhosis), the nodules are larger than 3 mm. The mixed cirrhosis consists of nodules with different sizes.

However, cirrhosis is defined by its pathological features on microscopy: (1) the presence of regenerating nodules of hepatocytes and (2) the presence of fibrosis, or the deposition of connective tissue between these nodules. The pattern of fibrosis seen can depend upon the underlying insult that led to cirrhosis. Fibrosis can also proliferate even if the underlying process that caused it has resolved or ceased. The fibrosis in cirrhosis can lead
to destruction of other normal tissues in the liver: including the sinusoids, the space of Disse, and other vascular structures, which leads to altered resistance to blood flow in the liver and portal hypertension.

As cirrhosis can be caused by many different entities which injure the liver in different ways, cause specific abnormalities may be seen. For example, in chronic hepatitis B, there is infiltration of the liver parenchyma with lymphocytes. In cardiac cirrhosis there are erythrocytes and a greater amount of fibrosis in the tissue surrounding the hepatic veins. In primary biliary cirrhosis, there is fibrosis around the bile duct, the presence of granulomas and pooling of bile. Lastly in alcoholic cirrhosis, there is infiltration of the liver with neutrophils.

Grading The different scores can be used are
- Child Pugh Classification.
- Discriminant factor
- Maddery Score.
- Glasgow Alcohol child Scale.

CASE STUDY – Patient name Mohan Kumar(Name Changed) 53 years old, farmer by occupation, Hindu by religion, belongs to the mid socio economic background admitted in the hospital 7th January 2016 with the following complaints.
- Yellowish discoloration of the conjunctiva and urine since two month.
- One episode of bleeding per mouth two and half month back.
- Feeling of malaise since two months.
- Both lower limb swelling since one and half month.
- Puffiness of the face and Loss of appetite since one month.
- Distension of the abdomen since one month.

- There is no premorbid such as diabetes mellitus, hypertension, Bronchial Asthma and intake of any NSAID’s and the corticosteroids.
- History of chronic alcohol intake whisky/Rum/Local Brands more than 25 years – 400-600ml per day comprising of the 30-45 gm/Day of the Ethanol content.
- During the course of the chronic alcohol intake poor intake of the food.

On Examination
1. Built – Well built
2. Nourishment – Under nourished
3. BMI – 19.5kg/m²
4. Pulse – 90/bm
5. Blood pressure – 100/60 mm of hg.
6. Respiratory rate – 19 cycles per minute
7. Lymphadenopathy - Present
8. Incertus – Present
9. Cyanosis - Absent
10. Edema – B/L pedal oedema.

Systemic Examination
1. Central Nervous System – Conscious, oriented to time/place/person, with preserved memory and higher mental function, no observed focal neurological deficit found.
2. Respiratory System – B/L EAE, With Basal creps and Fine Rhonchi (R>L)
3. Cardiovascular System – S₁ & S₂(+), NAS.
BS – Difficult to heard due to the massive abdominal dullness.
Shifting Dullness (+). Fluid Thrill (+)
Horse Shoe Shaped Dullness (+)
Abdominal Girth – 134 cm
5. MSK – Musculoskeletal System – B/L pitting pedal edema massive.

INVESTIGATIONS
- USG – Abdomen and Pelvis. – Hepatomegaly with architectural liver changes, Moderate ascites, portal hypertension and splenomegaly.
- UGIE – Upper GI Endoscopy. – Oesophageal Varices.
- LFT – Shown Below
- RFT All parameters WNL.
- Serum Electrolytes. - All parameters WNL.
- CBC - All parameters WNL.
- CXR – PA View – B/L pleural Effusion (R>L).

Diagnosis - LAENNEC CIRRHOSIS
As per the history taken and the clinical examination performed more towards the Cirrhosis of liver due to chronic alcohol Consumption manifesting as the hepatocellular failure – Jaundice, Ascites, Pedal edema, Puffiness of face, loss of appetite and Haememtosis due to the portal hypertension.
Treatment Proposed –
- Gomutra Haritaki. – 12 gm + 30 ml Fresh Gomutra for 3 days Later 12gm + 40 ml of Gomutra for 15 days.
- Dashanga Lepa on abdomen – 12 gm of Dashanga Lepa + Warm Water – Thin Even paste applied and left for 6 hours of duration.
- Shankha Bhasma(20gm) + GuduchiSattva(20gm) + GodantiBhasma(20gm) + MuktaBhasma(20gm) – 1 pinch TDS
- Aantrabastifor 4 days.

Supportive management such as
- Foot End elevation.
- Tab Liv 52 DS 2Tsf TDS
- SypNirocil 2 TsfTDS.

Probable Mode of Action of the proposed treatment.
- Gomutra Haritaki
In Ayurveda, cow urine (Gomutra) occupies a unique place and has been recognized as water of life or...
“Amrita”. In Sushruta Samhita, it has been described as the most effective substance of animal origin.

Cow urine has been granted US Patents (No. 6,896,907 and 6,410,059) for its medicinal properties, particularly as a bioenhancer and as an antibiotic, antifungal and anticancer agent. In India, drinking of cow urine has been practiced for thousands of years. According to Ayurvedic pharmacodynamics profile of cow urine and Haritaki (Terminaliachebula) has got the predominance of agni (fire), vayu(air) mahabhuta. Both are having laghu(light), ushna(hot), tikshna(sharp), rukshana(dry) gunas(properties) in both of them. GomutraHaritaki is used in condition where shodhana (purification) is needed.

- As laxative - Haritaki Is considered as the laxative and the help in the evacuation of the bowel two to three times helps in relieving of the bacterial overgrowth in the intestine and reduces the conversion time of the urea in to ammonia and reduces the chances of the hepatic encephalopathy.
- As Cholagogue – Gomutra is considered as the Cholagogue which helps in the excretion of the excessive bile and the salts and reduces the turnover metabolism of the Bilirubin which is prime for the increasing the Jaundice.

### SUCCESSIVE LAB INVESTIGATION AND THE CLINICAL CHANGES OBSERVED AS SHOWN BELOW –

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<tr>
<td>Total Bilirubin</td>
<td>22.04</td>
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<td>11.30</td>
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<td>Direct</td>
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<td>1.9</td>
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<tr>
<td>SGOT</td>
<td>111.6</td>
<td>136.3</td>
<td>200.0</td>
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<tr>
<td>SGPT</td>
<td>26.7</td>
<td>43.9</td>
<td>52.0</td>
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<td>ALKALINE PHOSPHATASE</td>
<td>151.9</td>
<td>140.0</td>
<td>179.0</td>
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<td>GAMMA GLUTYL TRANSFERASE</td>
<td>275.6</td>
<td>203.7</td>
<td>179.0</td>
<td>190.9</td>
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</tbody>
</table>

- Complete Resolution of the Bilateral Pitting edema.
- Complete resolution of the puffiness of the face.
- Dyspnoe on exertion has resolved completely.
- Abdominal Distension has been reduced from 134 cm to 93 cm.
- Yellowish Discoloration has been reduced to greater extent.

### CONCLUSION

Ayurveda has the treasure of the medical knowledge, which only requires the exploration and make it EBM – Evidenced Based Medicine. Present case is the live example of such clinical presentation where the irreversible things can be reversible.

### REFERENCES

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3. Charaka Samhita.
4. Introduction to Agni.
5. Wikipedia.

### Randomised clinical trial

- As Anti neoplastic/Anti-oxidant Action – Many RCT – Randomised clinical trial has been suggested that the Go mutra has the anti neoplastic activities which helps in the reduces the chances of the cirrhosis of liver turning in to the HCC- Hepato Cellular Carcinoma,
- Hepatic Regeneration – Haritaki has been explained as the Rasayan in the Ayurvedic text and the has the capacity for the regeneration of the heapatocytes.

### AantraBasti

In this procedure any part of small intestine of goat/sheep was taken and boiled with 2000ml of water reduced to 500ml. This filtrate was given as basti(enema) to the patient for four consecutive days.

Jalodara(Ascites) is accompanied with possible deficit of Pachkagni. This folklore preparation is based on the hypothesis that the pittat(agni) dharakala described by both Sushruta and Vagbhatta, may be the same as the mucosa of duodenum in particular and the small intestine in general, the administration of a crude aqueous extract of this should replace, at least, for the timebeing, the possible deficits of pachkagni- a deficiency which is stated to act as the main etiological and pathological feature of Udarroga.


13. Dwarakanath et al,"A brief report on the preliminary observation on the effect of Agnidhara kala sara or a composite aqueous extract of gastric and duodenal mucosa in cases of sotha (oedema) and jalodara (ascites) Antiseptic, July 57.