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CONCEPTUAL STUDY ON EFFICACY OF PALASHA KSHARA IN MANAGEMENT OF NON ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

Increasingly sedentary lifestyles and changing dietary patterns mean that the prevalence of obesity and insulin resistance has increased worldwide and fat accumulation in the liver is a common finding during abdominal imaging studies and on liver biopsy. In the absence of high alcohol consumption (typically, a threshold of <20 g/day for women and <30 g/day for men is adopted), this is called Non-alcoholic fatty liver disease (NAFLD). NAFLD is a wide spectrum of diseases characterized by fatty infiltration of the liver, simple steatosis, steatohepatitis, advanced fibrosis and cirrhosis.[1] NAFLD can be correlated to *Kaphaja Yakrutodara*. *Kapha Medo Dushti* occurs in the initial stage of NAFLD. This condition progresses to Non-Alcoholic Steato Hepatitis (NASH) due to inflammatory changes when *Pitta dosha* gets involved in the pathogenesis. Further fibrosis occurs which may lead to Cirrhosis when *Vata dosha* is involved. The management is breakdown of pathological factors like *Agnivaigunya*, *Srotorodha* and *Kaphamedodushti.* Restriction of consumption of fatty food, doing physical exercise and weight reduction form the first line of treatment. No agents specific for treatment of NAFLD is available. Since NAFLD is a multifactorial disease, single target based therapy has limited implications.Through the proper administration of *Ayurvedic* medications along with lifestyle modifications, progression of the disease and further complications can be prevented to a great extent. *Kshara prayoga* is indicated in *udara*. *Palasha* is considered as *kshara sreshta.*[2] *Palasha paneeya kshara prayoga* is mentioned by *acharya susrutha* in management of *Yakriddalyudara.*[3] Hence this study has been taken up to understand the role of *Palasha paneeya kshara* in management of NAFLD.

KEYWORDS: Non Alcoholic Fatty Liver Disease, *Yakrutodara, Palasha paneeya kshara.*

INTRODUCTION

Liver is the largest gland in the body. It weighs about 1600g in males and about 1300g in females. Liver occupies whole of the right hypochondrium and the greater part of the epigastrium, also extends into the left hypochondrium. Liver plays a key role in metabolism, control of infection and does the elimination of toxins and by-products of metabolism.[4]

NAFLD is considered to be hepatic manifestation of metabolic syndrome.

NAFLD is defined as the presence of hepatic steatosis with no evidence of hepatocellular injury in the form of ballooning of the hepatocytes.

When fat content exceeds 5% of total weight of liver or more than 30% of liver cells in a liver lobule are infiltrated with fat deposits, this condition is called as Fatty Liver.

Incidence of Non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steato-hepatitis (NASH) is increasing in last decade. NAFLD is estimated to effect 20-30% of the general population in western countries and 5-18% in Asia. One large European study found NAFLD to be present in 94% of obese patients, 64% of overweight patients and 25% of normal weight patients.[1] In India the prevalence of NAFLD and asymptomatic rise in liver enzymes is commonly found in adults and paediatric population. NAFLD is expected to increase in India owing to the life style and dietary habits. Obesity, dyslipidaemia, type 2 diabetes and hypertension are important risk factors.

Metabolic abnormalities including insulin resistance, impaired glycaemic control, and altered lipid metabolism have been hypothesized to contribute to the molecular

pathogenesis of NAFLD.

To diagnose the NAFLD, it is mandatory to exclude the secondary causes and alcohol consumption. The maximum limit for the alcohol consumption is 30g per day for men and 20g for women. In a person the alcohol consumption above these limits is an indication towards the alcoholic fatty liver disease (AFLD) or alcoholic steato-hepatitis (ASH). However, the damage to the liver due to alcohol depends upon many other co-factors like type of alcohol, duration of exposure, genetic pre-disposition and consuming patterns. But patients consuming alcohol in less dose as mentioned above may land in NAFLD due to metabolic risk factors which are more prominent causes than the alcohol.

There is no pharmacological agent officially approved to cure the condition.Therefore, life style modification through diet control and exercise are the preventive ways. Ayurvedic medication can improve the hepatic lipid metabolism, stop hepatic lipogenesis, regulate the mitochondrial dysfunction, modulate lipid metabolism by bile synthesis, modulate the hepatic inflammation through apoptosis and autophagy and correction of gut bacterial composition.

In comparison to modern pharmacological agents, herbal drugs are safe and effective. There are many herbal drugs and formulations that are quoted effectively in *Yakrutodara*.

Therefore this study is conducted to explore the effect of *Palasha kshara* in *Kaphaja Yakrutodara.*

Disease Review

Yakrit is one among the fifteen *Koshthangas* situated in the right side of the body below *Hridaya*. According to *Sushruta* and *Vagbhata*, *Yakrit* is developed from *matruja bhava* and derived from the *Raktaja* portion of *garbha*. According to *Acharya Arunadatta* the three *Bhavapadarthas*, i.e, *Samana vayu*, *Dehoshma*, and *Rakta dhatu* take part in the formation of *Yakrit*. *Yakri*t is the *moolasthana* of *Raktavaha srotas* and seat of *Ranjaka Pitta(Ranjakagni). Rasa dhatu* which comes to *Yakrut* and *Pleeha* gets coloured by *Ranjakagni*. It is also considered as the site of *bhutagnivyapara* and *malapitta nirmana.*

Yakritdalyodara is the term used by *Sushrutha. Acharya Sushrutha* has opined that *Yakritdalyodara* is same as that of *Plihodara* (enlargement of spleen). On palpation enlarged organs can be felt on left and right side of the *udara* in *Plihodara* and *Yakritdalyodara* respectively.

Nidana

Udara is said to be caused by factors such as intake of excessive *usna*, *ksara*, *vidahi*, *amla ahara*, improper *samsrajana karma*, intake of *snigdha*, *viruddhahara*, *karshyata(*emaciation) as a consequences of diseases like *Pliharoga*, *Arsas*, and *Grahani*, improper administration of *panchakarma* therapy, *vegadharana*, *srotodusti* and over nutrition *ati poorana* [over nutrition].[5]

Purvarupa

Loss of appetite, delayed digestion of foods that are *madhura, atisnigdha* and *guru*, food consumed causing *vidaha*, inability to appreciate the digestion and indigestion of the food, intolerance to over eating, *padashopha*, *balakshaya*, breathlessness even on slight exertion, *udara vriddhi* due to *pureesha nichaya* resulting due to *rukshata* or *udavarta*, *udaradmana* even after small meals, *rajijanma* and *valinasha.*[5]

Laxana

Daurbalya, arochaka, avipaka, varcograha, mutragraha, pipasa, angamarda, angasada, kasa, svasa, mrdu jvara, anaha, karsya, agninasha, parvabheda, asyavairasya, pain in the *kostha* due to *vata, tamahpravesha*. *Caraka* mentions appearing of *nila, harita, haridra* coloured lines on the abdomen, colour of abdomen may change to *arunavarna.*[6]

Types

SI No	*Yakrutodara*	*Laxana*
1.	*Vataja*	*Udavarta* *Ruja* *Anaha*
2.	*Pittaja*	*Moha* *Trut* *Dahana* *Jwara*
3.	*Kaphaja*	*Gourava* *Aruchi* *Kaatinya*

Samprapti Ghataka[7]

Dosha	*Kledaka kapha*
Dushya	*Rasa, rakta, Mamsa*
Agni	*Jtharagni, Dhatvagni*
Agnidusti	*Mandata*
Ama	*Agnijanya*
Srotas	*Swedavaha, Ambuvaha, Rasavaha, Raktavaha, Mamsavaha, Annavaha*
Srotodusti	*Sanga, Vimargagamana*
Udbhavasthana	*Amashayotta, Kostha*
Adhistana	*Udara*
Vyaktasthana	*Yakrit*
Rogamarga	*Abyantara*

Sancharasthana	*Sira*
Swabhava	*Chirakari*

Symptomatic comparision of *Kaphaja Yakrutodara* and NAFLD

Sl.No	***Kaphaja Yakrutodara***	**NAFLD**
1.	*Gourava*	Heaviness
2.	*Udaradmana*	Distention of abdomen
3.	*Aruchi*	Decreased appetite

Striking similarities are observed between the presentation of NAFLD and *kaphaja yakrutodara.*

Non-alcoholic fatty liver disease (NAFLD) is defined as accumulation of more than 5% of liver triglyceride without excess alcohol intake. NAFLD includes a spectrum of progressive liver disease ranging from fatty infiltration alone to fatty infiltration with inflammation and may progress to cirrhosis and primary liver cancer.

SI.NO	**Pathophysiological basis of hepatic steatosis**[8]
1.	Increased delivery of fatty acids to liver ➢ Obesity ➢ Starvation
2.	Increased synthesis of fatty acids in liver ➢ Excess Carbohydrate
3.	Increased mitochondrial beta-oxidation of fatty acids ➢ Creatinine deficiency ➢ Mitochondrial dysfunction
4.	Decreased incorporation of triglycerides into functional VLDL
5.	Impaired lipoprotein synthesis
6.	Impaired cholesterol esterification ➢ Choline deficiency ➢ Protein malnutrition
7.	Insulin resistance ➢ Increased lipolysis ➢ Hyperinsulinemia

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. 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. In reality, progression probably follows hepatocellular injury caused by a combination of several different '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-a 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.[1]

This should not be confused with acute fatty liver, which can occur in hepatic mitochondrial cytopathies, e.g. acute fatty liver of pregnancy, or in other situations, e.g. Reye's syndrome or drug toxicity (sodium valproate, tetracyclines), or with bacterial toxins (e.g. Bacillus cereus). In these, defective mitochondrial beta-oxidation of lipids leads to fat droplet accumulation in hepatocytes and microvesicular steatosis.[1]

NAFLD is frequently asymptomatic. It may be associated with symptoms similar to *kaphaja yakrutodara* like fatigue, loss of appetite, pallor and mild upper quadrant discomfort.

Investigations

Ultrasound is the first-line imaging test for patients with suspected steatosis.

There is no single diagnostic blood best for NAFLD. Serum alanine aminotransferase and Serum aspartate aminotransferase may be normal or moderately raised, usually less than twice the upperlimit of normal. Serum gamma-glutamyl transferase is often raised.

Grading of Steatosis[9]

	Steatosis Grade	Fatty Transformed Hepatocytes
0	Normal Liver	<5%
1	Mild	5-33%
2	Moderate	34-66%
3	Severe	>66%

Steatosis Grade		Ultrasonographic Features
0	Normal Liver	Echotexture of the liver is normal
1	Mild	Mildly hyperechoic liver Parenchyma, no vessel blurring, normal diaphragm visualisation.
2	Moderate	Moderate increase of liver echogenicity with slightly impaired appearance of the portal vein wall and the diaphragm
3	Severe	Marked increase of liver echogenicity with poor or no visualization of portal vein wall, diaphragm, and posterior part of the right liver lobe.

Treatment

Management of the NAFLD aims at reversal of causal factors namely weight reduction, lowering of serum lipids, exercise (improves insulin resistance) and medication to improve insulin resistance.

1. Modifications in diet: Loss of 3–5% of body weight improves steatosis and that greater weight loss (i.e., ≥7–10%) improves steatohepatitis and hepatic fibrosis. The benefits of modifying dietary macro nutrient contents (e.g., low-carbohydrate vs low-fat diets, saturated vs unsaturated fat diets) generally parallel changes in calorie consumption, suggesting that diet modifications are mainly beneficial because they reduce energy intake and improve obesity.

2. Exercises & increased physical activities: Exercise improves muscle insulin sensitivity, which improves the metabolic syndrome independent of weight loss. Both aerobic exercise and resistance training effectively reduce liver fat. At least 30 min of moderate-intensity aerobic exercise or resistance training five times per week is recommended.

3. The pharmacological interventions are antioxidants, insulin-sensitizing agents, lipid-lowering drugs, cytoprotective agents, and anti-inflammatory or antifibrotic drugs.

Ayurvedic Management

यकृति प्लीहवत् सर्वं तुल्यत्वाद्भेषजं मतम्॥[10]

Since the etiology and symptomatology of *plihodara* (splenomegaly) and *yakritodara* (hepatomegaly) are same, so all the treatment mentioned for *plihodara* can be adopted in *yakritodara*.

यकृद्दाल्येऽप्येष एव क्रियाविभागः।
विशेषतस्तु दक्षिणबाहौ सिराव्यधः ॥१५॥
मणिबन्धं सकृन्नाम्य वामाङ्गुष्ठसमीरिताम्।
दहेत् सिरां शरेणाशु प्लीहनो वैद्यः प्रशान्तये ॥१६॥[11]

In *yakruddalyudara* (enlargement of liver) too, the same treatment is employed particularly *siravyadhana* in *dakshina bahu*. Left wrist is slightly bent and the *Sira* coursing to *vamaangusta* should be cauterized with *shara (*red-hot arrow).

षट्पलं पाययेत् सर्पिः पिप्पलीर्वा प्रयोजयेत्।
सगुडामभयां वाऽपि क्षारारिष्टगणांस्तथा।[12]।

Patient of *yakruddalyudara* is treated by oral medication of *shatpala ghrita, pippali* (Piper longum), combination of *guda* and *haritaki* (Emblica officinalis), *kshara* (alkali obtained from ash of herbs) or *arishta* (fermented decoctions).

ततः संशुद्धदेहं समुद्रशुक्तिकाक्षारं पयसा पाययेत, हिङ्गुसौवर्चिके वा क्षीरेण, स्नुतेन पलाशक्षारेण वा यवक्षारं, किंशुकक्षारोदकेन वा बहुशः स्नुतेन यवक्षारं, पारिजातकेक्षुरकापामार्गक्षारं वा, तैलसंसृष्टं शोभाञ्जनकयूषं पिप्पलीसैन्धवचित्रकयुक्तं, पूतिकरञ्जक्षारं वाऽम्लस्नुतं विड्लवणपिप्पलीप्रगाढम् ॥१३॥[13]

After *shodhana* he should be made to drink *samudrashuktikshara* with milk; or *hingu* and *suvarchika* with milk; or *yavakshara* with decanted *kshara* of *palasha*; or *yavaksara* with *kimshuka ksharodaka* decanted many times; or *kshara* made out of *parijata, iksuraka* and *apamarga*; soup of *sobhanjanaka yusha* processed with oil and mixed with *pippali, saindhava* and *citraka*; or *putikaranja kshara* decanted with sour gruel and added with more quantity of *vida lavana* and *pippali*.

As mentioned by *sushruta* oral intake of different *ksharas* have been told in the management of *Yakrutodara*. One among them is *Palasha kshara*.

Different formulations mentioned in classics

- *Pippalyadi churna*
- *Vidangadi kshara*
- *Rohitakadi yoga*
- *Rohitaka ghrita*
- *Bhallataka modaka*
- *Pippalichitraka ghrita*
- *Maharohitaka ghrita*
- *Vardhaman pippali yoga*

Drug review

Palāśa plant grows all over India.
Botanical Name- Butea monosperma Lan-Kutze
Butea- Named after John Earl of Bute, Patron of Botany
Mono- One, sperma-seed
Family- Papilionaceae

Morphology

Habit- An erect tree grows upto 50 ft. height. Trunk is crooked.
Branches- Irregular, bark is rough, ash-coloured, young parts are tomentose.
Leaves-Compound, tri- foliate, petioles are 10 to 15 cm long, stipules are linear- lanceolate. Leaves deciduous, leaflets are coriaceous, the terminal leaflet is 10-20 cm long, and 10-15 broad, broadly obovate, from a cuneate base, lateral leaflets are smaller, 10 to 15 cm long and 7.5 10 10 cm wide, obliquely rounded at the base, glabrous above. Petiolules are 6 mm long and stout.Inflorescence- Raceme, 15 cm long.

Flower-Long pedicel, calyx densely velvety outside, dark olive green. Petals are orange or salmon coloured, 4 to 5 cm long standard petal is 25 cm broad, keel is semicircular, beaked and veined. Fruit-Pods, stalked, 12 to 20 cm long, 2-4 cm wide, thickened at the sutures.

Ganavargīkarana (Classical categorization)

Suśruta	*Rodhrādi gaṇa* *Muṣkakādi gana* *Ambasthādi gana* *Nyagrodhādi gana*
Bh.Pr.Ni	*Vaṭādi varga*
Caraka	Not mentioned

Kula- Aparajita kula

Properties of *Palasha*

Rasa	*katu, tikta, kashaya*
Veerya	*Ushna*
Vipaka	*Madhura*

Other Properties

Deepana, Yakrut uttejaka [Hepatic stimulant]

Palasha is one among the 23 *kshara dravya* as mentioned by *Sushruta.*

Properties of *Palasha kshara*

- *Kaphahara*
- *Agnijanana*
- *Gulma nashana*
- *Pleeha nashana*
- *Yakrutvriddi prashamana*
- *Srotoshodhana.*

Phytoconstituents:Seeds contain Palasonin, Aleuritic acid and stable oil (18%). Flowers yielded five flavonoid glucosides like butrin, Isobutrin, Coreospsin, Isocoreopsin, Sulphurein and two new compounds called Monospermoside and Isomono- permoside. Root bark afforded ß-sitosterol, Leucoantho- Cyandin, Tannic acid and Gallic acid (50%).[14]

Preparation of Palasha Kshara

All the *panchangas* of *Palasha* (*Butea monosperma)* will be collected, washed and cut into small pieces.

It will be dried completely and later burnt and the ash is collected. Obtained ash will be allowed for cooling.

For 1 part of ash 6 parts of water will be added and mixed well and it will be kept overnight. Next day morning liquid will be decanted and filtered through thick cloth.

Filtering process will be repeated for 21 times till clear liquid is obtained.

This liquid is subjected to heating in *mandagni* till all the water content gets evaporated leaving behind only solid salty white substance which will be deposited as flakes in the bottom of the vessel. This is known as *Kshara*.

Then it will be grounded to a fine powder. Obtained *Kshara* will be stored in air tight glass container.



Previous research outcomes of the studies conducted in the management of non alcoholic fatty liver disease:

A)An Open labelled single arm clinical study to evaluate the efficacy of *Palasha paneeya kshara* in the management of *Yakriddalyudara* with special reference to non-alcoholic fatty liver disease : 2017 - this study was conducted on 30 patients. Results showed improvement in liver function tests (LFTs), reduction in fatty deposits, and enhanced digestion and metabolism.The study suggests that *Palasha Kshara's deepana-pachana* (digestive & metabolic enhancing) and shodhana (detoxifying) properties help in liver detoxification and fat metabolism.[15]

B)The effect of *arogyavardhini vati* and *phalatrikadi Kvatha* in non-alcoholic fatty liver disease –case study - 2016.

Study showed significant improvement in liver function, fat metabolism, and detoxification.These formulations, known for their *Pitta*-balancing, hepatoprotective, and anti-inflammatory properties, help restore normal liver function and reduce fatty infiltration.[16]

C)*Patola katurohinyadi kwatha* in *kaphaja Yakriddalyudara* (non-alcoholic fatty Liver disease): A case study -2020.

The study reported that the administration of *Patola Katurohinyadi Kwatha* led to significant improvement in liver function tests and clinical symptoms like anorexia, indigestion and heaviness in the patient.[17]

D)A comparative clinical study on the role of *Virechana* with *uttama Shodhana churna* & *Avipattikara churna* in *yakritodara* with special refrence to fatty liver disease - 2017. The study included 60 patients, divided into two groups of 30 each. Group 1: Underwent *Virechana* therapy combined with *Uttama Shodhana Churna*. Group 2: Received *Virechana* therapy combined with *Avipattikara Churna*.The study concluded that *Virechana* therapy combined with *Uttama Shodhana Churna* and *Avipattikara Churna* was effective in improving liver function tests and clinical symptoms in patients with fatty liver disease.[18]

DISCUSSION

Yakrut is considered to be the seat of *pitta* governing digestion, transformation and metabolism of the food. When *agni* is dearranged *Ama* is produced, which leasd to variety of digestive issues. This includes *Yakrut rogas* like *yakrutodara. Yakrutodara* is corelated with NAFLD. It is a *Santarpana janya vyadhi* (~disease caused by over nourishment) where in accumulation of Ama leads to vitiation of *Kaphadosha* and inturn leads to improper formation and deposition of *Medodhatu* in *Yakrit.*

Yakrutodara is managed in *Ayurveda* by *Siravyadhana* over *dakshina bahu, Agnikarma* over *vama angusta, shatpala ghrita prayoga, shodana, abhyantara prayoga of Yavakshara, Parijathakshara, Apamarga kshara, Ikshurakakshara* and *Palasha kshara* as referred in *Sushruta samhita*. The *Phalashruti* of *Palasha kshara* mentioned by *Rasatarangini* is *Yakrudvriddhi prashamana, hence it is used in NAFLD.*

Many studies are being conducted on the efficacy of various *ksharas* in the management of *Yakrutodara*. *Kshara is having the* properties like *tridoshagnata, katu rasa, ushna virya, tikshna guna, pachana, shodhana, ropana, shoshana* and *lekhana guna and results in Yakrutvriddi prashamana* and *Srotoshodhana karma*. Hence *Palasha kshara* is selected for the present study.

CONCLUSION

Many clinical studies have been conducted on the efficacy of Kshara in the management of Yakrutodara.In the present study Palasha kshara is chosen and will be subjected for clinical trials.

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