



JAUNDICE AND AYURVEDIC APPROACH FOR ITS MANAGEMENT - A REVIEW ARTICLE

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ABSTRACT

Jaundice is a condition in which yellowness of skin, sclerae, mucous membranes, and excretions occurs due to hyper bilirubinemia and deposition of bile pigments. Equilibrium between bilirubin production and clearance is disturbed. Three types of jaundice like haemolytic, hepato-cellular and obstructive. In Ayurveda jaundice is described as *Kamala roga*. Pitta aggravating diet and regimen, the pitta so aggravated burn the *Rakta* and *Mamsa* to cause the disease *Kamala*. *Kamala* has been described in detail along with its etiology, patho -genesis, symptoms, compli-cations and management also. According to Acharyas Charak *Kamala Roga* is divided into two *Koshtashrita Kamala* and *Shakhashrita Kamala*. Some medicine are use full in jaundice like a *kwatha*, *vati*, *churna* or *arista* has been described during the description of treatment of *Kamala* in Ayurveda.

Keywords: Jaundice, Hyperbilirubinemia, *Kamala*, *Koshtashrita*, *Shakhashrita*, ayurvedic approach.

INTRODUCTION: Jaundice is yellowish discolouration of skin, sclera, mucous membranes and other tissues due to excess of bilirubin in the blood. The normal blood level is .5 – 1 mg/dl. Clinically jaundice is to be manifested when serum bilirubin rises above 3mg/dl. Approximately 30 mg of bilirubin is formed in the body every day 80% is derived from senescent erythrocytes and 20% from other sources¹. Bilirubin combines with albumin and this product is being insoluble in the water does not appear in urine. In haemolytic jaundice which most of the bilirubin is unconjugated, urine does not contain bilirubin. Three main phases are recognized in the metabolism of bilirubin. These are: [1] Entry into the liver cell, [2] Conjugation and [3] Excretion into the bile. After uptake, bilirubin is conjugate with glucuronic acid in the endoplasmic reticulum of hepatocytes which the help of

glucuronyl transferase and it reduces the level of enzyme that are responsible for certain forms of congenital hyperbilirubinemias like crigler-Najjar syndrome. Conjugated bilirubin in water – soluble and it freely passes in to urine. Bilirubin diglucuronide in the gut is acted upon by bacteria present in the distel small intestine and the colon and is converted in to urobilinogen. Uroblinogen is reabsorbed mainly from the intestine and to a small extent from the large intestine into the portal blood. It is subjected to enterohepatic circulation. The portion that is present in stool without being absorbed is called stercobilinogen. Both unconjugated and conjugated bilirubin stain tissues. Collagenous and elastic tissue have the maximum affinity for bilirubin and therefore, tissue rich in these are stained early and most deeply². In Ayurveda jaundice is described

as *Kamala roga*. *Kamala* has been described in detail along with its etiology, pathogenesis, symptoms, complications and management also.

AYURVEDA

REVIEW:

In Ayurveda jaundice is described as *Kamala roga*. *Kamala* has been described in detail along with its etiology, pathogenesis, symptoms, complications and management also. If the patient suffering from *pandu* indulges in *Pitta* aggravating diet and regimen, the *pitta* so aggravated burn the *Rakta* (blood) and *Mamsa* to cause the disease *Kamala* (jaundice). Acharya Charaka *Kamala roga* is divided into two types *Koshtashrita Kamala* and *Shakhashrita Kamala*³. (1) ***Koshtashrita Kamala*** which occurs as a result of *pitta vridhi* in is divided into two parts *Rakta dhatu* after the use of its aggravating causes has similarity with the mechanism of pre hepatic jaundice or Haemolytic jaundice. The term *Koshta* means “*Sharira Madhya*” or *Maha Srotasa*. Some amount of the accumulated *pitta* in the *Koshta* spread toward the *Shakha* and some amount stays at the gastro intestinal tract, due to function of *Vata Dosha*. As result the *Pitta* circulating in the *Shakha* produces yellow colour of eye, skin, nail and urine on the other hand *Pitta* stay in the *Koshta* (GIT) produces excessive yellow colour of stool. (2) ***Shakhashrita Kamala*** is produced due to the obstruction of normal *Pittavaha srotas* by *kapha* and *vata*, resulting in *pitta vridhi* in the *rakta dhatu*. Due to obstruction of *Pittavaha Srotasa* *Pitta* does not reach to the *Pakwashaya* which is responsible for the normal colour of stool. As a result “*Tila Pishta Sannibham Varchas*” (Clay colour stool) are produced⁴.

On the other hand major part of the

Pitta retained in the *Shakha* mixes with *Rakta Dhatu* (blood) and there is excretion of dark yellow colour of urine. While commenting upon the above classification, Cakrapani described *Koshtashrita Kamala* - *BahuPittaja*, *Paratantraj*, *Shakhashrita Kamala*- *AlpaPittaj*, *Swatantraja*, *Avarodhaja*. Charak also described *Kumbha Kamala*⁴ and *Halimaka*⁵ as *avastha visesha* of *Kamala* and *Pandu*. Acharya *sushruta* described ‘*Kamala*’ as synonym of *Pandu*. *Apanaki*, *Kumbhahvaya*, *Lagharaka* and *Alasakya* are also described as synonyms of *Pandu*⁶. *Kumbasaha* (*Kumbha Kamala*) is mentioned as a *bhedha* of *Kamala*. *Sushrut* described *Lagharaka* and *Alasakya* as *avaathaviseshas* of *Kamala*. Hepatocellular jaundice is very similar to *koshtashrita Kamala* and obstructive jaundice is very similar to *Shakhashrita Kamala*.

MODERNREVIEW:

Clinically useful classification of jaundice [Three types of jaundice]

1. Haemolytic jaundice / pre-hepatic jaundice/overproduction of bilirubin
2. Hepatocellular / hepatic jaundice / decreased hepatic uptake
3. Obstructive jaundice / post-hepatic jaundice

Causes of Haemolytic Jaundice: caused during the pre-hepatic phase is due to the excessive destruction (haemolysis) of red blood cells from various conditions. This rapid increase in bilirubin levels in the bloodstream overwhelms the liver's capability to properly metabolize the bilirubin, and consequently the levels of unconjugated bilirubin increase. Conditions which can lead to an increase in the hemolysis of red blood cells include: Malaria, sickle cell anaemia, Hereditary spherocytosis, Thalassemia, Glucose-6-

phosphate dehydrogenase deficiency (G6PD), drugs or other toxins, and Autoimmune disorders.

Causes of Hepatocellular Jaundice:

Jaundice caused during the hepatic phase can arise from abnormalities in the metabolism and/or excretion of bilirubin. This can lead to an increase in both unconjugated and/or conjugated bilirubin levels. Conditions with a hepatic cause of jaundice include:-

Acute or chronic hepatitis (commonly viral [hepatitis A, B, C, D, E] or alcohol related), Cirrhosis (caused by various conditions), Drugs or other toxins, Crigler- Najjar syndrome, Autoimmune disorders, Gilbert's syndrome, and Liver cancer.

Causes of Obstructive jaundice: Post hepatic jaundice is such type of jaundice in

which the cause lies in the biliary portion of hepatobiliary system. The major cause of post hepatic jaundice is extrahepatic biliary obstruction. Therefore it is also known as abdominal mass are also the representatives of obstructive jaundice⁷. Classification of obstruction on the basis of anatomical location-Upper third obstruction:- Polycystic Liver disease, oriental choangiohepatit, sclerosing cholangitis, Itrogenic, injury to the bile duct. Middle third obstruction:-Cystic fibrosa, intra biliary parasites, cyst. Lower third obstruction:-Pancreatic Tumor, Ampullary tum or, Diverticula, duodenal ulcer.

Symptoms of jaundice⁸

Tab. 1 Symptoms of Kamala Rog similarly in jaundice

In Ayurveda	In Modern
<i>Haridra netra</i>	Yellowness of conjunctiva
<i>Haridra twak</i>	Yellowness of Skin
<i>Haridra nakh</i>	Yellowness of nails
<i>Haridra anan</i>	Yellowness of buccal cavity
<i>Rakta-Peet sakrit mutro</i>	Yellow stool and urine
<i>Bhenk varna</i>	Greenish yellow tinge of body
<i>Daha</i>	Burning sensation of the body
<i>Avipak</i>	Indigestion
<i>Daurbalya</i>	Weakness
<i>Hrillas</i>	Nausea
<i>Chardi</i>	Vomitting
<i>Shohta</i>	Edema
<i>Jawar</i>	Fever
<i>Bhinna varcha</i>	Diarrhoea
<i>Tilpishta varcha</i>	Clay coloured stool
<i>Hikka and shwas</i>	Hiccough And cough
<i>Angmarda</i>	Bodyache

Differential Diagnosis:

- **Haemolytic jaundice [red blood cell destruction as a cause] symptoms and signs include**
- Weakness

- Pallor, and anaimia
- Total bilirubin incresease
- Uncounjugated bilirubin increase but counjugated bilirubin normal

- Counjugated bilirubin not present in urine
- Urobilinogen present
- Stool and urine colour normal
- Splenomegaly
- **Hepatocellular jaundice symptoms and signs include-**
- Fatigue
- Mild fever
- Muscle aches,
- Nausea and vomiting
- Total bilirubin increase
- Uncounjugated bilirubin and counjugated bilirubin increase
- Counjugated bilirubin present in urine
- Urobilinogen decrease
- Stool pale and urine colour dark(urobilinogen, stercobilinogen and conjugated bilirubin increase)
- Jaundice.
- **Obstructive jaundice [Biliary obstruction] symptoms and signs include**
- Light-colored stool,
- Dark urine,
- Itching,
- Pain in the right side of the abdomen,
- Uncounjugated bilirubin normal and counjugated bilirubin increase
- Counjugated bilirubin present in urine
- Urobilinogen decrease
- Stool pale and urine colour dark(urobilinogen, stercobilinogen and conjugated bilirubin increase)
- Nausea
- Vomiting

Investigation in jaundice:

- **Serum Bilirubin:** (0.2 – 1.2 mg%) – confirms diagnosis of Jaundice and its severity

- **Direct Bilirubin:** It is the proof of conjugating ability of Liver – high in obstructive jaundice

- **Indirect Bilirubin:** Level rises when there is high production or when conjugation fails

- **Alkaline Phosphatase:** (60-210 IU/L) – This enzyme is actively secreted into biliary canaliculi. Very high levels suggest obstruction^{9A}

- **Transaminases:** Serum glutamic oxaloacetic transaminase(SGOT), Serum glutamic pyruvic transaminase (SGPT) 10-40 IU/L – suggestive of hepatic cellular damage – Hepatocellular jaundice – in prolonged obstruction, increased levels suggest poor prognosis^{9B}

- **Gamma glutamyl transpeptidase (GGT):** (10-40 IU/L) – Although reasonably specific to the liver and a more sensitive marker for cholestatic damage than ALP, Gamma glutamyl transpeptidase (GGT) may be elevated with even minor, sub-clinical levels of liver dysfunction^{9C}.

- **Serum Albumin:** Important in cirrhosis of liver. An alternative to albumin measurements is pre-albumin, which is better at detecting acute changes TP=6-8g/dL

Alb=3.8-5 g/dL – It is decreased in Chronic Liver Disease

Glb=1.8-3.5 g/dL

- **Urinary Bilirubin and Urobilinogen:** Urinary bilirubin is present only in obstructive jaundice. High urobilinogen confirms that there is no obstruction

- **Tumor markers:** like CEA, CA 19-9 may be done if obstructive jaundice is diagnosed

- **USG Abdomin:** Organ of origin of mass-GB/ Liver/pancreas, Character of

mass – cystic/Solid/Acoustic shadow, CBD dilatation, Site of obstruction, Ascitis, Enlarged Liver, Stones, Hepatic metastasis Lymph nodes

- **CT scan abdominal** 1.Site of obstruction 2. Hepatic metastasis 3.Lymphnode involvement

MANAGEMENT OF JAUNDICE:

Nidana parivarjanam¹⁰:- Chikitsa is defined as avoidance of causative factors. Ayurveda basically being emphatic about “swastyasya swatyarakshan” give priority to prophylactic management. This is very much applicable in the case of Kamala.

Sodhan aushadhi for Kamala:

Snephan: The use of following Ghrits are said to be excellent for Snapan in Kamala Rog by different acharyas:- Kalyanak ghrit, Panchatikt Ghrit, Maha Tikta Grit, Pach Gavya Grita, Drakha Ghrit, Tilvak Ghrit.

Virechan: Kamala is one among them and is describe as Virechan sadhya vyadhi. Patient suffering from Kamala is given Tikt Dryva ukt Mardu virechana¹¹. Like a Gomutra with Milk, only cow milk. After the GIT is cleansed by the purgation. After purgation the use pathya in the patient.

Treatment of Shakhasrita Kamala¹²: The treatment with pungent, sharp, hot saline and extremely sour drugs is continued till the stool of the patient acquires the colour of pitta (yellow because of the presence of bile), and the vayu gets alleviated. When Pitta returns to its own habits, the stool gets coloured with Pitta (bile), and the patient prescribed earlier for the treatment

of Jaundice (*kosthasrita Kamala*) should be resumed.

B. Samnaousadhi for Kamala: Several herbal, mineral and preparation have been advocated in *Kamala roga*. They are-

1. Swaras: Triphala swaras, Amrita Swaras, Daruharidra Swaras, Nimba Patra Swaras, BhuNimba, Tamalaki Swarasa with honey¹³.

2. Singal Drug: Kutaki, Amrita, Kalemegha, Dharuharidra, Nimba, Shunthi, Bhunimba, Vasa, Bringraj, Punarnava, Bhumyamalki.

3. Compound Drug

Churnas: Navayasa churna¹⁴, Bhu Nimbadi churna¹⁵, Haritakiyadi churna¹⁶,

Vati: Arogyavardini vati, Kutki vati.

Gutika: MandooraVataka¹⁷.

Kwatha: Visaladi Kwatha¹⁸, Vasadi Kwatha¹⁹, Phalatrikadi Kwatha, Dasmuladi Kwatha.

Rasaoushadi: Sawarnamakshik Yoga²⁰, Yogaraj²¹, Punarnava Mandhura²², Mandura Bhasma²³.

Avaleha: Drakshadi leha²⁴, Amalakyadi leha²⁵, Dhatriyava leha²⁶, Dravyadi leha²⁷.

Asava&Arista: Dhatriyarista²⁸, Rohitakarista²⁹, Phalarista³⁰.

Loha: Trika trayadi loha, Dhatri Loha, Vidangadi loha, Tapyadi loha, Nish aloha, Yakrudari loha, Kamala ntak loha, Panchanana loha

Apathya Ahars and Vihar in Kamala:

Nidan can also be conveniently read under Apathyas. Pitta prakopa Ahara Viharas are described as Nidana of Koshtashrita Kamala .

	Aharas	Viharas
<i>Pathya</i>	<i>Jeerna Shali, shashtika, kodrava, Yava, Godhuma, Mudak, Adhaki, Masur, Jagal masras, Punarnava, Anjir, Guduchi,</i>	<i>Virechan Karma, Agnikarma, Dahan karma, Complit rest</i>

	<i>Muli, Bathua, Munka, Avala</i>	
<i>Apathya</i>	<i>Patra saka, Hingu, Pinyaka, Tambula, Sarshapa, sura, Amla Ras, Lavana Ras, Kulatha, Dustambu pana, Viruddhasana, Tikshana Ahara, Dadhi, Ghrita, Matsya and other Pitta vardaka Aharas Fatty diet and protein rich diet</i>	<i>Ativyayama, Diwaswapna, Swedana, Raktamokshana, Vamana, Dhumapana, Maithuna, Aayasam³¹</i>

DISCUSSION: Prevalence of Jaundice is increasing alarmingly due to excessive contaminated food and water, high fatty diet overcrowding, occupational conditions, stress and poor hygiene etc. Therefore, *Nidana parivarjana* has got a significant role to play in the management of the disease jaundice. Normally the liver cells absorb the bilirubin and secrete it along with other bile constituents. If the liver is diseased, or if the flow of bile is obstructed, or if destruction of erythrocytes is excessive; the bilirubin accumulates in the blood and eventually will produce jaundice. All above medicine are very effective in jaundice.

CONCLUSION: On the basis of above description and medicine it can be concluded that our ancient Acharyas had complete knowledge about the complete pathophysiology, types of *Kamala*, diagnosis and the line of treatment of *Kamala* (Jaundice). various principles of Ayurveda and many a formulations can be used according to Roga & Rogi bala. They were much about concerned towards the health of patient due to which there is description of a lot of medicines of jaundice according to the nature and nurture of patient.

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