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Case Report

Ayurveda Management of Acute Viral Hepatitis A: A case report

[1*]Vasant Patil, [2]Sudha.L, [3]Anuja Daga, [4]Ambalal Basava

ABSTRACT:

Background: Viral hepatitis is a liver inflammation caused by a virus, with the most common types being A, B, C, D, and E. Symptoms can range from mild to severe and may include fatigue, nausea, abdominal pain and jaundice (yellowing of the skin and eyes). It is a systemic infection affecting the liver, causing impaired metabolism. Acute Hepatitis A is transmitted almost exclusively by the fecal-oral route. Large outbreaks as well as sporadic cases have been traced to contaminated food, water and milk. **Clinical Findings:** A 26-year female patient presented with the complaints of yellowish discoloration of urine, stool, eyes, and oral mucosa, along with vomiting, fever for 2 days, which is gradually started with anorexia for 8 days. **Interventions:** *Vasaguluchyadi Kashaya, Patola Katu Rohinyadi Kashaya, Tab. Nirocil, Syp. Amlycure* DS, *Syp. Kalamegha* DS, *Avipattikara Churna* and Anuloma DS *were* administered orally. Diet and lifestyle restrictions are advised. **Outcome:** After 30 days of treatment, marked changes in the subjective and objective parameters were found. Anorexia, abdominal pain, vomiting, giddiness, yellowish eye-oral cavity-urine, and Mala, headache, constipation, fever, weakness were present with moderate to severe intensity. Following treatment, all these symptoms were completely resolved. Biochemical parameters: Total bilirubin 3.1↑ → 1.2↓, SGOT 381.5↑ → 34 ↓, SGPT 941.7 ↑ → 32↓, Alkaline phosphatase 190↑→ 90↓. **Conclusion:** Rational use of oral medications *with a* restricted diet in acute viral hepatitis A shown excellent results within a span of 30 days. This case highlights that the Ayurvedic line of treatment -*Pitta Rechana* (Cholagogue), *Pitta Shamana* (*Pitta* pacification), and *Yakritshodhana* (liver detox) helps in the effective management of acute viral hepatitis A.

KEYWORDS: Acute Viral Hepatitis A, Case Report, *Kamala, Koshta-Shakhashrita Kamala, Swatantra Kamala, Yakrit Shotha.*

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Corresponding Author Email:

ayurvasant@gmail.com

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1. INTRODUCTION

Acute Hepatitis A is transmitted almost exclusively by the faecal- oral route. Large outbreak as well as sporadic cases have been traced to contaminated food, water and milk. The virus is present in liver cells, Bile, stool and blood during the incubation period and in pre-icteric phase but viral shedding diminishes after the onset of jaundice. The mechanism by which HAV infection causes hepatitis A causes is poorly understood. An immunologic basis is suspected but the evidence in support is indirect in the form of immunologic markers such as IgM and IgG anti HAV. [1]

Acute Hepatitis A has a high incidence and is still a serious global public health concern. In 2017, there were an estimated 170 million new cases of AHA. In general, AHA does not develop to chronic hepatitis and is rarely fatal. Although chronic liver diseases do not raise the risk of HAV infection, concurrent underlying chronic liver disease (*e.g.*, HBV infection, HCV infection, cirrhosis, and fatty liver disease) has been associated with an increased risk of fulminant hepatitis when HAV infection occurs. Vaccination has limited AHA to a disease of adults instead of an early childhood disease in many regions of the world. [2] In children <6 years of age, most infections (70%) are asymptomatic and if illness does occur, it is usually anicteric. Among older children and adults, infection is usually symptomatic with jaundice occurring in> 70% of patients. After an average incubation period of 28 days (range, 15 to 50 days), most HAV-infected persons developed nonspecific constitutional signs and symptoms followed by gastrointestinal symptoms. Typically, these include fever, malaise, anorexia, nausea, abdominal discomfort, dark urine and jaundice all of which usually last <2 months. There is no evidence of chronic liver disease or persistent infection following infection. [3] Hence, early diagnosis helps to prevent post-icterus phase which ultimately progress on to chronic hepatitis.

Kamala is a condition leads to yellowish discoloration of the skin, eye, urine and stool associated with *Avipaka* (indigestion) *Dourbalya* (weakness) *Sada* (lassitude) *Aruchi* (Anorexia)and it is included under *Rakta Pradoshaja Vikara*.

Kamala is said to be of two types, *Shakhashrita Kamala* and *Koshtashrita Kamala*. [4] The clinical difference between the two types of *Kamala* is the color of feces. The fecal matter which is white in color and resembles the paste of sesame [5] is considered to be *Shakhashrita Kamala* which is due to *Kapha Avarana* and *Pitta* will not reach to the *Koshta* whereas *Kosthashrita Kamala* [6] is *Bahu Pitta Avastha* (excessive aggravated *Pitta*), *Mala Ranjana* (yellowish color of feces) occurs exceedingly leading to yellow-colored stool associated with other classical symptoms.

Kamala is also categorised into *Swatantra* (independent) and *Paratantra Vyadhi* (dependent) the one in which *Pandu Rogi* consumes excessive *Pittavardhaka Ahara* leading to *Kamala* is considered as *Paratantra* whereas *Kamala* which is manifested irrespective of *Pandu Roga* is considered as *Swatantra Kamala*. [7]

As per new NCISM BAMS syllabus it is correlated with *Yakritshotha*, although there is no direct classical

reference but symptoms of *Koshtashakashrita Kamala* can be considered under *Yakritshotha*.

No specific antiviral treatment for acute hepatitis A is available. Limited in vitro data suggest that sofosbuvir might have an anti-HAV effect; however, most studies of antivirals (such as ribavirin, amantadine or sofosbuvir) are from cell lines infected with specific HAV strains; therefore, further investigations are needed. [8] Considering the limitations in treatment of acute HAV in contemporary science an approach of *Koshtashakhashrita Chikitsa* was made. Hence, it is treatment challenging case study.

There are very few case reports on acute viral hepatitis management by Ayurvedic intervention; among these the present case having increased SGOT, SGPT along with Alkaline Phosphatase reduce to normal within short span of month indicating the uniqueness of this case.

2. CASE REPORT: A female patient aged 26 came to our OPD on 8/8/25 with complaints of sudden onset of yellowish discoloration of urine, stool, eye and oral mucosa along with vomiting, fever for 2 days, which initially started with the anorexia for 8 days. So, she consulted our clinic with no prior interventions.

Family/social/medical history- nil.

USG abdomen showed A-calculus cystitis.

Table 1: Clinical findings

Clinical findings	**Grade**
Anorexia	++
Abdominal pain	++
Vomiting	++
Giddiness	+++
Yellowish discoloration of eye, oral cavity, urine and stool	++
Headache	++
Constipation	++
Fever	++
Weakness	+++

Diagnosis: Clinical symptoms are inadequate to distinguish viral hepatitis types, therefore virus-specific serological testing is advised in individuals with presumed viral hepatitis. [9]

Table No 2. Differential Diagnosis

Parameter / Feature	**Hepatitis A**	**Hepatitis B (Acute)**	**Hepatitis C (Acute)**	**Hepatitis E**	**Drug-Induced Hepatitis (DILI)**
Onset pattern	Acute onset, prodromal symptoms followed by jaundice (seen in this case)	Usually requires exposure risk; may be insidious	Rarely acute severe; mostly chronic/insidious	Acute, resembles HAV	Depends on drug exposure; not seen here
Risk factors in case	Street/fast food consumption → fecal–oral exposure	No blood/sexual exposure history → absent	No risk factors (IV drugs, transfusion) → absent	Water/food contamination possible, but less common in adults without outbreak	No drug intake history → excludes
ALT / SGPT	Typically >1000 IU/L in	Can be >1000 in acute	Rarely this high in	Can be high but often	May be >1000 in

(941.7 IU/L)	acute HAV → MATCH	HBV → possible but risk factors absent → excluded	acute HCV → unlikely	less dramatic → possible but less likely	acetaminophen toxicity → unlikely (no drug use)
AST / SGOT (381.5 IU/L)	AST moderately high, ALT >> AST → typical HAV pattern	Elevated but requires exposure → excluded	Mild–moderate rise → does NOT match → excluded	Moderate rise → possible	Can be high but requires drug history → excluded
Bilirubin Total (3.1 mg/dL)	Mixed hyperbilirubinemia typical of HAV	Seen in HBV, but risk factors absent	Usually mild in acute HCV → excluded	Similar to HAV → possible	Severe mixed bilirubin common in cholestatic DILI → excluded (no drug exposure, ALP pattern differs)
ALP (190 IU/L)	Mild–moderate elevation → matches HAV	Mild elevation	Mild elevation	Mild–moderate elevation	Marked ALP elevation expected in cholestatic DILI → excluded
Albumin (3.7 g/dL)	Normal or near-normal → fits HAV (acute, not chronic)	May fall in acute severe HBV or chronic disease → not matching	Often low in chronic HCV → excluded	Usually normal; matches	Normal in some DILI but other features don't match
Stool color (yellow, NOT clay-coloured)	Supports HAV	Can be yellow	HCV normal	HEV stool often yellow	DILI, especially cholestatic type → clay-coloured stools, not seen → excluded
Age (26 years)	HAV increasingly affects adults → fits	HBV acute more common in unvaccinated or high-risk adults → exposure absent	HCV acute rare	HEV more common in pregnancy or outbreaks	No relevance

Final Diagnosis: Acute Viral Hepatitis A

3. INTERVENTION AND TIMELINE:

Table 3: List of *Shamanoushadhi*, Dose and *Anupana* with *Pathyapathya*.

Shamanoushadhi	Dose and *Sevana Kala*	*Anupana*	BT (08-08-2025)	AT1(18-08-2025)	AT2 (31-08-2025-7/09/25)
Vasaguluchyadi Kashaya	10ml BD, BF	*Ushna Jala*	✓	✓	✓
Patola Katu Rohinyadi Kashaya	10 TID, AF	*Ushna Jala*	✓	✓	✓
Tab. Nirocil	1 TID, BF	*Ushna Jala*	✓	-	-
Syp. Amlycure DS	10ml TID, BF	*Ushna Jala*	✓	✓	✓
Syp. Kalamegha DS	15ml BD, AF	*Ushna Jala*	✓	✓	✓
Avipattikara Churna	1tsf, BD, AF	*Ushna Jala*	-	✓	✓
Anuloma DS	1 HS, AF	*Ushna Jala*	✓	-	-
Do's: Sugarcane juice, Coconut water, Dry grapes, Pomegranate. *Don'ts*: Fried, hot, sour, excess of salt, wheat, Maida, spicy, green chillies.					

Follow-up and Outcome:

Table 4: Showing Clinical Features before and after Treatment.

Feature	08-08-2025	18-08-2025	31-08-2025	7-09-2025
Anorexia	++	+	-	-
Abdominal pain	++	+	+	
Vomiting	++	-	-	-
Giddiness	+++	-	-	-
Yellowish discoloration of eye, oral cavity, urine and stool	++	+	-	-
Headache	++	-	-	-
Constipation	++	-	-	-
Fever	++	-	-	-
Weakness	+++	+++	+	-

Table 5: LFT Parameters before and after treatment.

Parameter	BT (08-08-2025)	AT1 (18-08-2025)	AT2 (31-08-2025)
Bilirubin Total (mg/dl)	↑3.1	3.1	↓1.2
Bilirubin Direct (mg/dl)	↑1.7	↓1.4	↓0.6
Bilirubin Indirect (mg/dl)	↑1.4	1.6	↓0.6
SGOT (IU/Lt.)	↑381.5	↓80.0	34.0
SGPT (IU/Lt.)	↑941.7	↓92.0	32.0
Alkaline Phosphatase (IU/Lt.)	↑190.0	↓160.0	90.0
Total Protein (Gms %)	7.2	↓6.0	6.0
Albumin (Gms %)	3.7	4.0	4.0
Globulin (Gms %)	3.5	↓2.8	↓2.8

Adherence and tolerance:

Strict adherence to the medications and diet was employed by regular supervision with family member and reconfirmed during follow-up. Tolerance to the treatment was recorded by regular follow-up enquiry and patient tolerated well to all medications without any discomfort.

4. DISCUSSION:

In this case patient present with acute symptoms of Hepatitis A. Here *Yakṛit* (liver), *Pleeha* (spleen) and the *Koshta* are being involved leading to aggravation of *Kapha* and *Pitta* which obstruct the normal passage of the *Pitta* by *Kapha* leads to *Peeta Mutra, Purisha, Netra* and *Mukha* based on these *Lakshanas* the case was diagnosed as *Kosthashakashrita Kamala* as explained by Maharshi Charaka and also this case represents *Swatantra Vyadhi* as the patient was not diagnosed or having any *Lakshana* of *Pandu*. According to modern science, in pre-icteric phase patient was having anorexia, nausea, fatigue, nausea, vomiting with fever which shows elevation of transaminase. In icteric phase dark colored urine was due to bilirubinuria and abdominal discomfort was due to enlarged gallbladder. [10]

Patolakaturohinyadi Kashaya [11] contains *Patola (Trichosanthes dioica)*, *Katuki (Picrorhiza kurroa)*, *Chandana (Santalum album)*, *Guduchi (Tinospora Cardifolia)*, *Patha (Cissampelos pareira)*; it is having *Pitta-Kapha shamaka*, *Rechana, Yakritshodhana* (liver detox) *and* hepatoprotective, and antiviral.

Vasaguluchyadi Kashaya [12] contains *Vasa (Adhatoda Vasica), Guduchi, Triphala, Katuki, Bhunimba (Andrographis paniculata), Nimba;* it is having *Pittakapha Nashaka, does Pittashodhana,*

Yakritshodhana (liver detox), hepatoprotective, and antiviral properties.

Tab. Nirocil contain *Bhumyamlaki* (Phyllanthus Niruri) helps in normalize the liver function. WHV and HBV infections since replication of both these viruses takes place in the liver. The preliminary results indicate that there are one or more active materials in P. niruri that inhibit the replication of WHV in vivo and decrease the pathological effects of WHV on woodchuck liver. [13]

Syp. Amlycure DS contains mainly *Bhringaraja (Eclipta Prostrata)*, *Bhumyamlaki* (Phyllanthus Niruri), *Haritaki (Terminalia Chebula)*, *Guduchi*, *Kalamegha (Andrographis paniculata)*, *Katuki, Nimba*, *Sharapunkha* (*Tephrosia purpurea*); it is liver detoxifying and improves the hepatic function.

The hepatoprotective role of ADS-RF & ADS-ND forms of Amlycure DS might be due to available anti-oxidants tannins and flavonoids and also due to antimicrobial and anti-inflammatory actions. [14]

Avipattikara Churna contains *Triphala, Trivrut (Operculina turpethum), Vidanga (Embelia ribes), Musta (Cyperus rotundus)* and it does both *Shodhana* and *Shamana* of *Pitta dosha,* improves the appetite, digestion and liver function. [15]

All the palliative medicines administrated are having *Pitta Rechana* (Cholagogue), *Pitta shamana* (*Pitta* pacification) and *Yakritshodhana* (liver detox) properties. As per modern pharmacology, the oral medicines *Patolakaturohinyadi Kashaya, Vasaguluchyadi Kashaya,* Tab. Nirocil are having anti-viral, anti-pyretic, anti-inflammatory, cholagogue, and liver cleansing actions.

Hepatitis B patient having chronicity of 2 months was treated with Ayurveda drugs and *Virechana Karma* (therapeutic purgation). After the treatment for 13 months, the disease was cured. Improvement was observed based on hepatitis B virus deoxyribonucleic acid (DNA) polymerase chain reaction (PCR) (hepatitis B DNA PCR) (decreased from 3705.71 IU/ml to 50.0 IU/ml) and the values of AST (decreased from 140 IU/ml to 19.0 IU/ml) and ALT (decreased from 173 IU/ml to 28 IU/ml). The patient was stable and asymptomatic during the follow-up period of 4 months. [16]

The intervention of oral Ayurvedic medication for a period of 88 days, improvement was observed both symptomatically and diagnostically, as evidenced by the conversion of HBsAg to negative and marked reductions in biochemical parameters: serum bilirubin total decreased from 7.46 mg/dL to 0.88 mg/dL, SGPT decreased from 1082 U/L to 23.9 U/L, and SGOT decreased from 1240 U/L to 21.7 U/L. [17]

Limitations: We have not done IgM anti-HAV test, Anti-HCV antibody, and Anti-HEV IgM.

The rational selection and administration of Ayurvedic formulations along with strict diet will give definitive results in a short period denoting the potential in acute viral hepatitis.

5. CONCLUSION:

An acute case of *Koshta-Shakhashrita Kamala* (Acute Hepatitis A) was administered with oral medicines along with the diet and lifestyle advice for 30 days, resulting in marked improvement in both clinical and laboratory parameters. Anorexia, abdominal pain, vomiting, giddiness, yellowish eye-oral cavity- urine and Mala,

headache, constipation, fever, weakness were present with moderate to severe intensity. Following treatment, all these symptoms were completely resolved. Biochemical parameters- Total bilirubin 3.1↑→1.2↓, SGOT 381.5↑→34↓, SGPT 941.7↑→32↓, Alkaline phosphatase 190↑→ 90↓ which shows marked reduction after treatment. No adverse reactions were noted during the treatment. This case elucidates the importance of early diagnosis and skilled management of the disease, which significantly influence the outcome.

Abbreviations:

HAV- Hepatitis A Virus.

AHA- Acute Hepatitis A.

LFT- Liver Function test.

ALT- Alkaline Aminotransferase.

Declaration of Patient Consent – The authors confirm that they have acquired a patient consent form, in which the patient or caregiver has granted permission for the publication of the case, including accompanying images and other clinical details, in the journal. The patient or caregiver acknowledges that their name and initials will not be disclosed, and sincere attempts will be undertaken to safeguard their identity. However, complete anonymity cannot be assured.

Patient perspective - Patient was suffering from jaundice along with weakness and GIT upset. After starting the treatment, patient condition was improved steadily. After Complete treatment feeling normal, active and healthy, she was very happy with the recovery

Authors Details:

[1*]Consultant Ayurveda Physician Atreya Ayurveda clinic and Panchakarma center, Hubli

[2, 3, 4]PG Scholar, Dept of Panchakarma, Ayurveda Mahavidyalaya, Hubli

Authors Contribution:

Conceptualization and clinical management: VP, SL, AD, AB

Data collection and literature search: SL, AD, AB

Writing – original draft: SL, AD

Reviewing & editing: VP, SL, AD

Approval of final manuscript: All authors

Declaration of Generative AI

The authors declare this manuscript was written without the use of generative artificial intelligence tools. All the content, including text generation, data analysis and references was developed and reviewed by the author without assistance from AI technologies.

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