Ayurvedic management of acute viral hepatitis: A case report

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ABSTRACT

Acute viral hepatitis (AVH) has resemblance with Kamala mentioned in Ayurveda. Kamala is a disease of the *Raktavaha srotas* (a system which includes liver, spleen, blood vessels, and reticuloendothelial tissue) and dominant of pitta dosha. *Nitya virechana* (regular use of mild laxative) and *Pitta samana* is the principles to treat the condition in Ayurveda. Here, we report the case of a 20-year-old male diagnosed with AVH treated in accordance to the Ayurvedic principles reported, that is, *Mridu virechana* (regular use of mild laxatives), and *Pitta samana.* The total duration of the treatment including follow-up was 60 days. A complete remission of symptoms with a substantial reduction in bilirubin and hepatic enzymes observed following Ayurvedic treatment. Hence, presenting this case is an evidence to demonstrate the effectiveness of Ayurvedic treatment in AVH.

Key words: *Acute viral hepatitis, Avipattikar churna, Hyperbilirubinemia, Jaundice, Kamala*

Viral hepatitis is the most common form of hepatitis caused by A, B, C, D, and E virus. Viral hepatitis poses a major public health problem in the world with a death rate of 1.35 million deaths each year [1]. Although there is a paucity in the data of national disease burden, viral hepatitis shares a total of 3% among the communicable diseases burden to India [1]. Hepatitis A and E cause acute viral hepatitis (AVH), whereas B and C can cause chronic viral hepatitis, in addition to acute infection [2]. Hepatitis A and E are transmitted through feco-oral route, whereas the B and C are transmitted through parenteral route. Hepatitis B is preventable with the vaccine; however, there is no vaccine for hepatitis C [3]. The treatment of hepatitis A virus and hepatitis E virus is usually symptomatic and supportive, no specific antiviral treatment required for them [4]. In recent times, silymarin, interferon, and nucleoside analogues are practiced in the treatment of AVH [5].

In Ayurveda, hepatitis or jaundice is acknowledged as *Kamala roga*, caused due to impairment of pitta dosha and rakta dhatu. The condition occurs due to *Kostha gata Pitta, Pitta Rakta dusti*, and *Yakrit asrita Pitta dusti*. In this condition, aggravated pitta dosha further vitiates rakta and mamsa and produces Kamala. It has two variations, namely *Kosthasrita Kamala/Bahu pitta Kamala* and *Sakhasrita Kamala*. The chronic form of Kamala leads to Kumbha Kamala, halimaka, panaki roga. The diagnosis of Kamala consisting of an evaluation of the types of Kamala through examination of skin, mucous membrane, and stool color. Assessment of *Agni, Bala, Dosha vriddhi, Dhatus* involvement is required to decide the therapeutic measures. In Kosthasrita Kamala, *Tikta rasa* (drugs with bitter taste) *prayoga* and *virechana* are recommended. However, in Sakhasrita Kamala, *Kaphahara* treatment is required at initial stages to bring the pitta dosha from Sakha (skin and periphery) to Kostha (alimentary canal) [6]. Few studies have also proved the efficacy of Ayurvedic medicines in hepatobiliary disorders [7,8].

A detail documentation of the efficacy and safety of the Ayurvedic drug on Kamala has not yet reported distinctly. Hence, in the present communication, we are presenting a case of AVH (Kamala), intervened successfully with two Ayurvedic medicines on the basis of Ayurvedic principles.

CASE REPORT

A 20-year-old male of the suburban area of Bhubaneswar presented with loss of appetite, flatulence, low-grade fever, malaise, and yellowish discoloration of urine of the 3 days duration. He had a normal bowel habit, without any history of abdominal pain, dysuria, or vomiting. There was neither any history of any medication taken by him in the past 3 months nor any history of an epidemic of jaundice reported from the habitation of the patient. The patient was a college student and did not reveal any habit of smoking, alcohol consumption, blood transfusion, drug abuse, and hemodialysis. None of his family has any significant medical illness.

On examination, the patient had a general weakness and fatigue. He had a 38°C temperature with stable vital signs. Mild icteric tint detected on the sclera and undersurface of the tongue. However, pallor, edema, abdominal swelling, palmar erythema, pruritus, and weight loss were not observed. On abdominal

examination, moderate tender on epigastrium and mild tender hepatomegaly without any splenomegaly observed. No other any abnormality discovered through systemic examination. From the Ayurvedic perspective, the patient had nausea, yellowish discoloration of eyes, and dark urine with a normal stool color. *Agnimadya* (anorexia)*, pitta vriddhi, Madhyama Bala, Pitta Kapha Prakriti* also observed in the case.

The baseline investigation report showed that liver enzymes were elevated: Serum glutamic-oxaloacetic transaminase [SGOT] - 314.7 IU/L and serum glutamic-pyruvic transaminase [SGPT] - 323.4 IU/L; hyperbilirubinemia (total serum bilirubin - 4.83 mg/dl and direct bilirubin - 2.40 mg/ dl) with normal alkaline phosphatase 28.8 IU/L. The other hematological parameters were normal (total white blood cells - 8100/ µL, hemoglobin - 14 g/dl, differential leukocyte count neutrophil - 41%, lymphocyte - 52%, eosinophil - 7%, and platelet count - 210,000/µL). The viral marker for hepatitis B surface antigen (HBsAg) was found negative and no hepatomegaly detected in the ultrasound of the whole abdomen.

From the presenting features (constitutional symptoms) and investigation (hyperbilirubinemia and elevated hepatic enzymes), the case was diagnosed as *Bahu pitta Kamala* or *Kosthasrita Kamala* (hepatocellular jaundice/AVH). The patient was assessed clinically and also through laboratory investigation to ensure the clinical status and recovery. The following drugs were prescribed to the patient: Trikatu, triphala, trivrit, clove, mustaka, and M-liv tablet. The pharmacological basis of the drugs was given in Table 1 9-14]. The patient was strictly adhered to the drugs and regimen advised to him. No any adverse effect or any untoward event reported by him during the course of therapy. The patient recovered gradually from fever, malaise, and yellowish discolored of urine within 1 week of the treatment. However, loss of appetite, flatulence, and general weakness continued until the end of the 3rd week. The icteric tint started disappearing at the end of 2nd week of the treatment and disappeared after 3rd week. Liver size became normal at the end of the 2nd week treatment. A reduction in liver enzyme levels seen from 2nd week onward (SGOT - 218.5 IU/L and SGPT - 231.8 IU/L) and significant drop noticed (SGOT - 29.45 IU/L and SGPT - 24.75 IU/L) after 3 weeks of the treatment. Similarly, decrease in bilirubin level seen earlier, that is, from the 2nd week of therapy (total serum bilirubin - 2.91 mg/dl) and it became obsoletely normal (total serum bilirubin - 0.94 mg/dl) after 6 weeks of therapy. The other hematological parameters remain unchanged throughout the treatment period. No adverse effect of the prescribed drugs also observed in the case.

Table 1: Ayurvedic drugs prescribed to the patient and their pharmacological basis

Drugs	Manufacturer	Dosage	Ingredients	Indications as per Ayurvedic classics	Rationale of use in the case	Pharmacological basis of use
Avipattikara Churna Govinda Das. Bhaishajyaratnavali. 1st ed.. India: Chaukhambha Sanskrit Bhawan series; 2006	IMPCL, Moham, Uttarakhand	5 g twice daily after food	Trivrit, lavanga, trikatu, triphala, musta, vidanga, patra and ela	Amlapitta (hyperacidity/ functional dyspepsia), Malamutravivandha (constipation and obstruction in the flow of urine), Agnimandya (loss of appetite), prameha (diabetes), durnama (chronic diseases)	*Pittakaphasamana, Anulomana, Deepana, Virechana, Rasa prasadana, Raktaprasadana, Yakrita prasadana, Sukhaviracha*ka due to the presence of trivrit vide its *sheeta* and *manda gun*	*Trikatu* is hepatoprotective [9], *Triphala* is hepatoprotective[10] Trivrit is hepatoprotective[11] clove is antimicrobial, anti-inflammatory and hepatoprotective[12] Mustaka is hepatoprotective, antioxidant [13]
M-Liv tablet	IMPCL, Moham, Uttarakhand	2 tablets thrice daily befor food	*Eclipta alba* (Bhringaraj), *Phyllanthus niruri* (Tamalaki), *Picrorhiza kurroa* (Katuka), *Andrographis paniculata* (Kalmegh), *Mandoor bhasma* (Calcined iron slag), etc.	Agnimandya (loss of appetite), Yakritakarmakshaya (liver dysfunction), Yakritsotha (hepatitis), Kamala (jaundice), Madya sevanajanya yakritroga (alcoholic liver diseases), vasasanchayajanyaroga (fatty liver)	Yakrit prasadana	Most of the drugs exhibit hepatoprotective activity [14]

DISCUSSION

Kosthasrita Kamala, a disease caused due to vitiation of pitta dosha. Pitta dosha in normalcy is the responsible factor for proper digestion, metabolism, body complexion, maintenance of body temperature, and proper pigmentation of blood, urine and stool. In Kamala, the *Yakritasrita Pitta* is vitiated and its *vimargagamana* (regurgitation into blood) occurs. When intensified pitta gets

localized in Kostha, called *Kosthasrita Kamala* (hepatocellular jaundice), however, it affects Sakha (deeper Dhatus or tissues), when amalgamated with Kapha dosha (*Sakhasrita Kamala/* obstructive jaundice). Stool becomes abnormal in color, when *Kaphanubandha* is there and pitta present in *Sakha*. However, stool appears normal in color, when Pitta restricted to *Kostha* only. The normal yellow color of stool disappears and it looks white (*Tilapistanibhabarchah/Swetabarchah*) due to *vimargagamana* of pitta dosha. Aggravated pitta causes derangement in digestion and its excess accumulation causes regurgitation in to the blood and produces yellowish discoloration of skin, urine, nails, *Ajeerna* (dyspepsia), *Daurbalya* (weakness), and anorexia (*Aruchi*) [15].

Kamala is a disease of *Pitta* and *Raktadusti* origin. As per Ayurveda*, Yakrita* is the *moolasthana* (origin) of rakta, and pitta is the *mala* of *Rakta.* Due to *Ashrya-Ashrayisambhanda* (interdependency), elimination of pitta through *virechana* is essential to treat *Rakta Dusti,* and *Virechana* with *mridu and ruksha dravya* is beneficial in *Bahupitta Kamala*. Reduction of pitta through regular *virechana* can reduce the intensity of yellowish discoloration and can bring rapid recovery. The *Virechana karma* told in Ayurvedic classics for the treatment of Kamala may also be helpful in the prevention of hepatic encephalopathy and early cessation of carrier stage of virus. Sakhasrita Kamala is initially treated with *Katu rasa*, *Katu vipaka,* and *Ushna veerya* drugs to remove the kapha avarodha. In *Shakhagata Kamala* (deep seated, in tissues), *vriddha pitta* begins to move toward *Koshtha* (gastrointestinal tract). Once the *Pitta* reaches the *Koshtha*, it recolors the stool. At this stage, the patient is treated with *Tikta*, *Madhura rasa*, *Madhura vipaka*, *Sheeta veerya,* and *Anulomana* drugs which will help in pacifying the *vriddha pitta* and mitigate the *Kamala*. Improvement in symptoms, disappearing of icterus, drop in hepatic enzymes level to normal range, and reduction in the bilirubin level at different intervals of the treatment indicate the resolving state of the hepatic dysfunction.

The description of AVH or hepatocellular jaundice (viral) is similar to the *Ayurvedic* description of *Kamala*. Viral hepatitis A and E are usually self-limited and recover after 4 weeks of onset. However, in very few cases, fulminating hepatic necrosis or hepatic encephalopathy occurs. Viral hepatitis always associated with hyperbilirubinemia and elevated liver enzymes (SGOT and SGPT). Jaundice is clinically observed when bilirubin in the blood exceeds 2.3 mg/dl [16]. In *Bahu Pitta Kamala,* strict observation of diet and lifestyle is important because *Kaphavardhakaahara* can cause *agnimandya* and produces samakapha, which can cause sanga (obstruction) of srotas and vimarga gamana of Raktasrita Pitta, that can leads to Sakhasrita Kamala. Apart from *Samsodhanachikitsa, Samsamana* of vitiated pitta dosha is essential through the drugs with Tikta and Madhura rasa dominance [17].

In the present case, since the patient was negative for HBsAg, it was diagnosed as AVH (either A or E - virus origin). Other medical conditions such as gastritis, malaria, urinary tract infection, and biliary colic came in ambit of diagnosis, which differentiated from the condition through normal urinalysis, complete blood count and negative (malaria parasite) antigen test. Ultrasound sonography test abdomen and pelvis was done to rule out obstructive pathology. The stool color of the patient was normal, and he was treated in the line of Bahupitta*/Kosthasrita Kamala*. He was advised for bed rest and prescribed with two Ayurvedic medicines (Avipattikara Churna and M-Liv tablet) from the hospital. All the medicines prescribed to him are endowed with *Piitasamaka Ras*a (*Madhura, Tikta, and Kashaya), Madhura Vipaka, Sheeta Veerya, Anulomana,* and *mriduvirechaka* properties. He further directed to avoid *Pittavardhakaahara* and *Vihara* such as stale/sour salty, spicy, oily food, and drinks and also instructed to maintain proper hygiene and sanitation at home. Herein, the Avipattikara churna possesses the properties of Pitta elimination through *sukha virechana* (mild laxative) and the ingredients of M-liv fulfilled the *Pitta samana* as well as hepatoprotection. There are few cases of viral hepatitis treated with classical and proprietary Ayurvedic reported earlier by some scholars. In this study, we found a significant drop in bilirubin and enzyme level observed only after 2nd and 3rd week of commencement of treatment. Similar observations have also reported earlier with multiple drug regimens in hepatitis patient [18-20].

CONCLUSION

AVH is a major public health problem habitually with epidemic outbreaks. There is an ample scope to revalidate the Ayurveda and traditional medicine for the management of this disease. This case report endorses a step toward the revalidation of Ayurvedic medicine in AVH Kamala. Further, a well-structured clinical trial is recommended with an adequate sample size to establish the efficacy and safety.

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