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Case Study

ROLE OF *NITYA VIRECHAN* AND *PATHYA* IN CIRRHOTIC ASCITES

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ABSTRACT

Cirrhosis of liver is an imperceptible disease due to any of the various long standing liver diseases which gradually leads to scarring of the liver tissues. This scarring or fibrosis is the consequence from the healing of the injured liver cells. Cirrhosis is the most common cause of ascites and accounts for almost 85% of all cases. Cirrhotic ascites thus accounts as one of the major complications of Liver cirrhosis. Ayurvedic medicines give a promising result in the management of such decompensated liver disease. We hereby report a successfully managed case of a 53 year old male patient suffering from Cirrhotic Ascites. Post complication with hepatorenal syndrome and hepatogenous diabetes the patient was advised for liver and kidney transplant, as also his ascitic tapping was done multiple times; however he approached for alternative therapy in form of Ayurvedic medicine and was healed. Ascites can be correlated to *Jalodar* in Ayurvedic concept so the treatment protocol included the exclusive Ayurvedic treatment of *Nitya virechan* i.e., constant purgation mentioned as the principle treatment for *Jalodar* along with other supportive medications for liver malfunctioning. Therapeutic diet played an important role for the treatment. During and after treatment no adverse effects were found moreover patient was at comfort with the treatment and the distension of abdomen was negligible, patient also stopped taking insulin, while swelling was reduced remarkably.

INTRODUCTION

The term ascites denotes the pathologic accumulation of fluid in the peritoneal cavity where in normal condition there is no accumulation of fluid in males and around 20ml in females depending upon the stage of their menstrual cycle.[2] The most common cause of ascites is portal hypertension secondary to chronic liver disease which accounts for 80% of patients with ascites.[3] After the development of Cirrhotic ascites, only 50% of patients survive for 2 to 5 years.[4] The formation of ascites in the cirrhotic patient is caused by a complex chain of pathophysiological events involving portal hypertension and progressive vascular dysfunction.[5]

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Decompensated cirrhosis causes portal hypertension which leads to development of ascites and bleeding from esophagogastric varices whereas loss of hepatocellular function results in jaundice, coagulation disorders, and hypoalbuminemia and contributes to the causes of porto-systemic encephalopathy.[6] In a study it was seen that Ten years after diagnosis of Cirrhosis, the probability of developing decompensated cirrhosis and the survival probability rate were 58 and 47%, respectively.[7] The conventional treatment in patients who develop complications of liver disease and become decompensated is considered for liver transplantation.[8]

In Ayurveda, ascites is correlated to *Jalodar* mentioned as a subtype of *Udara Roga*. *Nitya virechan* i.e., continuous purgation keeping on the diet of milk alone can make one get cured of *Jalodar*.

Patient Information

A fifty three year old male Indian had presented to the OPD with a diagnosed case of cirrhosis of liver since almost a decade involuted with ascites and other numerous complications. On his first visit he initially complained of distension of abdomen with feeling of heaviness, decreased urine with dribbling micturition and frothy appearance, swelling at the peripheries, weakness, fatigue, lethargy, breathlessness on exertion and unsatisfactory evacuation.

Patient had portal hypertension. On inquisition it was found that the patient also suffered with upper GI bleeding for which he underwent endoscopy and was found to have oesophageal varices. As for treatment his Endoscopic variceal ligation was done and no bleeding henceforth was noticed. He had mild ascites since 2018 but the need for ascitic tapping multiple times was felt only after July 2021. He underwent abdominal tapping of total three times every fifteen days before visiting us. Simultaneously he developed hepatorenal syndrome and hepatogenous diabetes for which he started taking insulin. He even suffered from Covid 19 due to which he felt all the more weak, lethargic. He had anaemia and his Hb dropped to as low as 4.0gm% for which he underwent blood transfusion twice. Since the beginning of the disease patient had low platelet counts.

Clinical Findings

On examining the patient seemed a bit confused and acted aggressive at some point opposite to a drowsy episode with difficulty to rouse him. His GCS was 13 and looked pale, malnourished with distended abdomen. The BMI of patient was 28.2, his blood pressure was found to be raised 150/90mm Hg though was on antihypertensive drugs, and heart rate was normal 74/min. His RBS on admission was 124 and patient was on Inj. HIR 2 units thrice a day and Inj. Lantus 10 Units once a day.

On inspection, abdomen was distended; there was no engorgement of veins but the abdominal surface appeared shiny. Patient appeared pale and exhausted. The increased abdominal girth was clearly noted with bulging flanks; there was peripheral edema, signs of malnourishment and muscle wasting.

On palpation the abdominal wall appeared tensed with bilateral pitting pedal oedema.

On Percussion, shifting dullness and fluid thrill was positive.

Investigation showed thrombocytopenia and leukopenia, raised serum creatinine and blood urea levels.

Ashtavidha Pariksha

Nadi: Vatapittaj
Mutra: Samanya peeta varna
Mala: Amayukta
Jivha: Amayukta
Shabda: Spastha
Sparsha: Anushna
Druka: Samanya
Akruti: Madhyam

Time Line of Case/ Chronological order of complaints and events

Table 1: Time Line of Case

Health events/ Complaints	Duration/ Dates
NAFLD	2013
Portal Hypertension	2017
Upper GI bleeding i.e., oesophageal varices.	2018
Endoscopic variceal ligation	
Hepatorenal syndrome	
Distension of abdomen, feeling of heaviness in abdomen, decreased urine with dribbling micturition and frothy appearance, swelling at the Peripheries, weakness, fatigue, lethargy, breathlessness on exertion, unsatisfactory evacuation.	2019 – 2021 symptoms increased gradually
Hepatogenous Diabetes	Jan 2020
Increase in weight – 106kg	April 2020
Covid 19 +ve	June 2020
Daignosis of Ascitis	Oct 2020
Ascitic tapping - 3 times (at every 15 days interval)	After July 2021
Anaemia/ Hb – 4.0gm%/ Blood transfusion - twice	July – Aug 2021
Approached Ayurvedic management – Patanjali Ayurveda Hospital, Haridwar	August 2021

Time Line of the total no. of days of treatment

Table 2: Time Duration

30 days			
Hospitalization			**1st Follow up**
Admission		Discharge	
Day_1	$Day_2 \rightarrow Day_{13}$	Day_{14}	D30
10/08/21	22/08/21	23/08/21	8/9/21

Diagnostic Assessment

Table 3: Investigations (Diagnostic Assessment)

Investigations		**During the course of admission**			**First Follow up**
		10/8/21	17/8/21	21/8/21	8/9/21
BSL	FBS	160	98	75.40	80
CBC	Hb	-	8.6	11	9.6
	TLC	-	5.65	5.50	6100
	DLC: N L E M B	-	 65.1 20.4 6.5+ 7.6 0.4	 75.30 21.30 2.40 1 0.00	 72 18 06 04 00
	RBC count	-	2.87	3.54	3.02
	Platelet count	30000	35000	30000	45000
	Mean Platelet count	-	-	-	11.3
	Packed cell volume	-	-	33.70	24.7
	HCT	-	24.4	-	-
	MCV	-	85.0	95	81.8
	MCH	-	30.0	31	29.5
	MCHC	-	35.2	32.60	36.1
	RDW	-	-	15.20	16.5
LFT	S.billirubin (T)	-	0.6	0.95	0.69
	S.billirubin (D)	-	0.3	0.30	-
	S.Billirubin (ID)	-	0.3	0.65	-
	S.G.O.T.	-	6.0	40	30.1
	S.G.P.T.	-	6.3	11	13.3
	GGTP	-	-	103	-
	S. ALP	-	127.0	125	121
	S. tot Protein	-	4.8	5.61	4.92
	S. Albumin	-	2.9	3.30	3.07
	S.Globulin	-	1.9	-	1.85
	A/G Ratio	-	1.5:1	1.43	1.66
KFT	B. Urea	237.4	207.4	207	74
	S.Creatinine	5.6	4.96	4.86	2.50
	Sr. Uric Acid	10.3	8.5	7.2	5.95

	Sr. Calcium	5.2	5.3	7.8	9.89
	Sr. Sodium	138.8	138.1	137.2	136.33
	Sr. Potassium	4.6	4.1	4.3	4.21
	Sr. Total Protein	6.4	-	7.2	7.8
	Sr. Albumin	3.8	-	3.7	3.9
	Sr. Globulin	2.6	-	3.0	3.6
	A/G Ratio	1.4:1	-	-	1.3:1
Lipid Profile	Sr. Cholesterol	111	86.0	-	90
PT/ INR	PT	16	-	11.60	-
	Patient value			14.70	-
	PR	-	-	1.27	-
	INR	1.4	-	1.29	-

Assessment of Abdominal girth

Before Treatment

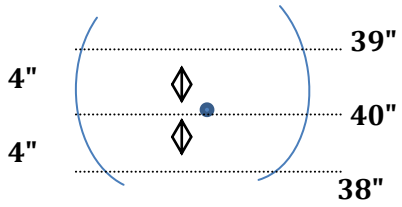

Fig 1. Abdominal girth before Treatment

After Treatment

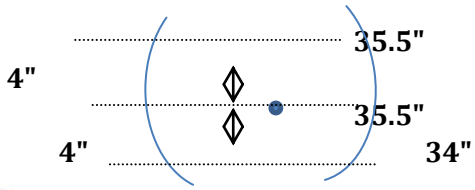

Fig 2. Abdominal girth after Treatment

Table 4: Ayurvedic Treatment

Medicine	**Dose**		**On Discharge**
Kayakalpa kwath *Sarvakalpa kwath* *Vrikkadoshahar kwath*	100ml twice a day	On empty stomach	☑
Kutki churna (100gm) *sweta parpati* (15gm)	1tsf twice a day	After meals	☑ SOS
Vrikkadoshahar vati	2 tab twice a day	Before meals	☑
Livamrit advance *Arogyawardhini vati* *Gokshuradi guggul* *Jalodarari rasa*	2 tab twice a day 2 tab twice a day 2 tab twice a day 1 tab twice a day	After meals	☑
Triphala churna (100gm) *Trivrut avleha* (50 gm) *Aragwadha phalamajja* (50 gm)	10gm + 10gm + 10gm	at 10 am	
Mukta Vati Extra power	2tab twice a day	Before meals	☑
Punarnavadi mandoor	2tab thrice a day	After meals	☑
Kaharva pishti (5gm) *Akika pishti* (5gm) *Jaharmohra pishti* (5gm) *Moti pishti* (2gm)	Twice a day	Before meals	
Tab. *Dengunil*	1tab thrice a day	After meals	☑
Nutrela whey protein	10gm twice a day	Any time of day	☑

Diet recommended during treatment period

Table 5: Diet

Recommended diet (*Pathya*)
Skimmed Cow Milk 300ml/day Preparation- cook 100ml skimmed cow milk with 10ml *Shunthi* powder Dose- 1tsf 100ml TDS
Yusha 120ml TDS (*Lauki, Tinda, Turai, Parwal, Kaddu, Kaccha papita barjhit* with *Hingu, Jeera & Trikatu* Powder)
Soaked Munakka – 100mg/day
Soaked and steam cooked dates – 2-3 /day
Soaked Anjeer – 2-3/day
Flax seeds – 1tsf BD
Total water intake including food – 1.5 litres/day
Patabandhan- All time expect after meals and during sleep

OBSERVATION AND RESULTS

The observation during the whole course is listed in table below.

	Before treatment	**During treatment**	**On Discharge**	**1st Follow up**
Pedal edema	+++	++	+	-
Abdominal girth	40"	Gradual decrease	35.5"	34”
Inj. HIR	2 Units thrice a day	Dose reduced gradually and stopped	Stopped	-
Insulin Lantus	20 unit OD	Dose reduced to 10 unit then stopped gradually Only on *Madhunashini vati*	*Madhunashini vati* 2 BD	*Madhunashini vati* 2 BD
Anti-Hypertensive drug – Cap. Depin	40mg twice a day	Dose reduced gradually and stopped Only on *Mukta vati* 2 tab BD	*Mukta vati* 2 BD	*Mukta vati* 2 BD
General condition	Poor	Fair	good	good
Meld Score	23	-	-	18

The result observed during the whole course of 1month was found to be satisfactory according to the patient condition. The patient took high dosage of allopathic medication which was either stopped or reduced to some extent. There was a tremendous change in the abdominal girth of the patient. On Examination no fluid was felt.

DISCUSSION

The conventional treatment of ascites is paracentesis i.e., excess fluid is removed from the abdominal cavity, but even after removal of this fluid during a course of time the fluid gets refilled and ascites reappears thus this abdominal tapping has to be done again and again. This whole procedure is very inconvenient and uncomfortable for the patient as also not feasible in terms of expenses.

Nitya virechan i.e., continuous purgation keeping on the diet of milk or some diet control is mentioned as the principal treatment for *Jalodar* in Ayurveda along with many other alternatives. Here we followed the same principal keeping the *Roga* and *Rogi bala* in mind and planning the therapy accordingly.

Patient was administered a combination of *Triphala churna, Aragwadhphala majja* and *Trivrut avleha*. All these drugs act as laxative. *Triphala* also shows anti-inflammatory, anti-bacterial and antioxidant properties along with laxative effect. This purgation helps the accumulated water in the abdominal cavity to get excreted via the *Guda marga*.

The patient was given only skimmed cow milk along with vegetable soup prepared with *Lauki, Tinda, Turai, Parwal, Kaddu, Kaccha papita barjhit* with *Hingu, Jeera & Trikatu Powder.* These all are *Supachya* and they provided proper nutrition during the whole course of treatment. It also helped to maintain the raised blood sugar levels.

Pattabandhan i.e., tying a cloth or a belt over the abdominal area helped *Vayuanuloman* and prevented from further distension thus reducing the abdominal distension.

Munnaka i.e., dry raisins were also added in the diet after sugar levels were maintained and Insulin was stopped. It worked as *Balya* i.e., energy booster and prevented the patient from landing into Hypoglycemia as the patient was on total diet restriction

Arogyavardhini vati, Jalodarari rasa, Sarvakalpa kwath, Kayakalpa kwath contain numerous key ingredients that help in the excretion of ascitic fluid and work as hepato-protective drugs. *Kutki, Makoy* and *Punarnava* are the major herbal contents found in most of these drugs. These drugs have hepato-protective, anti-inflammatory, *Agni* stimulant and diuretic properties.

Nutrela Whey Protein was given as a protein supplement to meet the protein demand as there was too much protein loss through fluid excretion.

Divya Livamrit advance contains *Bhumiamla, Bhringraj, Kutki, Giloy, Kalmegh, Makoy, Punarnava, Arjuna, Daru haldi* that work as hepato protective, diuretic and improve renal health, appetite and stimulate digestive fire. [9]

Before the admission of patient the abdominal girth was 40inches which was reduced to 35.5 inches with the impact of *Nitya virechan* during the course of hospitalization.

CONCLUSION

The ultimate treatment of cirrhotic ascites is most of the time liver transplantation, but through Ayurvedic medication one can escape such invasive treatment and prolong one's life.

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