# A study of anatomical changes in yakrit due to kamala with reference to ayurveda and modern science

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#### Abstract Introduction: Alcoholic Liver Disease: Anatomy The liver is the largest organ in the abdominal cavity and the most complex. It consists of a myriad of individual microscopic functional units called lobules. They are Kosthashakashrita kamala and Shakhashrita kamala. Koshtashakashrita kamala is clinically characterized by yellowish discolouration of skin, mucous membrane and even excreta and pathologically there is no obstruction to Pittamarga. Changes in Liver (Yakrit) in Various Types of Liver Diseases (Kamala): Chronic infections ultimately they cause the Liver Cirrhosis the most common causes of Liver cirrhosis are Chronic alcoholic intoxication, Hepatitis like Hepatitis B and Hepatitis C and Chronic ingestion of hepatotoxic drugs the common histo-pathological (i.e. Anatomical Changes are ) Grossly, with the naked eye, a cirrhotic liver appears nodular, "hub-nailed", on the external surface and nodular on the cut surface. The liver is usually indurated shrunken and vellowish-tan but it may be enlarged and yellow as in alcoholic fatty cirrhosis, rusty as in hemochromatosis or large and green as in biliary obstruction. liver contains complex parenchymal cells that perform multiple functions which are essential for life. The liver does not easily demonstrate dysfunction at least in its metabolic activities. This is because of enormous reserve capacity and marvelous regenerating power of the liver and only a small portion of the liver is enough to perform all the functions Exposure to dosha vitiating factors leads to vitiation of Pitta, especially its ushna-tikshnaguna, to cause disturbance in the normal physiology of the saumya, sneha quality of ojas which is already in circulation with raktadhatu. Along with this reduced the raktadhatu'sposhakras (required in formation of raktadhatu) is also reduced Conclusion: The Principles of the Ayurveda should be applied to treat and find out the causes and effect of treatment of Kamala (Jaundice or Liver diseases) and these results has to observed by histological (Anatomical features of Liver or Yakrit) for the better understanding of Kamla or Jaundice. Keywords: Anatomical changes in Yakrit, Kamala, Liver Cirrhosis.

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## **INTRODUCTION**

Alcoholic Liver Disease: Anatomy The liver is the largest organ in the abdominal cavity and the most complex. It

consists of a myriad of individual microscopic functional units called lobules. The liver performs a variety of functions including the removal of endogenous and exogenous materials from the blood, complex metabolic processes including bile production, carbohydrate homeostasis, lipid metabolism, urea formation, and immune functions. The liver is located in the right upper quadrant, between the fifth intercostal space in the midclavicular line down and the right costal margin. It weighs approximately 1800 grams in men and 1400 grams in women. The surfaces of the liver are smooth and convex in the superior, anterior and right lateral regions. Indentations from the colon, right kidney, duodenum and stomach are apparent on the posterior surface. The line between the vena cava and gallbladder divides the liver

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into right and left lobes. Each lobe has an independent vascular and duct supply. The lobes are divided into eight segments each containing a pedicle of portal vessels, ducts, and hepatic veins. The portal venous system extends from the intestinal capillaries to the hepatic sinusoids (Figure 8). This system carries blood from the abdominal gastrointestinal tract, the pancreas, the gallbladder and the spleen back to the heart (coursing through the liver). The largest vessel in this system is the portal vein, which is formed by the union of the splenic vein and superior mesenteric veins. The left gastric and right gastric veins and the posterior superior pancreaticoduodenal vein drain directly into the portal vein. The portal vein runs posterior to the pancreas and its extrahepatic length is anywhere from 5 to 9 centimeters. At the portahepatis, it divides into the right and left portal veins within the liver, and the cystic vein typically drains into the right hepatic branch. The portal vein supplies 70% of the blood flow to the normal liver, but only 40%of the liver oxygen supply. The remainder of the blood comes from the hepatic artery, and blood from both vessels mixes in the sinusoids. The liver receives a tremendous volume of blood, on the order of 1.5 liters per minute. This dual blood supply-from the portal vein and hepatic artery—allows the liver to be relatively resistant to hypoxemia. Unlike the systemic vasculature, the hepatic vascular system is less influenced by vasodilation and vasoconstriction. This is due to the fact that sinusoidal pressures remain relatively constant in spite of changes in blood flow. A classic example is hepatic vein occlusion resulting in high sinusoidal pressure and extracellular extravasation of fluid. To maintain a constant inflow of blood, hepatic artery blood flow is inversely related to portal vein flow. This appears to be hormonally mediated rather than neurally mediated, since it persists even in the transplanted liver <sup>1</sup>. In human being raktadhatu is given utmost importance. Among the dhatu, the diseases pertaining to rakta is more in incidence<sup>2</sup>. Kamala is a disease where in, the raktadhatu is vitiated primarily by Pitta dosha<sup>3</sup>. Change in the colour of skin, mucous membrane and even the excreta are characteristic feature of the disease. Two variants of kamala are noted on the basis of pathogenesis and clinical presentation<sup>1</sup>. They are Kosthashakashrita kamala and Shakhashrita kamala. Koshtashakashrita kamala is clinically characterized by yellowish discolouration of skin, mucous membrane and even excreta and pathologically there is no obstruction to Pittamarga<sup>1</sup>. Jaundice is a clinical manifestation which is clinically simulating kamala. Varied reasons results in Jaundice. Some of them are medically managed and some requires surgical intervention as a part of management. Surgical intervention is essential in cases where extrahepatic

obstruction is observed. Common conditions which require medical management are hereditary, hemolytic, metabolic  $etc^5$ . The infective. clinical toxic. manifestations in such conditions are similar to the descriptions of Kostashakhashrita kamala and of underlying causative factor. The incidence of such causes resulting in Jaundice in India is 2.37-3.15 per 1000 population<sup>6</sup>.A disease may be the outcome of one etiological factor or many. Similarly many causative factors result in a disease or many. The mode of each and every etiological factor in the manifestation of disease is different. Indulgences with Pitta prakopakanidana independently or after a Pittapradhanavyadhi such as Pandu are considered as nidana<sup>4</sup>. Kostashakhashrita kamala may end with fatal complications<sup>1</sup>. Any negligence from the part of patient or the physician may result in disastrous complications such as kumbakamala or even Yakrutodara

#### **MATERIAL AND METHODS**

Changes in Liver (Yakrit) in Various Types of Liver Diseases (Kamala): The majority of the Liver diseases presents as Jaundice (Kamala) the Most acute conditions of Liver causing Jaundice are all the hepatitis viruses i.e. Hepatitis A, B, E and Acute Drugs toxicity and Acute Alcoholic intoxication the Liver changes in acute conditions are temporary in Chronic infections ultimately they cause the Liver Cirrhosis the most common causes of Liver cirrhosis are Chronic alcoholic intoxication. Hepatitis like Hepatitis B and Hepatitis C and Chronic ingestion of hepatotoxic drugs the common histopathological (i.e. Anatomical Changes are ) Grossly, with the naked eye, a cirrhotic liver appears nodular, "hubnailed", on the external surface and nodular on the cut surface. Variation in size, color, shape and consistency is relevant and may help in the identification of the etiology. The liver is usually indurated shrunken and yellowish-tan but it may be enlarged and yellow as in alcoholic fatty cirrhosis, rusty as in hemochromatosis or large and green as in biliary obstruction. It is usually the privilege of the surgeon to inspect the liver in vivo, therefore he must acquaint himself with the gross changes of cirrhosis and develop the ability of detecting discolorations of possible neoplastic nodules in order to obtain adequate samples for histological examinationMicrondular cirrhosis: Small 2m nodules seperated by thin rather uniform fibroussepta usually due to a chemical agent as alcohol which diffuseuniformlythrought the liver. Macronodular cirrhosis: Larger nodules separated by wider scars and irregularly distributed throughout the liver usually due to an infectious agent such as viral hepatitis which does not diffuse uniformly throughout the liver. Presence of nodules and fibrous septa with effacement of the

lobular architecture. The nodules are of two types: type and Hyperplastic Regenerative Dissection Hypoplastic portal field: In a dissecting nodule. Notice presence of portal vein, portal artery but no bile duct. This case was interpreted as "vanishing duct syndrome". Regenerative nodules: These occur in micro and macro nodular cirrhosis. They arise in the midst of scars favored by the rich arterial blood of scar tissue. They are round nodules with a fibrous pseudo capsule with bile ductules due to obstruction of bile flow. They have embryonal type of cell plates, two cells thick, "twinning of cell plates". Nuclei are aligned at the sinusoidal pole of the plates. They often show focal cholestasis. They may undergo dysplastic and malignant changes. They compress the vessels of the capsule contributing to the perpetuation of the cirrhosis <sup>7</sup>These changes causes various symptoms and Clinical features like Portal hypertension, Hepatic encephalopathy which shows clinical features like Ascitis, Altered sensorium, Comatose, Smell to Breath, telenjactesia etc. these features has also been described in Ayurveda like Kamala has been classified as: KoshthashritaShakhashrita<sup>8</sup>In modern science jaundice is classified in three types: Haemolytic (Due to Lysis of RBCS and Excess Bilirubin Production), Obstructive (Obstruction to Common Bile Duct) Hepatocellular (Damage to Liver tissues)<sup>9</sup>. On the basis of samprati kamala gets manifested in two formskoshtaasritaandsakhaasrita kamala. Though in both type vikriti pitta play the role in sakhaasrita type the pitta which has been vitiated does not reach the koshta. So there is an underlying pathology of increased vatadosha leading to sakhagati of pitta along with an increased kapha stage crating a srotorodha. Thus not letting the normal pitta come back to koshta. Thus sakhaasraya kamala is a clear form of asayaapakarshajanyavyadhi. Form this total samprapti we can infer that mala ranjana does not occur in sakhaasraya. Kamala due to the absence of pitta in kostha thus, leading to condition of " svetavarchs" and it is a partial obstruction it certainly result in tilapishanibhavarchas. In classical reference another two chronic conditions as 'kumbha kamala' and 'haleemaka' are seen. Kumbhakamala is mentioned by AcharyaChakrapani as a bheda of koshtaasrita kamala, in which patient may present with condition of oedema associated with ascitis and bleeding tendencies. Haleemaka is being explained as vata- pitta dominant condition of kamala by Vaghbhataacharya and it is also known as alasaka.<sup>10</sup> CharakSamhita-According to CharakSamhita, Kamala is a clinical syndrome which develops after the panduroga. When a patient of Panduroga takes excessive paittikahar-vihar develops bahupittakamala. <sup>10,12</sup>ShushrutaSamhita<sup>11</sup>-According to ShushrutaSamhita, when patient of panduroga or person

affected with other diseases consumes amlaraspradhan and apathyakarahar develops kamala. Ashtang $Hridav^{13}$ -According to AshtangHriday, when pandurogi or person with excessive pitta consumes pittakarahar develops Samprapti: Koshthashakhashrita kamala<sup>10</sup>: kamala. SampraptiGhataka: 1. Dosha-Pitta. 2. Dushya-Rakta and Mansa, 3. Adhisthana-Kostha(Mahasrotasa) and shakha. Srotas-Rasavahasrotas, Raktavahasrotas, 4. Annavahasrotas. Purishvahasrotas. 5. Srotodushti-Atipravritti, Sanga, Vimargagamana.Shakhashritakamala <sup>13</sup>SampraptiGhataka: 1. Dosha – Pitta 2. Dushya -Rakta and mansa 3. Adhisthana- Kostha (mahasrotas) and shakha 4.Srotas - Rasavaha, raktavaha, annavaha, pureeshvahasrotas 5. Srotodushti – Atipravritti, sanga, vimarggamana.

#### DISCUSSION

liver contains complex parenchymal cells that perform multiple functions which are essential for life. The liver does not easily demonstrate dysfunction at least in its metabolic activities. This is because of enormous reserve capacity and marvelous regenerating power of the liver and only a small portion of the liver is enough to perform all the functions. About 75- 80% of liver, need to be out of function for any of the test to be positive. As is clear from the fact that the liver has been called "a silent organ," a diseased liver shows relatively few clinical signs unless the disease is severe or advanced. However, simple liver function tests using blood samples are widely available as part of routine health examination, providing opportunities for physicians to find abnormalities in liver function test results in daily clinical practice<sup>19</sup> This study should give a deeper insight on the intricate aspects on pathology of bahupitta kamala. :Normal complexion of skin grossly depends on two factors- Teja<sup>14</sup> and rakta<sup>15</sup> along with snehaguna<sup>16</sup> of ojas.<sup>17</sup> Pitta resides in raktadhatu.<sup>18</sup> Exposure to dosha vitiating factors leads to vitiation of Pitta, especially its ushna-tikshnaguna, to cause disturbance in the normal physiology of the saumya, sneha quality of ojas which is already in circulation with raktadhatu. Along with this reduced the raktadhatu'sposhakras (required in formation of raktadhatu) is also reduced. Decrease of both rakta and ojas hampers the normal complexion and hence pathological complexion appears. The abnormal complexion varies according to the doshic imbalance. Out of this imbalance of tridosha, aggravated pitta causes the panduvarnata complexion in pandu disease. Moreover, depreciation of the ten qualities of ojas is directly proportional to dhatu daurbalya.<sup>14</sup> (Many Acharyas agree that the condition of Bahupitta Kamala is preceded by Pandu. Hence, its discussion is invariably important too.).

### CONCLUSION

The Principles of the Ayurveda should be applied to treat and find out the causes and effect of treatment of Kamal (Jaundice or Liver diseases) and these results has to observed by histological (Anatomical features of Liver or Yakrit) for the better understanding of Kamla or Jaundice

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