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Case Report

Successful Ayurvedic Management of Alcoholic Liver Disease: A Case Report

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Abstract

Alcohol is the major cause of liver disease. Liver is an important and largest gland of the human body, it detoxifies various metabolites, synthesize various proteins and produces various biochemicals necessary for digestion. Alcoholic liver disease could be due to long term alcohol consumption, including fatty liver, alcoholic hepatitis, and chronic hepatitis with liver fibrosis or cirrhosis. Fatty liver is present in more than 90% of daily as well as binge drinkers. Although fatty liver will develop in any individual who consumes a large quantity of alcoholic beverages over a long period of time but this process is transient and reversible if timely care is taken. The prognosis of severe alcoholic liver disease is very poor, the mortality of patients with alcoholic hepatitis concurrent with cirrhosis is nearly 60% at 4 years. The conventional treatment mainly includes steroid therapy in alcoholic hepatitis, fluid tapping for ascites and in end stage cirrhosis of liver with last choice of liver transplantation. However, Ayurveda has options to liver transplantations. According to Ayurveda it can be correlated with *Yakridalyodara*. The treatment protocol mentioned in Ayurveda is mainly *Nityavirechana* (Medicated purgation) for this disease along with various oral medications. Here is a case report of a diagnosed case of Alcoholic liver disease, presented with main symptoms: nausea, vomiting, swelling in bilateral foot, weakness in the body, reduced appetite, gradually weight reduction and semisolid stool with frequency of 6-7 times/ day. Patient had history of Reduced appetite and frequent vomiting. Treatment was done on the basis of Ayurvedic principles and after 2 months of treatment, there was found significant recovery of the patient subjectively as well as objectively. After 7 months of treatment patient got complete improvement, and free from any complications and follow-up of patient is still going on regularly.

Keywords: Alcoholic liver disease, Ascites, *Jalodara*, *Nityavirechana*, Hepatoprotective Drugs.

INTRODUCTION

ALD may take the form of acute involvement (alcoholic hepatitis) or chronic liver disease (steatosis, steatohepatitis, fibrosis and cirrhosis). The severity and prognosis of alcohol-induced liver disease depends on the amount, pattern and duration of alcohol consumption, as well as on the presence of liver inflammation, diet, nutritional status and genetic predisposition of an individual[i]. Their progression also depends on the pattern of alcohol intake at mealtimes results in a lower risk of liver disease than consumption at other times; fitful, intermittent drinking is less dangerous than a continuous consumption of alcohol[ii]. Liver is the main organ site where alcohol metabolism takes place. The Alcohol Metabolism [iii] and Pathogenesis of inflammatory changes in alcoholic liver disease is described well[iv]. Alcohol is metabolized in the liver in three ways: (1) by the enzyme alcohol dehydrogenase (ADH); (2) by cytochrome P-4502E1 (CYP2E1); and (3) by mitochondrial catalase. Only the first two pathways are of practical significance-ADH finds use in the degradation of limited quantities of alcohol, while alcohol-induced CYP2E1 takes place in excessive alcohol intake [v]. Both enzymes convert alcohol to acetaldehyde, which is in part responsible for the liver injury too. However, the process of liver injury is much more complex resulting from biochemical, genetic, cellular, immunological and humoral disorders in connection with the intake and metabolism of excessive quantities of alcohol. A major role is played there by oxidative stress (which is mainly due to alcohol-induced CYP2E1), by simultaneous shortage of antioxidants in the hepatocytes and, last but not least,

by acetaldehyde alone and altered balance of many cytokines-mainly tumor necrosis factor (TNF)-α [vi]. Alcohol intake of more than 50 ml per day by men will have high risk of ALD.[vii] Alcoholic Hepatitis (AH) is a syndrome of necro- inflammation of liver due to heavy alcohol intake and occurs in 10-35% of such patients. Patients with more severe AH usually present with fever and signs of hepatocellular failure such as jaundice, ascites, encephalopathy and cirrhotic changes presents with jaundice, ascites and peripheral edema[viii]. Jaundice is usually due to failure of hepatocytes to excrete bilirubin resulting into conjugated hyperbillirubinemia.

CASE HISTORY

A 35-year-old, non- hypertensive, non-diabetic, male patient approached the Kayachikitsa OPD on 5 july 2019, with chief complaints of nausea, vomiting, generalized weakness, loss of appetite, gradual weight reduction and semisolid stool with frequency of 6-7 times/ day and tremors in hand since 1 month.

On Examination

General examination reveals lean and thin built of the patient. On examination, weight was 45 kg, pulse rate was 88/minute and blood pressure was 110/70 mm Hg.

On inspection

No abdominal distension was observed. No visible veins, Umblicus centrally placed.

On palpation

Abdomen was soft with mild tenderness in right hypochondrium region and hepatomegaly - 2 finger was present.

On percussion

Dull note was present over the area extended right lobe of liver.

The USG abdomen revealed Hepatomegaly. Liver function tests presented with elevated SGOT, SGPT. The Satva (mental status) of the patient was Avar and Samhanana (Body compactness) was of Madhyama. The patient Prakriti was Vata Pradhana Pittaja.

Past History

Patient had history of Reduced appetite and nausea and frequent vomiting since 1 month. He was known alcoholic and was consuming minimum 180-360 ml alcohol approx. daily since 3 years. Cessation of alcohol for 10 days during treatment but later on he again continued alcohol consumption. He could not withdraw from alcohol consumption. his parents and family members got worried and at last get him for the ayurvedic treatment as the ray of last hope. So Patient visited us for Ayurvedic management. This condition is compared with Yakriddalyuara in Ayurveda[ix].

Diagnosis

In past history it is clear that patient had the excessive intake of food(excess of Spicy,nonveg,) and drink(alcohol) having Ushna, teekshna, katu, amla, viruddh [x] properties causes vitiation of pitta and this excess of pitta vitiates the Rakta dhatu (blood) and this vitiated blood along with excess of pitta [xi] → seated between twak and mamsa dhatus of body → due to impure vitiated rakta dhatu [xii] →Increases the size of spleen and liver (yakritdalyodara)[xiii],[18] → Ultimately if not controlled it may leads to jalodara (ascitis). Based on clinical presentation and examination with radiological and laboratory tests; the patient was diagnosed with Jaundice, Alcoholic liver disease with moderate ascites.

Treatment adopted

From 05-07-2019 to 29-07-2019

S. No.	Drug name	Dose	Anupana
1	Syrup Himcocid	10 ml TDS before meal	-
2	Syrup Liv 52	10 ml TDS after meal	-
3	Arogyavardhini Vati	250 mg BD after meal	Luke warm water
4	Drakshasava	30 ml BD after meal	Equal amount of water

From 30-07-19 to 29-08-2019

S. No.	Drug name	Dose	Anupana
1	Syrup Himcocid	10 ml TDS before meal	-
2	Avipattikar churna	5gm TDS after meal	Luke warm water
3	Cap. Cytozen	2cap. BD after meal	
4	Sutshekhar Ras	250 mg BD after meal	Water
5	Dhanyak hima	Throughout the day	

From 30-08-2019 to 26-09-2019

S. No.	Drug name	Dose	Anupana
1	Syp Liv 52	10 ml TDS	
2	Arogyavardhini Vati	250 mg BD after meal	Luke warm water
3	Cap. Cytozen	1cap. BD after meal	
4	Sutshekhar Ras	250 mg BD after meal	Water
5	Ashwagandharishta, Draksharishta	20 ml each after meal	Equal amount of water

Further the above last medication was continued for a period of 2 months and then investigations done which shows the LFT values within normal range. Then patient advised to follow the healthy diet and counselling done properly to avoid intake of alcohol. Patient is on regular follow up having no withdrawal symptoms.

DIET PLAN DURING TREATMENT

Along with above medications, restricted diet[xiv] which included 1 liter of boiled milk per day in divided doses was advised in diet as it is Sadya Santarpaka (Instant source of energy), Snigdha Virechaka (Mild Laxative) and is a good source of protein. 250 ml of Mudga Akrita-Yusha[xv] twice daily is indicated as per the status of agni of the patient. Mudga also is rich source of protein and easily digested. This diet was followed by patient for 5 months with some alteration according to the condition of the patient like some fruits like apple, papaya was also advised.

OBSERVATIONS AND RESULTS

Table-1: Objective parameters

Parameters	Before starting Ayurvedic treatment on 5.07.2019	After 18 days of treatment (30.07.2019)	During treatment 13.09.19	During follow up 01.10.19	During follow up 04.12.19
Haemoglobin (gm/dl)	11.9	-	-	10.8	10.0
TLC (per cumm)	11,100	-	-	9,800	9,300
Total Bilirubin (mg/dl)	0.89	-	-	0.96	0.92
Direct Bilirubin (mg/dl)	0.32mg/dl			0.36	0.28
Indirect Bilirubin(mg/dl)	0.57mg/dl			0.60	0.64
SGOT(IU/L)	340.7	116.64	115.82	71.45	38.16
SGPT(IU/L)	510.6	157.02	89.56	69.56	48.35
Alkaline phosphates (IU/L)	82	-	-	- 78	
Total Protein(gm/dl)	6.16	-	-	-	6.01
HbsAg	Negative	-	-	-	-
USG	USG abdomen revealed Hepatomegaly (mild)	-		-	NAD

DISCUSSION

The treatment principle of Udara Roga is Nitya Virechana and Deepana Chikitsa. Nitya Virechana was planned to eliminate the Samchita Dosha (Accumulated Toxins). As the patients is of Madhyama Bala, (moderate strength), Avara Satva (low mental Strength) and Kapha-Pitta Prakriti so Haritaki churna was administered along with Luke warm water.

Haritaki churna contains Haritaki (Terminalia chebula) which have thermogenic, purgative, antioxidant, adaptogenic, antiprophylactic, antipyretic, hepatic stimulant and hepatoprotective[xvi] properties.

Guduchi has hepato-protective properties that prevents fibrous changes and promotes regeneration of parenchymal tissue[xvii].Guduchi plays an important role in normalization of altered liver functions (ALT, AST)[xviii].

Cap. Cytozen contains 56.25 mg of Mandur bhashma, 112.5mg of Arogyavardhani rasa, 67.5mg each of Kakamachi (Solanum nigrum Linn.) fruit and Chitraka (lumbago zeylanica Linn.) root, 112.5mg each of pericarp of Triphala (Terminalia chebula Retz., Terminelia bellerica (Gaertn.) Roxb. And Embelica officinalis Linn.), 337.5mg each of Punarnva (Borhavia diffusa Linn.) root, Kumari (Aloe barbadensis (L.)

Burm.f.) leaf, Kasni (Cichorium intybus Linn.) seed, Katuki (Picrorhiza kurrooa Royle ex. Benth) rhizome, Bhunimba (Andrographis paniculata (Burn.f.) Wall.) whole plant. Punarnva (Boerhaavia diffusa) is preferred in the management of Shotha (Swelling), and Pandu (Anemia) [xix]. It also has Hepato-protective Properties and help in decreasing albuminuria and increasing serum protein [xx]. It also exhibits anti-inflammatory activity, thus help in modulating inflammatory responses[xxi].

Katuki possesses hepato-protective anti-viral and anti-oxidants activities[xxii]. Kalmegha (Bhunimba) is the potent drug, which reverses the altered hepatic biochemical parameters [xxiii]. Amalaki has hepato-protective activity and helps in elevating the serum protein levels[xxiv].

Amalaki possess antioxidant activity and could be an important dietary source of Vitamin C, which is a powerful water-soluble antioxidant [xxv] and helps in increasing iron absorption from the gut[xxvi]. One study on Pippali suggests that, piperine gets absorbed very quickly across the intestinal barrier through the intracellular pathway. It may modulate membrane dynamics due to its easy partitioning thus helping in efficient permeability across the barriers [xxvii]. In addition, Pippali is said to be bio-availability enhancer of the drug[xxviii], which further helps in easy assimilation of the drug components. Hence, it counteracts poor digestion and absorption usually reported in patients of geriatric anemia. It has also immunomodulator activity[xxix]. Therefore, it improves general health and immunity vigor, luster of the skin etc. in patients having anaemia.

Mandura Bhasma (incinerate form of iron-Fe_2O_3), the main component is the activator of the formulation and is the chief responsible component for the pharmacodynamics of Punarnava Mandura. The ferric and ferrous fractions of Mandura provide sufficient amount of iron to the living matter, which is needed for normal erythropoiesis [xxx]. Upon administration of (Nimba) Azadirachta indica, it stabilizes the levels of Serum glutamate oxaloacetate transaminase (SGOT), Serum Glutamate Pyruvate Transaminase (SGPT), Alkaline Phosphatase (ALP), Serum bilirubin and elevates total protein amount. Thus, this plant clearly notifies the improvement of the functional status of liver cells [xxxi]. The various experimental studies on Curcuma longa has shown its different activities such as hepatoprotective, Anti-inflammatory, Anti-cancerous, hypo-lipidemic, Gastro-protective, Hypoglycemic and Anti-Alzheimer effect. It is a cholagogue, stimulating bile production in the liver and encouraging excretion of bile via the gallbladder[xxxii].

Berberin a known compound from berberis aristate plant, shows anti-hepatotoxicity, immunomodulator activity[xxxiii]. With the intervention of Nitya virechana (Daily therapeutic purgation), Shamana (conservative treatment) and Maanasika chikitsa for strengthen the mental status by proper counselling. Alcohol intake was withdrawn, appetite was improved after 5 months of completion of treatment, the condition of the patient was good. Appetite and general health were improved. Significant reduction in Liver function markers i.e. SGOT and SGPT were observed.

CONCLUSION

Looking to the response of the present case and also similar other cases, with integrative management yields excellent results as compared to palliative and symptomatic conventional treatment alone. Along with Ayurvedic systematic classical approach with support of essential conventional medicines to manage the acute symptoms will be more productive in terms of better management and prognosis of CLD/ALD.

Abbreviations: ALD- alcoholic liver disease, USG-Ultrasonography, CECT-contrast-enhanced computed tomography, SGOT- Serum Glutamic Oxaloacetic Transaminase, SGPT- Serum glutamic pyruvic transaminase , GGT- gamma-glutamyl transpeptidase, PT- Prothrombin time , INR- international normalized ratio , TLC- Total leucocyte count , cm- Centimeter, Kg- Kilogram, gm- gram, ml- milliliter, BID- Bias a day, am- Anti Meridiem, pm- Post Meridiem, TID-thrice a day, Cap.- Capsule, Tab. Tablet, CBC-Complete Blood Count, HbsAg -Hepatitis B surface antigen, Hep C- hepatitis C, mg- milligram, dl-Deciliter, Cumm-cubic milimeter, L- liter, IU-international units, Sec- Second, HPF- 'High Performance Fortran.

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