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Case Report
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Ayurvedic management of chronic liver disease with portal hypertension and hepatic encephalopathy - A case report

Abstract

Chronic liver disease (CLD) account for millions of deaths worldwide every year. Hepatitis C virus (HCV), hepatitis B virus (HBV), non-alcoholic fatty liver disease (NAFLD) and alcohol-related liver disease (ALD) are the most common causes of CLD. Ascites, variceal hemorrhage, portal hypertension, infections, acute kidney injury (AKI), hepatorenal syndrome (HRS), spontaneous bacterial peritonitis (SBP), frailty etc are the complications of CLD and associated with liver-related mortality and morbidity. The present case report deals with a patient who has been suffering with CLD and its associated complications including ascites, portal hypertension, edema of lower limbs, diabetes, thrombocytopenic purpura, hepatic encephalopathy, and renal impairment. *Ayurvedic* diagnosis of *Jalodara* has been made and the patient has been treated accordingly. Liv-52 tablets, *Samshamani Vati, Sudarshana Ghana Vati* and *Punarnavashtaka Kwath* have been prescribed for 4 months along with dietary restrictions. Patient got clinically significant improvement that is evident in hematological and biochemical parameters also. *Ayurvedic* treatment is safe, cost effective and seems to be promising in the management of CLD and its complications. To generalize the present study findings, further research with large sample size is required.

Keywords: *ayurveda,* alcohol-related liver disease, ascites, *jalodara*, NAFLD, purpura

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Abbreviations: CLDs, chronic liver diseases; ALD, alcohol-related liver disease; AKI, acute kidney injury; HRS, hepatorenal syndrome; NAFLD, non-alcoholic fatty liver disease; HBV, hepatitis B virus; HCV, hepatitis C virus

Introduction

There has been an increase in the prevalence and incidence of both acute and chronic liver diseases (CLDs) all over the globe compromising the quality of life and causing life threatening complications.[1] Chronic liver disease (CLD) account for millions of deaths worldwide every year. Hepatitis C virus (HCV), hepatitis B virus (HBV), non-alcoholic fatty liver disease (NAFLD) and alcohol-related liver disease (ALD) are the most common causes of CLD. Ascites, variceal hemorrhage, portal hypertension, bacterial infections, acute kidney injury (AKI), hepatorenal syndrome (HRS), spontaneous bacterial peritonitis (SBP), frailty, and acute on chronic liver failure (ACLF) are the complications of CLD that contribute to liver-related mortality and morbidity.[2] In India, liver diseases are recognized as major public health priority. CLDs in India have been increasing progressively since 1980 compared with other countries in Asia. Adoption of a western diet, misuse of alcohol, sedentary habits, and other cultural-lifestyle changes are the causes for a spectrum of liver diseases in India.[3]

Ayurveda (an ancient Indian system of medicine) has been proved efficacious in the management of uncomplicated cirrhotic ascites with betterment in quality of life of the patient. Reversal of liver pathology at cellular level could happen with *Ayurvedic* treatment.[4] Studies have shown that *Ayurvedic* interventions such as *Virechana* (therapeutic purgation), internal medicines, diet, fluid and salt restrictions could improve the clinical profile, liver and renal functions, prothrombin time, hemoglobin etc in ascites patients with decompensated cirrhosis.[5] The present case report deals with a female patient diagnosed as having CLD with multiple complications and/or comorbidities such as portal hypertension, splenomegaly, cholelithiasis with cholecystitis, renal dysfunction, purpura with thrombocytopenia, diabetes mellitus (DM) and hepatic encephalopathy (HE). The patient came to our hospital for *Ayurvedic* treatment with the hope for better and sustained relief. Written informed consent has been taken from the patient for the publication of the present case report and associated images.

Case description

A 55 year old female was presented (06.05.2023) with distension of abdomen, lower limb edema, intermittent fever, urine incontinence, constipation, loss of appetite, purpuric skin lesions on left antecubital fossa and right forearm (Figure 1) since 10.03.2023. The patient came with the diagnosis of CLD with portal hypertension, ascites, splenomegaly, cholelithiasis & cholecystitis, renal dysfunction, thrombocytopenic purpura, DM and HE. The investigation reports have revealed low platelet count, decreased hemoglobin, elevated serum total, direct and indirect bilirubin levels, increased Serum glutamic oxaloacetic transaminase (SGOT) or Aspartate transaminase (AST) levels and decreased serum albumin levels (Table 1) (28.04.2023). USG abdomen and pelvis has revealed mild ascites, coarse and heterogenous liver parenchymal echotexture, relatively enlarged caudate lobe of liver and slightly irregular liver surface indicating chronic liver parenchymal disease; Mild splenomegaly, recanalization of paraumbilical vein, dilated portal vein and splenic vein, multiple dilated collaterals at splenic hilum indicating portal hypertension; small and atrophic ovaries (17.03.2023). Mode of onset was insidious with progressive deterioration of liver functioning. The patient got hospitalized twice (25.03.2023 to 31.03.2023 & 19.04.2023 to 28.04.2023) and received treatment for CLD and HE. Patient has been taking antihypertensives, hepatoprotective, health supplements to improve liver and gall bladder functioning, antibiotics, laxatives and digestion improving medications at the time of visit (06.05.2023). Due to frequent hospitalizations (for the management of HE), high cost of conventional drugs, adverse drug reactions and non-satisfactory relief, patient has opted *Ayurveda* for sustained and better relief.

Table 1 Investigation reports

Investigation	17.03.2023	25.03.2023	26.03.2023	27.03.2023	29.03.2023	30.03.2023	04.04.2023	05.04.2023	16.04.2023	19.04.2023	28.04.2023	08.05.2023	27.08.2023	Units
Liver														
Serum bilirubin total	3.78	5.04		6.54			3.5					2.36		mg/dl
Serum bilirubin conjugate (direct)	1.05	2.59					2				1.9	0.7	0.68	mg/dl
Serum bilirubin unconjugated (indirect)	2.73	2.45					1.5				1.3	1.66	3.37	mg/dl
SGOT/AST	58.7	59.1									42.1		47.48	U/L
SGPT/ALT	41.68													
Alkaline phosphatase							328						227	IU/L
HBsAg	Non reactive	Non-reactive						Non reactive						
Renal														
Blood urea		72.3	78.4	97	102.3	80.2						18.8		mg%
Serum creatinine		1.86	1.82	1.7	1.23	1.01								mg%
Serum uric acid				4.95	7.56									mg%
BUN/Creatinine ratio										32.9				
Lipid profile														
Serum cholesterol				97.6										mg%
HDL				17										mg%
LDL				67.18										mg%
Total/HDL cholesterol ratio				5.74										
Hematological														
Hemoglobin			9.7	10	7.9	8.2	8.3		9		8.4	9.1	8.3	gm%
RBC count			3.35		2.74	2.84	2.86		3.47		2.98		3.3	millions/cu mm
TLC			19080	17350										cu mm
PCV			29.8	31.1	23.2	24.2	25.7		27.9		27.3	28.1	25.6	%
Platelet count			65000	73000	61000	64000	55000		69000		47000	71000	86000	mcL
ESR												33		mm/hr
MCH									25.94				23.6	pg/cell
MCHC											30.77		30.5	g/dL
Serum proteins														
Total protein		5.01			5.35		5.6							gm%
Serum albumin		3.1			2.54		3.1			3.2	3.2	2.3	2.92	gm%
A/G ratio				0.821	0.904								0.63	%
Serum electrolytes														
Sodium				130.3	131.6	131.8	132			133.3				mmol/L
Potassium					3	2.84				3.3				mmol/L
Chloride							95.6							
Bicarbonate		18.9												mmol/L
Others														
HIV I & II		Non reactive												
Anti HCV		Non reactive							Negative					
PCO2		30.7												mmHg
TCO2		20												mmol/L
HEV IgM								Negative						
Serum calcium		8.36		7.9	7.74	8.37	8.2			8.37				mg%
Random blood sugar												250.5		mg/dL
Urine														
Albumin				Trace										
Pus cells				20-25										HPF

#The values shown in blue and red colours denote below and above the normal levels respectively

Subject is a graduate, married, homemaker and has a physically active lifestyle. Subject was non-alcoholic, non-smoker, vegetarian and doesn't have history for any drug abuse. No drug and food allergy have been reported. Patient's medical history, past surgical history, family medical history, social and medication history have not revealed any abnormality. No significant toxicological data of interest has been found. The subject has appeared to be her stated age, hygienic, alert and came by wheel chair. The subject looked pale including conjunctiva without icterus. Subject has protruded or distended abdomen and febrile (body temperature was 100.0 F). Blood pressure was 150/80 mm Hg; body weight was 75 kg; SpO2 level 98%; pulse rate 87/minute; other vital signs were within normal limits. There was no lymphadenopathy. Clubbing of fingers, leukonychia and palmar erythema were absent. Dusky red to violaceous, multiple ecchymotic skin lesions/macules of different sizes with ill-defined borders have been seen on left antecubital fossa and right forearm (Figure 1). Bilateral pitting edema was elicited on dorsum of both feet (Figure 1). The purpuric skin lesions were non progressive and persistent. No epistaxis or bleeding from any natural orifices was found. There was no visible peristalsis and dilated blood vessels over abdomen. Palpation of abdomen has revealed soft, non-tender, with splenomegaly and hepatomegaly. Shifting dullness was elicited on percussion of abdomen.

Based on patient's history, clinical examination findings, and investigation reports, the patient was diagnosed as having CLD with DM, ascites, portal hypertension, anemia, thrombocytopenic purpura, renal impairment, and cholecystitis with cholelithiasis. *Ayurvedic* diagnosis of *Jalodara*/*Yakrutodara* has been made and treatment was planned accordingly. As the patient was not suitable for Shodhana (body cleansing) procedures due to multiple complications/ comorbidities with low physical strength/stamina, only Shamana

(pacifying) treatment has been given along with dietary restrictions. No specific assessments have been done except frequent laboratory investigations (platelet count, liver and kidney function tests, and other hematological & biochemical indices). Takra Pana (intake of butter milk) has been advised along with salt restriction. *Ayurvedic* treatment has been planned and implemented in a flexible way as per the clinical requirement. *Ayurvedic* management protocol aims at improving functioning of the liver and to manage associated complications (Table 2). Patient was treated at OPD (out-patient department) level and assessed frequently (once in a month or as required) during treatment.

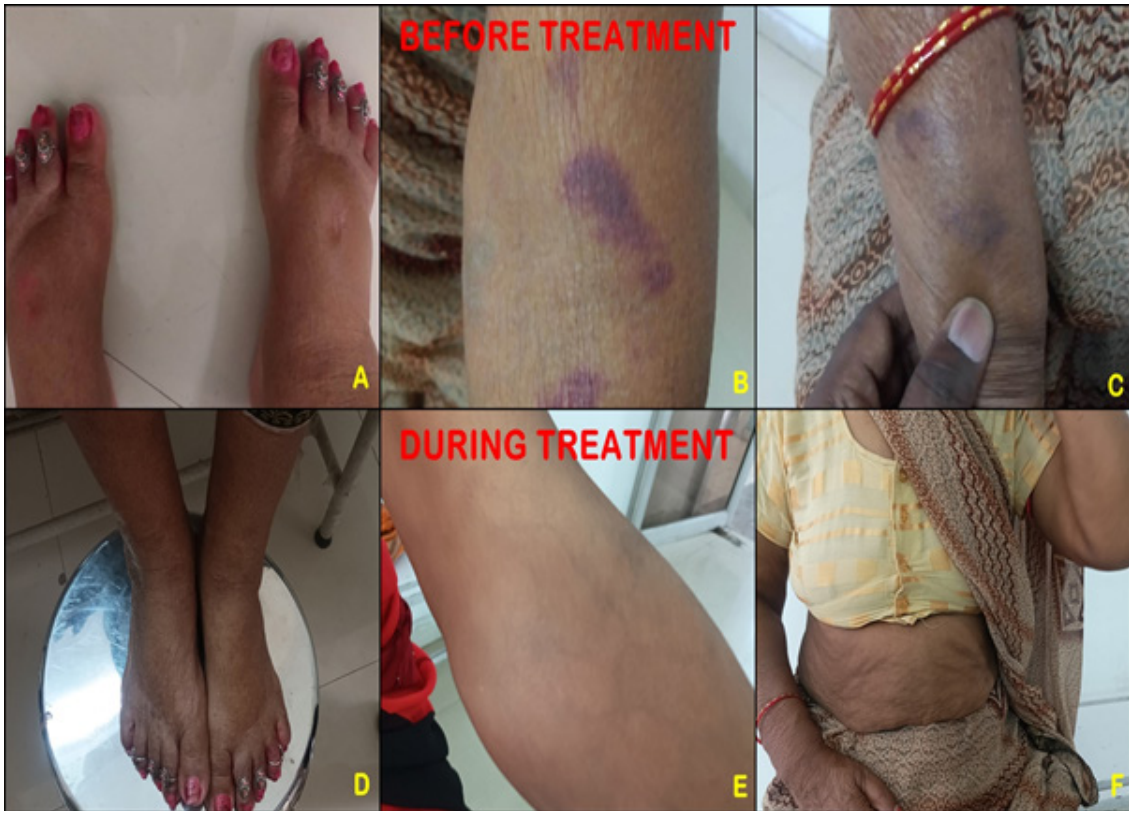

Figure 1 Improvement in clinical signs**.**

A-Pitting edema in both feet; B-Purpuric skin lesions on left antecubital fossa; C-Purpuric skin lesions on right forearm; D-Resolution of pitting edema in both feet; E-Disappearance of purpuric skin lesions on left antecubital fossa; F-Resolution of ascites and purpuric skin lesion on right forearm;

Table 2 Intervention

Duration	Medicine	Dose	Frequency
06.05.2023 to 04.07.2023	1. Liv-52 tablets	500 mg	Twice daily after meals
	2. *Samshamani Vati*	500 mg	Twice daily after meals
	3. *Sudarshana Ghana Vati*	500 mg	Twice daily after meals
05.07.2023 to 02.09.2023	1. Liv-52 tablets	500 mg	Twice daily after meals
	2. *Samshamani Vati*	500 mg	Twice daily after meals
	3. *Punarnavashtaka Kwath*	80 ml	Twice daily before meals

Discussion

Some of the most common etiological factors of CLD such as HBV, HCV, alcohol misuse and hepatocellular cancer (HCC) have been excluded in the present case based on the clinical findings and investigation reports. Diabetes is seen in 11.7% of newly diagnosed CLD patients at the time of diagnosis. Diabetes as a comorbidity is more prevalent in central and western regions of India where NAFLD is also common. In present case also, diabetes has been detected at the time of diagnosis of CLD. CLDs are progressive in nature and clinically unapparent for longer periods. They can become elicited only when liver failure sets in, often associated with the dysfunction of other vital organs.[6] Similarly the patient was unaware of CLD progression until complications like HE manifested.

Portal hypertension is part of a dynamic process triggered by CLD and it is characterized by an increase of portal venous pressure due to end-stage liver disease (ESLD). Complications such as the formation of ascites, HE, and hyperdynamic circulation involving splanchnic and peripheral vessels associated with dysfunction of various vital organs.[7] Portal hypertension and associated pathological findings have been observed in the present case also. HE is a reversible

syndrome of impaired functions of the brain often seen in patients with CLD/ESLD. It denotes brain atrophy or a reversible metabolic encephalopathy or edema of the brain or any combination of these conditions. It is highly recurrent and associated with poor survival rate. Disorientation, inappropriate behaviour, acute confusional states, somnolence, agitation, stupor and finally coma may develop in patients with HE. Hyponatremia is also a risk factor for the development of HE in CLD patients.[8] In present case also the patient got hospitalized twice due to severe bouts of HE associated with hyponatremia.

Ascites comes under the broad spectrum of *Udara Roga* (diseases of abdomen) in *Ayurveda*. *Udara Roga is a multifactorial disease*. Elaborate description is available regarding medical and surgical management of Ascites in *Ayurvedic* literature. Diet restriction plays an important role in the management Udara Roga. *Ayurvedic* management include improving appetite & digestion, daily therapeutic purgation (Nitya Virechana), hepatoprotective drugs and dietary specifications in Ascites. [9] Ascites can be correlated to *Jalodara* in *Ayurveda*.[5] The present case was diagnosed as *Jalodara* and Udara Roga Chikitsa (treatment) has been implemented. Dietary restrictions for salt and sweets have been implemented. Takra Pana is advised. Nitya Virechana has not been done due to Avara Bala (low physical strength/stamina) of the patient. Treatment was focused on mainly to relieve the signs and symptoms and to improve the functions of the liver. The patient has been taking *Ayurvedic* medicines since 4 months (from 06.05.2023 to 02.09.2023). Treatment protocol has been changed once according to the requirement (Table 2).

Liv-52 tablets have been prescribed to improve the liver functions and also to manage associated complications. Liv-52 is a polyherbal formulation consisting of *Mandura Bhasma* (incinerated sludge iron), *Tamarix gallica* and herbal extracts of *Cichorium intybus, Capparis spinosa, Terminalia arjuna, Solanum nigrum,* and *Achillea millefolium*. Liv-52 is having diuretic, anti-oxidative, anti-inflammatory and immunemodulatory properties. It is known to decrease ascites, serum AST and ALT and has hepatoprotective effect in cirrhotic patients.[10] In present case also similar improvement has been observed in liver function tests (Table 1). *Samshamani Vati* and *Sudarshana Ghana Vati* have been prescribed to tackle fever and for improving liver functions. *Samshamani Vati* is also known as *Guduchi Ghana Vati* and it is prepared from the aqueous extract of *Tinospora cordifolia*. *Tinospora cordifolia* possesses antimicrobial, anti-oxidant, antitoxic, anti-diabetic, hepatoprotective, and immunomodulatory properties.[11]

Sudarshana Ghana Vati is a polyherbal formulation used in the management of fever, malaria, liver and spleen diseases. *Kiratatikta (Swertia chirata*) is the major ingredient (50% of the total proportion of the ingredients).[12] *Shamshamani Vati* and *Sudarshana Ghana Vati* both act as an immune booster and in infectious diseases.[13] Fever got completely relieved with these medications in the present case. Punarnavashtaka Kwath (PK) is quoted in Bhaishajya Ratnavali and it is used in hepatic disorders. It consists of *Punarnava* (*Boerhaavia diffusa* Linn), *Katuki* (Picrorhiza kurroa Royle ex Benth), *Guduchi* (*Tinospora cordifolia*), *Shunthi* (*Zingiber officinalis* Rosc*), Daruharidra* (*Berberis aristata* DC), *Haritaki* (*Terminalia chebula* Retz), *Nimba* (*Azadirachta indica* A. Juss), and *Patola* (*Tricosanthes dioica* Roxb) plants. Due to its antioxidant effects, it protects hepatocytes from CCl_4-induced liver damage in rats. PK has shown significant hepatoprotective effect. PK has decreased serum AST, ALT, alkaline phosphatase, and bilirubin levels and increased serum protein levels in rats.[14] PK has been prescribed in present case to manage pitting edema, portal hypertension, ascites and to improve liver functions.

The patient has been taking *Ayurvedic* treatment from 06.05.2023 to 02.09.2023 (and continuing) and in between treatment was revised once on 05.07.2023. The patient has been taking medicines regularly from 06.05.2023 to 02.09.2023 (and continuing). Fever has subsided and good improvement was reported in urinary incontinence and constipation. The purpuric skin lesions on left antecubital fossa and right forearm were completely disappeared within three days of *Ayurvedic* treatment (from 06.05.2023 to 08.05.2023) (Figure 1). Pitting edema in the both feet and edema of lower limbs were also completely disappeared within the first 3 days of *Ayurvedic* treatment (Figure 1). Good relief has been observed in distension of abdomen/ ascites. Only mild improvement in appetite has been reported. The patient was able to walk and got the ability to do her daily activities (patient came by wheelchair before starting *Ayurvedic* treatment and unable to do her daily activities). Some improvement has been observed in the liver function tests as shown in Table 2 (08.05.2023). No recurrence of bouts of HE reported during *Ayurvedic* treatment (from 06.05.2023 to 02.09.2023). Serum bilirubin levels (total, direct and indirect) were maintained around the upper normal limit. Good improvement was observed in renal function tests and serum electrolytes. Blood sugar levels were still high and platelet count has been reached 71000/mcL (Table 1). Other haematological and biochemical parameters were almost stable without further deterioration though they were not within a normal range.

No adverse effects have been reported with any *Ayurvedic* formulations taken by the patient during treatment period. The patient got satisfactory improvement with *Ayurvedic* medicines within first 3 days especially in purpuric skin lesions, constipation, fever, urine incontinence and pitting edema of lower limbs. The patient was stable with improved quality of life (though not measured quantitatively) and satisfied with her condition. *Ayurvedic* treatment seems to be promising in the management of CLD and its associated complications such as thrombocytopenic purpura, ascites, portal hypertension, recurrent bouts of HE, impaired liver and kidney functions etc. *Ayurvedic* medicines used in the present work are cost effective also.

Conclusion

The present case report deals with a patient who has been suffering with CLD and its associated complications including ascites, portal hypertension, edema of lower limbs, diabetes, thrombocytopenic purpura, hepatic encephalopathy, and renal impairment. Liv-52 tablets, *Samshamani Vati, Sudarshana Ghana Vati* and *Punarnavashtaka Kwath* have been prescribed for 4 months along with dietary restrictions. Patient got clinically significant improvement that is evident in hematological and biochemical parameters also. *Ayurvedic* treatment is safe, cost effective and seems to be promising in the management of CLD and its complications. Further research is required to authenticate the present study findings.

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None.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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