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LIVER FUNCTION TESTS AND ITS AYURVEDIC INTERPRETATIONS

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ABSTRACT

Liver function tests are the biochemical parameters for the evaluation of various liver disorders. Correct interpretation of the levels provide direct clues regarding the underlying etiology and pathology. The set of tests includes serum bilrubin-direct/conjugated and indirect/unconjugated, liver enzymes like transaminases(SGOT, SGPT), Alkaline Phophatase (ALP), Gamma glutamyl transferase, Proteins-albumin, globulin, Prothrombin time, alpha feto protein. The aim of investigations are to detect hepatic abnormality, identify specific cause and measure the severity of liver damage. Ayurveda explains the involvement of *Yakrit* in several disease conditions like *Kamala* and its types *(Koshtaasritha, Shakhaasrita), Pittaja Pandu, Yakritdora, Madatyaya* and *Raktapitta*. In various *Yakrit vikaras* (liver disorders) there will be involvement of *ranjaka pitta, rakta dhatvagni, mala roopi pitta* and *dhamani* arising from the *Yakrit*. The assessment of various biochemical parameters and its interpretation to understand disease pathology is a significant contribution of contemporary science. Therefore liver function tests can be utilized to understand various *Yakrit vikaras* in a better way and plan appropriate treatment.

Keywords Jaundice, Bilirubin, Biochemical tests, *Yakrit vikaras, Kamala*

INTRODUCTION: The main aim of investigations are to detect hepatic abnormality, measure the severity of liver damage, define structural effects on the liver, identify specific cause and investigate possible complications. There is no single test by which the liver can be stated to be functioning normally. Indeed, many tests detect liver damage rather than liver function. Therefore several tests are usually undertaken in each patient, and in some instances the individual test must be repeated. Liver function tests (LFTs or LFs) are groups of clinical biochemistry, Laboratory blood assays designed to give information about the state of a patient's liver. It helps in the initial detection and management of liver diseases and the tests are frequently termed as "Liver function tests".

Several biochemical tests are useful in the evaluation and management of patients with hepatic dysfunction. These tests can be used to detect the presence of liver disease, distinguish among different types of liver disorders, gauge the extent of known liver damage and to follow the response to treatment.

Yakrit is the *moolasthana* of *rakta vaha srotas* that maintains the quality as well as quantity of rakta. *Kamala* is a *rakta pradoshaja vyadhi* that can be correlated to jaundice in contemporary science. Jaundice is the yellowish discoloration of

skin, sclera and mucus membrane due to excess of circulating bilirubin. It is typically seen when the level of bilirubin in the blood exceeds 2.5-3mg/dl. Jaundice may be prehepatic(due to hemolysis), hepatic (due to intrinsic liver disease) or cholestatic (due to either intrahepatic cholestasis or post hepatic biliary tract obstruction). The *pratyatma lakshana*(cardinal feature) of *Kamala* is *peeta netra mootra twak nakha*(yellowish discoloration of the eyes, urine, skin, nails). According to Charaka there are two types of *Kamala Koshtasrita* which can be clinically correlated to prehepatic and hepatic causes of disease and *Shakhasrita Kamala* which can be correlated to Post hepatic/Obstructive jaundice. Liver function tests is one such parameter that helps in diagnosing and understanding the underlying liver pathology. In fact this can be utilized to understand *Kamala* in a better way and plan appropriate treatment.

Liver function tests are a helpful screening tool which are an effective modality to detect hepatic dysfunction. Most of the laboratories usually employ a battery of tests in serum for initial detection management of liver diseases and these tests are frequently termed "Liver function tests" which includes the following parameters

Total bilirubin, Direct (Conjugated) bilirubin, Indirect (Unconjugated) bilirubin, Serum glutamate oxaloacetate transaminase (SGOT) or AST (Aspartate aminotransferase), SGPT (Serum glutamate pyruvate transaminase) or ALT(Alaninine transaminase), Alkaline phosphatase, Total protein, albumin, globulin and A/G ratio.

Classification of Liver function tests: Tests used in the study of patients with liver disease can be classified according to the specific function of the liver involved. These include

I. Tests of the liver's capacity to transport organic anions and to metabolize drugs- Serum bilirubin, urine bilirubin(bile pigment), urobilinogen etc.
II. Tests that detect injury to hepatocytes (serum enzyme tests)- Aminotransferases(SGOT, SGPT), alkaline phosphatase, gamma-glutamyl transpeptidase etc
III. Tests of the livers biosynthetic capacity- Serum proteins, albumin, prealbumin, serum ceruloplasmin, alpha-1 antitrypsin, alpha feto protein, prothrombin time.[2]

Table1 test Normal Range:

TEST	NORMAL RANGE
Total Bilirubin	0-1 mg/dl
Direct Bilirubin (Conjugated)	0-0.35 mg/dl
Indirect Bilirubin (Unconjugated)	0.2-0.65 mg/dl
SGOT/AST	10-50 IU/L
SGPT/ALT	10-50 IU/L
Alkaline Phosphatase	40-125 U/L
Total Protein	6.6-8.7 g/dl
Albumin	3.5-5.5 g/dl
Globulin	0.9-2.0 g/dl

DISCUSSION

Hemolytic or Prehepatic Jaundice: Jaundice in this case is caused by rapid increase in the breakdown and destruction of the red blood cells (hemolysis) overwhelming the livers ability to adequately remove the increased levels of bilirubin from the blood and as a result there is an increase of unconjugated (indirect) bilirubin in the plasma. Excessive breakdown of red cells may be due to abnormalities within the cells (Intrinsic) such as Hereditary spherocytosis, various hemoglobinopathies etc or due to factors external to the cells (extrinsic) such as hemolytic disorder of the new born, autoimmune disorders etc.

In hemolytic jaundice SGOT is either normal or mild to moderately increased, because quite amount of SGOT present in the red blood cells. SGPT and alkaline phosphatase are usually normal. Total protein, albumin, globulin and A/G ratio are normal. There is moderate to marked increase of urobilinogen in urine because of increased conjugation of bilrubin by the liver, consequently increased quantity of bile pigments enters the intestine. As a result increased quantity of stercobilin is formed making the feces very dark and more urobilinogen is formed and increased amount excreted through kidney making urine dark yellow.[3]

In Ayurvedic perspective Prehepatic/ Hemolytic Jaundice can be correlated with *Paratantra Koshtashakhastrita Kamala.* A person suffering from *Pandu roga* indulges in *hetus*(etiological factors) that causes morbidity of *Pitta dosha* leads to the manifestation of *Paratantra Koshtashakhasrita Kamala.* There will be morbid *pitta* and *rakta* within the *Yakrit* and abnormality of *raktadhatwagni* leads to excess generation of *mala pitta*. It will be clinically characterized by jaundice and discoloration of stools due to excess accumulation of *mala pitta* in *shakha* and *koshta* respectively. There will be involvement of *ranjaka pitta* which is indicated by the reduced hemoglobin in the blood and morbid *raktadhtatwagni* is indicated by increased unconjugated bilirubin/Indirect bilirubin in the blood.[4]

Hepatic or Hepatocellular Jaundice: This type is due to disease or damage of the parenchymal cells of the liver.

Conditions in which there is defective conjugation**:** Here there is reduction in the number of functioning liver cells, so that there is impairement in the liver function as seen in chronic hepatitis or there may be a specific defect in the conjugation process as in Gilberts disease (UDP-glucuronyl transferase deficiency or Crigler-Najjer syndrome. In chronic hepatitis because of decreased functioning of liver cells there is decreased ability of liver cells to conjugate bilirubin and as a result there is increased unconjugated bilirubin in serum is observed. Synthetic function of liver is also affected in chronic hepatitis resulting in low normal to mild decrease of albumin levels in the serum. Moderate to marked decrease of albumin is observed in liver failure and cirrhosis of liver. Aminotranferases level decrease compared to acute hepatitis reaching mild to moderate elevations. The level of alkaline phosphatase may be normal or moderately elevated.

Conditions such as infective (Viral hepatitis) and toxic jaundice (Carbon tetrachloride necrosis): In this type of Jaundice there is extensive damage to liver cells and also considerable degree of intrahepatic obstruction. In these conditions there is increase of conjugated/direct and unconjugated bilirubin/Indirect in almost equal

proportion. Hepatic inflammation and necrosis in acute hepatitis, toxic injury or ischemic injury results in the leakage of enzymes AST (SGOT) and ALT (SGPT) from liver into circulation. Value of AST and ALT may cross 20 times than upper limit. In acute viral hepatitis, alkaline phosphatase is usually either normal or moderately increased. Hepatitis A may present a cholestatic picture with marked and prolonged itching and elevation of alkaline phosphatase. Total protein, albumin and A:G ratio are normal till the synthetic function of the liver is normal. Prothrombin time is prolonged.[5]

Destrruction of liver cells release the enzymes with consequent rise in their values in plasma. In obstructive jaundice and more so in acute hepatitis, the serum levels of SGOT and SGPT rise to very high levels (300-1500 units). Chronic hepatitis may produce moderate elevations of serum transaminases.[6]

This variety of *Kamala* is considered as *bahu pitta kamala* since it is characterized by excess production of *mala pitta*. *Jwara* as an *Oupasargika roga* can be considered as an *utpadaka hetu*(predisposing factors)in the manifestation of *Koshtashakastrita kamala*(Infective hepatitis) In this type morbid *pitta* is present in both *koshta* and *shakha*. Altered functioning of *raktadhatwagni* can be identified by increased levels of conjugated and unconjugated bilirubin associated with marked elevation of transaminases (SGOT/AST, SGPT,ALT).The clinicial features are yellowish discoloration of eyes, skin, nails and face of the patient, burning sensation, weakness and anorexia.[7]

Cholestatic Jaundice: In this type there is disproportionate rise in alkaline phosphatase compared to aminotransferases. The bilirubin can be prominently elevated in both hepatocellular and cholestatic conditions and therefore is not necessarily helpful in differentiating between the two. Normal to moderate elevation of aminotransferases rarely exceeds 500 U/L. Alkaline phosphatase is elevated often > 4 times of upper normal limits. Diseases that predominantly affect hepatocyte secretions (eg: Obstructive disease) will be accompanied by elevations of alkaline phosphatase levels like duct obstruction, primary sclerosing cholangitis and primary biliary cirrhosis (PBC) are some examples of diseases in which elevated alkaline phosphatase levels are often predominant over transaminase level elevations. Total protein and albumin are usually normal till the alteration of synthetic function of liver.

Obstructive Or Posthepatic Jaundice: In this the cholestasis is extrahepatic, due to interference with the normal passage of bile to the duodenum. This includes atresis of the main bile duct, a gall stone in the common bile duct, a tumor of the bile duct, bile duct stricture (following earlier surgery), extrinsic tumors compressing the major bile ducts (such as carcinoma of the head of the pancreas).Due to cholestasis there is increased level of conjugated bilirubin in serum due to decreased excretion into the bile duct or backward leakage of the pigment.[8] There is decreased/no excretion of bile into the intestine and as a result decreased or little amount of stercobilinogen is formed thus making the faeces pale. Similarly there is decreased/no excretion of urobilinogen in urine. Color of urine is dark yellow because of the presence of conjugated bilirubin in urine. There will be marked elevation of Alkaline phosphatase and Gamma glutamyl transferase to several

times the normal level after several days of bile duct obstruction. The highest levels alkaline phosphatase elevation often greater than 1000 U/L or more than six times the normal value are found in diffuse infiltrative diseases of the liver such as infiltrating tumors and fungal infections. AST and ALT are normal to moderately increased.

In this type of *Kamala mala pitta* forms normally owing to the action of *raktadhatwagni* but the clearance of *mala pitta* is affected and it gets accumulated in *Shakha* and manifests as *Shakhasrita kamala*. Here the vitiated kapha along with vitiated vata causes vimargagamana of pitta from koshta to shakha. The vitiated vata causes *ashayaapakarsha* of *ranjaka pitta* into the *shakha* producing *pitta vruddhi lakshanas* in *Shakhas* The patient develops *sweta varchas*(clay colored stools) since *malaranjana* is not taking place properly. Increased conjugated bilirubin levels are due to altered functioning of *raktadhatwagni* and rise in alkaline phosphatase and gamma glutamyl transferase are due to obstruction to the clearance of *mala pitta*.[9/]

Hepatic Cirrhosis/Portal Hypertension:

World wide the most common cause of cirrhosis is excessive alcohol consumption and prolonged viral hepatitis A, hepatitis B, hepatitis C, biliary damage or obstruction and post-surgical biliary strictures will also result in hepatic cirrhosis. In liver cirrhosis induced progressive and widespread death of hepatocytes associated with inflammation and fibrosis ultimately leads to loss of normal liver architecture It is a complication of various forms of liver diseases such as hepatitis, hepatomas, chronic alcoholism that involves loss of hepatocytes and irreversible scarring (fibrosis) of the liver. Portal hypertension is defined as hepatic venous pressure gradient above 5 mm Hg. It results from a combination of increased flow into the portal circulation and/or increased resistance to portal blood flow.[10]

According to Ayurveda it can be correlated with *Kumbha Kamala*. When *Kamala* perisists for a long period of time then it will result in *Kumbha Kamala.* It can be considered as a complication of improperly managed *koshtaashrita Kamala*. *Atipravruti* and *Vimargagamana* of *doshas* will occur. The clinical manifestations are *Krishna peeta varchas and mootra* (black and yellow colored stools and urine), *shotha* (edema), *rakta chardi* (bleeding tendency), *aruchi* .[11]

Variations of liver function tests seen in this condition are Bilrubin (Total & Direct-elevates slightly), Aminotransferase(AST and ALT moderately elevated, with AST> ALT), Alkaline phosphatase (slightly elevated but less than 2-3 times), Gamma-glutamyl transferase (GGT elevated, correlates with AP level), Albumin level falls, Globulin increases, Prothrombin time increases..Prolongation of PT is also suggestive of poor long term outcome in chronic liver disease.

Hepatoma/Hepatocellular Carcinoma:

The most common type of primary liver cancer usually occurring in patients with cirrhosis. *Yakrit vruddhi/udara* may occur by *shonitha vruddhi.* According to Charaka *dusta shonitha vruddhi* takes place because of *raktadusthikara hetu* and *dusti* of other *srotas*. While describing the *samprapti* of *achyuta yakrit vruddhi* Charaka mentions *rasa* and *rakta vaha sroto dusti* results in *rakta vruddh*i there by *Yakrutodara*. According to Chakrapani *mamsavaha srotodusti* is responsible for *rakta vruddhi*.[12]An alpha-feto Protein

(AFP) value above 400-500 µg/L has been considered to be diagnostic for hepatocellular carcinoma in patients with cirrhosis. A high AFP concentration >400 µg/L in HCC is associated with greater tumor size, bilobar involvement, portal vein invasion and a lower median survival rate.

CONCLUSION: Several biochemical tests are used in the evaluation and management Yakrit vikaras(Hepatic disorders) and Liver function tests is one such parameter that helps in understanding liver activity, recognize inflammation and impairment of the liver. *Paratantra Koshtashaksrita Kamala* includes hemolytic anemias and various hemoglobinopathies like Thalassemmia and Sickle cell disease and it is indicated by increased unconjugated bilirubin/Indirect bilirubin in the blood whereas *Swatantra Koshtashakhasrita Kamala* includes various causes of hepatic/hepatocellular jaundice like viral hepatitis, alcoholic hepatitis and it is indicated by marked elevation of transaminases(SGOT,SGPT). *Shakhasrita Kamala* includes various causes of Post hepatic/Cholestatic Jaundice such as biliary atresia, acute cholangitis and cholangiocarcinoma and serum alkaline phosphatase and gamma glutamyl levels increases. In *Kumbha Kamala* as well as in *Asadhya Kamala* i.e hepatic cirrhosis and hepatic failure conditions there will be a significant fall in albumin levels and prolongation of prothrombin time.

Several markers of liver disease are available but in many cases their usefulness is limited by insufficient sensitivity or specificity. In addition, significant liver damage may already have been occurred in patients who have normal findings on liver function tests. However in most of the cases LFTs indicate impaired excretion of bilirubin , possible hepatocellular injury(ALT, AST) or interruption of bile flow or cholestasis (ALP, GGT) .Therefore a fundamental understanding of abnormalities in liver enzymes is important to assist clinicians to develop a rational approach in the evaluation of various Yakrit vikaras(liver diseases).

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