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A CRITICAL REVIEW ON ALCOHOLIC LIVER DISEASE AND ITS MANAGEMENT

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ABSTRACT

Alcohol is one of the most common causes of chronic liver disease worldwide, with consumption continuing to increase in many countries.[1] Patients with alcoholic liver disease (ALD) may also have risk factors for other liver diseases (e.g. coexisting NAFLD or chronic viral hepatitis infection), and these may interact to increase disease severity.[1] Alcoholic liver disease (ALD) is defined by histological lesions in the liver that can range from simple hepatic steatosis to more advanced stages such as alcoholic steatohepatitis, cirrhosis, hepatocellular carcinoma and liver failure.[2] ALD is still a leading cause of liver related morbidity and mortality rising constantly worldwide. There is no universally accepted theraphy to prevent or reverse ALD.[2] *Ayurveda* being the oldest healing science has a better role includes rejuvenation, regeneration, detoxification, and lifestyle modification which helps to stop the progression of Alcoholic liver diseases and prevent them. It includes like use of hepatoprotectives, hepatic stimulant, virechana, *Rasayana* drugs, *Tikta* and *Madhura Rasayukta* drugs, *Mootrala* drugs and protein supplementation has better outcome. *Ayurveda* vividly described liver diseases in the context of '*Yakrit roga'* in classical texts. There is no description of *Yakrit vikara* as a separate chapter in *Ayurvedic* classics, only *Bhavprakash* mentioned it as a separate chapter.[3]

KEYWORDS: Simple hepatic steatosis, alcoholic steatohepatitis, cirrhosis, hepatocellular carcinoma, *Rasayana drugs, Mootrala drugs, Yakrit vikara.*

INTRODUCTION

Alcohol is a psychoactive substance and has been widely used in many cultures for centuries. The prevalence of ALD has increased in the last years, parallel with the increasing alcohol consumption in the western world as well as in Asian countries.[4] The threshold for developing ALD is intake of more than 80 g/day for more than 5 years in men and intake of more than 40 g/day for more than 5 years in women.[1] The average alcohol consumption of a man with Cirrhosis is 160 g/day for over 8 years.[1] Abstention from alcohol may reverse the early stage of ALD to a normal condition. The treatment for ALD with conventional medicines, mainly pharmaceutical medications, has limited success with side-effects. Recently natural medicines, which mainly apply herb-derived agents, are emphasized as alternative therapies to manage the various alcohol-related liver diseases. According to *Ayurveda*, liver is root of *Raktavaha srotas* and *pitta* is by-product of *Rakta*, so excess intake of Alcohol, sour and salty foods increases *pitta* and damage the architecture of *Raktavaha srotas* and create chronic liver disease and complications.[5] *Samhana* or physical propionate of individual part of body is an indication of healthy liver and body. The *kaphaja prakriti* people are more susceptible to fatty liver whereas *pitta prakriti* people are more susceptible to ALD.[5]

AIMS AND OBJECTIVES

To study about Alcoholic liver disease and its correlation in detail.

MATERIAL AND METHODS

1. Classical text books of Ayurveda.
2. Text book of modern science.
3. Previously published research article.

VYUTPATI

The word ''Mada'' signifies intoxification while "Atyaya" means excess so Madatyaya literally translates to ''exceesive intoxication" or "alcoholism".

DEFINITION

As per *Sharangdhara Samhita purva khanda* 4/22, *Madya* is a drug possessing *Tamoguna* predominantly and cause dearrangement of mind are called *Madyakar.*

''Madatyaya roga'' in Ayurveda refers to a condition directly translating to ''alcoholism'' essentially describing the detrimental health effects on body due to exceesive consumption of alcohol which is considered as Tridoshaja vyadhi in ayurvedic texts.

***NIDANAS* (Sushruta Samhita uttaratantra 47/15-16)**

AAHARAJA NIDANA

- *Avidhi madhyapana, Nirjala Madyapana* (Inappropriate consumption of alcohol or without water)
- *Matraadhikaya madhyapana* (Excessive consumption of alcohol without considering matra, bala and vidhi)
- *Niranna koshta Madhyapana* (consuming alcohol with empty stomach)
- *Kshuda* (consumption of alcohol during hunger will be absorbed quickly)
- *Ajeernaavastha* (consuming alcohol during indigestion)
- *Krisha-shareera* (lean body)
- *Pramitashana* (habit of eating any one taste dominated food only increases vata dosha)

VIHARAJA NIDANA

- *Vyayama, bharavehan and udhvagamana* (consumption of alcohol after heavy exercise or after carrying heavy weights or after exceesive walking)
- *Vegaavarodha* (consumption of alcohol after suppressing the natural urges)
- *Ushna, Grishma ritu* (consumption of alcohol during summer season or after person exhausted by heat)
- *Pitta prakarti* (consumption of alcohol by person having *pitta prakarti*)

MANSIKA NIDANA

Person afflicted from *krodha* (anger), *bhaya* (fear), *shoka* (grief) or *alpa satwa* (who have less control over their mind).

RISK FACTORS OF ALD[1]

- **Drinking pattern**:- Liver damage is more likely to occur in continuous rather than intermittent or 'binge' drinkers as this pattern gives the liver a chance to recover. It is therefore recommended that people should have at least two alcohol-free days each week. The type of beverage does not affect risk.
- **Gender**:- The incidence of ALD is increasing in women who have higher blood ethanol levels than men after consuming the same amount of alcohol. This may be related to the reduced volume of distribution of alcohol.
- **Genetics**:- Alcoholism is more concordant in monozygotic than dizygotic twins. While polymorphisms in the genes involved in alcohol metabolism, such as aldehyde dehydrogenase, may alter drinking behaviour. The patatin-like phospholipase domain-containing 3 (PNPLA3) gene, also known as adiponutrin, has been implicated in the pathogenesis of both ALD and NAFLD.
- **Nutrition**:- Obesity increases the incidence of liver-related mortality. Ethanol itself produces 7 kcal/g (29.3 kJ/g) and many alcoholic drinks also contain sugar, which further increases the calorific value and may contribute to weight gain. Excess alcohol consumption is frequently associated with nutritional deficiencies that contribute to morbidity.

Table 1: **Amount of alcohol in an average drink.[1]**

Alcohol type	% Alcohol by volume	Amount	Units* 1 unit = 8 gram
Beer	3.5 9	568 ml 568 ml	2 4
Wine	10 12	125 ml 750 ml	1 9
Alcopops	6	330 ml	2
Sherry	17.5	750 ml	13
Vodka/rum/gin	37.5	25 ml	1
Whisky/ brandy	40	700 ml	28

ETIOPATHOGENESIS

Excessive consumption of *Madya* with inadequate diet and all other causative factors leads to vitiation of *tridosha* causing *annavaha srotodushti* and *agnimandhya* leads to production of *Apachita* and *Dushita* aahara rasa which causes dhatu *shaithilya* and *ojo vikrutti*. The *tikshana, ushna* and *amla gunas* of *Madya* causes *vata dushti* and *Ruksha* and *laghu* guna of *Madya* vitiate *vata* dosha leading to *Majja dhatu kshaya*. Excessive alcohol consumption causes *Meda dhatu kshaya* which leads to *balahani* (loss of strength). When *pitta dushti* occurs causes inflammation and a reduction in absorption of nutrients especially vitamin B1 (Thiamine) and other metabolic abnormalities. When *vata dushti* occurs causing symptoms of nutritional deficiency and weakness etc. When it inflicts *Manovaha srotas,* where *satva guna* is reduced and *raja- tama guna* increases due to alcohol consumption produces symptoms of *vikshaya* as *updrava*.

PATHOPHYSIOLOGY[1]

Alcohol reaches peak blood concentrations after about 20 minutes, it may be influenced by stomach contents. It is metabolised almost exclusively by the liver via one of

two pathways.

Approximately 80% of alcohol is metabolised to acetaldehyde by the mitochondrial enzyme, alcohol dehydrogenase. Acetaldehyde is then metabolised to acetyl-CoA and acetate by aldehyde dehydrogenase. This generates NADH from NAD (nicotinamide adenine dinucleotide), which changes the redox potential of the cell. Acetaldehyde forms adducts with cellular proteins in hepatocytes that activate the immune system, contributing to cell injury.

The remaining 20% of alcohol is metabolised by the microsomal ethanol- oxidising system (MEOS) pathway. Cytochrome CYP2E1 is an enzyme that oxidises ethanol to acetate. It is induced by alcohol, and during metabolism of ethanol it releases oxygen free radicals, leading to lipid peroxidation and mitochondrial damage.

The release of endotoxin into the blood because of increased gut Permeability, leading to release of tumour necrosis factor alpha TNF-a) and interleukin 1 (IL- 1), IL-2 and IL-8 from immune cells. All of these cytokines have been implicated in the pathogenesis of Iiver fibrosis.

Stages of ALD[9] are

The *yakrit vridhi* is a pathological process by vitiation of *dosha*, decrease *agni*, production of *ama* and obstruction of *srotasa*. Excess intake of fat diet and progressively developed obesity, fatty dietary habit less physical exercise leads to increase blood lipid and vitiated fat metabolism, which produce more free fatty acids (FFA), decrease digestive power of fat tissues and all *tridosha* are aggrevated and localized in liver. Due to increase in unctuous property and decrease of hot property triggered to produce more *kapha* and free fatty acids leads to liver enlargement, which may correlate as steatosis.

Hepatic FFA directly block the channels and portal hypertension resulted as blockage of intra and extra hepatic duct leads to accumulation of bile and jaundice. Increased accumulation of *pitta* due to hot and liquid properties triggered fibrosis. Excess consumption of alcohol increased *pitta* and reduces watery part and vitiated *vata* to reduce the size of liver mass. Vitiated *pachaka pitta* due to hot property effect the functions of *ranjaka pitta*, increased pitta that evaporate the watery content and damage the architecture of liver, hence degeneration and dryness of liver stated. Due to dry property vitiated *vata* further may change the parenchyma of liver leads to liver cirrhosis.

CLINICAL FEATURES[10]

ALD has a wide clinical spectrum, ranging from mild abnormalities of LFTs on biochemical testing to advanced cirrhosis. There are mainly 3 types of ALD:-

Alcoholic fatty liver (Alcoholic steatosis)

- Asymptomatic.
- Occasionally, discomfort in right upper quadrant with tender hepatomegaly, nausea and jaundice
- Progression to cirrhosis uncommon.
- Modest elevations of AST. ALT and GGTP
- Occasionally, elevated bilirubin.
- Elevated triglycerides, cholesterol.
- Ultrasound shows fatty liver
- Biopsy if done shows accumulation of fat in perivenular hepatocytes and later in entire hepatic lobule
- It has a good prognosis and cessation of alcohol for 3 months results in normalisation of pathological changes.

Alcoholic Hepatitis (Alcoholic Steatohepatitis)

- Many asymptomatic
- Fever, rapid onset of jaundice, abdominal discomfort and proximal muscle wasting
- Hepatomegaly.
- Features of chronic liver disease like spider angiomata, palmar erythema, chapped lips and gynaecomastia
- In severe cases, portal hypertension, ascites and variceal bleed can occur without cirrhosis
- Non hepatic manifestations of alcohol toxicity including polyneuropathy, cardiomyopathy and a history of pancreatitis may be present.
- AST and ALT elevated usually <400 IU.
- AST:ALT ratio >2
- Elevated bilirubin.
- Mild increase in alkaline phosphatase
- Reduced albumin
- Prolonged prothrombin time.
- Leucocytosis, elevated C-reactive protein.
- Biopsy shows ballooning degeneration of hepatocytes with leucocyte infiltration. Mallory bodies often present.

- Potentially reversible but many progresses to cirrhosis.
- Poor prognosis Maddrey discriminant function >32. 4.6 × (patient's prothrombin time - control prothrombin time) + serum bilirubin.

Alcoholic cirrhosis

- Cirrhotic changes is characterised by widespread necrosis of liver cells and extensive fibrosis that distorts hepatic architecture and leads to obstruction of blood flow which eventually leads to portal hypertension and hepatocellular damage. As a result of it, ascites, jaundice, hepatic encephalopathy, oedema, coagulopathy and metabolic abnormalities occurs.
- Alcoholics safe limits of alcohol are 200 g and 140 g of alcohol per week in males and females respectively.
- 10 g of alcohol equals 30 ml of whisky, 100 ml of wine and 250 ml of beer.

- Ingestion of alcohol 180 g/day increases the risk of developing cirrhosis 25 times.
- Signs of hepatocellular failure are

1. Jaundice (due to failure of bilirubin metabolism and intrahepatic cholestasis)
2. Ascites (due to portal HTN and hepatic insufficiency)
3. Diminished body hair, gynaecomastia and Testicular atrophy (due to hyperoestrogenism)
4. Spider naevi (due to arteriolar changes induced by hyperoestrogenism)
5. Palmar erythema (due to increased peripheral blood flow)
6. Dupuytren's contracture (due to fibrosis of palmar aponeurosis caused by local microvessel ischaemia and platelet and fibroblast derived growth factors that promote fibrosis.)
7. Clubbing (due to development of pulmonary arteriovenous shunts leading to hypoxaemia)
8. White nails (due to hypoalbuminaemia)
9. Anaemia (can occur due to nutritional deficiency of vitamin B12 and folate; direct bone marrow suppression by alcohol; haemolysis due to hypercholestrolaemia on RBC membrane)

Table 2: Child-Pugh Score or Child-Turcotte-Pugh Score for severity of liver disease.

Parameter	Point 1	Point 2	Point 3
Encephalopathy	None	1-2	3-4
Ascites	None	Mild or controlled with diuretics	Moderate despite diuretics
Prolongation of prothrombin time (in secs)	<4	4-6	>6
Serum albumin (g/dl)	>3.5	2.8-3.5	<2.8
Serum bIlirubin(mg/dl)	<2	2-3	>3

CP class A: Points 5-6; CP class B : 7-9; CP class C: >9 (range 5-15)

Investigations

- **Complete blood picture**

1. Anaemia
2. Leucopenia and thrombocytopenia (due to bone marrow suppression by alcohol)
3. Acanthocytosis spur like projections on RBC

- **Liver function test**

1. Serum albumin decreased (due to impairment of hepatic protein synthesis)
2. Serum globulin increased (stimulation of reticuloendothelial system)

- **Transaminases**

1. SGOT raised
2. SGPT raised but less than 300 units
3. AST:ALT ratio is more than 2
4. Alkaline phosphatase may be mildly raised.

- **Prolonged prothrombin time** (due to reduced synthesis of clotting factors)
- **Raised blood ammonia** (due to diminished hepatic clearance)
- **Metabolic abnormalities** (like hyponatraemia; hypokalemia)
- **Ultrasonographic examination**

1. Liver size small and coarse echotexture
2. Macronodules
3. Hypertrophied caudate lobe
4. Portosystemic collaterals
5. Ascites

- **Fibroscan** (to determine amount of fibrosis)
- **Liver biopsy** confirms cirrhosis.

MANAGEMENT

Life long abstinence is the best advice and is effective at preventing progression, hepatic decompensation and death once cirrhosis is present.

Cognitive behavioral therapy (CBT) and medications called benzodiazepines can ease withdrawal symptoms in a person with alcohol dependency. People with severe alcohol dependency may stay at an inpatient rehabilitation facility for closer monitoring. Ongoing therapy may then be required to prevent a relapse into drinking alcohol. Medications can also prevent relapse, such as: acamprosate, vivitrol (naltrexone), topamax (topiramate), baclofen and disulfiram. Corticosteroids or pentoxifylline may help reduce inflammation in people with acute alcoholic hepatitis. Other medications that show potential for treatment and are currently being studied include: probiotics and antibiotics; stem cell therapy and medicines that target the inflammation pathway. The treatment for ALD with conventional medicines, mainly pharmaceutical medications, has limited success with side-effects.

Recently natural medicines, which mainly apply herb-derived agents, are emphasized as alternative therapies to manage the various alcohol-related liver diseases.

Nidana parivarjana

It is the first and foremost way to treat any illness. Consumption of alcohol over prolonged period is the prime causative factor of alcoholic liver disease. Cessation of alcohol consumption is the single most

important treatment and prognostic factor. Life-long abstinence is the best advice.

Diet restrictions[11]

Salt and sour foods should be avoided as it increases *pitta*. Milk is a complete food of all nutrients and easily digestible is a diet of choice. *Mamsa rasa* can be maintained as protein supplementation for non-vegetarian patients. Intake of water should be limited to 800-1200 ml. *Takra* and *Laja Manda* may be the diet of choice to balance *pitta* in stomach and reduces acidity. *Yakrit kshaya* patients should eat 6-8 times in small quantity as their *agni* is low.

Lifestyle modifications

Pranayama (breathing exercise), *asana* (yogic posture) and *dhyana* (meditation) are very popular in management of ALD. In this phase, *Ashtanga yoga* helps patients build strong will power that helps them achieve sobriety by balancing the mind and body while executing various positions *(asana*).

Regular practice of the asanas like *Bhujangasana, ardha Matsyendrasana, dhanurasana, trikonasana, balasana* etc. helped the patient by improving neurobehavioural energy, decrease craving for alcohol, decrease peer pressure and decrease withdrawal symptoms so practicing yoga and *dhyana* helps in prevention and progression of ALD.

Shodana chikitsa

Shodhana can be done in *bahu dosha avastha*, considering *dosha avastha* and *bala* of patient. Emesis, purgation, and letting of blood are to be done.

Virechana acts by producing lightness in body, elimination of dosha, *agni deepana, vayu anulomana* and *srotoshodhana.*

Vamana helps in pacification of *Kapha, Vata* and *Meda* helpful in reducing weight, total cholesterol levels and serum triglycerides levels as per condition of a patient, purification therapy can be implemented.

In *Ayurveda*, liver problems are mainly associated with an imbalance of *pitta dosha*, so *nitya virechana* is considered a therapeutic method to pacify *pitta*. *Nitya virechana* can stimulate the digestive system, which can be beneficial for patients with ALD and helps in cleansing the body toxins accumulated in liver and also eliminates excess fluid accumulated in abdominal cavity due to liver damage.

Shamana chikitsa[12,13]

Single drugs and their actions

Katuka (Picorrhiza kurora)	Immunomodulator, hepatoprotective, hypolipidaemic, antioxidant, anti-inflammatory
*Guduchi (*Tinospora cordifolia)	Hepatoprotective and hepatostimulative liver tonic
Dronapushpi (Leucas aspera)	anti-inflammatory, analgesic, *rasayana* properties, *deepana, pachana*
Punarnava (Boerhavia diffusa)	Improves metabolism, diuretic, antioxidant
Moolaka (Raphanus sativus*)*	Detoxification, anti-inflammatory, *rasayana*
Alabu (Water gourd)	Antioxidant, anti-inflammatory
Haritaki (Terminalia chebula*)*	Detoxification, *rasayana*, improves metabolism
Vibhitaki (Terminalia bellarica*)*	Antioxidant, detoxification, anti cancer
Amalaka (Emblica officinalis)	Anti oxidant, anti inflammatory
Patola (Trichosanthus dioca)	Detoxification and improves metabolism
Bhunimba (Androgarphis paniculate*)*	Hepatoprotective, *rasayana*, antioxidant, anti-inflammatory
Kharjura (Phoenix dactylifera)	Anti hyperlipidaemic, improves digestion
Kakamachi (Solanum nigrum)	Liver stimulant, anti oxidant, balance *tridosha*, lipid lowering agent

Formulations used in ALD with their actions

Phalatrikadi kwatha	Hepatoprotective, detoxification
Kumari asava	*Deepana, pachana, vata-kaphaghna*
Triphala tablets	Hepatoprotective, anti- hyperlipidaemic, detoxification, anti-inflammatory
Arogyavardhini vati	*Deepana, pachana, tridoshaghna,* detoxification
Kakamachi swarasa	Liver stimulant, anti oxidant, balance *tridosha*, lipid lowering agent
Guduchi kwatha	Hepatoprotective and hepatostimulative liver tonic
Shweta parpati	Diuretic, anti-inflammatory, analgesic
Punarnava mandora	*Rasayana*, anti-inflammatory, *pittaghna*
Triphala mandora	Hepatoprotective, anti- hyperlipidaemic, detoxification, anti-Inflammatory
Pravala panchamrita rasa	*Vata-kaphaghna,* indigestion, diuretic
Mukta panchamrita rasa	For treating jaundice
Pravala pishti	Anti-inflammatory, diuretic
Mukta pishti	Anti-inflammatory, anti-psychotic, lipid lowering agent
Godanti bhasma	*Rasayana, deepana, pachana*
Rohitakarishta	For treating *kamala, yakrit vriddhi*
Dronapushpi swarasa	anti-inflammatory, analgesic, *rasayana* properties, *deepana, pachana*
Punarnava swarasa	*Rasayana*, anti-inflammatory, *pittaghna*
Kharjuradi mantha	Anti hyperlipidaemic, improves digestion

Rasayana chikitsa

Rasayana acts by enhancing body natural defense mechanism, promoting cellular rejuvenation, longevity and enhancing tissues structural integrity and resilience. Rasayana drugs having Deepana, *Pachana*, *Lekhana* property like *Triphala*, *Guggulu* etc can be given.

Shastra chikitsa

The role of liver transplantation in the management of ALD remains controversial. It requires a 6 month period of abstinence from alcohol before a patient is considered for transplantation. The outcome of transplantation for ALD is good and if the patient remains abstinent there is no risk of disease recurrence.

***UPDRAVA* (Sushruta Samhita uttaratantra 47/22)**

Hikka associated with *jwara*, *vamathu*, *vepathu*, *Parshwashoola*, *kasa* and *Bhrama*.

***ASADHYA LAKSHANAS* (Sushruta Samhita uttaratantra 47/22)**

- *Heepottaraushtiham* (Pressing of lower lip inside)
- *Atisheetam Amandadaaham* (Feeling of cold outside and burning inside the body)
- *Tailaprabhaasyam* (oiliness on face)
- *Jihva, ushtiha, dantamasitamvaaneel* (blackish or bluish discoloration of tongue, lips and teeth)
- *Peeta-nayana rudhirataa* (yellowish or reddish discoloration of eyes).

DISCUSSION

Acharya charaka has also given the properties of *madya in sutrasathana chapter 24* which are *laghu* (lightness), *ruksha* (dryness), *tikshna* (penetrating), *ushna* (hot), *sukshma* (diffusing), *amla* (sour), *vyayama* (absorbs without undergoing digestive changes), *asukari* (fast acting), *vikashi* (breaks internal tissue barriers) and *vishada* (cleaning the channels). The *pachaka pitta* has *dravyatva* vitiation due to excess intake of *madya*, it effects *ranjaka pitta* in liver. The *ushnaguna* of *ranjaka pitta* reduces *kapha/meda* and vitiate *vata* and cause *yakrit kshaya*. Alcohol is described as *amritam*, when consumed adequately considering *bala, matra, vidhi* and with wholesome diet; if consumed improperly provides intoxication symptoms in body. 80% of alcohol is mostly absorbed in intestines and metabolized in liver and shows toxic effect on liver if consumed excessively by producing hepatotoxicity. Drugs having *tridosha shamaka* effect and which increases *oja* and *bala* having *deepana, pachana, anulomana, virechana,mutrala, raktavardhak, raktashodhak, rasayana* properties, hepatostimulant drugs supplementation of protein will be used in treating Alcoholic liver disease.

CONCLUSION

Common medicinal plants that we consume daily for maintaining liver health are garden mint (pudina), coriander (dhaniya), cumin seeds (jeera), asafoetida (*hingu*), turmeric (*haridra*), ginger, garlic etc. Diet, exercise and medicinal treatment using herbs is an essential component of *Ayurveda*. A well balanced diet, good sleep and healthy lifestyle which includes doing regular exercise, meditation and *pranayama* is important for healthy liver. *Shamana* theraphy or specific *panchkarma* therapies will be administered based on individuals dosha imbalance and severity of the condition it helps in decrease progression of alcoholic liver disease.

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