

## MANAGEMENT OF HEPATOCELLULAR JAUNDICE IN AYURVEDA – A CASE REPORT

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### ABSTRACT:

Hepatocellular jaundice arises because of damage to hepatic cells. Jaundice is a condition in which yellowness of skin, sclerae, mucous membranes, and excretions occurs due to hyper bilirubinemia and deposition of bile pigments. It occurs when the ability of the liver to conjugate normal amounts of bilirubin into bilirubin diglucuronide is significantly reduced by inadequate intracellular transport or enzyme systems. In Ayurveda jaundice is described as *Kamla roga*. *Kamala* has been described in detail along with its etiology, pathogenesis, symptoms, complications and management also. In Ayurveda *kamala roga* is divided into two parts and Hepatocellular jaundice is very similar to *kosthashrita kamla*. A lot of medicines either in form of *kvatha*, *vati*, *churna* or *arista* has been described during the description of treatment of *kamla* in Ayurveda. Here a case report of a 17 1/2- Years-male having jaundice with increased prothrombin time is being presented who was treated with Ayurveda effectively.

**Key Words:** Hepatocellular jaundice, *Kamala*, Hyperbilirubinemia.

### INTRODUCTION:

Jaundice also known as icterus. Hepatocellular (hepatic) jaundice can be caused by acute or chronic hepatitis, hepatotoxicity, cirrhosis, drug-induced hepatitis and alcoholic liver disease. Hepatocellular jaundice occurs from an inability of the liver to transport bilirubin into the bile, occurring as a consequence of parenchymal liver disease. Bilirubin transport across the hepatocytes may be impaired at any point between uptake of unconjugated bilirubin into the cells and transport of conjugated bilirubin into the canaliculi. In addition, swelling of cells and edema resulting from the disease itself may cause obstruction of the biliary canaliculi. In Hepatocellular jaundice the concentration of both unconjugated and conjugated bilirubin in the blood increase, perhaps because of the variable way in which bilirubin transport is disturbed. The severity of jaundice, other clinical features may vary with the underlying disease.<sup>[1]</sup>

### Signs and symptoms<sup>[2]</sup>

- Yellowish discoloration of skin
- Yellowish discoloration of mucus membranes
- Yellowish discoloration of sclera
- Yellowish discoloration of urine
- Anorexia

- Nausea, vomiting etc.

**CASE HISTORY-**A 17 1/2-years-male named Ranjeet Kumar, came on 05/2/15 in Varanasi (UP) with the complaint of-

- Decreased appetite
- Nausea
- Vomiting

### On examination

Yellowish discoloration of sclera and mucous membrane.

For this problem he was advised for laboratory investigation.

**Table 1. Liver Function Tests**

Test	Results on 05 / 2 / 15	Normal Value
Bilirubin total	5.9 mg/dl	Upto 1 mg/dl

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Bilirubin (Direct)	3.8 mg/dl	Upto .2 mg/dl
Bilirubin (Indirect)	2.1 mg/dl	Upto .8 mg/dl
SGOT (AST)	1276.1 U/L	Upto 37 U/L
SGPT (ALT)	1694.8 U/L	Upto 40 U/L
Alk. Phosphatase	387.6 U/L	98-279 U/L
Protein Total	7.4 gm/dl	6.6-8.7 gm/dl
Albumin	3.9 gm/dl	3.5-5.0 gm/dl
Globulin	3.5 gm/dl	2.7-3.7 gm/dl
A/G ratio	1.1:1	2:1

According to this table patient was suffering from hepatocellular jaundice as both direct and indirect bilirubin was increased along with high transaminase enzymes SGPT is more than SGOT.

#### Ayurvedic medication advised- On 5/2/15

- *Phalatrikadi ghan vati* 3 tab TDS
- *Punarnavastka ghan vati* 2 tab TDS
- *Arogyavardhni vati* 3 gm
- *Punarnava Mandur* 3 gm

This dose divided into 8 parts. One dose BD

First this treatment was given for 4 days. After that LFT was done again

**Table 2. Liver Function Tests**

Test	Results on 9/2/15	Normal Value
Bilirubin total	4.9 mg/dl	Upto 1 mg/dl
Bilirubin	3.1 mg/dl	Upto .2 mg/dl
Bilirubin	1.8 mg/dl	Upto .8 mg/dl
SGOT (AST)	1836.9 U/L	Upto 37 U/L
SGPT (ALT)	2851.7 U/L	Upto 40 U/L
Alk. Phosphatase	228.3 U/L	98-279 U/L

#### HBsAg- NEGATIVE

After this treatment was advised with some change as

- *Phalatrikadi ghan vati* 3 tab TDS
- *Punarnavastka ghan vati* 2 tab TDS
- *Arogyavardhni vati* 10 gm
- *Punarnava Mandur* 10 gm

This dose divided into 20 parts. One dose BD

- *Avipattikar churna* 5gm BD

This treatment was advised for 15 days. Along this treatment LFT was repeated again and again. In between it was also seen that patient was going towards hepatic failure as prothrombin time was

increased but after the completion of treatment patient was absolutely healthy. All complaints were resolved.

#### RESULTS:

**Table 3. Liver Function Tests**

Test	On 12/2/15	On 16/2/15	On 20/2/15	Normal Value
Bilirubin total	4.5 mg/dl	2.8 mg/dl	0.8 mg/dl	Upto 1 mg/dl
Bilirubin (Direct)	2.7 mg/dl	1.8 mg/dl	0.1 mg/dl	Upto .2 mg/dl
Bilirubin (Indirect)	1.8 mg/dl	1.0 mg/dl	0.7 mg/dl	Upto .8 mg/dl
SGOT (AST)	248.2 U/L	92.7 U/L	32.7 U/L	Upto 37 U/L
SGPT (ALT)	396.6 U/L	107.8 U/L	52.8 U/L	Upto 40 U/L
Alk. Phosphatase	-	182.4 U/L	161.4 U/L	98-279 U/L
Prothrombin Time	22 seconds	-	10 seconds	11-16 seconds

Now patient is healthy till now.

#### DISCUSSION:

The pigment causing jaundice is called bilirubin. It is derived from hemoglobin that is released when erythrocytes are hemolyzed and therefore is constantly being formed and introduced into the blood as wornout or defective erythrocytes are destroyed by the body. 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. Patients with severe jaundice are at risk for encephalopathic changes that produce confusion, impaired mentation, and altered levels of consciousness. The potential for injury is increased and demands vigilance and safety measures to protect the patient.<sup>3</sup> Plasma protein show characteristic changes. Plasma albumin level is low but plasma globulins are raised due to an increased formation of antibodies.<sup>4</sup> *Avipattikar churna* is effective in hyperacidity. It prevents nausea, vomiting, and pacifies abdominal pain also<sup>5</sup> It has purgative property due to presence of *Nisotha (Operculina turpenthum)* as *kamla* is *pittapradhana vyadhi* and the *virechana* (Purgation) is the line of treatment in *pittapradhana vyadhi*. *Bayavidanga*<sup>6</sup> (*Embelia ribes*) in *Punarnava mandura* strengthen to the liver. *Arogyavardhini vati* is used in the treatment of

acute and chronic fever, obesity, liver disorders, and anorexia. It improves digestion power, clears waste products from the body. It acts as cardiac tonic. It is also used as co prescription with other medicines in different conditions. It acts as a catalyst<sup>7</sup>.

#### CONCLUSION:

On the basis of above description it can be concluded that our ancient *Acharyas* had complete knowledge about the complete pathophysiology, types of *kamala* and the line of treatment of *kamala* (Jaundice). 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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