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An Integrative Ayurvedic and Conventional Approach to Clinically Interpreted *Ruddhapatha Kamala*: A Case Report**Vishwajeet Govindrao Mehetre*¹, Savita Arjun Wale²**Professor¹, Kayachikitsa Department, Assistant Professor², Rognidan Department

Vasantdada Patil Ayurvedic Medical College, Sangli, Maharashtra, India

Corresponding Author: vgmehetre@gmail.com*ABSTRACT**

Background: *Kamala* is an Ayurvedic clinical entity characterized by yellowish discoloration of the eyes, urine, and body tissues. *Ruddhapatha Kamala* is described in the context of obstruction of the normal movement of aggravated *Pitta* by *Kapha* and provides a conceptual Ayurvedic correlation for jaundice associated with impaired biliary flow. This case illustrates the application of this Ayurvedic diagnostic assessment alongside contemporary clinical and biochemical assessment.

Case Presentation: A 20-year-old male presented with yellowish discoloration of the eyes and urine, decreased appetite, and leg cramps, with no significant past medical history. Examination revealed icterus and clay-coloured stool. Ultrasonography demonstrated mild hepatosplenomegaly, with

a liver span of 15.1 cm and splenic length of 12.8 cm, along with mild pseudothickening of the gallbladder wall. Ultrasonography did not document definite structural biliary obstruction. Initial investigations revealed total serum bilirubin of 10.19 mg/dL (direct 6.48 mg/dL; indirect 3.71 mg/dL), SGOT 311 IU/L, and SGPT 330 IU/L. Urinary bile salts and bile pigments were positive, while HBsAg and HIV were negative. Based on the clinical presentation, Ayurvedic assessment, biochemical findings, and imaging, the condition was interpreted as *Ruddhapatha Kamala*. The patient received conventional supportive treatment concurrently with Ayurvedic medicines, including *Arogyavardhini Vati*, *Kamdudha Vati*, *Triphala Churna*, *Trikatu Churna*, *Sharpunkha-Amalaki Churna*, and *Bhumyamalaki Swarasa*. By Day 5, total bilirubin decreased to 5.30 mg/dL and SGOT

to 67 IU/L, although SGPT was 337 IU/L; urinary bile salts, bile pigments, and urobilinogen were absent. By Day 8, total bilirubin further decreased to 2.94 mg/dL, with marked symptomatic improvement and minimal residual weakness.

Conclusion: This case demonstrates substantial clinical and biochemical improvement during integrative management of a patient interpreted as having *Ruddhapatha Kamala*. The case highlights the potential utility of integrating Ayurvedic pathophysiological assessment with objective biochemical and imaging parameters.

Keywords: *Ruddhapatha Kamala*; *Kamala*; Jaundice; Biliary flow; Ayurveda; Integrative management; Case report

INTRODUCTION

Jaundice is a clinical manifestation of disturbed bilirubin metabolism and generally becomes apparent when serum bilirubin exceeds approximately 2.5 mg/dL. Based on the site of the underlying pathology, jaundice is broadly classified as prehepatic, hepatic, or posthepatic. Prehepatic jaundice is predominantly associated with increased bilirubin production, hepatic jaundice with impaired hepatocellular processing or excretion, and posthepatic jaundice with impaired bile formation or biliary drainage obstruction. Dark urine and pale or clay-coloured stools are characteristic manifestations of impaired bile flow.^{[1],[2]}

In Ayurveda, *Kamala* is described in association with *Pandu Roga* and is primarily related to vitiation of *Pitta*. When aggravated *Pitta* progresses towards the *Koshta*,

Koshtashrita Kamala develops. The obstruction of the normal movement of *Pitta* by *Kapha* is described in the pathogenesis of *Ruddhapatha Kamala*, also referred to in relation to *Shakhashrita Kamala*.^{[3],[4]} From a contemporary clinical perspective, this may be considered a conceptual correlation in which *Pandu*, *Koshtashrita Kamala*, and *Ruddhapatha Kamala* broadly correspond to prehepatic, hepatocellular, and impaired biliary-flow patterns of jaundice, respectively. Such correlations represent interpretative clinical parallels and should not be considered direct equivalence between Ayurvedic and contemporary diagnostic classifications.

Biochemical evaluation is essential for determining the pattern and severity of hepatobiliary dysfunction. Serum bilirubin, aminotransferases, alkaline phosphatase, and gamma-glutamyl transferase, interpreted together with clinical findings and imaging, help distinguish predominantly hepatocellular from cholestatic or obstructive patterns. In particular, disproportionate elevation of cholestatic enzymes relative to aminotransferases favours a cholestatic pattern, whereas marked aminotransferase elevation is more suggestive of hepatocellular injury.^[2]

The present case describes a young adult with clinical and biochemical features interpreted as *Ruddhapatha Kamala* based on Ayurvedic clinical assessment, in whom no definite structural biliary obstruction was demonstrated on the available ultrasonographic examination. The case documents the integration of Ayurvedic assessment with contemporary clinical,

biochemical, and imaging parameters and reports the subsequent clinical and laboratory course during concurrent conventional and Ayurvedic management.

CASE PRESENTATION

A 20-year-old male presented to the outpatient department of Vasantdada Patil Ayurvedic Medical College, Sangli, with a primary concern of yellowish discoloration of the eyes and urine. The patient also reported decreased appetite and painful cramps in both lower limbs. There was no significant past medical history or previously diagnosed chronic systemic illness reported.

The history did not reveal any significant previous episode of jaundice or other hepatobiliary disease. There was no relevant family history and documented history of previous hospitalisation or specific treatment for a similar illness. No significant psychosocial factors relevant to the presenting illness were documented. On 18 August 2026, physical examination revealed:

General and Physical Examination

The relevant clinical findings at presentation were:

Table 1. Clinical Findings on Initial Presentation

Clinical parameter	Finding
General appearance	Icterus present
Eyes	Icterus present
Oedema	Absent
Cyanosis	Absent
Clubbing	Absent
Respiratory rate	16/min
Blood pressure	100/50 mmHg
Temperature	98°F
Stool	Clay-coloured
Urine	Yellowish/dark yellow

Ayurvedic Clinical Examination

Ayurvedic assessment was performed using *Asthavidha Pariksha*. The findings were:

Table 2. Findings of *Ashtavidha Pariksha* at Initial Assessment

<i>Asthavidha Pariksha</i>	Observation
<i>Nadi</i>	<i>Vata-Kaphaja</i>
<i>Jihva</i>	<i>Prakruta</i>
<i>Druk</i>	<i>Peetavarni</i>
<i>Mala</i>	<i>Shwetavarni</i>
<i>Shabda</i>	<i>Spashta</i>
<i>Akruti</i>	<i>Madhyama</i>
<i>Mutra</i>	<i>Peetavarni</i>
<i>Sparsha</i>	<i>Anushna</i>

The clinical findings were subsequently correlated with biochemical investigations and ultrasonographic findings to assess the nature of the jaundice and to arrive at an integrated clinical interpretation.

Diagnostic Methods

At presentation, the patient underwent detailed physical examination and

Asthavidha Pariksha. Laboratory evaluation included liver function tests (LFT), serum bilirubin fractions, serum AST (SGOT), ALT (SGPT), and urine examination for bile salts, bile pigments, and urobilinogen. Screening investigations for HBsAg and HIV were also performed. Abdominal ultrasonography was undertaken to assess hepatic and biliary morphology.

Table 3. Laboratory Diagnostic Findings on Day 1

Laboratory Investigation	Day 1 Value (18 August 2026)	Reference/Interpretation
Total Bilirubin	10.19 mg/dL	Markedly elevated
Direct Bilirubin	6.48 mg/dL	Elevated; direct-predominant hyperbilirubinaemia
Indirect Bilirubin	3.71 mg/dL	Elevated
Aspartate Aminotransferase (AST/SGOT)	311 IU/L	Markedly elevated
Alanine Aminotransferase (ALT/SGPT)	330 IU/L	Markedly elevated

Random Blood Sugar	98 mg/dL	Within expected range
Urine Bile Salts	Positive	Suggestive of conjugated bilirubin excretion
Urine Bile Pigments	Positive	Suggestive of conjugated hyperbilirubinaemia
Urine Urobilinogen	12 mg/dL	Elevated
Urine Albumin	3+	Positive
HBsAg	Negative	No laboratory evidence of hepatitis B surface antigen
HIV	Negative	Non-reactive

Imaging Findings

Abdominal ultrasonography showed mild hepatosplenomegaly, with a liver span of 15.1 cm and splenic length of 12.8 cm, along with mild pseudo-thickening of the gallbladder wall. No definite structural cause of biliary obstruction was identified on the available ultrasonographic examination.

The Day 1 investigations demonstrated marked hyperbilirubinaemia with a direct-predominant pattern, accompanied by substantially elevated AST and ALT levels. Urinary bile salts and bile pigments, together with clay-coloured stool and dark-yellow urine, supported the clinical assessment of impaired biliary flow. However, definitive imaging evidence of mechanical obstruction, a specific obstructive aetiology, could not be conclusively established.

Final Diagnostic Interpretation

Considering the clinical presentation, *Asthavidha Pariksha* findings, biochemical abnormalities, urine findings, and imaging,

the case was clinically interpreted from the Ayurvedic perspective as *Ruddhapatha Kamala*, characterised by features suggestive of impaired biliary flow.

From the contemporary clinical perspective, the case represented acute jaundice with predominantly direct hyperbilirubinemia and elevated aminotransferases, accompanied by clinical features suggestive of impaired bile flow, without demonstrated structural biliary obstruction on ultrasonography.

Therapeutic Intervention

The patient was managed with a concurrent conventional and Ayurvedic therapeutic approach during the hospital stay. The conventional treatment consisted primarily of supportive pharmacological therapy, while Ayurvedic management was directed towards the clinically assessed condition of *Ruddhapatha Kamala*. No surgical intervention was undertaken.

5.2 Conventional Pharmacological Intervention

The following conventional medications were administered during hospital management:

Table 4. Conventional Therapeutic Interventions During Hospitalisation

Intervention	Dose / Strength	Route	Frequency	Duration
Normal saline (NS) with Pantoprazole	100 mL	Intravenous	Twice a day	For 7 days, Initial supportive management
DNS with Eldervit, subsequently D10%	500 mL	Intravenous	Twice a day	For 7 days, administered during initial treatment
Monocef (Ceftriaxone)	1 g in 100 mL NS	Intravenous	Every 12 hours	For 7 days, Administered during hospitalisation
Emeset	4mg Stat dose	Intravenous	Stat	Administered initially once

Ayurvedic Pharmacological Intervention

The Ayurvedic regimen consisted of:

Table 5. Ayurvedic Therapeutic Regimen Administered During Hospitalisation

Ayurvedic intervention	Dose	Frequency	Route	Duration
<i>Triphala Churna</i>	5 g	At bedtime (HS)	Oral	For 7 days
Nirocil syrup	2 teaspoons	Twice daily (BD)	Oral	For 7 days
<i>Arogyavardhini Vati</i>	2 tablets	Twice daily (BD)	Oral	For 7 days
<i>Kamdudha Vati</i>	2 tablets	Twice daily (BD)	Oral	For 7 days
Himcocid SF	1 teaspoon	Twice daily (BD)	Oral	For 7 days
Looze syrup	10 mL	At bedtime (HS)	Oral	For 7 days
<i>Trikatu Churna</i>	5 g	Twice daily (BD)	Oral	For 7 days

<i>Sharpunkha-Amalaki Churna</i>	10g	Twice daily (BD)	Oral	For 5 days
<i>Bhumyamalaki swaras</i>	10ml	Twice daily (BD)	Oral	For 5 days

Clinical Course and Response to Integrative Management During Hospitalisation

The patient was admitted on 18 August 2026 with clinically evident icterus, yellowish discoloration of urine, clay-coloured stool, decreased appetite, and painful cramps in the lower limbs. Physical examination revealed icterus without oedema, cyanosis, or clubbing. Baseline biochemical investigations demonstrated marked hyperbilirubinaemia, with a total serum bilirubin of 10.19 mg/dL, including direct bilirubin of 6.48 mg/dL and indirect bilirubin of 3.71 mg/dL. Hepatic enzymes were elevated, with AST (SGOT) of 311 IU/L and ALT (SGPT) of 330 IU/L. Urine examination was positive for bile salts and bile pigments, with elevated urobilinogen. The clinical findings of direct-predominant hyperbilirubinaemia, dark-yellow urine, clay-coloured stool, and positive urinary bile constituents were suggestive of impaired bilirubin excretion and altered bile flow. However, ultrasonography demonstrated only mild hepato-splenomegaly without definite evidence of biliary duct dilatation or structural obstruction.

The patient received concurrent conventional supportive management and Ayurvedic intervention as shown in Table 4 and Table 5. On Day 1, *Triphala Churna*, *Nirocil syrup*, *Arogyavardhini Vati*, *Kamdudha Vati*,

Himcocid SF, *Looze syrup*, and *Trikatu Churna* were administered. On Day 2, stool had not been passed, and urine remained dark yellow; therefore, the Ayurvedic regimen was modified by adding *Sharpunkha–Amalaki Churna* and *Bhumyamalaki Swarasa*. Following this modification, bowel evacuation occurred on the same day, and vital parameters remained stable.

Subsequently, progressive clinical improvement was observed. Yellowish discoloration of the eyes and urine gradually reduced, appetite improved, and leg cramps subsided. Serial biochemical assessment performed on 22 August 2026 (Day 5) demonstrated a marked decline in serum bilirubin levels, with total bilirubin decreasing from 10.19 mg/dL to 5.30 mg/dL, direct bilirubin from 6.48 mg/dL to 2.48 mg/dL, and AST from 311 IU/L to 67 IU/L. Urinary bile salts, bile pigments, and urobilinogen were no longer detected. ALT increased slightly from 330 IU/L to 337 IU/L, and this finding was retained in the report to provide a transparent representation of the biochemical response profile. These diagnostic findings are presented in Table 6.

Further investigations performed on 25 August 2026 (Day 8) demonstrated continued improvement, with total bilirubin reducing to 2.94 mg/dL, direct bilirubin to 1.51 mg/dL, and indirect bilirubin to 1.43 mg/dL. These biochemical changes corresponded with

substantial clinical recovery. The patient had recovered clinically, with only minimal residual weakness and was discharged on 25

August 2026. Ayurvedic medication was continued for a further 5 days at home.

Table 6. Date-wise Clinical and Laboratory Findings

Date / Clinical Day	18 Aug 2026 – Day 1	22 Aug 2026 – Day 5	25 Aug 2026 – Day 8
Clinical findings	Icterus present; yellowish/dark-yellow urine; clay-coloured stool; decreased appetite; leg cramps	Reduction in yellowish discoloration; improvement in appetite and leg cramps; clinical condition stable	Clinically recovered with minimal residual weakness; discharged in stable condition
Total bilirubin (mg/dL)	10.19	5.30	2.94
Direct bilirubin (mg/dL)	6.48	2.48	1.51
Indirect bilirubin (mg/dL)	3.71	2.82	1.43
AST/SGOT (IU/L)	311	67	---
ALT/SGPT (IU/L)	330	337	---
Urine bile salts	Positive	Absent	---
Urine bile pigments	Positive	Absent	---
Urobilinogen	12 mg/dL	Absent	---
Clinical interpretation	Baseline biochemical evidence of significant hyperbilirubinaemia with a predominantly direct component and elevated aminotransferases and Urobilinogen	Marked reduction in total bilirubin and AST; ALT showed a slight increase compared with baseline.	Further reduction in serum bilirubin with favourable short-term clinical recovery

RESULTS

The patient showed progressive clinical and biochemical improvement during the 8-day

hospitalisation while receiving concurrent conventional and Ayurvedic management.

At baseline, total bilirubin was 10.19 mg/dL (direct: 6.48 mg/dL), with AST 311 IU/L and

ALT 330 IU/L. Urine was positive for bile salts and bile pigments, with urobilinogen of 12 mg/dL. By Day 5, total bilirubin decreased to 5.30 mg/dL, direct bilirubin to 2.48 mg/dL, and AST to 67 IU/L. Urinary bile salts, bile pigments, and urobilinogen were no longer detected; ALT showed a slight increase to 337 IU/L.

By Day 8, total bilirubin further decreased to 2.94 mg/dL (direct: 1.51 mg/dL; indirect: 1.43 mg/dL). Clinically, icterus and dark-yellow urine reduced, appetite improved, and leg cramps subsided. The patient was discharged in a stable condition with minimal residual weakness. No adverse events were documented. As conventional and Ayurvedic therapies were administered concurrently, the observed improvement cannot be attributed to any single intervention.

DISCUSSION

Jaundice represents a clinical manifestation of disturbed bilirubin metabolism and may arise from prehepatic, hepatocellular, or posthepatic processes. In the present case, the initial serum bilirubin was 10.19 mg/dL, with a direct bilirubin of 6.48 mg/dL, accompanied by elevated AST (311 IU/L) and ALT (330 IU/L). The clinical presence of icterus, yellowish urine, clay-coloured stool, and urinary bile salts and bile pigments suggested altered bilirubin handling and impaired bile flow. However, abdominal ultrasonography demonstrated only mild hepatosplenomegaly along with mild pseudo-thickening of the gallbladder wall, without documented biliary duct dilatation or a definite structural obstructive lesion. Therefore, the case is best interpreted as jaundice with clinical features suggestive of

impaired biliary flow, rather than radiologically confirmed mechanical obstructive jaundice.

From the Ayurvedic perspective, *Kamala* is described in association with *Pandu Roga* and is predominantly related to derangement of *Pitta*. Classical descriptions distinguish *Koshtashrita Kamala* from *Ruddhpatha Kamala*, in which the normal movement of aggravated *Pitta* is considered to be obstructed by *Kapha*.^{[5],[6]} In the present case, the clinical findings - including *Peetavarni Druk*, *Peetavarni Mutra*, *Shwetavarni Mala*, and *Vata-Kaphaja Nadi*- were interpreted within this Ayurvedic framework. The diagnosis of *Ruddhpatha Kamala* should therefore be understood as an Ayurvedic clinical interpretation, rather than as a direct equivalent of contemporary radiologically confirmed biliary obstruction.

Integrative Therapeutic Approach

The patient received concurrent conventional and Ayurvedic treatment. This is an important feature of the case because the clinical improvement occurred in the context of integrative management, and not after an isolated Ayurvedic intervention.

The conventional regimen included intravenous fluids, pantoprazole, ceftriaxone, and ondansetron (Emeset). Pantoprazole is a proton-pump inhibitor that irreversibly inhibits the gastric H^+/K^+ -ATPase and suppresses gastric acid secretion; its role in this case was therefore supportive rather than directly hepatoprotective.^[7] Ceftriaxone is a bactericidal β -lactam antimicrobial that inhibits bacterial cell-wall synthesis.^[8] Ondansetron is a 5-HT₃ receptor antagonist used primarily for the prevention and

treatment of nausea and vomiting; it similarly represents symptomatic supportive treatment rather than a specific treatment for jaundice. The intravenous fluids were intended to provide supportive hydration and metabolic support during the acute illness.

Ayurvedic Therapeutic Rationale

The Ayurvedic regimen comprised *Triphala Churna*, Nirocil syrup, *Arogyavardhini Vati*, *Kamadudha Vati*, Himcocid SF, Looze syrup, and *Trikatu Churna*. On the following day, *Sharpunkha–Amalaki Churna* and *Bhumyamalaki Swarasa* were added in response to persistence of dark-yellow urine and failure to pass stool.

From an Ayurvedic therapeutic perspective, the regimen appears to have been directed toward correction of *Pitta–Kapha* imbalance, restoration of gastrointestinal function, and support of hepatic metabolism.

Arogyavardhini Vati is particularly relevant to the present case because it has a long-standing Ayurvedic indication in disorders involving the liver and metabolism. Experimental studies have reported hepatoprotective effects of *Arogyavardhini* in chemically induced hepatic injury, including reductions in serum AST and alkaline phosphatase and histological evidence of hepatic protection in animal models.^{[9],[10]}

Trikatu Churna, traditionally composed of *Zingiber officinale*, *Piper longum*, and *Piper nigrum*, may theoretically support digestion and metabolic processing through its classical *Deepana* and *Pachana* properties. Experimental research has also demonstrated hepatoprotective, antioxidant, anti-inflammatory, and anti-apoptotic effects of

Trikatu in an experimental model of alcohol-induced liver injury.^[11]

Bhumyamalaki, used in this case as *Bhumyamalaki Swarasa*, is traditionally employed in Ayurvedic practice for liver disorders. Contemporary experimental literature suggests antioxidant and hepatoprotective activities of *Phyllanthus* species. However, clinical evidence remains inconsistent. A double-blind randomized controlled trial of *Phyllanthus niruri* in mild-to-moderate alcoholic hepatitis did not demonstrate significant improvement in liver enzymes or bilirubin, although an increase in total antioxidant levels and an appetite-related benefit were reported.^[12]

Triphala was included in the initial regimen. Its traditional rationale includes regulation of gastrointestinal function and elimination; however, contemporary clinical evidence does not establish a specific therapeutic effect of *Triphala* on acute jaundice or liver injury. A systematic review of clinical studies found no significant effect of *Triphala* on liver-function tests.^[13] Accordingly, its role in the present case should be presented as part of the broader Ayurvedic therapeutic strategy rather than as an independently established hepatoprotective treatment.

The addition of *Sharpunkha–Amalaki Churna* and *Bhumyamalaki Swarasa* occurred when the patient had not passed stool and continued to have dark-yellow urine. The subsequent passage of stool and later clinical improvement temporally followed this modification.

The principal basis for reporting this case is the convergence of an Ayurvedic clinical interpretation of *Ruddhapatha Kamala* with

contemporary findings of jaundice characterised by direct-predominant hyperbilirubinaemia, urinary bile pigments and bile salts, clay-coloured stool, and elevated aminotransferases. The marked decline in bilirubin and AST, together with clinical recovery during treatment, provides a clinically meaningful observation.

CONCLUSION

This case highlights the potential value of integrating Ayurvedic clinical assessment with contemporary biochemical and imaging evaluation in a patient clinically interpreted as *Ruddhapatha Kamala*. The patient demonstrated substantial clinical and biochemical improvement during concurrent conventional and Ayurvedic management, with serum total bilirubin decreasing from 10.19 mg/dL to 2.94 mg/dL over the observed period. The case also demonstrates the importance of distinguishing an Ayurvedic clinical concept of impaired *Pitta* movement from radiologically confirmed mechanical biliary obstruction. While the observed response is clinically encouraging, the concurrent use of multiple interventions and the single-case design prevent attribution of benefit to any individual therapy.

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