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Conceptual study of Non-Alcoholic Fatty Liver Disease and its management in Ayurveda

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ABSTRACT

Non-alcoholic Fatty liver Disease (NAFLD) is a condition with excessive fat deposition in liver on refers to a broad spectrum of disorders characterized by fatty infiltration of the liver steatosis, steatohepatitis and cirrhosis. It is a growing cause of liver injury globally and is present in developed and developing countries. NAFLD is a broad term which describes the build up of excessive fat in the liver cells (hepatocytes) in the absence of excessive alcohol intake. It is a growing worldwide, due to obesity and insulin resistance leading to liver accumulation of triglycerides and free fatty acids. NAFLD encompasses a spectrum of liver Pathology with different clinical prognoses, from the simple accumulation of triglycerides within hepatocytes to more progressive steatosis with associated hepatitis, fibrosis, cirrhosis and in some cases hepatocellular Carcinoma. It is a global epidemic that ranges from isolated hepatic steatosis (NAFL) to Steatosis Plus inflammation (NASH) with or without fibrosis. Ayurveda described different types of Non-Communicable diseases, and NAFLD is one among them. In Ayurveda NAFLD can be interpreted as a *Santarpanotha Vikara* caused by *Kapha Medo Dushti*, getting *Sthanasamshraya* in *Yakrit*, which is *Raktavaha Srotasmula* and *Pittasthana*. In the initial stage of NAFLD, *Kapha Meda Dushti* occurs when *Pitta* gets involved in the pathogenesis, inflammatory changes occur which leads to the next stage of disease i.e., Non alcoholic steatohepatitis (NASH), When *Vata* comes involved fibrosis occurs which may end up in cirrhosis. So, the management should be the breakdown of pathological factors like *Agnivaigunya*, *Srotorodha* and *Kaphamedo Dushti*. This review will give a better knowledge of etiopathogenesis.

Key words: *Non Alcoholic Fatty Liver Disease (NAFLD), Non Alcoholic steatohepatitis (NASH), Yakrit Vikara, Santarpanajanya Vyadhi.*

INTRODUCTION

Non alcoholic fatty liver disease of one of the most common liver diseases worldwide, usually emerges due to lipid accumulation, mostly triglycerides in hepatocytes. Fat exists in the liver naturally, but if it surpasses 5-10% of the liver's total weight, it can result in fatty liver. NAFLD is a common condition characterized by excess of fat in the liver which ranges from simple steatosis to steatohepatitis, cirrhosis and hepato cellular carcinoma (HCC) in the absence of excessive alcohol intake. NAFLD consists of steatosis and Non-alcoholic steatohepatitis (NASH). Steatosis is the accumulation of fat in the liver and steatohepatitis is a condition accumulation of fat 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%. Among obese patients, the frequency of NASH ranges from 12.6% to 30.4%. In Ayurveda, NAFLD is assigned as *Yakrit Roga* (liver disease) and *Medoroga* (Obesity). Descriptions of *Yakrit* related diseases are minimum in Ayurveda. As per Ayurveda it is very difficult to use distinctive nomenclature to denote the word Non Alcoholic Fatty Liver Disease, NAFLD is a lifestyle disorder, based on its *Nidana*, *Lakshana* and *Samprapti* we have equated it with *Yakritudara*.

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Fatty liver

- Fatty Liver is a reversible condition where in large amount of fat accumulate in liver cells via the process of steatosis. When fat content exceeds 5% of total weight of liver or more than 30% of liver cell in a liver lobule are with fat deposit, they condition is called as fatty liver.
- Etiology of fatty liver mainly falls under two Categories: First category comprises the conditions with excess fat which imparts increased workload to liver for metabolizing fat. Second category involves conditions of liver cell damage in which fat can not be metabolized due to liver cell injury.
- Fatty Liver disease (FLD) includes Alcoholic liver disease (ALD) & Non alcoholic fatty liver disease (NAFLD). Ludwig et al. (1980) proposed the term NAFLD. NAFLD occurs without the consumption of alcohol in quantities which can potentially damage the liver.

There are different types of involvement of the liver Starting from accumulation of fat in hepatocytes, to a liver with inflammation and hepatocyte injury (Non Alcoholic steatohepatitis) leading to fibrosed state and ultimately to hepatocellular carcinoma.

Materials and Methods

Ayurvedic classics, compendia, clinical medicine texts & related websites were consulted and reviewed for the present work.

- Non Alcoholic Fatty Liver Disease (NAFLD) is an umbrella term for a range of liver disorders characterized by macro vesicular hepatic fat accumulation (steatosis), signs of hepatocyte injury mixed inflammatory cell infiltrate and variable hepatic fibrosis leading to cirrhosis.
- NAFLD itself has benign prognosis, but Non-alcoholic steatohepatitis (NASH) is a potentially serious form of NAFLD, marked by liver inflammation, which may progress to scarring & irreversible damage.
- Approximately, 20% to 30% of adults in the general population in western countries NAFLD and its prevalence increases to 70% to 90% among persons who or obese or have diabetes.
- As per AASLD guidelines the following risk factors are associated with NAFLD. Established associations of NAFLD in common conditions include obesity, type2 DM, dyslipidaemia, metabolic syndrome polycystic ovary syndrome. Lifestyle factors like lack of exercise & diet factors causing fatty liver. High intake of saturated fatty acids, trans fats, refined carbohydrates, sweetened beverages, dietary fructose all have an associated with NAFLD.

Epidemiology and risk factors

Prevalence of the disease is estimated to be around 9-32% in the general Indian population, with a higher incidence rate amongst obese and diabetic patients. NAFLD is more common in men. The current estimates are the prevalence of NAFLD is approximately 20% in general population making it most common liver disease in united states. NAFLD is strongly associated with obesity, dyslipidaemia, insulin resistance and type 2 diabetes mellitus. Recently NAFLD is seems to be related with obesity, diabetes, hypertension, hyperlipidaemia & metabolic syndrome.

NAFLD in Ayurvedic view

In Ayurveda is not directly given details about NAFLD. So as per Ayurveda it is very difficult to use distinctive nomenclature to denote the word Non Alcoholic Fatty Liver Disease, NAFLD is a lifestyle disorder, based on its *Nidana*, *Lakshana* and *Samprapti* we have equated it with mainly *Yakritudara* / *Yakritdalyudara*, *Yakrit Vikara* (as per *Dosha* predominance).

Nidana

Aharaja Hetu (Dietary factors)

- In NAFLD there is accumulation of fat in liver and metabolic disorders like obesity, diabetes mellitus, insulin resistance and dyslipidaemia are associated factors of NAFLD so we have considered *Santarpanajanya Vyadhis* and *Ahara-Vihara* which causes *Agnimandya*, *Rasa*, *Rakta* and *Medo Dushti* important factors in etiology of *Yakrit Vikara.*

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- Excessive ingestion of foods having the qualities like *Snigdha*, Guru, *Picchila*, *Sheeta* and *Madhura*, *Amla*, *Lavana Rasa* results in disorders related to *Kapha* and *Medas*. For example - Excess intake of excessive spicy, oily, salty, sour fatty diet (high intake of red meat, processed meat, sugar sweetened beverages, trans fatty acids and sodium)

Viharaja Hetu

Avyayama, Divaswapna, *Adhyasana*, *Atichintana*, *Vyadhikarshanat*, *Vegadharana*, *Aatapa* and *Anila Sevana*, *Deshakalaritiviparyad*, Lack of exercise, Lack of physical activities, irregular dietary habit, prolonged sitting working lifestyle.

Causative factors of NAFLD

Heavy fat rich diet, junk food, soft drinks, sedentary lifestyle, metabolic syndrome (Obesity, Diabetes Mellitus, Dyslipidaemia), drugs (eg: Corticosteroids, Aspirin, Tetracycline) etc. are considered to be the major etiological factors of NAFLD.

Etio-pathogenesis - Modern and Ayurvedic perspective

Liver can be compared with *Yakrit* mentioned in Ayurvedic classics. *Yakrit* is the seat of *Ranjaka Pitta* - which transforms *Apya Rasa Dhatu* to *Rakta Dhatu*. It is *Mulasthana* of *Raktavahasrotas*; also related to *Raktavahi* and *Mamsavahi Dhamani. Yakrit* is an important *Koshthanga*, which intimately related to *Rasa*, *Rakta* and *Mamsa Dhatus* and plays an important role in *Dhatu Parinama*.

Liver is the largest gland and heaviest organ in the body weighing about 1200-1500 gm in adults. It is situated in the abdominal cavity below the diaphragm mainly in the right hypochondrium. Liver is the commonest site for accumulation of fat because it plays central role in fat metabolism. Depending upon the cause and amount of accumulation fatty change may be mild and reversible, or severe producing irreversible cell injury band cell death. Disturbance of lipid metabolism in liver due to various etiological factors leads to fatty liver.

As per Ayurvedic concept, heavy fat rich diet, soft drinks and sedentary lifestyle are responsible for the *Dushti* of *Annavaha*, *Rasavaha*, *Raktavaha*, *Medavaha* and *Purishavaha Srotas*.

The pathogenesis of NAFLD was originally described by the "two-hit theory" or multifactorial. First hit is represented by an accumulation of fatty acids and triglycerides in liver cells (hepatocytes). The second hit is represented by chronic stress such as enhanced lipid peroxidation, generation of reactive species (ROS), Endoplasmic reticulum stress (ERS) and by-products of exacerbated pro-inflammatory responses in fatty-liver.

The components of first hit include release of free fatty acids from central adipose tissue, along with adipokines drain into the portal vein as well as causing insulin resistance. These process result in reduced hepatic fatty acid oxidation and increased fatty acid synthesis.

Insulin resistance

↓

Increase in Triglycerides synthesis & Hepatic uptake of FFA

↓

Steatosis

↓

FFA oxidation leads to increase free radical formation

↓

Hepatocellular injury

↓

Oxidative stress or lead to progression of proinflammatory cytokines

↓

Hepatic inflammation

↓

Chronic inflammation leads to fibrosis

The first hit - hepatic triglyceride accumulation or steatosis, increases susceptibility of the liver to injury mediated

The second hit - such as inflammatory cytokines / mitochondrial dysfunction and oxidative stress which in turn leads to steatohepatitis or fibrosis

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Fatty liver occurs as a result of increased fat import into hepatocytes and reduced fat export. Insulin resistance causes hepatic steatosis, which also perpetuates insulin resistance. Subsequent activation of TNF-α, oxidant stress through the production of reactive oxygen species and production of endotoxin then result in inflammation and eventually fibrosis.

NAFLD is a *Santarpanajanya Vyadhi* (Disease cause by over nourishment) having *Nidana* (causative factors) and *Samprapti* (pathogenesis) similar to *Sthaulya*. Initial pathology lies at *Agnivikruti* which leads to the formation of *Apakwa Anna Rasa* (improperly formed digestive end product) which again leads to the vitiation of *Kaphadosha* and unequal formation and deposit of *Meda* in *Yakrit*. This condition is called as Fatty Liver. Vitiated *Kapha* and *Meda* results in *Srotorodha* which provokes *Vata*. Vitiated *Vata* again results in *Agnivikruti* and this cycle repeats.

When *Pitta* gets involved in the pathogenesis, hepatocytes have inflammatory changes and the disease progresses to the next stage i.e, NASH. When *Vata* comes into the picture, fibrosis occurs and the condition may progress to its drastic end stages cirrhosis, ascites, hepato cellular carcinoma.

Clinical presentation of NAFLD

Most patients with NAFLD are asymptomatic. Diagnosis most often follows incidental detection of raised liver enzymes or of Fatty Liver on ultrasound. These abnormalities are usually picked up during evaluation for dyspepsia, malaise or fatigability, prior to medical procedures like organ donation or routine health examination. A smaller fraction of patients experience symptoms indicative of more serious liver diseases and may develop pruritus, anorexia and nausea. The development of ascites, anasarca, variceal haemorrhage or symptoms of hepatic encephalopathy indicates decompensated cirrhosis. Jaundice occurs late in the course of NASH and indicates advanced liver disease. No specific symptoms can distinguish NAFLD or NASH from other type of liver diseases. The majority of patients have one or more risk factors for metabolic syndrome, such as type 2 Diabetes, obesity, hypertension or hyperlipidaemia. Clinical examination is often unremarkable though nearly half the patients have mild hepatomegaly. Jaundice or signs of liver failure are absent.

Mainly NAFLD has two types of presentations

1. Obese NAFLD: Fatty liver in obese people which is more common and having better prognosis
2. Lean NAFLD : Fatty Liver in lean people, less common but comparatively worse prognosis

NAFLD hardly exhibits signs or symptoms, especially in the initial stages. Hence it is usually detected during causal blood investigation or a routine examination. Its presentation can be manifested independently or in association with some disorder. Therefore, NAFLD can be considered as an initial stage of *Agni* derangement which leads to several metabolic diseases like *Prameha, Sthoulya* etc. However, symptoms like *Klama*, *Tiktashyata*, *Utklesha*, *Udara* Shula, *Agnimandya*, *Ajeerna* and *Adhmana* can be seen.

In the initial stage of NAFLD, patients present with heaviness and distention of abdomen, increased or decreased appetite, constipation or diarrhoea, malaise distention can be clearly compared with *Ajirna*. In obese persons, the presentation exactly resembles with that of *Sthaulya*. When NAFLD progress to next stage, clinical profile of NASH is persistently similar with *Amlapitta* (Gastro-esophageal Reflex Disease) in which patients present with sour eructation, burning sensation of chest and abdomen, distention of abdomen, tastelessness and loose stools. Patient may pass stools frequently without proper digestion as in *Grahani* (Sprue). When the condition progresses to Fibrosis and Cirrhosis, systemic features indicative of more serious liver disorders such as *Pandu* (Anaemia, Fatigue, Altered sensorium), *Kamala* (yellowish sclera, skin & urine), *Raktapitta* (haematemesis) will be more evident. Finally, it ends up in one among *Ashtamahagada* (8 major diseases) i.e., *Udara* (Ascites); *Yakriddalyudara* is being more site specific.

Diagnosis

The presence of NAFLD has been suspected in those presenting with abnormal liver blood tests or evidence of fatty changes on ultrasound. However, the full

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spectrum of NAFLD (from simple steatosis to steatohepatitis, cirrhosis and liver-related morbidity) can also be present with normal liver tests. It should be suspected as a cause of asymptomatic elevation of amino transferases (AST) and alanine aminotransferases (ALT) or isolated elevation of Gamma Glutamyl Trans peptidases (GGT). ALT is normally higher than AST. Elevated ALP levels are seen in about 30% cases.

Ultrasound may show liver steatosis as a hyper echogenic image, i.e., 'bright liver'. Radiologic techniques used to evaluate NAFLD include ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance spectroscopy (MRS) and fibroscan. These radiologic modalities are accurate in detecting moderate to severe hepatic steatosis and none is able to distinguish simple steatosis from NASH or to determine the stage of hepatic fibrosis. Liver biopsy is diagnostic, but may not be routinely required. It allows semi-quantitative assessment of fat deposition and associated necro-inflammation and fibrosis. Typical histologic features of NAFLD predominate in perivenular regions, i.e., Zone of 3 hepatic acinus and include the presence of macro-vesicular steatosis, lobular neutrophilic inflammation, presence of Mallory bodies, ballooning degeneration, lipogranuloma and pericellular fibrosis.

Management

NAFLD is an increasingly recognized clinico-pathological condition that may progress to end stage liver disease. The clinical implications of NAFLD are derived mostly from its common occurrence in the general population, as well as its potential to progress to cirrhosis and liver failure. No established pharmacological treatment is available for NAFLD in modern medicine. The main goals of treatment are to improve steatosis and to prevent the progression of the disease. Being a disease associated with insulin resistance and metabolic syndrome, insulin-sensitizing agents are expected to alter the pathophysiological mechanisms of NAFLD. Metformin and the thiazolidinedione group of antidiabetic agents are the most studied medications in this group. For normalising blood cholesterol statins have been opted. To counter oxidative stress, antioxidants like N-acetyl cysteine, and vitamin E is the drug of choice.

Treatment of NAFLD is still evolving with no single drug clearly shown to be effective. Several empiric treatment strategies such as dietary restriction, physical exercise and weight reduction from the first line of treatment. Hence, there is a search for alternative treatment modalities in other systems of medicine, which is safe and cost-effective. Ayurveda has immense potential in the management of Non-Communicable Diseases, and NAFLD is one among them.

Points to be focused in the treatment of NAFLD are *Agnivikruti*, vitiation of *Kapha* and *Meda* the *Mula Sthana* of *Raktavaha Srotas*, *Srotorodha* and vitiation of *Vata*. So, treatment of NAFLD are *Agnideepana* (Stimulation of digestive fire), *Rookshana* (Dryness and into a blockage of channels pacification of *Kapha*, *Meda* any less therapy). *Srotosodhana* (Removal of the management of Fatty Liver. Here, the treatment principle and should be the first line of treatment *Sthaulya*. When Fatty Liver progresses to next stage and *Dhatus* get involved, treatment should be directed towards *Prasadana* (purification) of *Rasa* and *Rakta* and also *Yakritshothahara*. *Samshodhana*, *Samshamana*, *Ahara* and *Achara* will help in the reversal of fatty changes of liver and prevention of further complications.

Nidana Parivarjana

Avoidance of *Sleshma Medokara Ahara Vihara* helps to reduce its incidence.

Shodhana

Virechana is the most suitable *Shodhana Karma* in Liver disorders. As *Ashraya Sthana* of the disease is *Yakrit* which is *Raktavaha Srotomula.* As Non-alcoholic fatty liver disease is a *Santarpanajanya Vyadhi, Ruksha Virechana* using *Churnas*, *Kashayas* etc. should be given. *Virechana* is indicated in excessive *Dosha* accumulation and *Srotorodha*. It acts as *Agnideepana* and *Stotoshodhana. Churnas* like *Patoladi*, *Hapushadi*, *Narayana*, *Avipattikara* different preparations of

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Erandataila, *Trivrit Avaleha*, *Mishraka Sneha*, *Harityakadi Ghrita* can be used judiciously for this purpose.

Samshamana

Pachana, *Deepana* and *Rookshana* should be done in the first stage of *Amavastha* for pacifying the *Kapha* and *Ama* from the *Pittasthana*. Use of *Katu Tikta Rasa* that becomes *Katu Vipaka* may be useful as these have *Sneha-Meda-Kleda-Shoshana* properties. The use of *Lekhaniya* drugs helps in the removal of *Sanchita Ama* and accumulated fat from hepatocytes. *Medahara Chikitsa* can also be adopted. While coming to *Shamana Chikitsa*, single herbs like *Sharapunkha*, *Bhoomiamalaki*, *Katurohini*, *Rohitaka*, *Guduchi*, *Vasa*, *Haritaki* and *Pippali* can be used safely for the effective management of NAFLD. All these drugs are proven hepato-protectives in experimental and clinical trials. Formulations like *Kashaya* (*Vasaguduchyadi*, *Phalatrikadi*, *Drakshadi*, *Patola Katurohinyadi*), *Arishta* (*Sudarshanaarishta*, *Rohitakarishta*, *Pippalyasava*), *Churna* (*Hinguvachadi*, *Vaiswanara), (Abhraka Bhasma Arogyavardhini Rasa, Punarnava Mandoora)* are well known for their hepatoprotective, hypolipidaemics and haematinic properties, and can be effectively used for the management of NAFLD.

Rasayana

As *Ama* is *Dhatuleena*, *Rasayana Chikitsa* can also be adopted. *Rasayana* drugs having *Deepana*, *Pachana*, *Lekhana* property may be selected. *Triphala*, *Shilajatu*, *Guggulu* etc. are good example for this. *Guggulu* is effective in reducing hyperlipidemia. Guggulsterone has been reported to reduce serum cholesterol and triglyceride and has cardioprotective action. *Triphala* and its constituents show valuable hepato-protective activity. Extract of *Triphala* showed significant protection against acute liver toxicity induced by high doses of drugs and chemicals.

Disharmony in the relationship between human being and ecosystem is the major causative factor of all diseases especially Metabolic syndrome, which mainly arises out of faulty diet and lifestyle. Therefore, diet and lifestyle have a major role in the causation, prevention and management of NAFLD. Diet should be modified as per 'Eight Rules of Eating' by Acharya Charaka. Yoga and Pranayama can be included in the daily routine for better results.

Pathya and *Apathya*

Pathya

Ahara - Eat freshly prepared food and eat only when hungry and after evacuation of *Mala*. Having *Katu, Tikta Rasa*, *Laghu, Ruksha Guna* etc. must be incorporated as a part of the diet.

Vihara - Consistent physical exercise like brisk walking daily half an hour in fresh air. Regular doing exercise increases the power of digestion, lightness to the body, increases capacity to do work, removes excess fat and provides stability. *Yoga - Dhanurasana, Bhujangasana, Gomukhasana, Pranayama, Ardhamatsyendrasana, Kapalabhaati.*

Apathya

Ahara - Foods having *Guru, Snigdha* and *Pichila Gunas* should be avoided. *Abhishyandi Aharas* like *Dadhi, Ghrita, Navadhanya, Pishtanna* etc. should be avoided. Avoid reheated & untimely food excessive spicy, oily, salty, sour, fatty diet, pickles, junk food, chocolates, bakery items, artificial sweeteners, ice-creams, jams.

Vihara - Sedentary life style, excessive & day time sleeping, smoking, suppression of natural urges.

CONCLUSION

NAFLD is an increasingly recognized clinico-pathological condition that may progress to end stage liver disease in which the conventional medicine fails to establish effective management strategy. Ayurvedic treatment modalities have immense potential in the management of these kind of lifestyle disorders. NAFLD is rapidly becoming the most common liver disease worldwide. Due to the *Aharaja* and *Viharaja Nidanas*, *Kapha Doshakopa* occurs in the body. This leads to *Jatharagnimandya* and the formation of *Ama* i.e., improperly formed *Rasadhatu*. This *Samarasa* circulates throughout the body by the action of *Vyanavayu*, reaches *Yakrit* for the process of *Dhatu Parinama* since *Yakrit* is the seat of *Ranjaka Pitta* and *Raktavaha-Srotomula*. This causes *Avayavadushti*

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(vitiation of *Yakrit*). *Medadhatu* accumulation in liver is the main concern of the NAFLD therefore the treatment should focus on correcting *Agnimandya*, *Kapha* and *Pitta Shamaka Chikitsa*, and *Medohara Chikitsa*. For the breaking of pathogenesis of NAFLD, *Deepana* (carminative), *Pachana* (digestive), *Kaphahara* (kapha alleviating) and *Medohara* drugs (fat reducing) and *Rasayana* (rejuvenating) therapy for preventing further progression are essential. A practical treatment protocol including *Virechana*, *Shamanoushadha* and *Pathyasevana* with due importance to particular bodily constitution can help in the proper management of the disease.

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