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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*.[1] 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*.[1] 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".[2] 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.[2] Now a days, due to modernization and urbanization, people moves towards the comfort and sedentary lifestyles, they are particularly much prone for getting life style disorders.[3] On the basis of etiopathogenesis, lifestyle disorders can be correlated with *Santarpanjanya 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.[3] *Ayurveda*, with its comprehensive range of medicines, can cure the disease at an early stage and prevent serious complications.[4] Non-alcoholic fatty liver disease (NAFLD) consists of steatosis and non-alcoholic 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%.[5]

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)

यदा स्त्रिया दोषप्रकोपणोक्त-मासेवमानाया दोषाः प्रकुपिताः शरीरमुपसर्पन्तः शोणितगर्भाशयावुपपद्यन्ते, न च कात्स्न्येन

शोणितगर्भाशयौ पन्ति तदेयं गर्भं लभते स्त्रीः तदा तस्य गर्भस्य मातृजानामवयवानामन्यतमोऽवयवो विकृतिमापद्यत एकोऽथवाऽनेके, यस्य यस्य ह्यवयवस्य 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 स्निग्धैर्मधुरैर्गुरुपिच्छिलैः। नवान्नैर्नवमद्यैश्च मांसैश्चानूपवारिजैः II गोरसैर्गौडिकैश्चान्नै पैष्टिकैश्चातिमात्रशः। (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। चिन्ताशोकभयक्रोधदुःखशय्याप्रजागरैः॥(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, NR1I2, 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)[6,7]

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.[8] 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.[9,10] In a retrospective study, cigarette smoking was found to be an independent risk factor for the onset of NAFLD.[11] The use of tobacco predisposes a person for the development of insulin resistance.[12-14]

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.[15] 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.[15]

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 domain-containing protein 3.[8] 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.[16]

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.[17]

Obstructive sleep apnoea

Obstructive sleep apnea (OSA) is characterized by complete or partial airway obstruction caused by pharyngeal collapse during sleep.[18] 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.[18]

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*.[1] 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.[1]

The probable Samprapti may be

Nidana sevana (Vidahi and *abhishyandhi ahaara)*
↓
Kapha and *pitta gets dushita Jatharagnimandyata*
(Impaired fatty acid metabolism)
↓
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
↓
Yakritmeda[1]

.

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.[19] 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.[19] 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.[19]

Lakshana

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

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.[19]

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.[21,22] The macrovesicular terminology is used when large lipid droplets inhibit the cytoplasm and displace the nucleus.[21] Lipid droplets are comprised of a core of triacylglycerols with or without cholesterol esters and a peripheral monolayer of phospholipids.[23] Inactive PNPLA3 has been shown to accumulate on the surface of lipid droplets and is linked to an increase in macrovesicular steatosis.[24]

Table 1: Steatosis score.[25]

Assessed the quantities of large or medium-sized lipid droplets (0-3)	**S0**: < 5% **S1**: 5%-33% **S2**: 34%-66% **S3**: > 67%

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.[19] 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.[19]

Table 2:- Brunt grading and staging of non-alcoholic steatohepatitis.[25]

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

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.[19] 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]

Fibrosis, when seen in NAFLD, has a characteristic appearance with early lesions showing a perisinusoidal deposition in zone 3.[21] 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.[21] 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.[21] 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’’.[26]

Table 3: Fibrosis stage.[27]

Fibrosis stage	**F0**: No relevant fibrosis **F1:** 1a - mild zone 3 perisinusoidal fibrosis 1b - moderate zone 3 perisinusoidal fibrosis 1c - portal fibrosis **F2**: Zone 3 perisinusoidal fibrosis with periportal fibrosis **F3:** 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.[19]

Complications

The most important complications of NAFLD are:-

- Portal hypertension such as variceal haemorrhage
- Liver cirrhosis
- Hepatocellular carcinoma.[19]

DIAGNOSIS

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

- 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

Table 4:- Grading of NAFLD in USG on the basis of macro vesicular steatosis.[29]

Gradings	Macrovesicular steatosis	USG Findings
Grade 0	Upto 5%	Normal appearance
Grade 1	5-30%	-Mild fatty infiltration (<30% of liver parenchyma) -Slight increase in echogenicity -Normal liver texture
Grade 2	30-50%	-Moderate fatty infiltration (30-50% of liver parenchyma) - Moderate increase in echogenicity - Slightly coarse liver texture
Grade 3	50-70%	-Severe fatty infiltration (50-70% of liver parenchyma) - Significant increase in echogenicity - Coarse liver texture - Possible loss of definition of liver vessels
Grade 4	More than 70%	-Very severe fatty infiltration (>70% of liver parenchyma) - Marked increase in echogenicity - Very coarse liver texture - Poor definition of liver vessels

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.[30]

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

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.[32] Drug-related hepatic injury is due in part to mitochondrial toxicity resulting in inhibition of beta oxidation, oxidative phosphorylation, and mitochondrial respiration.[33] Since beta oxidation is one of the main ways lipids are metabolized, drug induced inhibition results in the accumulation lipids within the hepatocytes.[33]

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.[34]

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.[35] 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.[36]

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.[19] No drug is recommended at present though several drugs like metformin, pioglitazone, ursodeoxycholic acid (UDCA), pentoxyphylline, Vitamin E and atorvastatin have shown some promise.[19]

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

संतर्पयति यः स्निग्धैर्मधुरैर्गुरुपिच्छिलैः। नवान्नैर्नवमद्यैश्च मांसैश्चानूपवारिजैः II

गोरसैर्गौडिकैश्चान्नै पैष्टिकैश्चातिमात्रशः । वेष्टाद्वेषी दिवास्वप्नशय्यासनसुखे 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| ऊर्ध्ववैवानुलोमंचयथादोषंयथाबलम् ॥(Cha. Su 16/16)

Shodhana can be done in *bahu dosha avastha*, considering *dosha avastha* and *bala* of patient.

शस्तमुल्लेखनंतत्र विरेको रक्तमोक्षणम्(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*

- सक्षौद्रश्चाभयाप्राशः (Cha. Su. 23/8)

- *Triphaladi kwatha*

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

- तक्राभयाप्रयोगैश्च त्रिफलायास्तथैवच । अरिष्टानां प्रयोगैश्च (Cha. Su. 23/17)

- *Triushnaadi mantha*

त्र्यूषणं त्रिफला क्षौद्रं क्रिमिघ्नमजमोदकम् । मन्थोऽयं सक्तवः सर्पिर्हितो लोहोदकाप्लुतः ॥ (cha. Su.23/18)

- *Vyoshadhya Saktu*

मुस्तमारग्वधः पाठा त्रिफला देवदारु च । श्वदंष्ट्रा खदिरो निम्बो हरिदे त्वक् च वत्सकात्। व्योर्य विडङ्गं शिग्रूणि त्रिफलां कटुरोहिणीम्। बृहत्यौ हे हरिदे हे पाठामतिविषां स्थिराम् ।हिङ्गु केबुकमूलानि यवानीधान्यचित्रकात् । सीवर्चलमजाजों च हयां वेति 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), *sharapankhadi 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.[37]

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.[37]

Life style modifications

प्रायो रूक्षान्नसेवनम् (Cha. Su. 23/9)

प्रशातिका प्रियश्च श्यामाकायवका यवाः । जूर्णाह्वाः कोद्रवा मुद्गाः कुलत्थाश्चकमुद्रकाः ॥

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

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

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 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.

***Pathya and Apathya Pathya Ahara*[38]**

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	*Draksha*, casted apple, pomegranate, apple, Kiwi,papaya	Orange, lemon, mango, watermelon
Vegetables	*Potala*, *shigru*, beans, *Sahijan*, brinjal, potato, *Jeevanti*, 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 *Ayurveda* treatment. More experience and Data publication is a must-have this year. with one another, taking into account that bariatric surgery is ever-increasing 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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