

**ABSTRACT** Yakrita (liver) is a sthana of Bhootagni, many functions occur in liver. It is mool of rakta vaha srotas. Kamala is the one of main disease of Yakrita. Liver is the also a place of ranjaka pitta.[1] In Ayurveda Yakrita (liver) is a pitta sthana, influence of ama with pitta has to be understood. Pitta is sneham and it is produced as a mala of rakta. This has to be eliminated properly. In Kumbha Kamala the dosh-dushya sammurchna takes place in liver itself i.e., between pitta and rakta. In Ayurveda treatment principal is use of tikta rasa ruksha virechana and better to avoid ghritam, tailam, avasam and arishtam, here only tikta rasa dominant shaman chikitsa was done. In Kamala, there is loss of desire to do anything. Kama means different type of desires of the body and mind. Desires especially regarding the diet and physical activity of the body are minished. This paper discusses a diagnosed case of asymptomatic Hepatitis-B, patient seen in the OPD of Kayachikitsa department of Patanjali Ayurveda Hospital, Haridwar. This patient was effectively treated with Shamana Chikitsa by the combination of Arogyavardhini vati, Punarnavadi Mandoor, Totala kwath, Livamrit advance, Livogrit. These Ayurveda formulations normalized the raised viral load.

KEYWORDS : Kumbha Kamala, Hepatitis B, Arogyavardhini vati, Punarnavadi Mandoor, Livogrit

# INTRODUCTION

Acharya Charaka has described Kamala in the diseases caused due to the morbid doshas situated in Rakta Dhatu i.e., Rakta pradoshaja vikara. It is of three types (a)Koshtagat kamala (Hepatocellular) (b).Sakagata kamala or Ruddhapatha kamala(Obstructive). (c).Paratantra kamala (Haemolytic). So, It is clear from the above observations that Kamala is a vyadhi of Raktavaha srotasa (raktaja roga).[2] Also, in the samprapati (pathogenesis) of Kamala roga, Acharya Charaka has mentioned that when the anaemic patient (pandu rogi) indulges in the Paittika Ahara and Vihara, then morbid Pitta causes Dagdha of Rakta and Mamsa and ("Asriga Mamsama Dagdhva Rohgaye Kalpate") produces Kamala, specifically Kosthaashrita Kamala.[3]

The *pitta dosha* gets vitiated by two means, one is *achaya-poorvaka* (without accumulation phase), and another is *chaya-poorvaka* (with accumulation phase). In *achaya-poorvaka* phase, there is no accumulation of *doshas*, they directly get aggravated (*prakopa*) and manifests the symptoms of *kamala* which can probably be co-related with Acute Hepatitis B.

On the other hand, in *chaya- poorvaka* phase, firstly there is accumulation of *pitta dosha*, which lie dormant for a long period in *sanchaya-awastha*. When *pitta dosha* get favourable conditions, it progressively leads to *prakopa* and then *prasara-awasthas*. If still not controlled, then it progresses to *sthana-sanshraya awastha*, where there is appearance of *poorvarupas*,(prodromal features) after which there is *vyakta-awastha* with manifestation of various signs and symptoms of *kamala* and ultimately leads to *Bheda-awastha* (complication phase) of *Kamala* called as *Kumbh-kamala* which can probably be co-related with Chronic Hepatitis 'B'[4].

Hepatitis B infection is caused by Hepatitis B virus, a DNA virus which can cause acute or chronic infection hepatitis B. [5] HBV is a DNA virus classified in the virus family Hepadnaviridae. Humans are the only known natural host. HBV enters the liver via the bloodstream, and replication occurs only in liver tissue.[6] Hepatitis B virus (HBV) is a 42 nm enveloped partially double stranded DNA virus with a 3200 base pair genome classified as hepadavirus type 1. India falls in intermediate endemicity group with a prevalence of 2 to 4 % in general population. Prevalence is higher in high-risk population like professional blood donars, health care workers and patients with chronic renal failure on haemodialysis, thalassemia and haemophilia. There are 10 genotypes (GT)–A to J. GT-B is common in aisa.

Carriers of HBV are at increased risk of developing cirrhosis, hepatic decompensation, and hepatocellular carcinoma (HCC).[12] Although most carriers will not develop hepatic complications from chronic hepatitis B, 15% to 40% will develop serious sequelae during their lifetime.[7] Chronic viral hepatitis refers to hepatic inflammation or necrosis with hepatotropic viruses - hepatitis B.

Hepatitis B carrier is a term used to describe those who have hepatitis B surface antigen (HBsAg) in the blood for more than 6 months. Most of them have no symptoms and are unaware of their status as Hepatitis B carrier. Unfortunately, these otherwise healthy people can infect others without knowing it.[8]

There are certain such disorders which are discussed thoroughly in our classical texts, but their direct pathological or clinical relationship with hepatitis B has not been mentioned and cannot be purely co-related. Some conditions in particular, fairly reasonably similar with Hepatitis B in their clinical pictures are *Kamala, Koshtha-ashrita kamala, Shakha-ashrita kamala, Koshtha-shakha-ashrita kamala, Koshtha-ashrita kamala, Koshtha-ashrita kamala, Koshtha-ashrita kamala, the raktavaha srotasa whose moolas are Yakrita (liver) and pleeha (spleen). When pitta dosha gets vitiated, it leads to dushti of rakta and mamsa dhatus. In Chronic Hepatitis B (Kumbha-kamala)* 

Incubation period of HBV ranges from 30-180 days with a mean of 60-90 days. Progression to chronicity is as high as 95% in neonates compared to only to only 1-5% in adults. HBV is non-cytopathic and liver injury secondary to host – mediated immune response. Depening upon the interaction of host immunity and viral replication chronic hepatitis B can be divided into 5 phases, all of which are not necessarily seen in all patients and may not always develop sequentially. (a). Immune Tolerant Phase, (b).Imuune Clearance Phase, (c).Inactive Residual Phase, (d). HBsAg Negative Hepatitis, (e).HBsAg Negative Phase [9]

In modern science there is treatment are still limited, as no drugs is consistently able to eradicate hepatitis B infection completely. The goals of treatment are HBsAg sero-conversion, reduction in HBV-DNA and normalization of the LFTs. The indication for treatment is a high viral load in presence of active hepatitis, as demonstrated by elevated serum transaminases / histological evidence of inflammation fibrosis. [10]

## MATERIALS AND METHODS

A 26 year old male patient, came to OPD of Kayachikitsa deparment of

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### Patanjali Hospital and was asymptomatic.

Patient was accidently diagnosed with hepatitis B recently when he went for routine blood donation.

History of Present illness - Patient was asymptomatic. Patient has not taken any medication yet after being diagnosed. He came to our hospital for management of hepatitis B.

Past History - No H/O DM, Asthma, HTN and No H/O alcohol addication.

### Therapeutic intervention-

## 1. Internal Ayurvedic medication

| 1. Internary un veure incureation |                          |                  |          |  |  |
|-----------------------------------|--------------------------|------------------|----------|--|--|
| S.                                | DRUGS PRESCRIED          | DOSE             | ANUPANA  |  |  |
| No.                               |                          |                  |          |  |  |
| 1.                                | Totala Kwath             | 100ml BD, Empty  |          |  |  |
|                                   |                          | stomach          |          |  |  |
| 2.                                | Livogrit Tablet          | 2 tab BD, Before | Lukewarm |  |  |
|                                   |                          | meal             | water    |  |  |
| 3.                                | Puunarnavadi Mandoor     | 2 tab BD, Before | Lukewarm |  |  |
|                                   |                          | meal             | water    |  |  |
| 4.                                | Aarogyavardhini Vati     | 1 tab TDS, After | Lukewarm |  |  |
|                                   |                          | meal             | water    |  |  |
| 5.                                | Livamrit Adavance Tablet | 1 tab TDS, After | Lukewarm |  |  |
|                                   |                          | meal             | water    |  |  |

Duration of intervention-5 months

## **Probable Mode Of Action Of Medicines**

**Livogrit** is a tri-herbal *Ayurveda* formulation is prepared by mixing aqueous extracts derived from three plants, *Boerhavia diffusa L*. (Nyctaginaceae), *Phyllanthus niruri L*. (Euphorbiaceae), and *Solanum nigrum L*. (Solanaceae) in 2:1:1 ratio, respectively and have been earlier shown to effectively revert carbon tetrachloride (CCl<sub>4</sub>)-induced hepatocellular injuries in rats and HepG2 cells [11]

In our efforts to comprehensively address the hepatocurative potential of Livogrit, our current study has been conducted in zebrafish (*Danio rerio*) model of liver toxicity. The similarities towards mammals in terms of cellular mechanisms, enzyme activation, disease pathology, biochemistry during liver toxicity have established zebrafish as promising experimental model [12].

Although TAA administration is lethal to *in vivo* model organisms but researchers still get a wide window of time course to evaluate the mechanism of damage in hepatocytes. TAA-induced model organisms are generally exploited to address the recovery rate of a potential hepato-protective compound.

In our study, we performed a time and dose dependent screening to determine an effective dose of *Livogrit* and ascertain a time frame that shows the recovery from hepatotoxicity. Transaminase enzymes, metabolites, proteins and electrolytes are reliable biochemical parameters to assess the functionality in liver metabolism [13]

*Shyonak Oroxylum indicum* (Family: Bignoniaceae) or *Sonapatha* is a medium-sized deciduous tree distributed throughout Southeast Asia including India. Ayurveda, the oldest Indian traditional system of medicine recognizes Sonapatha as a multipurpose medicinal source. Therefore, Sonapatha has been used in many Ayurvedic formulations, especially its stem and root barks. The Sonapatha root and stem bark decoction of the root bark is used in fever, colic, constipation, indigestion.,The decoction of the leaves is used to treat flatulence, headaches, ulcers, dyspepsia etc., [14]

**Punarnava Mandoor** balances *kapha* and *pitta*. It is effective in treating liver diseases as it detoxifies liver by eliminating excess fluids and toxins. Most of the drugs in *Punarnava Mandoor* that is *Triphala*, *Trikatu*, *Chitraka*, *Vidanga* and *Pippalimula* are having appetizer, digestive and carminative properties. Hence it improves digestive power and ultimately absorption of nutrition and drug also. The components like *Trivita*, *Haritaki* and *Danti* act as purgative/laxative which help in relieving constipation. [15]

*Arogyawardhini Vati* is *rasaushadhi* mentioned in *Ayurveda* Formulary it is mainly indicated in treatment of jaundice, liver disorders, and various skin disorders. It consists of *Haritaki* (Terminalia chebula Retz.), *Bibhitaka* (Terminalia bellirica Roxb.),

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Amalaki (Emblica officinalis Gaertn.), Silajatu Suddha (Asphaltum), Guggulu Shuddha (Commiphora wightii), Eranda (Ricinus communis Linn.), Katuka (Picrorrhiza kurroa Royle ex Benth.), leaf juice of Nimba (Azadirachta indica A. Juss) and minerals like Shuddha Rasa (purified Published online in Shuddha Gandhaka (purified sulfur), Lauha Bhasma (iron compound in ash form), Abhraka Bhasma (mica in ash form), and Tamra Bhasma (copper compounds in ash form). Vati has deepan, pachan, stotoshodhan, yakrita pliha shothhar, tridoshashamak properties. Being deepan and pachan it acts on mandagni in the yakrit which leads to agnivardhan and formation of new cells in liver. Stotoshodhan relives the obstructed strotas occurred due to kapha and pitta. Yakrit pliha shothahar property relives the shotha in yakrit. Research conducted on it proves its choleretic, antiinflammatory and antiviral action. Hence this drug is widely used in the hepatic disorders.[16]

## 2. Dietary changes and Yoga/Pranayam

Pathya – Light, less oily, easily digestible, fruits, green vegetables, kulattha yusha, bittergourd, loki, methi, takra.

Apathya – Fried food, fatty, abhishyandi diet, alcohol, salt, soft drinks, meat,tea

Pranayam-Kapalbhati, Anulom-Vilom, Bhramri, Udgith, Ujjai.

**Asanas** – Utthanaasan, Pawanmuktasana, Shashankaasan, Mandalasana.

#### 3. Observation Of Investigation Report

| S.NO.    | HBV DNA               |  |
|----------|-----------------------|--|
| 14/7/23  | 8590 IU/ML (POSITIVE) |  |
| 23/11/23 | 00-00IU/ML (NEGATIVE) |  |

## **Reports Before Treatment**







#### **Reports After Treatment-**



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## DISCUSSION

Hepatitis B is mainly caused due to Hepatitis B virus and can be correlated with kamala. Kamala is a disease which occurred due to vitiation of the pitta dosha dominantly. Samprapti ghatak includes Pitta dosha, Rakta, Mansa dushya, mahastrotas as a adhishthan. In Avurveda various formulations are described for the management of kamala. In this case study formulations like, Arogyavardhini vati, livogrit, Totala kwath and Punarnava mandoor, Livamrit advance are used for the management of kamala. All these herbomineral formulations contain the drugs having tikta rasa, dipana, pachana, rechan, pittakaphashamaka, and yakruttotejaka and rasayana properties. Probable mode of action of medicines

## RESULT -

Formulations used in this case study helps in improving reduction in values of liver function test with improvement in viral load of patient. Hence it can be concluded that Hepatitis B can be successfully treated with formulations described in Ayurveda. They mainly contain drugs which act on liver and helps in improving the functioning of liver. This single case study hence proves the efficacy of these drugs.

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