EFFECT OF INTEGRATED PARACENTESIS AND ORAL VARDHAMAN PIPPALI RASAYAN (VPR) THERAPY IN ASCITES DUE TO ALCOHOLIC LIVER CIRRHOSIS: A CASE REPORT

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ABSTRACT

Alcoholic Liver Cirrhosis induced Ascitis is a condition with poor prognosis. Liver transplant is only the best option available but it is difficult and costly affair. Conservative management may not be effective in many cases. The condition correlates with Jalodara which is a complication of Yakritodar in Ayurveda. It has also mentioned as one of the Ashtamahagad (~Diseases that are difficult to treat). Case report- 38 years old Male patient presented with progressive abdominal distension with aching pain over whole abdomen with stretching, swelling mostly over lower limbs, dyspnea, jaundice, sometimes hematemesis, loss of appetite, generalized weakness. On clinical examination Shifting dullness, pitting edema over lower limbs, fluid thrills, everted umbilicus in advance stage with crepitations in chest were present. Diagnosis was done with history, clinical signs of liver disease, Ultra sonography (USG) of abdomen and supportive laboratory tests. Integrated surceal and pharmacological intervention in the form of Paracentesis and Oral Vardhaman Pippali Rasayan (VPR) Therapy were given after initial emergency paracentesis. Schedule was followed for 3 months with only boiled cow milk was advised in diet with total restriction of normal diet and daytime sleeping. Remarkable relief in terms of reduction in abdominal girth, swelling reduced in each follow up with significant improvement in clinical features and biochemical parameters and reduction in fluid was observed. Conclusion: Integrative treatment of ALD with Vardhaman Pippali rasayan and...
emergency Paracentesis can improve quality of life of these patients. Clinical trials on integrative treatment are warranted.

**KEYWORDS:** Liver Cirrhosis, Jalodar, Paracentesis, Vardhaman Pippali Rasayan, Integrative approach.

**INTRODUCTION**

Ascitis is an accumulation of free fluid in the peritoneum which is diagnosed after its progressive stage of swelling of abdomen with weight gain, aching pain all over abdomen with stretching, bloated feeling in abdomen, dyspnoea or orthopnoea in massive collection.[1] About 90% of patients found to have Ascitis due to Liver Cirrhosis, abdominal neoplasm, CHF or abdominal TB[2] and out of which 75% are due to Alcoholic Cirrhosis only.[3] Daily consumption of 60-80 gm of ethanol in type of any alcoholic beverage for at least 10 years is likely to develop Liver Cirrhosis.[4] In case of Cirrhosis, Ascitis is developed due to high hydrostatic pressure of sustained Portal Hypertension along with decreased serum albumin due to low hepatocellular mass which results in low osmotic pressure and further lead to develop Ascitis.[5] Liver Cirrhosis is a consequential conversion of alcohol into fatty acids which initially results in Alcoholic Fatty Liver followed by hepatocytes destruction due to lipid vacuoles and proliferation of myofibroblasts and fibrocytes which finally results in fibrosis of liver[6] and Micronodular Cirrhosis.[7] In Ayurvedic classisc, Ascitis due to Alcoholic Liver Cirrhosis can be correlated to Jalodar as a complication of Yakritodar.[8]

Vidahi, Ushna, Tikshna Aahar (Alcohol) lead to Pitta and Rakta Prakop which further causes Jalodar (Ascitis). Jalodara is advance stage of Udar and all types of Udar are due to prolonged Agnimandya[9] (Anorexia). So it is very important to provoke appetite. Vardhaman Pippali is indicated in Plhodar[10] and can be given in Yakritodar as symptoms and treatment of both is same.[11] Acharya Sushrut and Acharya Charak have advocated to go for Shastrakarma[12][13] (~Paracentesis) in case of Ascitis. So it also play important role in the treatment. But only paracentesis is just symptomatic management and due to recurrence, it is also not effective one. It just gives temporary relief in emergency. So combine effect of paracentesis and VPR is effective one.

In Modern science, line of management in Cirrhosis is just symptomatic with no significant effect as route cause of fibrosis is irreversible. Paracentesis as per Ayurveda along with VPR restrict the recurrence of fluid. VPR is seen to get out of the condition as it restricts the further insult of hepatocytes by fibrosis and also possibly helps in rejuvenation of the damaged area.
The current case study provides further scope for the clinical trials on large sample size in such cases to generate better outcome in the field of medical science.

**Patient Information**

38 years old Male Farmer patient belonging to Patna was brought to OPD of Kayachikitsa on 6 January 2016 by his relatives. He was then admitted to MKC (Male Kayachikitsa Ward) on bed no. 37. He was discharged after 20 days of treatment.

**Symptoms**

He was presented with breathlessness, altered sensorium since 2 weeks, yellowish discolouration of sclera and skin, gross abdominal distension, edema over both lower limbs since 3 months, nausea, vomiting, anorexia, malaise since 4 months.

**Past Medical History**

He was suffered from same complaints previously before one year without haemetemesis and was admitted to private hospital. He got relief at that time and was discharged. But further he continued his habit and again suffered from same complaints mentioned above. He was taken to private hospital but didn’t get relief and so he was brought to Kayachikitsa OPD for Ayurvedic treatment. from private hospital for Ayurvedic management on suggestion of private gastroenterologist as a last hope due to poor prognosis of patient.

**Personal History**

His relatives told about his history of daily alcohol intake since last 12 years. He was used to take 400-500 ml alcohol daily and no intake of proper diet.

**Family History**

No significant facts were found.

**Past Intervention & Outcome**

He was taken to private medical college for the first time and he was improved that time as his relatives told. But over a period of one year, he developed same complaints and again taken to private hospital. He was given Inj. Metronidazole, Inj. Ceftriaxone, Inj. Keplin, IV fluid DNS, Oxygen inhalation and protein diet, etc at private hospital, but didn’t get any improvement 2nd time.
Clinical examinations on admission

1. General Examination: General condition of patient was poor. He was afebrile and lethargic with blood pressure - 102/70 mmHg, pulse rate - 96/min, respiratory rate - 26/min, weight - 42 kg and icterus (+++).

2. Physical Examination: There was weakness of both upper limbs with emaciation and gross pitting edema over both lower limbs (+++).

3. Systemic Examination: CNS: Conscious with mild disorientation, DTR (Deep Tendon Reflexes) were normal.

CVS: No any abnormality detected.

RS: Bilateral air entry was present but diminished at basals along with fine crepitations at bilateral basal zones.

P/A: Everted umbilicus and huge abdominal distension with stretched and shiny skin were seen on inspection. No organomegaly was seen on palpation due to tense abdomen, Shifting dullness and fluid thrills were present. Other systemic examinations were within normal limits.

Timeline of the patient given below.

<table>
<thead>
<tr>
<th>Visited to private hospital on 3/1/15</th>
<th>Relevant Past Medical History and Interventions: Past history of breathlessness, altered sensorium, yellowish discolouration of sclera and skin, gross abdominal distension, edema over both lower limbs, nausea, vomiting, anorexia, malaise before 1 year. He was admitted that time in private hospital of Patna and was diagnosed as a Alcoholic Liver Cirrhosis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient was admitted in MKC on 6/1/16</td>
<td>Complaints at the time of visit</td>
</tr>
</tbody>
</table>
IgM, Anti HEV IgM, Anti HCV were Negative  
**Autoimmune Hepatitis profile** (18/11/15): AMA, ANA, LKM-1 were negative  
**Quantiferon TB Gold**: Negative  
PT-INR: Test- 31.5 sec, control- 13 sec, INR- 2.88  
**Ascitic Fluid R/M**: Transudate in appearance, White cell count- WNL, Protein 2.4 g/dL, ADA- 15 IU/L  
**USG Abdomen** (19/11/15): Mild hepatomegaly with diffuse grade 2 fatty infiltrations and coarse echotexture, portal vein dilated and mild splenomegaly 16.8 cm  

| He was discharged on request on 12/1/16 | No any complaints (Abdominal distension, Swelling on both legs were reduced) | Blood investigations (CBC, LFT, RFT) were advised but not done by patient  
**CT Abdomen** (9/1/16) at S.S.Hospital, BHU: Antero-posterior diameter of liver was 15 cm, left lobe atrophy with compensatory hypertrophy of right lobe and gross ascitis with mild splenomegaly | VPR therapy continued as previous |
|---|---|---|---|
| 1<sup>st</sup> visit on 21/1/16 due to complaints | Burning sensation in abdomen, burning micturition, Fever (Abdominal distension, Swelling on both legs were reduced)  
R/S- Chest was clear, no crepitations were present | Blood investigations had done on 20/1/2016 at private lab of Varanasi  
**CBC**: Hb- 9.7 gm/dL, Platelets- 78000/ cu.mm, TLC- 11950 and DLC – within normal limits  
**LFT**: Total bilirubin- 3.1 mg/dl, direct bilirubin- 1.9 mg/dl, Total protein- 6.9 g/dl, albumin- 2.2 g/dl, ALT- 27 U/L, AST- 84 U/L, ALP- 184 U/L  
**RFT**: Creatinine- 1.9 mg/dL, Urea- 60 mg/dL, Serum electrolytes were within normal limits  
**Urine R/M**: 2-3 Epithelial cells, no proteins,  
**USG Abdomen** (20/1/16): Mild hepatomegaly with fatty infiltrations and moderate Ascitis | VPR therapy continued as previous with tapering dose by 1 Pippali  
Oral Kamdudha ras 250 mg twice a day for 3 days |
| 4<sup>th</sup> visit on 9/2/16 | Dryness of mouth, mild burning sensation in abdomen and throat (Abdominal distension, Swelling on both legs were reduced, improved appetite) | No investigations were advised | VPR therapy continued as previous  
Oral Kamdudha ras 250 mg and Honey for local application at oral cavity twice a day for 3-4 days |
<p>| 5&lt;sup&gt;th&lt;/sup&gt; visit on | Generalised weakness, mild | Investigations were lost by the patient | VPR therapy |</p>
<table>
<thead>
<tr>
<th>Date</th>
<th>Symptoms</th>
<th>Blood Tests</th>
<th>VPR Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>29/2/16</td>
<td>abdominal distension</td>
<td>Blood investigations were done on <strong>15/3/2016</strong> at S.S.Hospital, BHU</td>
<td>continued as previous</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>CBC:</strong> Hb- 10.2gm/dL, Platelets-96000/cu.mm, TLC- 10840 and DLC – within normal limits</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td><strong>LFT:</strong> Total bilirubin- 2.9mg/dl, direct bilirubin- 1.7mg/dl, Total protein- 7.2g/dl, albumin- 2.9g/dl, ALT- 35U/L, AST-56U/L, ALP- 160U/L</td>
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<tr>
<td></td>
<td></td>
<td><strong>RFT:</strong> Creatinine- 1.7mg/dL, Urea-45mg/dL, Serum electrolytes were within normal limits</td>
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<td></td>
<td></td>
<td><strong>USG Abdomen</strong> (15/3/16): Mild hepatomegaly with changed nodular echotexture and moderate Ascitis</td>
<td></td>
</tr>
<tr>
<td>6th visit</td>
<td>Dizziness, dryness of mouth</td>
<td>Blood investigations had done on <strong>28/3/2016</strong> at S.S.Hospital, BHU</td>
<td>VPR therapy continued as previous</td>
</tr>
<tr>
<td>on 16/3/16</td>
<td>BP- 112/70mmHg, PR-78/min</td>
<td><strong>CBC:</strong> Hb- 11.2gm/dL, Platelets-95000/cu.mm, TLC- 10900 and DLC – within normal limits</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Urine R/M:</strong> 2-3 Epithelial cells, no proteins</td>
<td></td>
</tr>
<tr>
<td>7th visit</td>
<td>Burning micturation, mild</td>
<td>Blood investigations had done on <strong>6/4/2016</strong> at S.S.Hospital, BHU</td>
<td>VPR therapy continued as previous</td>
</tr>
<tr>
<td>on 28/3/16</td>
<td>feverish (Abdominal distension,</td>
<td><strong>CBC:</strong> Hb- 11.8gm/dL, Platelets-110000/cu.mm, TLC- 9300 and DLC – within normal limits</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Swelling on both legs were</td>
<td><strong>LFT:</strong> Total bilirubin- 1.6mg/dl, direct bilirubin- 0.8mg/dl, Total protein- 7.1g/dl, albumin- 3.2g/dl, ALT- 32U/L, AST-52U/L, ALP- 143U/L</td>
<td></td>
</tr>
<tr>
<td></td>
<td>reduced)</td>
<td><strong>RFT:</strong> Creatinine- 1.5mg/dL, Urea-32mg/dL, Serum electrolytes were within normal limits</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td><strong>USG Abdomen</strong> (7/4/16): Mild hepatomegaly with corse echotexture and no free fluid in peritoneal cavity</td>
<td></td>
</tr>
<tr>
<td>Last visit</td>
<td>Generalised weakness,</td>
<td>Blood investigations had done on <strong>3/9/2016</strong> at S.S.Hospital, BHU</td>
<td></td>
</tr>
<tr>
<td>on 7/4/16</td>
<td>(Abdominal distension,</td>
<td><strong>CBC:</strong> Hb- 10.8gm/dL, Platelets-110000/cu.mm, TLC- 9300 and DLC – within normal limits</td>
<td></td>
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<tr>
<td></td>
<td>Swelling on both legs were</td>
<td><strong>LFT:</strong> Total bilirubin- 0.8mg/dl, direct bilirubin- 0.8mg/dl, Total protein- 7.1g/dl, albumin- 3.2g/dl, ALT- 32U/L, AST-52U/L, ALP- 143U/L</td>
<td></td>
</tr>
<tr>
<td></td>
<td>vanished, improved appetite)</td>
<td><strong>RFT:</strong> Creatinine- 1.5mg/dL, Urea-32mg/dL, Serum electrolytes were within normal limits</td>
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<tr>
<td></td>
<td></td>
<td><strong>USG Abdomen</strong> (7/4/16): Mild hepatomegaly with corse echotexture and no free fluid in peritoneal cavity</td>
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</tbody>
</table>

On 5/9/16, brother of the patient informed me by phone that he expired on 3/9/16. Relative threw the all investigations of patient after his death. **Cause of death:** Patient again started to drink alcohol, didn’t follow the medication and Diet regimen after one month of last follow up.
Diagnostic Assessment

Diagnostic Methods
Blood investigations had done on 5/1/2016 at private hospitals

CBC: Hb- 8.8gm/dL, Platelets- 61000/cu.mm, TLC and DLC – within normal limits

LFT: Total bilirubin- 3.2mg/dl, direct bilirubin- 2.1mg/dl, Total protein- 6.8g/dl, albumin-2.1g/dl, ALT- 22U/L, AST- 92U/L, ALP- 155U/L

RFT: Creatinine- 1.7mg/dL, Urea- 40mg/dL, Serum electrolytes were within normal limits

RBS: 108mg/dL.

Hepatitis profile: HbsAg, Anti HAV IgM, Anti HEV IgM, Anti HCV were Negative

Autoimmune Hepatitis profile (18/11/15): AMA, ANA, LKM-1 were negative

Quantiferon TB Gold: Negative

PT-INR: Test- 31.5sec, control- 13sec, INR-2.88

Ascitic Fluid R/M: Transudate in appearance, White cell count- WNL, Protein 2.4g/dL, ADA- 15IU/L.

USG Abdomen(19/11/15): Mild hepatomegaly with diffuse grade 2 fatty infiltrations and course echotexture, portal vein dilated and mild splenomegaly 16.8cm.

CT Abdomen(12/1/16): Anteroposterior diameter of liver was 15cm, left lobe atrophy with compensatory hypertrophy of right lobe and gross ascitis with mild splenomegaly.

Diagnostic Challenges: No affords were made to diagnose the condition as patient was brought with sufficient investigations. And also he did necessary investigations as early as possible.

Differential Diagnosis
1. Abdominal TB: In ascitic fluid examination, no lymphocytes were raised, no protein raised, no glucose reduced, no ADA raised so ruled out
2. Baterial Peritonitis: TLC was normal in CBC and Ascitic fluid findings were also not suggestive so ruled out
3. Abdominal carcinoma: No relevant findings suggesting to carcinoma found so ruled out
4. Autoimmune Hepatitis: Autoimmune profile was normal so ruled out
5. Viral Hepatitis: Viral markers for hepatitis were negative so ruled out
THERAPEUTIC INTERVENTION

Type of Intervention
Initially Surgical and Conservative management was done for 2 days as patient was not fully oriented. From 3rd day, Pharmacological intervention was done as Oral VPR Therapy.

Administration of Intervention
Initially Paracentesis (900mL) was done and apply cotton cloth tightly around the whole abdomen (as mentioned Ayurveda) on first day of admission. Then patient was given loose enema every 12 hourly, nebulisation with duolin and budecort (1 respule of each) every 6 hourly, moist oxygen inhalation at 4-5 L/min and other conservative management as Injectable Antibiotics, Diuretics every 12 hourly and IV fluids 24 hourly with moniteration.

After 2 days, patient became oriented and was planned for Oral VPR Therapy for one year in 3 stapes.
1st day: 3 Pippali(crushed) boiled in cow milk twice day orally
2nd day: 4 Pippali twice a day with same method
3rd day: 5 Pippali twice a day with same method
4th day: 6 Pippali twice a day with same method
5th day: 7 Pippali twice a day with same method

Then dose of 7 Pippali twice a day was continued for next 10 days. From 16th day, dose was tapered by one Pippali per day upto 3 Pippali as per above schedule followed by no oral VPR for next 10 day. This was a aschedule of one month and was continued for 3 months.

Diet planning: Only boiled cow milk was allowed in diet for first 6 months, Peya and cow milk for next 3 months and Kodo or Sava rice with cow milk for last 3 months. Complete restriction of normal diet, drinking water and daytime sleeping.

Changes in Intervention: Kamdudha Ras 250mg (sos) for burning sensation, Honey for local application at oral cavity for dryness and Trinpanchamul Kashay 40mL for burning micturation and feverishness during follow ups.

OBSERVATIONS AND RESULTS
Clinician Outcome: After treatment of 3 months, it was found that patient improved with dramatic change. No Ascitis was observed after 3 months and CBC, LFT and RFT were within normal limits which are shown in tables given below.
Patient Outcome: Patient was satisfied with improvement in complaints with no abdominal distension, no swelling over limbs, no breathlessness.

Follow up Diagnostics & Other Test

Table 1: Showing physical measurements of abdominal girth from umbilicus, both thighs, both calf regions and both feet from their mid points in centimeters.

<table>
<thead>
<tr>
<th>Date</th>
<th>Umbilicus</th>
<th>Right Thigh</th>
<th>Left Thigh</th>
<th>Right Calf</th>
<th>Left Calf</th>
<th>Right Foot</th>
<th>Left Foot</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/1/16</td>
<td>115</td>
<td>58</td>
<td>59.5</td>
<td>39</td>
<td>39.5</td>
<td>28</td>
<td>29</td>
</tr>
<tr>
<td>11/1/16</td>
<td>111</td>
<td>56</td>
<td>58</td>
<td>37</td>
<td>37.5</td>
<td>27.5</td>
<td>28</td>
</tr>
<tr>
<td>21/1/16</td>
<td>108.5</td>
<td>55.5</td>
<td>56.5</td>
<td>36.5</td>
<td>35.5</td>
<td>26</td>
<td>27</td>
</tr>
<tr>
<td>9/2/16</td>
<td>104</td>
<td>54.5</td>
<td>55</td>
<td>34</td>
<td>34.5</td>
<td>26</td>
<td>26.5</td>
</tr>
<tr>
<td>29/2/16</td>
<td>100.5</td>
<td>54</td>
<td>54.5</td>
<td>33.5</td>
<td>34</td>
<td>25.5</td>
<td>25.5</td>
</tr>
<tr>
<td>16/3/16</td>
<td>96.5</td>
<td>53.5</td>
<td>53.5</td>
<td>33.5</td>
<td>33.5</td>
<td>24.5</td>
<td>25</td>
</tr>
<tr>
<td>28/3/16</td>
<td>91.5</td>
<td>53</td>
<td>53</td>
<td>33</td>
<td>33.5</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>7/4/16</td>
<td>88</td>
<td>52</td>
<td>52.5</td>
<td>32</td>
<td>32</td>
<td>23</td>
<td>23.5</td>
</tr>
</tbody>
</table>

Table 2: Ultra Sonography of the abdomen showing progressive decrease in Ascitis.

<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>20/1/16</td>
<td>Mild hepatomegaly with fatty infiltrations and moderate Ascitis</td>
</tr>
<tr>
<td>15/3/16</td>
<td>Mild hepatomegaly with changed nodular echotexture and moderate Ascitis</td>
</tr>
<tr>
<td>6/4/16</td>
<td>Mild hepatomegaly with coarse echotexture and no free fluid in peritoneal cavity</td>
</tr>
</tbody>
</table>

Table 3: Haematological investigations including CBC, LFT and RFT showing improvement after each follow up.

<table>
<thead>
<tr>
<th></th>
<th>5/1/16</th>
<th>20/1/16</th>
<th>15/3/16</th>
<th>6/4/16</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLC (per cu.mm)</td>
<td>11,000</td>
<td>11,950</td>
<td>10,840</td>
<td>9,300</td>
</tr>
<tr>
<td>Hemoglobin (gm/dL)</td>
<td>8.8</td>
<td>9.7</td>
<td>10.2</td>
<td>11.8</td>
</tr>
<tr>
<td>Platelets (per cu.mm)</td>
<td>61,000</td>
<td>78,000</td>
<td>96,000</td>
<td>1,10,000</td>
</tr>
<tr>
<td>Sr.Total Bilirubin (mg/dL)</td>
<td>3.2</td>
<td>3.1</td>
<td>2.9</td>
<td>1.6</td>
</tr>
<tr>
<td>Sr.Direct Bilirubin (mg/dL)</td>
<td>2.1</td>
<td>1.9</td>
<td>1.7</td>
<td>0.8</td>
</tr>
<tr>
<td>Sr.Total Protein (mg/dL)</td>
<td>6.8</td>
<td>6.9</td>
<td>7.2</td>
<td>7.1</td>
</tr>
<tr>
<td>Sr.Albumin (mg/dL)</td>
<td>2.1</td>
<td>2.2</td>
<td>2.9</td>
<td>3.2</td>
</tr>
<tr>
<td>Sr.ALT (mg/dL)</td>
<td>22</td>
<td>27</td>
<td>35</td>
<td>32</td>
</tr>
<tr>
<td>Sr.AST (mg/dL)</td>
<td>92</td>
<td>84</td>
<td>56</td>
<td>52</td>
</tr>
<tr>
<td>Sr.ALP (U/L)</td>
<td>155</td>
<td>184</td>
<td>160</td>
<td>143</td>
</tr>
<tr>
<td>Sr.Creatinine (mg/dL)</td>
<td>1.7</td>
<td>1.9</td>
<td>1.7</td>
<td>1.5</td>
</tr>
<tr>
<td>Sr.Urea (mg/dL)</td>
<td>40</td>
<td>60</td>
<td>45</td>
<td>32</td>
</tr>
</tbody>
</table>

Intervention Adherence & Tolerability

It was very difficult to console the patient that he must to follow diet regimen and not to start regular diet. He was trying to neglect medication whenever he started burning sensation. He used to ask for water when he felt dryness of mouth.
Adverse Events
Sometimes patients suffered from burning sensation in abdomen, dryness of mouth, burning micturation, feverishness, dizziness.

DISCUSSION
Strength in Approach
There is only conservative management in modern science for Cirrhotic Ascitis as Paracentesis which lead to recurrence of Ascitis. So Ayurvedic management aims at removal of fluid along with protection of hepatocytes. Integrative Paracentesis and Oral VPR Therapy showed significant effect by this principle.

Limitations
Diet planning is major factor which patients generally don’t follow due to strict milk diet only. Patients mainly feel burning sensation and dryness sometimes, so patients give up the medication. So further study is needed to overcome adverse factors and limitations. Also, this was a case study observed in single patient. So there is further need of clinical trial on large sample size to evaluate effect, side-effects and the exact mechanism of improvement.

Discussion of Literature
Ascitis is developed as a complication of Liver Cirrhosis which involves 3 sequential stages due to alcoholic cause (a) Alcoholic Fatty Liver (b) Alcoholic Hepatitis and (c) Alcoholic Cirrhosis. It’s pathogenesis can be corelated to Samprapti (pathogenesis) of Jalodar in Ayurveda given below.
Pathogenesis of Ascitis in terms of Ayurveda\textsuperscript{[15]}

\textbf{Pathogenesis of Ascitis in terms of Ayurveda}

\textit{Vidahi, Ushna, Tiksha Aahar} (Alcohol)

\textit{Pitta & Rakta Prakop}

\textit{Agnimandya}

Further (\textit{Pitta, Rakta}) & (\textit{Kapha} Dushti)

\textit{Ambuvaha & Swedavaha Srotavarodh}

\textit{Vikrit Meda Sanchay} (Fatty Liver)

\textit{Tiryak Gati of Ambu & Sweda}

\textit{Yakrit Shoth} (Hepatitis)

Mixing of \textit{Ambu} & \textit{Sweda} with \textit{Aahar Rasa}

\textit{Yakritodar} (Liver Cirrhosis)

\textit{Jalodar} (Ascitis)

On the first day of admission, Shastrakarma (Paracentesis) was done and then cotton cloth was applied around the abdomen tightly to prevent recurrence of ascitic fluid. Acharya Charak and Sushrut have advised Shastrakarma for Jalodar followed by Vastra Aavestana\textsuperscript{[16]} (to encircle cloth around the abdomen). So Paracentesis followed by tight application of cloth around the abdomen restricts the recurrence of the fluid. This is the unique method mentioned in Ayurveda for Paracentesis. Removal of Ascitic fluid reduced tension over diaphragm and helped to get relief in breathlessness. It was a plan for acute management. Nebulization and oxygen inhalation were part of supportive management at initial part. VPR was started from 3\textsuperscript{rd} day when patient became oriented. It was given by modified plan of oral administration to avoid complications due to overdose. Acharya Charak has recommended to start VPR with 10 Pippali per day and to increase dose by 10 on each day. But no previous study has been done with such large dose, so it was decided to give with low dose by modified plan.
Previous study of Meera Antiwal et al. and Rajpoot Ranjana showed significant effect in Jalodar with such low dose plan.[17]-[18] Principle of treatment in Jalodar is oral administration of Tikshna, Ushna, Dipaniya and Kaphahar medication[19] along with cow milk[20] in diet. Dry Pippali has all such properties. It is a Katu Rasatmak, Tikshna and Ushna Virya dravya so help to improve appetite.[21] Hepatoprotective activity[22] and rejuvenation[23] property of Pippali by restriction to fibrosis were studied by Prashant Sahu et al. and Suresh Kumar et al. By virtue of Tikshna guna, it stimulates hepatic activity and help to stop further fibrosis and also promote erythropoisis, thrombopoisis and albumin formation. Nitya Virechana is recommended in all types of Udar.[24] Virechana is internal cleansing therapy and detoxify the body and opens various channels (Srotas)[25] of it. In Jalodar, Ambuvaha and Swedavaha srotas are blocked.[26] So Virechana opens the closed channels and help to reduce Ascitis. Pippali has Rechan property[27] and Vardhaman Pippali pattern lead to 5 to 6 motions per day.[28] It was even observed in this patient as he gave that history of loose motions.

After starting of VPR, patient complained of burning sensation in abdomen and throat along with dryness of mouth on 5th day. At that time, he was asked to reduce one Pippali for that day and to continue same dose for next 10 days. He was given Kamduha ras for burning sensation each time intermittently and advised to take cow milk as much as possible. Also he was asked to apply honey[29] at oral cavity for dryness. After 7 days of admission, he was discharged from MKC on his request and advised to follow all instructions. He visited to Kayachikitsa OPD as per his convenience after 15 to 20 days of interval. Integrated Paracentesis and Oral VPR Therapy were showing their effect in terms of improvement as reduction in abdominal distension, swelling of lower limbs and breathlessness, improved appetite and feeling of recovery by patient. Readings showing improvement are given in tables 1, 2 and 3 above in terms of haematological investigations, ultra sonography and physical measurements. Patient was almost improved with 3 month VPR therapy and asked to continue medication and diet schedule for one year as per advice.

CONCLUSION

By this case study, it is found that Integrated Paracentesis and Oral VPR Therapy have excellent potency to reverse the condition and to extend life expectancy of patient for several years. Its possibly due to Principle of treatment in Jalodar is withdrawal of fluid followed by application of cloth tightly around the abdomen and oral administration of Tikshna,
Ushna, Dipaniya and Kaphahar medication\textsuperscript{19} along with cow milk\textsuperscript{20} in diet. Dry Pippali has all such properties. It has Virechak property essential for removal of toxins and to purify internal channels. It also has hepatoprotective activity\textsuperscript{22} and rejuvenation\textsuperscript{23} property by virtue of which it restricts further fibrosis and hepatocellular insult.

Take Away Lessons
Approach for Ayurvedic management in such chronic cirrhotic cases helped to raise faith in Ayurvedic medical science which is necessary for future inventions.

Patient Perspective
When patient was brought to Kayachikitsa Department, S. S. Hospital BHU, relatives of patient had no more expectations due to poor condition of patient. But when patient become oriented after 2 days, they felt a hope for better improvement. Further, patient was consoled for the VPR and he became ready to follow all the instructions related to medication and diet regimen. He followed instructions for 10-12 days and when he started complaints of burning sensation and dryness of mouth, he became disappointed. He used to ask for water and normal diet but his wife didn’t listen to him as she was instructed at hospital. Patient continued the treatment with patience and started getting improvement which made him to believe in further medication. After 3 months, he was very thankful to us and rest of hospital staff for saving his life. His successful treatment made him to believe in Ayurveda also.

Informed Consent
Patient got improvement but he continued his habit of alcohol just after of one month. He again developed previous symptoms and he expired on 3/9/16. So the consent is signed by his brother which is attached with the case report. Relevant investigations of the patient were thrown out by his relatives after the death of patient and so scanned photographs of investigations are not presently available. Though it is the weakness of the case study, all the facts about patient are true.

REFERENCES


