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# NON-ALCOHOLIC FATTY LIVER DISEASE AND ITS MANAGEMENT- A CRITICAL REVIEW

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# ABSTRACT

Public health is facing an increasing problem due to the harmful impacts of non-alcoholic fatty liver disease (NAFLD), as diabetes and obesity are becoming more common place globally. In the Western world, non-alcoholic fatty liver disease (NAFLD) is most prevalent. Metabolic diseases such as central obesity, dyslipidemia, hyperglycemia, and chronic abnormalities in liver function tests are intimately linked to non-alcoholic fatty liver disease (NAFLD). Generally, NAFLD is a term used to describe a wide range of liver disease, including fibrosis, inflammatory processes, and hepatocellular injury. *Ayurveda* being the oldest healing science has vividly described liver diseases in the context of '*Yakrit roga'* in classical texts. NAFLD can be interpreted as a '*Santarpanjanya vikara'* with *kapha medho dushti* and its *sthanasamsraya* in *Yakrit*, which is said to be *Raktavaha srotomoola* and *Pittasthana*.<sup>[1]</sup> There is no description of *Yakrit vikara* as a separate chapter in *Ayurvedic* classics, only *Bhavprakash* mentioned it as a separate chapter. Description of *Yakritdalyodara* is found while describing *Pleehodara* in the *Brihat trayees*.<sup>[1]</sup> The few suggested evidences for practice and production more information to help stop the progression of fatty liver diseases and to prevent them.

KEYWORDS: Yakrit roga, Santarpanjanya vikara, Kapha medho dushti, Yakritdalyodara, Pittasthana.

# INTRODUCTION

Ayurveda is considered by many scientists to be the oldest healing science. In Sanskrit, Ayurveda means "The Science of Life." Ayurvedic knowledge originated in India more than 5,000 years ago and is often called the "Mother of All Healing".<sup>[2]</sup> The concept of Ayurveda is to promote health and prevention of disease, and Ayurveda in daily life aims at maintaining harmony between nature and the individual to ensure optimal health.<sup>[2]</sup> Now a days, due to modernization and urbanization, peoples move towards the comfort and sedentary lifestyles, they are particularly much prone for getting life style disorders.<sup>[3]</sup> On the basis of etiopathogenesis, lifestyle disorders can be correlated with Santarpanajanya Vyadhi caused mainly due to vitiation of Kaphadosha and Medodhatu in terms of their vriddhi. Modern science has their limitation to manage the lifestyle disorders and their treatments are cost effective.<sup>[3]</sup> Ayurveda, with its comprehensive range of medicines, can cure the disease at an early stage and prevent serious complications.<sup>[4]</sup> Non-alcoholic fatty liver disease (NAFLD) consists of steatosis and nonalcoholic steatohepatitis (NASH). Steatosis is the accumulation of fat in the liver, and steatohepatitis is a condition with inflammation. NAFLD is a growing epidemic worldwide due to increasing obesity, with

prevalence in the general population ranging from 11.2% to 37.2%.<sup>[5]</sup>

# AIMS AND OBJECTIVES

To study about NAFLD 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.

# AYURVEDIC CONCEPT AND MODERN CONCEPT

# NIDANA

In *Ayurveda*, etiological components can be explained under *beejadushti* (genetic factors), *aharaja hetu* (dietary factors), *viharaja hetu* (habit factors) and *manasika hetu* (psychogenic factors).

## Beejadushti (Genetic factors)

यदां रिजया दोषप्रकोपणोक्ता-मासेवमानाया दोषाः प्रकुपिताः शरीरमुपसर्पन्तः शोणितगर्भाशयावुपपद्यन्ते, न च काञ्येन शोणितगर्भाशयौ पन्ति तदेयं गर्भ लभते रुजीः तदा तस्य गर्भस्य मातृजानामवयवानामन्यतमोऽवयवो विकृतिमापद्यत एकोऽधयाऽनेके, यस्य यस्य हावयवस्य बीजे बीजभागे वा दोषाः प्रकोपमापद्यन्ते, तं तमवयवं विकृतिराविशति । (cha.sh. 4/30) When a woman uses aggravating factors, the doshas get vitiated and in course of spreading reach the ovum and uterus but donot affect them entirely. She conceives but the foetus gets damaged in one or more maternally derived organs; that part is affected with morbidity in the gene, wholly or partially, of which the doshas are vitiated. When in its ovum the gene concerned with uterus is damaged, the progeny becomes sterile; when a part of this gene is affected, a putrified child is born.

# Aharaja hetu (Dietary factors)

संतर्पयति यः रिजन्धेर्मधुरेर्नुरुपिच्छितैः। नवान्नैर्नवमहद्यैश्च मासैश्वानूपवारिजैः ॥ गोरसैगौडिकैश्वान्नै पैष्टिकैश्वातिमात्रशः । (ch. Su.23/3,4)

One who saturates himself excessively with unctuous, sweet, heavy, slimy substances, new cereals, fresh wine, meat of marshy and aquatic animals, milk and its products, jaggery and flour preparations.

# Viharaja hetu (Habit factors)

चेष्टाद्वेषी दिवास्वप्नशय्यासनसुखे रतः। (Cha. Su. 23/4)

*Cheshtadveshi* (Person who dislike any kind of activity) *Divaswapna* (habituate day sleep)

Shaya asana sukha rati (comfort in sitting at one place for long time)

# Manasika hetu (Pshchogenic factors)

\* मात्रयाऽप्यभ्यवहृतं पथ्यं चान्नं न जीर्यति । चिन्ताशोकभयक्रीधदःखशय्याप्रजागरैः ॥(cha. Vi. 2/9)

As per *Charak acharya*, even though the food is taken in proper quantity, according to *Ahara Vidhivisheshayathana*, will not be digested properly if the person is affected by worry, grief, fear, anger, uncomfortable bed and vigil.

# Causes of NAFLD as per modern Genetics

Approximately 7 categories of genes have been associated with NAFLD.

- hepatic lipid export/oxidation in steatosis (PNPLA3, TM6SF2, NR112, PPAR-alpha, PEMT, MTTP, APOC3 and APOE);
- glucose metabolism and insulin resistance (ENPP1/IRS1, GCKR, SLC2A1, GOAT, TCF7L2 and PPARG);
- steatosis-hepatic lipid import or synthesis (SLC27A5, FADS1, and LPIN1);
- steatohepatitis-oxidative stress (HFE, GCLC/GCLM, ABCC2 and SOD2);
- steatohepatitis-endotoxin response (TLR4 and CD14);
- cytokines (TNF and IL6); and
- fibrosis (AGTR1 and KLF6)<sup>[6,7]</sup>

# **Dietary Factors and lifestyle**

Diet has been thought of as an independent risk factor for the development of NAFLD, specifically, a diet high in fats.<sup>[8]</sup> It has been shown, through energy restriction and manipulation of dietary macronutrients, namely, restriction of carbohydrates, fat, or enrichment with monounsaturated fatty acids, that dietary modifications can reduce metabolic syndrome.<sup>[9,10]</sup> In a retrospective study, cigarette smoking was found to be an independent risk factor for the onset of NAFLD.<sup>[11]</sup> The use of tobacco predisposes a person for the development of insulin resistance.<sup>[12-14]</sup>

# Metabolic syndrome and Type 2 Diabetes Mellitus

Metabolic syndrome is a conglomerate of cardiovascular risk factors which predispose a person to developing type II diabetes and cardiovascular disease.<sup>[15]</sup> The current diagnostic criteria require having 3 of 5 of the following factors: Triglycerides 150 mg/dL or greater, high-density lipoprotein cholesterol of less than 40 mg/dL in men and less than 50 mg/dL in women, hyperglycemia (fasting glucose of 100 g/dL or greater), an increased waist circumference (defined by population specific data), and hypertension (systolic blood pressure of 130 mmHg or greater or diastolic blood pressure of 85 mmHg or greater. In fact it has been stated that the incidence of NAFLD increases with increasing number of metabolic syndrome criteria met.<sup>[15]</sup>

# Ethnic differences

When genetic investigation by genome wide association done, it was noted that Hispanics had a two fold higher liver fat content if they possessed the homozygous PNPLA3 allele (patatin-like phospholipase domaincontaining protein 3<sup>-[8]</sup> The PNPLA3 gene family has been shown to affect lipid metabolism and patients who harbor this polymorphism were found to have increased hepatic fat content, triglyceride stores, and inflammation. In fact, the mutation of PNPLA-3 gene has revealed more severe histologic features of NAFLD in those carrying the mutation.<sup>[16]</sup>

## Polycystic ovarian syndrome

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder in reproductive aged women and is typically characterized by obesity and insulin resistance. Hence, women with PCOS are at a heightened risk of developing T2DM.<sup>[17]</sup>

# **Obstructive sleep apnoea**

Obstructive sleep apnea (OSA) is characterized by complete or partial airway obstruction caused by pharyngeal collapse during sleep.<sup>[18]</sup> The pathogenic mechanisms that underpin this association is believed to be due to the alteration of gas exchange (repetitive hypoxemic and hypercapnic events), termed chronic intermittent hypoxia, which can lead to an increase in pro-inflammatory cytokines, endothelial dysfunction, oxidative stress, metabolic dysregulation, and finally insulin resistance.<sup>[18]</sup>

## Samprapti

In Ayurveda, Fatty liver may be understood in light of *Yakritodara* and *Medoroga*. *Meda dhatu* in its natural state maintains *Snighata* and provides *bala* to the body

Excessive intake of these *ahara viharas* leads to *Jatharagni mandyata* and decrease the *Medodhatwagni*.<sup>[1]</sup> If the *Meda Dhatwagni* is deregulated then there is disharmony of distribution of *Baddha Meda* (visceral fat) and *Abaddha Meda*(circulating fat) and that excessive *Meda* deposits in various parts of the body including *Yakrit* which impairs its proper function. From this phenomenon, it can be said that *Meda* not only creates *Sthaulya* it can create *Yakritmeda* too.<sup>[1]</sup>

# The probable Samprapti may be

Nidana sevana (Vidahi and abhishyandhi ahaara)

Kapha and pitta gets dushita Jatharagnimandyata (Impaired fatty acid metabolism)

 $\downarrow$  *Aama* formation due to *Apakwa anna rasa* 

↓ *Apachita dhatu* formation

Samadhatu Formation Vitiated Mamsa Dhatu and Vasa (Upadhatu of Mamsa)

↓ Vitiated *Meda Dhatu* due to *Medodhatwagnimandyata* 

Increased production of *Abaddha Meda* or *Durmeda* Causing *Srotavarodh* in *Raktavaha*, *Mamsavaha* and *Medovaha Srota KaphaAvarana* in *Pitta sthana* (liver)

Accumulation of *Meda* and *Vasa* (fat or triglycerides) in Yakrit as Vasa is Upadhatu of Mamsa Sneha Guna and Kledaka Guna will increase and Ushna Guna will decrease in Yakrit

↓ Mandagni, Aruchi, Avipaka,utklesha, chardi, hridkantadaha will be seen

#### $\downarrow$ Yakritmeda<sup>[1]</sup>

# PATHOGENESIS

The initiating events in NAFLD are based on the development of obesity and insulin resistance, leading to increased hepatic free fatty acid flux. This imbalance between the rate of import/synthesis and the rate of export/catabolism of fatty acids in the liver leads to the development of steatosis.<sup>[19]</sup> This may be an adaptive response through which hepatocytes store potentially toxic lipids as relatively inert triglyceride. A 'two-hit' hypothesis has been proposed to describe the pathogenesis of NAFLD, the 'first hit' causing steatosis that then progresses to NASH if a 'second hit' occurs.<sup>[19]</sup> In reality, progression probably follows hepatocellular injury caused caused by a combination of several 'hits' including.

- Oxidative stress due to free radicals produced during fatty acid oxidation.
- Direct lipotoxicity from fatty acids and other metabolites in the liver
- Endoplasmic reticulum stress
- Gut-derived endotoxin

• Cytokine release (TNF-alpha etc.) and immune mediated hepatocellular injury.

Cellular damage triggers cell death and inflammation, which leads to stellate cell activation and development of hepatic fibrosis that culminates in cirrhosis.

Other factors contributing to steatosis like pregnancy (Acute fatty liver), Reye syndrome, Drug toxicity (sodium valproate and tetracycline) and small bowel bacterial overgrowth leads to defective mitochondrial beta-oxidation of lipids leads to fat droplet accumulation in hepatocytes and microvesicular steatosis.<sup>[19]</sup>

# Lakshana

NAFLD can be considered as an initial stage of *Agni* dearrangement which leads to several metabolic diseases like *Prameha*, *Sthoulya* etc<sup>.[20]</sup> However, symptoms like Utklesha (Nausea), chardi (vomiting), Aruchi(Loss of appetite) Udara Shoola (Abdomen pain) and Hridkantadaha (Epigastric discomfort) can be seen.<sup>[5]</sup>

# SYMPTOMS

Most patients have no symptoms at the time of diagnosis, although some have fatigue, malaise and a sensation of fullness in the upper abdomen. Other features may include daytime sleepiness and auto-nomic disturbances. Hepatomegaly is the only sign in most patients. Many patients are obese.<sup>[19]</sup>

# STAGES OF NAFLD

NAFLD has mainly 4 stages

**Steatosis:**- when fatty infiltration is more than 5% with or without mild inflammation. The terminology of microvesicular steatosis denotes the accumulation of innumerable lipid droplets with the hepatocyte nucleus remaining essentially in its original location<sup>-[21,22]</sup> The macrovesicular terminology is used when large lipid droplets inhibit the cytoplasm and displace the nucleus.<sup>[21]</sup> Lipid droplets are comprised of a core of triacylglycerols with or without cholesterol esters and a peripheral monolayer of phospholipids.<sup>[23]</sup> Inactive PNPLA3 has been shown to accumulate on the surface of lipid droplets and is linked to an increase in macrovesicular steatosis.<sup>[24]</sup>

Table 1: Steatosis score.<sup>[25]</sup>

Assessed the quantities of large or medium-sized lipid droplets (0-3)	<b>S0</b> : < 5% <b>S1</b> : 5%-33% <b>S2</b> : 34%-66% <b>S3</b> : > 67%
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**Non-alcoholic steatohepatitis (NASH):-** When defined histologically, based on combination of 3 lesions (steatosis, hepatocellular injury and inflammation) which mainly arises in acinar zone 3 distribution.<sup>[19]</sup> NASH specific features include hepatocyte ballooning degeneration with or without acidophil bodies or spotty necrosis and a mild, mixed inflammatory infiltrate. These can be accompanied by Mallory-denk bodies (also

known as Mallory hyaline). perisinusoidal characteristic feature of NASH.<sup>[19]</sup>

GRADE	STAGES		
	• Steatosis up to 66%,		
Crada 1 (mild)	• occasional ballooning in zone 3, scattered polymorphs with or without		
Graue I (IIIIu)	lymphocytes,		
	mild or no portal inflammation		
	Any degree of steatosis		
Cuada 2 (madauata)	obvious ballooning predominantly in zone 3		
Grade 2 (moderate)	• intralobular inflammation with polymorphs and chronic inflammation and		
	mild to moderate portal inflammation		
	Panacinar steatosis		
Creada 2 (Correra)	• ballooning, and obvious disarray predominantly in zone 3 intralobular		
Grade 5 (Severe)	inflammation with scattered polymorphs with or without		
	chronic and mild to moderate portal inflammation		

 Table 2:- Brunt grading and staging of non-alcoholic steatohepatitis.

Fibrosis:- Liver fibrosis is the excessive accumulation of extracellular matrix proteins including collagen. In liver, injured hepatocytes releases peroxide products, insulin like growth factor (IGF-1) and transforming growth factor (TGF- alpha) and platelets releases epidermal growth factor (EGF) causing activation of stellate cells.<sup>[19]</sup> Once they are active, they can perpetuate their own activation by TGF-beta 1 and PGDF (platelet derived growth factor) through autocrine loops. TGF beta 1 stimulating production of collagen matrix and acts as inhibitors of collagen breakdown.[19] MMP-2 and MMP-9 (Metalloprotinease) are inactivated by tissue inhibitors (TIMP 1 and TIMP 2) which leads to increase in fibrosis. Inflammation also contributes to fibrosis and release of IL-6 and IL-13 (interleukins). Activate stellate cells also produce endothelin-1 which may contribute to portal hypertension.[19]

deposition in zone 3.<sup>[21]</sup> Collagen fibers may be seen to encircle hepatocytes with more progressed lesions. Periportal fibrosis develops after the perisinusoidal fibrosis and is demonstrated as trapping of hepatocytes around the portal area and extension of short strands of collagen into the parenchyma.<sup>[21]</sup> Bridging fibrosis may eventually form single bands between the portal area and central vein without hepatocyte trapping or island formation. Evidence suggests that portal fibrosis in association with pericentral fibrosis is a necessary component for bridging fibrosis to develop.<sup>[21]</sup> t Masson trichrome stain can highlight the fibrosis and are useful in identifying early fibrosis of steatohepatitis. On the other hand, the active steatohepatitis changes may disappear in cirrhosis as well, resulting in a diagnosis of "cryptogenic cirrhosis".<sup>[26]</sup>

Fibrosis, when seen in NAFLD, has a characteristic appearance with early lesions showing a perisinusoidal

 Table 3: Fibrosis stage.<sup>[27]</sup>

	<b>F0</b> : No relevant fibrosis			
	<b>F1:</b> 1a - mild zone 3 perisinusoidal fibrosis			
	1b - moderate zone 3 perisinusoidal fibrosis			
Fibrosis	1c - portal fibrosis			
stage	<b>F2</b> : Zone 3 perisinusoidal fibrosis with			
8	periportal fibrosis			
	<b>F3:</b> Bridging fibrosis			
	F4: Cirrhosis			

**Cirrhosis:-** increasing fibrosis finally leads to cirrhosis. The most severe stage of NAFLD, which is characterised by diffuse hepatic fibrosis and nodule formation. This can lead to liver failure and hepatocellular carcinoma.<sup>[19]</sup>

# Complications

The most important complications of NAFLD are:-

- Portal hypertension such as variceal haemorrhage
- Liver cirrhosis
- Hepatocellular carcinoma.<sup>[19]</sup>

# DIAGNOSIS

Factors to be assessed in the evaluation of a patient with suspected non-alcoholic fatty liver disease<sup>[28]</sup>

- Personal and family history of diabetes, hypertension and CVD
- Alcohol use: < 20 g/d (women), < 30 g/d (man)
- Waist circumference, BMI, change in body weight
- Hepatitis B/C infection
- Liver enzymes

(Laboratory features include intermittent elevation in ALT and AST with AST:ALT <1. This ratio increases as fibrosis advances).

- History of steatosis-associated drug use
- Fasting blood glucose, hemoglobin A1c
- Serum total and HDL-cholesterol, triglycerides
- Undertaken due to clinical suspicion
- Ultrasound
- Hemochromatosis testing: Ferritin levels increased in 20-50% of patients and transferrin saturation
- Celiac disease: IgA and tissue transglutaminase
- Thyroid disease: TSH level (T3/T4)

- Polycystic ovarian syndrome
- Wilson's disease: Ceruloplasmin
- Autoimmune disease: ANA, AMA, SMA (ANA: Anti-nuclear antibody; AMA: Anti-mitochondrial antibody; SMA: Anti-smooth muscle antibody) identified in nearly 25% of patients and may be associated with more advanced fibrosis.
- Alpha-1 antitrypsin deficiency: Alpha-1-antitrypsin level
- Liver biopsy remains the best diagnostic tool for confirming fatty liver disease

Gradings	Macrovesicular steatosis	USG Findings		
Grade 0	Upto 5%	Normal appearance		
		-Mild fatty infiltration (<30% of liver parenchyma)		
Grade 1	5-30%	-Slight increase in echogenicity		
		-Normal liver texture		
		-Moderate fatty infiltration (30-50% of liver parenchyma)		
Grade 2	30-50%	- Moderate increase in echogenicity		
		- Slightly coarse liver texture		
		-Severe fatty infiltration (50-70% of liver parenchyma)		
Crede 2	50 700/	- Significant increase in echogenicity		
Grade 5	50-70%	- Coarse liver texture		
		- Possible loss of definition of liver vessels		
		-Very severe fatty infiltration (>70% of liver parenchyma)		
Course La A	More than 70%	- Marked increase in echogenicity		
Grade 4		- Very coarse liver texture		
		- Poor definition of liver vessels		

# Table 4:- Grading of NAFLD in USG on the basis of macro vesicular steatosis. [29] Considered Macrosoftenergies USC Findings

# **DIFFERENTIAL DIAGNOSIS**

The conditions that can also cause hepatic steatosis include.

Alcoholic liver disease:-The distinction of alcoholic liver disease and NASH can simply be done with

detailed history taking for the affirmation of alcohol use. In fact, the risk of developing alcohol related cirrhosis increases greatly with consumption of more than 60-80 gm/day for more than 10 yrs in men and more than 20 yrs in women.<sup>[30]</sup>

Table 5: Histologic	comparison	of non-alcoholic	fatty liver	disease/non-alcoholic	steatohepatitis	and	alcoholic
liver disease. <sup>[31]</sup>	-		•				

Characteristic	NAFLD and NASH	Alcoholic liver disease
Disease severity	Mild	Varying
Mallory denk bodies	Poorly formed	Well formed
Glycogenated nuclei	Common	Less common
Ductular proliferation	Less prominent	More prominent
Fibrosis/cirrhosis	Less common	More common
Sclerosing hyaline necrosis	None/rare	Present
Phlebosclerosis	None/rare	Present
Canalicular cholestasis	None/rare	Present
Foamy degeneration	None/rare	Present

**Drug induced hepatic steatosis results mainly from drugs like** Methotrexate, amiodarone, tetracycline, glucocorticoids, tamoxifen, chemotherapeutics, and nucleoside analogues causing exuberant accumulation of intracellular phospholipids due in part by a drug therapy that has lasted several weeks to months.<sup>[32]</sup> Drug-related hepatic injury is due in part to mitochondrial toxicity resulting in inhibition of beta oxidation, oxidative phosphorylation, and mitochondrial respiration.<sup>[33]</sup> Since beta oxidation is one of the main ways lipids are metabolized, drug induced inhibition results in the accumulation lipids within the hepatocytes.<sup>[33]</sup>

Anti retroviral agents :- In fact it has been reported that half of patients with human immunodeficiency virus (HIV) who undergo testing for liver test aberrations have concurrent NAFLD, which can result from HIV itself or the HAART therapy used in treatment.<sup>[34]</sup>

**Hepatitis-C:-** HCV, especially genotype 3a, has been reported to up-regulate the expression of fatty acid synthase in infected hepatocytes leading to increased fatty acids, impaired beta oxidation and reduced export of triglycerides.<sup>[35]</sup> As a part of its pathogenesis, HCV causes the inhibition of the microsomal triglyceride transfer protein, which is involved in the release of triglycerides from hepatocytes and as a consequence leads to triglyceride accumulation.<sup>[36]</sup>

# MANAGEMENT

There is no single intervention that is proven to be effective in the treatment of NAFLD. NAFLD Treatment includes control of weight, diabetes and hyperlipidaemia.<sup>[19]</sup> No drug is recommended at present though several drugs like metformin, pioglitazone, ursodeoxycholic acid (UDCA), pentoxyphylline, Vitamin E and atorvastatin have shown some promise.<sup>[19]</sup>

As NAFLD can be considered as *Santarpanjanya vyadhi*, *Apatarpana* treatment can be done. A practical treatment protocol include *shodhana*, *shamana* and lifestyle modification can help in proper management of NAFLD.

# Nidana parivarjana

रांतर्पयति यः स्निम्धेर्मधुरैर्गुरुपिच्छितैः। नवान्नेर्नवमहद्यैश्च मांसैश्चानूपवारिजैः ॥ गोरसैगोडिकैश्चान्ने पैष्टिकैश्चातिमात्रशः । चेष्टाद्वेषी

दिवास्वप्नशय्यासनसूखे रतः ||(Cha. Su. 23/3-4)

Food articles with excess unctuous, sweet, heavy, and viscous substances, freshly harvested food grains, wines with the flesh of wetland and aquatic animals with cow's milk and its products, the products of gur (jaggery) and with articles prepared of flour, dislikes movements (lazy) mean one should remain physically active, day sleeping, overindulgence in lounging, and lying in soft beds (*luxurious and mattress*) are to be avoided.

## Shodhana

बहुदोषस्य लिङ्गानि तस्मै संशोधनं हितम्। ऊर्ध्वचैवानुलोमंचयथादो षंयथाबलम् ||( Cha. Su 16/16)

Shodhana can be done in bahu dosha avastha, considering dosha avastha and bala of patient. शस्तमुल्लेखनंतत्र विरेको स्त्रामीक्षणम् (Cha. Su. 23/8)

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.

According to the condition of a patient, purification therapy can be implemented.

## Shamana chikitsa

Based on etiopathogenesis, the choice of drugs which have *Tikta, Kashaya rasa* and having properties of *Deepana, Pachana, Lekhana* can be given. Single herbs like *Sharapunkha, Bhoomiaamalaki, Katuki, Guduchi,*  Haritaki, Vasa and Pippali can be utilised for shamana chikitsa.

Some Aushadha yogas

• सक्षौद्रश्वाभयाप्राशः (Cha. Su. 23/8)

# • Triphaladi kwatha

त्रिफलार्ञ्वधं पाठां सप्तपर्णं सवत्सकम् । मुस्तं समदनं निम्बं जलेनोत्क्वथितंपिबेत् || (cha.Su.23/10)

- तकाभयाप्रयोगैश्च त्रिफलायारतथैवच । अरिष्टानां प्रयोगैश्च (Cha. Su. 23/17)
- Triushnaadi mantha
   त्र्यूषणं त्रिफला क्षौद्रं क्रिमिध्नमजमोदकम् । मन्थोऽयं सक्तवः सर्पिर्हितो लोहोदकाप्लुतः ॥ (cha. Su.23/18)
- Vyoshadhya Saktu

मुस्तमारुवधः पाठा त्रिफला देवदारु च । श्वदंष्ट्रा खदिरो निम्बो हरिदे त्वक् च वत्सकात् । व्योर्य विडङ्गं शिग्रणि त्रिफलां कटुरोहिणीम्। बृहत्यी हे हरिदे हे पाठामतिवियां स्थिराम् |हिड्ङ्ग केबुकमूलानि यवानीधान्यचित्रकात् । सीवर्चलमजाजों च हयां चेति चूर्णयेत् ॥(Cha. Su.23/19)

Herbs and formulations that are found effective in NAFLD can be *Katuki*(Picrorhiza kurrora), *Bhumi amalaka* (Phyllanthus niruri), *kirata tikta* (Andrographis paniculata), *sharapankhad*i *churna*, *katukyadi churna* etc.

# 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 *Bhallataka, Triphala, Shilajathu, Guggulu* etc can be given.<sup>[37]</sup>

## Yoga and Asanas

Vyayama (physical exercise) like brisk walking 3-4 kms/day. Yogic exercises like *Surya namaskara, pranayama (Kapalbhati and Bhastrika)*, two sitting asana (*Ardha matsyendrasana* and *Gomukhasana*), two lay down asana (*Dhanurasana* and *Balasana*) and *dhyana* have showed significant results Yoga mainly helps in decreasing blood glucose levels, total cholesterol levels and weight reduction. Asanas can correct metabolic functions and healing by reducing stress and anxiety.<sup>[37]</sup>

## Life style modifications

प्रायो रूक्षान्नसेवनम् (Cha. Su. 23/9)

प्रशातिका प्रियश्च श्यामाकायवका यवाः । जूर्णाह्वाः कोद्रवा मुद्राः कुलत्थाश्वक्रमुद्रकाः ||

आढकीनां च बीजानि पटोलामलकैः सह। भोजनार्थं प्रयोज्यानि पानं चानु मधूदकम् ||

अरिष्टांश्चानुपानार्थे मेद्रोमांसकफापहान् | (Cha. Su. 21/25-26) व्यायामश्चोपवासश्चधूमाश्च रुवेदनानि च || (Cha. Su. 23/8) व्यायामनित्योजीर्णाशी यवगोधूमभोजनः |(Cha. Su. 23/25)

used as a drink, and wines that are eliminative of fat,

flesh, and Kapha should be consumed. Exercise, fasting,

smoking, and sudation are beneficial. Daily exercise or

eating only after the previous meal has been digested,

and regular intake of barley and wheat is effective.

The diet should be mostly composed of dry foods, such as Prashatika, Italian millet, sanwa millet, wild barley, barley, great millet, common millet, green gram, horse gram, Cakramudgaka, pigeon pea seed mixed with wild snake gourd, and emblic myrobalan. Hydromel should be

Pathya and Apathya Pathya Ahara<sup>[38]</sup>

 Table 2: Pathya (wholesome diet) and Apathya (Unwholesome diet) for Liver Food items.

Food items	Pathya (Wholesome diet)	Apathya (Unwholesome diet)
Cereals	Rice, wheat, Oats, Barley	Newrice, riceflour
Pulses	Greengram, Redgram, Lentil	Sesame, Chickpea, Kidney beans, black Lentin
Fruits	<i>Draksha</i> , casted apple, pomegranate, apple, Kiwi,papaya	Orange, lemon, mango, watermelon
Vegetables	<i>Potala, shigru</i> , beans, <i>Sahijan</i> , brinjal, potato, <i>Jeevanti</i> , punarnava, Radish, Carrot, beet root	chilly, bitterguard, pickle, pumpkin, all leafy green vegetables(Saka)
Milk product	Ghee, milk, cheese	Curd, sugar mixed milk products, paneer
Beverages	Gomutra, Asava, Aristha	Soda, cold drinks, salted water

# CONCLUSION

As the obesity epidemic spreads and comorbid conditions like Type 2 Diabetes become more common, as well as NAFLD, treatment for these patients has become more complicated still. There are some treatments, but these are not enough high-quality studies that compare the various treatments. Officially, there is no pharmacological agent is in use. Approved. As a result, preventing the progression of fatty liver by way of life change and Ayurveda medicine are entirely acceptable by individuals now daily. Diet, yogic intervention, Panchakarma therapy, and medication are all components of Avurveda treatment. More experience and Data publication is a must-have this year. with one another, taking into account that bariatric surgery is everincreasing use, potential investigations the remaining queries regarding the relationship between insulin progression of fibrosis, resistance, and fatty liver should become readily available soon.

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