ISSN : 2249- 5746
International Journal of Ayurvedic and Herbal Medicine 7:6 (2017) 2948–2955
Journal homepage:http://www.interscience.org.uk
DOI:10.18535/ijahm/v7i6.05
Impact Factor: 4.415

Non Alcoholic Fatty Liver Disease- An *Ayurvedic* Pragmatic Approach with Its Management

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Abstract:
The term Non-alcoholic Fatty Liver Disease (NAFLD) refers to a broad spectrum of disorders characterized by fatty infiltration of the liver, steatosis, steatohepatitis, and cirrhosis. Epidemiological studies suggest prevalence of NAFLD in around 9% to 32% of general population in India with a higher prevalence in those with obesity and diabetes. No established pharmacological treatment is available for NAFLD in conventional medical science. Several empirical treatment strategies such as dietary restriction, physical exercise and weight reduction form the first line of treatment. *Ayurveda* has immense potential in the management of Non-Communicable Diseases, and NAFLD is one among them. Purpose of the present study was to formulate a practical *Ayurvedic* protocol for the management of NAFLD. *Ayurvedic* classics, compendia, clinical medicine texts & related websites were consulted and reviewed for the present work. In the initial stage of NAFLD, *Kapha Medo Dushti* occurs. When *Pitta* gets involved in the pathogenesis, inflammatory changes occur which leads to the next stage of the disease, ie. Non-Alcoholic Steato Hepatitis (NASH). When *Vata* comes into the picture, fibrosis occurs which may end up in Cirrhosis. The management should be the breakdown of pathological factors like *Agnivaigunya, Srotorodha and Kaphamedodushti*. A practical treatment protocol including Purgation, Pacification and Lifestyle modification with due importance to particular bodily constitution can help in the proper management of the disease. Through the proper administration of *Ayurvedic* treatment protocol along with lifestyle modification, progression of the disease and further complications can be prevented upto a great extent.

Key words : *Non- Alcoholic Fatty Liver Disease, Non-Alcoholic Steato- Hepatitis, Sthoulya, Yakrit*

Introduction

Liver is the largest gland and heaviest organ in the body, weighing about 1200-1500gm in adults. It is situated in the abdominal cavity below the diaphragm mainly in the right hypochondrium, extending across the epigastrium to the left hypochondrium. It is an extremely vascular organ, which receives about 30% of resting cardiac output. Liver is an essential organ of the body, which acts as a well equipped biochemical laboratory where practically metabolism of all the nutritional substances takes place. 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 and cell death. Disturbances of lipid metabolism in liver due to various etiological factors lead to Fatty Liver.

Fatty liver is a reversible condition wherein 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 cells in a liver lobule are with fat deposit, this condition is called as Fatty Liver. Etiology of Fatty Liver mainly falls under two categories (Table No:1). 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 cannot be metabolized due to liver cell injury

Conditions with excess fat usually manifest as Non – Alcoholic Fatty Liver Disease (NAFLD). It is the Hepatic complication of Metabolic Syndrome which occurs when fat is deposited in the liver due to causes other than excessive alcohol use. 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 Steato-Hepatitis (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 have NAFLD and its prevalence increases to 70% to 90% among persons who or obese or have diabetes. NASH can progress to cirrhosis and end-stage liver disease and is projected to be the leading cause of liver transplantation by 2020. NAFLD is overall associated with an increased cardio metabolic risk. (API – page no 885 Introduction)

NAFLD is a disease of affluent societies which increases in prevalence in proportion to the rise in obesity. It has become the most common cause of chronic liver disease after Hepatitis B, hepatitis C and alcohol. (Davidson) The prevalence of NAFLD has increased over last few years. The global epidemic of obesity and diabetes in all age groups may be partly responsible for the increase . Urbanisation and associated changes such as sedentary lifestyle, fat rich diet and a higher inherited tendency for Diabetes mellitus make Indians more prone to Metabolic Syndrome or insulin resistance and its manifestations , such as NAFLD and NASH. The majority have one or more risk factors for metabolic syndrome, such as Diabetes mellitus, obesity, hypertension or hyperlipidaemia. The risk of NAFLD was the highest in those with BMI > 25kg/m^2. Both Fatty Liver and NASH have been reported in all age groups, including children, with highest prevalence in the age group of 40-49 years, with equal frequency in males and females[i]. (API – 885 Epidemiology)

Aims and Objectives

1. To define NAFLD in terms of *Ayurvedic p*erspective
2. To formulate a practical *Ayurvedic* protocol for the management of NAFLD

Materials and Method

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

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 the *Mulasthana* (site of origin) of *Raktavahasrotas;* also related to *Raktavahi & Mamsavahi Dhamani*. *Yakrit* is an important *Koshthanga,* which intimately related to *Rasa, Rakta* and *Mamsa Dhatus* and plays an important role in *Dhatu Parinama.*

Heavy fat rich diet, junk food, soft drinks, sedentary lifestyle, Metabolic syndrome (Obesity, Diabetes Mellitus, Dyslipidaemia), drugs (eg: Cortico steroids, Aspirin, Tetracyclin) etc. are considered to be the major etiological factors of NAFLD. Etiology of Fatty Liver has been discussed in Table No:1. As per Ayurvedic concept, heavy fat rich diet, soft drinks and sedentary lifestyle are responsible for the dushti of

Annavaha, Udakavaha, Rasavaha, Raktavaha, Medovaha and *Pureeshavaha Srotas (Caraka). Ajirna (*Indigestion*), Sthaulya (*Obesity*)* and *Prameha (*Diabetes Mellitus*)* which occurs due to the vitiation of *Annavaha, Rasavaha* and *Medovaha Srotas* acts as *Nidanarthakara Rogas* (Diseases which cause another diseases) which may result in the manifestation of Fatty Liver.

Several mechanisms have been postulated to explain the pathogenesis of NASH. The currently favoured is 'Multiple Hit Hypothesis'[i]. The current two- hit hypothesis explains why not everyone with fatty liver disease develops hepatic fibrosis. The 'first hit' results in steatosis (fatty liver), which is only complicated by inflammation if a 'second hit' occurs. Leptin , which is an appetite reducer and a fibrogenic in vitro, is probably then needed to cause hepatic fibrosis. 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 processes result in reduced hepatic fatty acid oxidation and increased fatty acid synthesis. Pathogenesis of Fatty Liver has been depicted in Figure No:1.

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 – alpha, oxidant stress through the production of reactive oxygen species and production of endotoxin then result in inflammation and eventually fibrosis. Factors including leptin are probably needed for fibrosis[vii].

NAFLD is a *Santarpanajanya Vyadhi* (Disease cause by over nourishment) having *Nidana* (Etiology) and *Samprapti* (Pathogenesis) similar to *Sthaulya.* Initial pathology lies at *Agnivikruti* (Vitiation of digestive mechanism) which leads to the formation of *Apakva Anna Rasa* (Improperly formed digestive end product) which again leads to the vitiation of *Kaphadosha* and unequal formation and deposit of *Meda* (fat tissue) in *Yakrit*. This condition is called as Fatty Liver. Vitiated *Kapha* and *Meda* results in *Srotorodha* (blockage of channels) 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 ie. NASH. When *Vata* comes into the picture, fibrosis occurs and the condition may progress to its drastic end stages Cirrhosis, Ascites, Hepato cellular Carcinoma and also pave way to other metabolic complications. Major factors responsible for the etio-pathogenesis of NAFLD include vitiation of *Samanavayu, Apanavayu, Pachakapitta, Ranjakapitta, Kledaka kapha , Rasa Rakta Medo dhatu* and *Pureesha*. *Annavaha, Udakavaha, Rasavaha, Raktavaha , Medovaha* and *Pureeshavaha* are the *Srotas* involved in the causation and manifestation of NAFLD.

Clinical Presentation

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 fatiguability, 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, anasacra, 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[i].

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

In the initial stage of NAFLD, patients present with heaviness and distention of abdomen, increased or decreased appetite, constipation or diarrhea, malaise and belching. This condition can be clearly compared wit *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* (Gasto - eosophageal 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) ie. *Udara* (Ascites) ; *Yakriddalyudara* is being more site specific.

Diagnosis

Diagnosis of NAFLD is strongly suggested when metabolic syndrome is present and other specific etiologies of liver disease and excessive alcoholic consumption (>20g/day) have been excluded. It should be suspected as a cause of asymptomatic elevation of amino transferases, although it can be presented with normal or fluctuating aspartate aminotransferases (AST) and alanine aminotransferases (ALT)[i]. or isolated elevation of Gamma Glutamyl Transpeptidases(GGT). Unfortunately , there is no single diagnostic blood test. ALT is normally higher than AST. Elevated ALP levels are seen in about 30% of cases. It is important to differentiate NAFLD, which does not require follow-up , from NASH. Elevated serum transaminases greater than twice the upper limit of normal and the presence of the metabolic syndrome are useful predictors of NASH[vii].

Ultrasound may show liver steatosis as a hyper echogenic image , ie. 'bright liver'. Radiologic techniques used to evaluate NAFLD include ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance spectroscopy(MRS) and fibroscan (tissue elastography). 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, ie. 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.

Treatment

There is no established treatment for NAFLD in conventional medical science. Treatment is usually directed towards optimizing body weight , which is safe and improves histological disease activity in NASH. Initial approach involves dietary modification based on metabolic profile (obesity, diabetes, hyperlipidemia, hypertension) and getting patients to increased levels of physical activity. Even small changes in body weight (0.5 to 3kg) can achieve improvement in histological changes and reversal of insulin resistance. Various other treatment modalities used for NAFLD have included treatment for risk factors like diabetes mellitus and hyperlipidaemia, and use of insulin sensitizing agents such as biguanides (metformin), thiazolidinediones (rosiglitazone, pioglitazone), antioxidants and various hepato protective agents. Other agents like pentoxifylline, telmisartan, L- carnitine and vitamin E are being evaluated.

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. 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 form 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* at the *Mula Sthana* of *Raktavaha Srotasa, Srotorodha and* vitiation *of Vata. Agnideepana* (Stimulation of digestive fire), *Rookshana* (Dryness therapy), *Srotosodhana* (Removal of blockage of channels) , pacification of *Kapha, Meda* and *Vata* should be the first line of treatment in the management of Fatty Liver. Here, the treatment principle adopted is almost similar to that of 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* (Relieves hepatic inflammation)). Judicious administration of all the four limbs of treatment ie. *Samsodhana, Samshamana, Ahara* and *Achara* will help in the reversal of fatty changes of liver and prevention of further complications.

Virechana is the most suitable S*hodhana Karma* in Liver disorders. . It is considered as the best remedy for *Pittaja & Raktaja Roga* and also for GIT disorders like *Gulma, Arsha*(Haemorrhoids), *Kamala* (Jaundice), *Chardhi* (Vomiting), *Pleeha* (Disorders of spleen), *Pakwasaya Vyatha*(Disorders of Large Intestine), *Koshthaga krumi* (Intestinal worms), *Shakrit Graha* (Constipation). It is Indicated in excessive *Dosha* accumulation *& Srotorodha* . It acts as *Agnideepana and Srotoshodhana* . Different *Churna, Taila, Ghrita, Avaleha* preparations mentioned in classical texts and available in markets are widely used for this purpose. *Churnas* like *Patoladi, Hapushadi, Narayana, Avipatti*, different preparations of *Erandataila, Trivrit Avaleha, Misraka sneha, Haritakyadi ghrita* can be used judiciously for this purpose.

While coming to *Shamana chikitsa*, single herbs like *Sharapunkha, Bhoomiamalaki, Katuki, Guduchi, Haritaki, Vasa* 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)* , Herbo- mineral preparations (*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.

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 Caraka*. Yoga and Pranayama can be included in the daily routine for better results. *Pathyapathya* which can be advised to the patient during the course of the therapy has been discussed in Table No:2

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. As per *Ayurvedic* concepts, *Agni, Kapha, Vata dosha, Anna vaha Srotasa, Rasa, Rakta, Meda Dhatu & Srotasa , Yakrit* are the cornerstones of *Samprapti* of Non-Alcoholic Fatty Liver Disease. . 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. Through the proper administration of

Ayurvedic treatment protocol along with lifestyle modification, progression of the disease and further complications can be prevented upto a great extent.

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Figures

NORMAL LIVER

DIET

⇧FA INFLUX
⇩FA OXIDATION
FIRST HIT ⇧FA SYNTHESIS
⇩VLDL ASSEMBLY
INSULIN RESISTANCE

FATTY LIVER

TNF –ALPHA
OXIDANT STRESS
SECOND HIT ENDOTOXIN
IMMUNE FACTORS

NASH

⇩⇦ OTHER FACTORS EG: LEPTIN

FIBROSIS

Figure No:1 Pathogenesis of Fatty Liver

Tables

Table No:1 Etiology of Fatty Liver

Conditions with excess fat	Liver cell damage
Obesity	Alcoholic Liver Disease (most common)
Diabetes mellitus	Starvation
Congenital hyperlipidaemia	Protein calorie malnutrition
	Chronic illness (e.g. TB)
	Acute fatty liver in late pregnancy
	Hypoxia (Anemia, cardiac failure)
	Hepatotoxins (Carbon tetrachloride, chloroform, ether, aflatoxin)
	Drug induced liver cell injury (methotrexate, CCl_4,steroids, halothane anaesthetic, tetracycline etc.)
	Reye's syndrome

Table No:2 *Pathya* & *Apathya* for NAFLD

Factor	Do's	Dont's
Diet Control	Eat freshly prepared food Eat only when hungry and after evacuation of Mala (waste products). Cereals & Pulses – Red rice, barley, green gram Vegetables – Bitter gourd, snake gourd, drumstick, curry leaves, coriander, ginger, garlic Fruits – Pomegranate, orange, dry fruits like risins Luke warm water minimum 8 glasses/day, buttermilk Gruel prepared with *Trikatu, Panchakola*	Avoid reheated & untimely food Excessive spicy , oily, salty,sour, fatty diet, pickles Basmati rice, Pulses like Black gram, Yellow gram, Fish, Meat preparations Full fat milk, curd Potato, Cauliflower Junk food, Aerated drinks, Chocolates, Ice creams, Bakery items, Artificial sweeteners, jams
Lifestyle modification	Consistent Physical exercise like brisk Walking daily half an hour in fresh air *Yoga – Dhanuraasana, Gomukhaasana, Ardhamatsy- endraasana, Bhujangasana* *Pranayama – Kapaalabhaati*	Sedentary life style Excessive & day time sleeping Smoking Alcohol consumption