

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

K. S. Girhepunje<sup>\*1</sup>, Varsha Gupta<sup>2</sup>, Rajesh Jain<sup>3</sup>, Amit Nakanekar<sup>4</sup> and O. P. Singh<sup>5</sup>

<sup>1</sup>Junior Resident-III, Dept. of Kayachikitsa, Faculty of Ayurveda, IMS, BHU, Varanasi.

<sup>2</sup>Junior Resident-I, Dept. of Rachana Sharir, Faculty of Ayurveda, IMS, BHU, Varanasi.

<sup>3</sup>Junior Resident, Dept. of Kayachikitsa, Faculty of Ayurveda, IMS, BHU.

<sup>4</sup>Assistant Professor, Dept. of Kayachikitsa, Govt. Ayurvedic College, Nagpur.

<sup>5</sup>Professor, Dept. of Kayachikitsa, Faculty of Ayurveda, IMS, BHU, Varanasi.

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### \*Corresponding Author

**Dr. K. S. Girhepunje**

Junior Resident-III, Dept. of  
Kayachikitsa, Faculty of  
Ayurveda, IMS, BHU,  
Varanasi.

### 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 surgical 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.<sup>[1]</sup> About 90% of patients found to have Ascitis due to Liver Cirrhosis, abdominal neoplasm, CHF or abdominal TB<sup>[2]</sup> and out of which 75% are due to Alcoholic Cirrhosis only.<sup>[3]</sup> 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.<sup>[4]</sup> 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.<sup>[5]</sup> 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<sup>[6]</sup> and Micronodular Cirrhosis.<sup>[7]</sup> In Ayurvedic classisc, Ascitis due to Alcoholic Liver Cirrhosis can be correlated to *Jalodar* as a complication of *Yakritodar*.<sup>[8]</sup> *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*<sup>[9]</sup> (Anorexia). So it is very important to provoke appetite. *Vardhaman Pippali* is indicated in *Plihodar*<sup>[10]</sup> and can be given in *Yakritodar* as symptoms and treatment of both is same.<sup>[11]</sup> *Acharya Sushrut* and *Acharya Charak* have advocated to go for *Shastrakarma*<sup>[12]-[13]</sup> (~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 2<sup>nd</sup> 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.**

Visited to private hospital on 3/1/15	<b>Relevant Past Medical History and Interventions:</b> 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.		
<b>Visits</b>	<b>Complaints at the time of visit</b>	<b>Diagnostic Tests</b>	<b>Interventions</b>
Patient was admitted in MKC on 6/1/16	1. Breathlessness, Altered sensorium since 2 weeks 2. Yellowish discolouration of sclera and skin, Gross abdominal distension, Edema over both lower limbs since 3 months 3. Nausea, Vomiting, Anorexia, Malaise since 4 months.	Blood investigations had done on <b>5/1/2016</b> at private hospitals <b>CBC:</b> Hb- 8.8gm/dL, Platelets- 61000/cu.mm, TLC-11,000 and DLC – within normal limits <b>LFT:</b> 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 <b>RFT:</b> Creatinine- 1.7mg/dL, Urea- 40mg/dL, Serum electrolytes were within normal limits <b>RBS-</b> 108mg/dL <b>Hepatitis profile:</b> HbsAg, Anti HAV	1. Paracentesis 2. Nebulization with Duoline & Budecot every 6 hourly For initial 2 days  VPR oral therapy was started from 8/1/16 with Diet regimen of cow milk

		<p>IgM, Anti HEV IgM, Anti HCV were Negative</p> <p><b>Autoimmune Hepatitis profile</b>(18/11/15): AMA, ANA, LKM-1 were negative</p> <p><b>Quantiferon TB Gold:</b> Negative</p> <p>PT-INR: Test- 31.5sec, control- 13sec, INR-2.88</p> <p><b>Ascitic Fluid R/M:</b> Transudate in appearance, White cell count- WNL, Protein 2.4g/dL, ADA- 15IU/L</p> <p><b>USG Abdomen</b>(19/11/15): Mild hepatomegaly with diffuse grade 2 fatty infiltrations and coarse echotexture, portal vein dilated and mild splenomegaly 16.8cm</p>	
He was discharged on request on 12/1/16	No any complaints (Abdominal distension, Swelling on both legs were reduced)	<p>Blood investigations (CBC, LFT, RFT) were advised but not done by patient</p> <p><b>CT Abdomen</b>(9/1/16) at S.S.Hospital, BHU: Antero-posterior diameter of liver was 15cm, left lobe atrophy with compensatory hypertrophy of right lobe and gross ascitis with mild splenomegaly</p>	VPR therapy continued as previous
1 <sup>st</sup> visit on 21/1/16 due to complaints	<p>Burning sensation in abdomen, burning micturition, Fever (Abdominal distension, Swelling on both legs were reduced)</p> <p>R/S- Chest was clear, no crepitations were present</p>	<p>Blood investigations had done on <b>20/1/2016</b> at private lab of Varanasi</p> <p><b>CBC:</b> Hb- 9.7gm/dL, Platelets- 78000/cu.mm, TLC- 11950 and DLC – within normal limits</p> <p><b>LFT:</b> Total bilirubin- 3.1mg/dl, direct bilirubin- 1.9mg/dl, Total protein- 6.9g/dl, albumin- 2.2g/dl, ALT- 27U/L, AST- 84U/L, ALP- 184U/L</p> <p><b>RFT:</b> Creatinine- 1.9mg/dL, Urea- 60mg/dL, Serum electrolytes were within normal limits</p> <p><b>Urine R/M:</b> 2-3 Epithelial cells, no proteins,</p> <p><b>USG Abdomen</b>(20/1/16): Mild hepatomegaly with fatty infiltrations and moderate Ascitis</p>	<p>VPR therapy continued as previous with tapering dose by 1 Pippali</p> <p>Oral Kamdudha ras 250mg twice a day for 3 days</p>
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	<p>VPR therapy continued as previous</p> <p>Oral Kamdudha ras 250mg and Honey for local application at oral cavity twice a day for 3-4 days</p>
5 <sup>th</sup> visit on	Generalised weakness, mild	Investigations were lost by the patient	VPR therapy

29/2/16	abdominal distension		continued as previous
6 <sup>th</sup> visit on 16/3/16	Dizziness, dryness of mouth BP- 112/70mmHg, PR-78/min	Blood investigations were done on <b>15/3/2016</b> at S.S.Hospital, BHU <b>CBC:</b> Hb- 10.2gm/dL, Platelets- 96000/cu.mm, TLC- 10840 and DLC – within normal limits <b>LFT:</b> 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 <b>RFT:</b> Creatinine- 1.7mg/dL, Urea- 45mg/dL, Serum electrolytes were within normal limits <b>USG Abdomen(15/3/16):</b> Mild hepatomegaly with changed nodular echotexture and moderate Ascitis	VPR therapy continued as previous  Honey for local application at oral cavity twice a day for 3-4 days
7 <sup>th</sup> visit on 28/3/16	Burning micturation, mild feverish (Abdominal distension, Swelling on both legs were reduced)	Blood investigations had done on <b>28/3/2016</b> at S.S.Hospital, BHU <b>CBC:</b> Hb- 11.2gm/dL, Platelets- 95000/cu.mm, TLC- 10900 and DLC – within normal limits <b>Urine R/M:</b> 2-3 Epithelial cells, no proteins	VPR therapy continued as previous  Trinpanchamul Kashay 40mL twice a day till burning sensation vanishes
Last visit on 7/4/16	Generalised weakness, (Abdominal distension, Swelling on both legs were vanished, improved appetite)	Blood investigations had done on <b>6/4/2016</b> at S.S.Hospital, BHU <b>CBC:</b> Hb- 11.8gm/dL, Platelets- 110000/cu.mm, TLC- 9300 and DLC – within normal limits <b>LFT:</b> 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 <b>RFT:</b> Creatinine- 1.5mg/dL, Urea- 32mg/dL, Serum electrolytes were within normal limits <b>USG Abdomen(7/4/16):</b> Mild hepatomegaly with coarse echotexture and no free fluid in peritoneal cavity	VPR therapy continued as previous
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.	<b>Cause of death:</b> 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 3<sup>rd</sup> 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 budesonide (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 monitoring.

After 2 days, patient became oriented and was planned for Oral VPR Therapy for one year in 3 stages.

1<sup>st</sup> day: 3 *Pippali*(crushed) boiled in cow milk twice a day orally

2<sup>nd</sup> day: 4 *Pippali* twice a day with same method

3<sup>rd</sup> day: 5 *Pippali* twice a day with same method

4<sup>th</sup> day: 6 *Pippali* twice a day with same method

5<sup>th</sup> day: 7 *Pippali* twice a day with same method

Then dose of 7 *Pippali* twice a day was continued for next 10 days. From 16<sup>th</sup> day, dose was tapered by one *Pippali* per day upto 3 *Pippali* as per above schedule followed by no oral VPR for next 10 days. This was a schedule 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 micturition 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 Ascites 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.**

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

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

<b>20/1/16</b>	Mild hepatomegaly with fatty infiltrations and moderate Ascitis
<b>15/3/16</b>	Mild hepatomegaly with changed nodular echotexture and moderate Ascitis
<b>6/4/16</b>	Mild hepatomegaly with coarse echotexture and no free fluid in peritoneal cavity

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

	5/1/16	20/1/16	15/3/16	6/4/16
TLC (per cu.mm)	11,000	11,950	10,840	9300
Hemoglobin (gm/dL)	8.8	9.7	10.2	11.8
Platelets (per cu.mm)	61,000	78,000	96,000	1,10,000
Sr.Total Bilirubin (mg/dL)	3.2	3.1	2.9	1.6
Sr.Direct Bilirubin (mg/dL)	2.1	1.9	1.7	0.8
Sr.Total Protein (mg/dL)	6.8	6.9	7.2	7.1
Sr.Albumin (mg/dL)	2.1	2.2	2.9	3.2
Sr.ALT (mg/dL)	22	27	35	32
Sr.AST (mg/dL)	92	84	56	52
Sr.ALP (U/L)	155	184	160	143
Sr.Creatinine (mg/dL)	1.7	1.9	1.7	1.5
Sr.Urea (mg/dL)	40	60	45	32

### Intervention Adherence & Tolerability

It was very difficult to console the patient that he must 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**

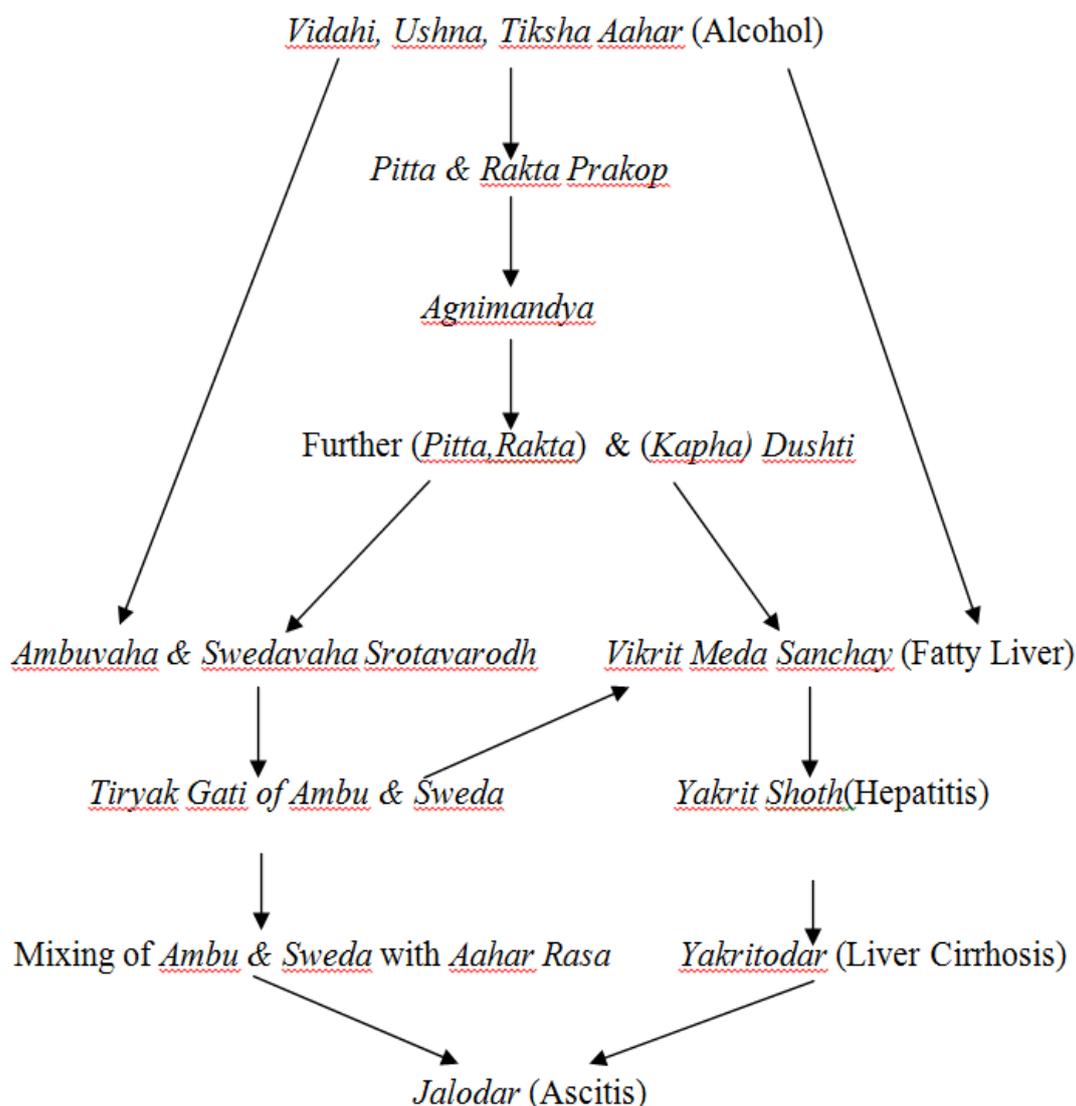
There is only conservative management in modern science for Cirrhotic Ascitis as Paracentesis which lead to recurrence of Ascitis. So *Ayurvedic* management aims at removal of fluid along with protection of hepatocytes. Integrative Paracentesis and Oral *VPR* Therapy showed significant effect by this 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<sup>[14]</sup> due to alcoholic cause (a) Alcoholic Fatty Liver (b) Alcoholic Hepatitis and (c) Alcoholic Cirrhosis. It's pathogenesis can be correlated to Samprapti (pathogenesis) of *Jalodar* in *Ayurveda* given below.

Pathogenesis of Ascitis in terms of Ayurveda<sup>[15]</sup>

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*<sup>[16]</sup> (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<sup>rd</sup> 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.<sup>[17]-[18]</sup> Principle of treatment in *Jalodar* is oral administration of *Tikshna*, *Ushna*, *Dipaniya* and *Kaphahar* medication<sup>[19]</sup> along with cow milk<sup>[20]</sup> in diet. Dry *Pippali* has all such properties. It is a *Katu Rasatmak*, *Tikshna* and *Ushna Virya dravya* so help to improve appetite.<sup>[21]</sup> Hepatoprotective activity<sup>[22]</sup> and rejuvenation<sup>[23]</sup> 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 erythropoiesis, thrombopoiesis and albumin formation. *Nitya Virechana* is recommended in all types of *Udar*.<sup>[24]</sup> *Virechana* is internal cleansing therapy and detoxify the body and opens various channels (Srotas)<sup>[25]</sup> of it. In *Jalodar*, *Ambuvaha* and *Swedavaha* srotas are blocked.<sup>[26]</sup> So *Virechana* opens the closed channels and help to reduce Ascitis. *Pippali* has *Rechan* property<sup>[27]</sup> and *Vardhaman Pippali* pattern lead to 5 to 6 motions per day.<sup>[28]</sup> 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 5<sup>th</sup> 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 *Kamdudha ras* for burning sensation each time intermittently and advised to take cow milk as much as possible. Also he was asked to apply honey<sup>[29]</sup> 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*<sup>[19]</sup> along with cow milk<sup>[20]</sup> 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<sup>[22]</sup> and rejuvenation<sup>[23]</sup> 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.

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