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Conceptual study of Fatty Liver Disease through Ayurvedic perspective

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Abstract

Introduction: Liver is vital organ for metabolism. Fatty Liver Disease (FLD) is characterized by macro vesicular hepatic fat accumulation (steatosis). The prevalence of fatty liver disease in India is found to be as high as 9 to 32%and it mainly observed in overweight, obese, diabetic, pre-diabetic cases. Fatty liver disease is usually silent diseases with few or no symptoms. It is accidently diagnosis during abdominal ultrasound and abnormal liver function test. There are no medicines that have been approved to treat NAFLD. Therefore, an attempt has been made to study etiopathogenesis of Fatty liver disease through ayurvedic perspective.

Methods: A literature review was conducted by reading of classical text of Ayurveda and research articles. Discussion is done with experts in this field.

Discussion and conclusion: Fatty liver disease is *santarpanjanya vyadhi. Rasa, Rakta, Medo dushti hetu* are causative factor for developing Fatty liver disease. *Kapha* and *pitta* are affected *dosha, Rasa, Rakta,* and *meda* are *dushya, Agnimandya, dhatvagnimandya* are plays role in

pathogenesis of fatty Liver disease. *Yakritvriddhi, agnimandya, jwar, pandurpeetvarnata* are symptoms occurs in fatty liver disease. *Rakta Prasadana, Medolekhana, agni vardhak* and *yakrit balya dravya* will be line of treatment for fatty liver disease.

Keyword: Fatty liver disease, *Yakritvikara, raktadushti, medodushti, agnimandya.*

Introduction

Liver performs multiple functionsin the body like digestion, metabolism, regulation, detoxification, storage, synthesis, and excretion. Liver diseases can be caused by multiple reasons such as infections or by autoimmune reactions or drug toxicity.

Fatty Liver Disease (FLD) is characterized by macro vesicular hepatic fat accumulation alone (steatosis) or accompanied by sign of hepatocyte injury.[1] The prevalence of fatty liver disease in India is found to be as high as 24%[2] and mainly observed in overweight, obese, diabetic, pre-diabetic cases.[3] There are two main types: Non-alcoholic fatty liver disease (NAFLD) and Alcoholic fatty liver disease (ALD). The cause of non-alcoholic fatty liver disease (NAFLD) is unknown.[4] However, NAFLD is commonly associated with Metabolic Syndrome, obesity, diabetes, hyperlipidaemia, rapid weight loss and overuse of medicine like steroids.[5] Both NAFLD and ALD disease are usually silent diseases with few or no symptoms. It isusually accidently diagnosed during abdominal ultrasound and abnormal liver function test and blood count. Starting from a simple steatosis, FLD can proceed to steatohepatitis and fibrosis. Cirrhosis and/or hepatic carcinoma can be its terminal complications.[6]

The most important part of treating alcohol-related fatty liver disease is to stop alcohol intake. There are no approved medicines to treat NAFLD. Reducing weight can help in reducing fat in the liver, inflammation, and fibrosis.[7] pharmacological options such as Metformin, Vitamin E, fish oil, Orlistat (an inhibitor of gastric and pancreatic lipase), and Sibutramine are used for treatment of fatty liver disease but evidence behind these pharmacological modalities is weak, more studies are needed.[8]

As per Ayurveda, a disease occursdue to imbalance in the equilibrium of *doshas* (*Vata, Pitta & Kapha*), *Agni* (digestive fire), *Dhatu* (*Rasa, rakta* etc) and *Mala* (*Purish, Mutra & Sweda*). Liver can be correlated with *yakrit*. *Yakrit* is formed from *raktadhatu*.[9] *Yakrit* and *Pleeha* are

moolsthana of *raktavaha srotasa*. *Rakta dhatu* is developed by the action of *ranjaka pitta* on *rasa dhatu*, in *yakrit*.[10] During the process of metabolism and formation of *rakta dhatu*, *Kandara* (tendons) and *sira*(veins) are formed as *upadhatu* and *pitta* is formed as *mala* (byproduct).[11] So *Rakta, Pitta, Yakrit* are inter-related to each other.

Lipid or Fat can be correlated with *meda dhatu*. In Fatty Liver disease there is abnormal collection of fatty tissue in the liver. An abnormal production and collection of *meda dhatu* is clearly visualized, in Fatty Liver disease.

Therefore, an attempt has been made to study etiopathogenesis of Fatty liver disease through ayurvedic perspective.

Review of Literature

- Detail review of Fatty liver disease in allopathy texts and online studies.
- Detail review of *Yakritroga* from Ayurvedic classical text and available online platform and studies conducted in various institutes.
- Review of previous research work related to this subject will be taken.

Aim: Detail study of etiopathogenesis of fatty liver disease (FLD) through ayurvedic perspective.

Objectives:

1. To study etiological factors of Fatty Liver Disease through ayurvedic perspective.
2. To study pathogenesis of Fatty Liver Disease through ayurvedic perspective.
3. To study line of treatment for Fatty Liver Disease through ayurvedic perspective.

Methods:

- A literature review was conducted using reading of classical text of Ayurveda and research articles.
- Discussion is done with experts in this field.

Discussion:

Fatty liver disease

Fatty liver disease (steatosis) is a common condition caused by excessive collection of fat which is more that 5 to 10 % of liver weight. There are two main types:

- Non-alcoholic fatty liver disease (NAFLD)
- Alcoholic fatty liver disease (ALD)

Fatty liver disease commonly observed in Pre diabetic and diabetic condition, rapid weight gain and weight loss, metabolic

syndrome, high blood pressure, abnormal levels in lipid profile, prolong use of specific drugs: corticosteroids, Anti tuberculin drugs, exposure to infection like Hepatitis B, C, A, exposure to toxins, Intestinal microbiota dysbiosis.

Pathology

- Non-alcoholic fatty liver disease (NAFLD)

  Insulin Resistance, genetic and epigenetic factors, mitochondrial dysfunction, endoplasmic reticulum stress, microbiota, chronic low-grade inflammation, and dysfunction of adipose tissue all represent synchronic causes for development and progression of NAFLD.[12]

  - Role of diet

    Excessive calory intake and low energy consumption cause for development of obesity. Fructose is a pro-inflammatory lipogenic factor able to cause oxidative stress and TNF-α overproduction.[13]

  - Role of Intestinal Microbiota

    The intestinal microbiota, maintain the energy balance by the process of fermentation of resistant starch and non-starch polysaccharides in short-chain fatty acids (SCFA), and promoting their absorption by the intestinal epithelium. The intestinal microbiota is also able to produce enzymes that catalyze the conversion of dietary choline into toxic compounds (e.g., methylamines). In the liver, methylamines are transformed into trimethylamine-N-oxide, that induce inflammation and hepatic damage.[14]

  - Role of Visceral Adipose tissue:

    Adipose tissues secrets hormones, such as adipokines, (leptin and adiponectin). Adiponectin acts as a stimulator of insulin action in the peripheral tissues, leptin and resistin inhibit the sensitivity to insulin action.Obesity and Insulin resistance causes hypertrophy in adipocyte. The reduced levels of adiponectin and the increased levels of leptin can induce steatosis which leads to inflammation and fibrogenesis.[14]

  - Insulin Resistance (IR)

Due to an unhealthy lifestyle there is increase in the level of calories which causes IR by the increase in visceral adipose tissue and the consequent release of FFA, TNF-α, and adipokines. It causes an increase in hepatic lipogenesis and an inhibition of adipose tissue lipolysis, with a subsequent elevated flow of fatty acids in the liver. [15]

- Role of low degree chronic Inflammatory factor

  Increased levels of FFA, lipotoxicity, IR, dysfunction of peripheral adipose tissue, and endotoxemia secondary to elevated intestinal permeability can induce a low-degree chronic inflammatory state, which seems relevant in the pathophysiological mechanisms of NAFLD and NASH. Two main inflammatory pathways, JNK-AP-1 and IKK-NF-κB, are critically involved in the development of the chronic inflammation occurring during NAFLD

- Role of Mitochondrial dysfunction

  The mitochondrial and peroxisomal functions are not able to manage the increase in lipid flow, respiratory oxidation collapses, thus leading to impairment in lipid homeostasis, generation of toxic metabolites, and overproduction of reactive oxygen species (ROS). These molecules determine oxidative stress and contribute to hepatic necro-inflammatory processes and the worsening of mitochondrial damage.

- Alcoholic fatty liver disease (ALD)
- The pathogenesis of ALD can be conceptually divided into 1) Ethanol mediated liver injury, 2) Inflammatory Immune response to injury, 3) Intestinal permeability and microbiome changes.[16]

1. Ethanol mediated liver injury

   Alcohol (ethanol) is absorbed from the stomach and small intestine. Alcohol is catabolised in the liver. A cytoplasmic enzyme Alcohol dehydrogenase oxidizes alcohol into acetaldehyde. Acetaldehyde dehydrogenase (ALDH), a mitochondrial enzyme, then oxidizes

acetaldehyde into acetate. These oxidative reactions generate hydrogen, which converts nicotinamide-adenine dinucleotide (NAD) to its reduced form (NADH), increasing the redox potential (NADH/NAD) in the liver. The increased redox potential inhibits fatty acid oxidation and gluconeogenesis, promoting fat accumulation in the liver.

2. Inflammatory Immune response to injury

Cell necrosis and apoptosis result in hepatocyte loss, and subsequent attempts at regeneration result in fibrosis. Stellate (Ito) cells, which is blood channels (sinusoids) in the liver, proliferate and transform into myofibroblasts, producing an excess of type I collagen and extracellular matrix. As a result, the sinusoids narrow, limiting blood flow. Fibrosis narrows the terminal hepatic venules, compromising hepatic perfusion and thus contributing to portal hypertension. Extensive fibrosis is associated with an attempt at regeneration, resulting in liver nodules. This process culminates in cirrhosis.

3. Intestinal permeability and microbiome changes.

Alcohol changes gut permeability, increasing absorption of endotoxins released by bacteria in the gut. In response to the endotoxin's liver macrophages (Kupffer cells) release free radicals, increasing oxidative damage.

Ayurvedic Perspective

Hetu (Causative Factor)

Kapha Pitta Prakopak aahara vihara,Rasa Rakta and *Medo dushtijanya ahara vihar* are causative factor for developing Fatty liver disease.[17]

	Rakta dushti hetu	*Medo dushti hetu*	*Rasa Dushti Hetu*
1	*Vidahi annapan* (Spicy products)	*Medur padarth* (Excessive intake of non-veg)	*Guru* (Heavy to digest food)
2	*Snigdha*	*Madya sevan*	*Sheeta*

	(Fatty products)	(Excessive intake of alcohol)	(Cold products)
3	*Ushna* (Warm substance)	*Avyayam* (Lack of exercise)	*Atisnigdha* (Fatty products)
4	*Drava* (Excessive liquid)	*Diwaswap* (Daytime sleep)	*Atimatra* (Excessive quantity of food)
5	*Atap sevan* (Excessive sun exposure)		*Chintyanam cha atichintanat* (Excessive thinking and stress)
6	*Anal sevana* (Excessive work near fire)		

Samprapti (Pathogenesis)

Ayurveda has its basic framework and its own understanding of pathogenesis (Samprapti) of diseases. Ayurveda considered *Dosha, Dhatu, Mala, Srotasa-vaigunya, Agni* and *Aama* are *Vyadhi ghataka*, the basic component of body responsible for development of disease.

Fatty liver disorder is mainly observed in *sthula* (obese) person that is in *kapha prakriti* (genetic and epigenetic factor). *Avyama* (Lack of exercise), *diwaswap* (daytime sleep), *mansa and varuni sevan* (excessive intake of non-veg and alcohol) can causes *medo dushti*. It causes *jatharagni mandya* (weakness of digestive power). This affected *jatharagni* produces *aama* (undigested products) and *apachita ahara rasa*. This *aama* causes *srotorodh* (obstruction in channels). It affects nourishment of *dhatus* and *mala*. *Meda dhatvagnimandya* occurs it produces more *abaddha meda dhatu* (FFA, abnormal lipid molecules). That *abaddha meda dhatu* get circulated with *aahara rasa*. This *abaddha meda* get accumulated wherever it finds *sroto-vaigunya* (affected organ).

Person who excessive consumes *vidahi* (spicy) *snigdha* (fatty dietary products) *ushna* (hot substance) *drava* (excessive liquid) *atapa sevan* (excessive exposure to sun) *anal seven* (excessive work near fire) causes vitiation in *rakta dhatu*. *Yakrit* is *moolsthan* of *raktadhatu*. *Rakta*

dhatu is developed by the action of *ranjaka pitta* on *rasa dhatu*, in *yakrit*.[5] During the process of metabolism and formation of *rakta dhatu, Kandara* (tendons) and *sira* (veins) are formed as *upadhatu* and *pitta* is formed as *mala* (by-product). So *Rakta, Pitta, Yakrit* are get affected.

This *abaddha meda* is started accumulating at *Yakrit pradeshi* that increases *baddha meda* (adipose tissue) in *yakrit*. Due to excessive collection of *baddha meda* (adipose tissue) swelling occurs in *yakrit* which leads into *yakritvriddhi* (enlargement of Liver). *Yakritvriddhi* creates *udarshool* (pain in abdomen & right hypochondriac region). This affected *yakrit* produces vitiated *rakta dhatu* and *pitta*. *Agni* (digestive power) is depending upon good quality of *Pitta*. Vitiated *pitta* affects *agni* and produces *agnimandya* (abnormal digestive system). Due to *agnimadya, apachitaahar rasa* (undigested food) is produced which produces symptoms like *Aruchi* (loss of appetite), *Avipak* (indigestion), *drava malpravrutti* (semisolid or loose stool). Vitiated *rakta* gives discoloration to skin like pallor or yellowish discoloration. Due to lack of nourishment of other *dhatus balaksheenata* (weakness) is seen.

***Lakshana* (Symptoms)**

1. *Yakritviddhi* (Swelling at liver)
2. *Udarshool* (Pain in abdomen)
3. *Yakritpradeshi shool* (Rt hypochondric region pain)
4. *Aruchi* (loss of appetite)
5. *Avipaka* (indigestion)
6. *Manda jwara* (mild fever)
7. *Daha* (burning sensation)
8. *Shithil Malapravrutti* (semisolid stool)
9. *Bala ksheena* (weakness)
10. *Atipandu* (pallor)
11. *Twak Pittata* (yellowish discoloration of skin)[18]

***Upadrava* (Complication)**

1. *Kamala* (Jaundice)
2. *Udara* (Ascites)
3. *Pandu* (Anaemia)
4. *Hridroga* (cardiac disease)
5. *Prameha* (diabetes)
6. *Raktapitta* (bleeding disorders)

***ChiktsaTatva* (Treatment principle)**

1. *Nidan Parivarjan:* Avoid causative factors
2. *Rakta Prasadana dravya*: Drug which purifies Blood Like *Sariva, manjishta* etc.

3. *Medoghna or Lekhaniyadravya*: Drug which reduces Fat Like *Triphal, Trikatu,* etc.
4. *Agni pradeepak dravya*: Drug which improves digestive fire Like *Sunthi, Marich, Pippali,* etc.
5. *Yakrit balya dravya*: ug which strengthen Liver Like *Bhrigraj, Kutaki,* etc.

Conclusion:

As per ayurveda, patho-physiology of fatty liver disease is *Kapha* and *Pitta* are vitiated *doshas, Rasa, rakta, meda* are *dushta dhatu*. *Annavaha, raktavha* and *medovha srotasa* are affected *srotasa*. *Yakrit* Pradesh is *stansanshraya*. *Agnimandya* and *dhatvagnimandya* are *agnidushti*. These factors plays role for development of Fatty liver disease.

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