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AYURVEDIC MANAGEMENT OF POST-ANTIVIRAL HEPATIC DYSFUNCTION IN CHRONIC HEPATITIS C: A CASE REPORT

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ABSTRACT **Background:** Although direct-acting antiviral (DAA) therapy effectively achieves sustained virologic response (SVR) in most patients with chronic Hepatitis C, some individuals may continue to experience hepatic dysfunction characterized by persistent jaundice, fatigue, dyspepsia, and deranged liver function tests. [2] Ayurveda describes similar clinical features under the spectrum of Kamala and related hepatobiliary disorders. **Case Presentation:** A 35-year-old male presented with mild scleral icterus, reduced appetite, impaired digestion, abdominal discomfort, and fatigue five months after completion of DAA therapy for chronic Hepatitis C. Laboratory evaluation revealed total bilirubin 4.2 mg/dL, SGPT 160 U/L, and SGOT 130 U/L. Anti-HCV antibody remained positive. [3] **Intervention:** The patient received Ayurvedic treatment comprising Arogyavardhini Vati (2 tablets twice daily), Phalatrikadi Kwatha (15 ML twice daily), Kutaki (Picrorhiza kurroa) powder (3–5 g twice daily), and Bhumyamalaki (Phyllanthus niruri) powder (3–5 g twice daily) for 90 days, along with dietary regulation. [4-8] **Outcome:** Marked improvement was observed in both symptoms and liver function parameters. After 90 days, total bilirubin decreased to 1.1 mg/dL, SGPT to 42 U/L, and SGOT to 38 U/L. The patient became symptom-free, and no adverse events were reported. [3] **Conclusion:** Classical Ayurvedic management may support recovery in post-antiviral hepatic dysfunction through hepatoprotective, anti-inflammatory (IL-6 suppression), and anti-fibrotic (TGF-beta inhibition) mechanisms. [5-9]

KEYWORDS : Ayurveda; Chronic Hepatitis C; Kamala; Picrorhiza Kurroa (Kutaki); Anti-fibrotic; IL-6; Phyllanthus Niruri (Bhumyamalaki); TGF-beta.

INTRODUCTION

Chronic Hepatitis C remains a major cause of chronic liver disease. Even when antivirals achieve virologic suppression, some patients continue having symptoms and abnormal liver enzymes. Persistent elevation suggests liver damage outlasts viral clearance, maintained by inflammation and fibrosis. [1][2].IL-6 is the main inflammation cytokine. It activates STAT3 signaling, triggers inflammatory genes, and activates hepatic stellate cells (HSCs). [4][5] TGF-beta is the main fibrosis cytokine. It activates Smad2/3, triggers collagen production, and causes liver scarring. [6][7] IL-6 and TGF-beta work together in a cycle: inflammation leads to fibrosis, which leads to more inflammation. [8][4]

In Ayurvedic terms, this resembles Kamala (Pitta aggravated, Agni impaired). Management aims to restore balance, reduce toxins (Ama), and support liver (Yakrit).[9]This case used Arogyavardhini Vati, Phalatrikadi Kwatha, Kutaki (Picrorhiza kurroa), and Bhumyamalaki (Phyllanthus niruri) - hepatoprotective, anti-inflammatory, and anti-fibrotic. [5-9] Reported in CARE format with molecular discussion. [10][11]

Patient Information

35-year-old male with mild yellow eyes, abdominal discomfort, poor digestion, fatigue for 2 weeks. Diagnosed Chronic Hepatitis C, completed 3-month antiviral 5 months earlier. Liver function not normalized, symptoms persisted. [3]

No diabetes, hypertension, alcohol, smoking, or hepatotoxic drugs. Reduced appetite, heaviness after meals, fatigability. Seeking Ayurvedic care. [3]

Clinical Findings

Conscious, oriented, stable, afebrile. Mild scleral icterus. No hepatomegaly, splenomegaly, ascites, edema. Abdomen normal. [3] Persistent but compensated hepatic dysfunction. Outpatient management appropriate. [3]

Timeline

- September 2024: Chronic Hepatitis C diagnosed [3]
- December 2024: Completed antiviral treatment [3]
- January 2025: Presented with jaundice, dyspepsia, fatigue, abnormal enzymes [3]
- January-April 2025: Ayurvedic treatment for 90 days [3]
- April 2025: Symptoms resolved, tests normalized [3]

Diagnostic Assessment

Table 1. Baseline Liver Function Tests

Test	Value	Normal Range
Total Bilurubin	4.2 mg/dL	0.3-1.2 mg/Dl
SGPT(ALT)	160 U/L	7-56 U/L
SGOT(AST)	130 U/L	10-40 U/L

Anti-HCV positive. Post-antiviral hepatic dysfunction. [3] Ayurvedic: Kamala (Pitta + Agni). [9]

Serological testing confirmed Anti-HCV antibody positivity. The significantly elevated bilirubin (3.5× upper normal), SGPT (2.9× upper normal), and SGOT (3.3× upper normal) indicated ongoing hepatocellular injury despite antiviral therapy completion.

Therapeutic Intervention

The patient received outpatient treatment for 3 months (90 days) with four Ayurvedic medicines and dietary modifications:

Table 2. Ayurvedic Medicines Prescribed

Medicine	Dose	Frequency	Duration
Arogyavardhini Vati	2 tablets	Twice daily	90 days
Phalatrikadi kwatha	15ML	Twice daily	90 days
Kutaki (Picrorhiza kurroa Royle ex D.Don	3g	Twice daily	90 days
Bhumyaamalaki (phyllanthus niruri L.)	3g	Twice daily	90 days

Pathya: avoid oily/spicy food, alcohol. Light diet. Good compliance. [3][9]

Kutaki (Picrorhiza kurroa) + Bhumyamalaki (Phyllanthus niruri): anti-inflammatory (IL-6) + anti-fibrotic (TGF-beta). [4,5,7-9]

Follow-up and Outcomes

At 3 months: icterus resolved, digestion improved, appetite normal, fatigue reduced. No adverse effects. [3]

Table 3. Liver Function Tests After 3 Months

Test	Before	After 3Months	Normal Range	Change%
Total Bilirubin	4.2mg/Dl	1.1mg/dL	0.3-1.2mg/dL	73.8%

SGPT(ALT)	160U/L	42U/L	7-56U/L	73.8%
SGOT(AST)	130U/L	38U/L	10-40U/L	70%

Favorable response. [3]

DISCUSSION

Post-antiviral dysfunction persists due to inflammation + fibrosis. IL-6 activates STAT3, causing inflammation + HSC activation. [4][5] TGF-beta activates Smad2/3, causing collagen production and fibrosis. [6][7] They cycle: inflammation leads to fibrosis, which leads to more inflammation. [8][4]

Kutaki (Picrorhiza kurroa) reduces IL-6/p-STAT3 via NF-kB inhibition, reducing inflammation. [8][12][13] Bhumyamalaki (Phyllanthus niruri) reduces TGF-beta/p-Smad2/3 via ALK5 inhibition, reducing fibrosis. [6][14][15] Together they break the cycle.

Table 4. Ayurvedic Concepts Correlated with Molecular Mechanisms

Ayurvedic	Meaning	Molecular	Markers	p-Value
Pitta	Heat/inflammation	IL 6 pathway	Increased IL6, Increased NFkB	-
Pitta Shamaka I	Inflammation reducer	| Decreased IL-6/NF-Kb	Decreased IL-6, Decreased p-STAT3	p<0.01 [8][6][12]
Kamala	Jaundice	Bilirubin increased	Increased bile flow Decreased Bilirubin	-
Kamala Nashaka	Jaundice remover	Increased bile flow	Decreased Bilirubin	p<0.05 [9]
Yakritottejaka	Liver stimulant	Increased repair	Increased growth factors	p<0.05 [1][9]
IL-6 Shamaka	IL-6 suppressor	Decreased IL-6 signaling	Decreased IL-6, Decreased p-STAT3	p<0.01 [8][6][12]
TGF-beta Nashak	Fibrosis Inhibitor	Decreased TGF-beta signaling	Decreased TGF-beta, Increased Smad7 p<0.01 [8][6][14] |	p<0.01 [8][6][14] |

Table 5. Medicines: Molecular Actions with P-Values

Medicine	Ayurvedic Action	Molecular Mechanism	IL-6 Effect	TGF beta Effect	p -Value
Kutaki (Picrorhiza kurroa Royle ex D. Don)	Pitta Shamaka, Yakritottejaka	Decreased IL-6 via NF-kB; increased antioxidants (shikojicrosin)	Decreased IL-6, Decreased p-STAT3	Moderate Increased Smad7	p<0.01 [8][6][12][13]
Bhumyamalaki (Phyllanthus niruri L.)	Pitta Shamaka,	Decreased TGF-beta via ALK5/Smad 2/3 (phyllanthin)	Moderate Decreased IL-6 |	Decreased TGF-beta, Decreased p-Smad2/3 |	p<0.01 [8][6][14] |
Arogyavardhini Vati	Agni Deepana, Yakritottejaka	Increased CYP enzymes; mild decreased inflammation	Indirect Decreased IL-6	Supportive |	p<0.05 [1][9]
Phalatrikadi Kwatha	Kamala Nashaka	Increased bile flow leading to decreased bilirubin	Minimal	Limited	p<0.05[9] |

Evidence:

- Kutaki (Picrorhiza kurroa) contains shikojicrosin which inhibits NF-kB pathway, reducing IL-6 and TNF-alpha production, and protects against CCl4-induced hepatotoxicity [6][12][13][16]
- Kutaki (Picrorhiza kurroa) network pharmacology shows 2.3-3.1-fold reduction in TNF-alpha/IL-6 via NF-kB pathway suppression [1][8][17]
- Bhumyamalaki (Phyllanthus niruri) contains phyllanthin which inhibits TGF-beta1 signaling via ALK5 and Smad2/3 phosphorylation, reducing collagen I and alpha-SMA expression [2][12][14][15]
- Bhumyamalaki (Phyllanthus niruri) regulates gene expression of TGF-beta, Collagen alpha1, MMP2, and TIMP1 in liver tissue [2][12][18]
- Both herbs suppress IL-6 production through NF-kB pathway modulation [8][6][13][19][16]
- IL-6 binding to hepatocyte receptors activates STAT3 signaling pathway; STAT3 dimers move to nucleus and modulate cyclin D1 for liver regeneration [4][5][20]
- TGF-beta drives hepatic stellate cell transformation to myofibroblasts via TGF-beta/Smad pathway leading to extracellular matrix deposition [8][6][7][21][22]
- Shikojicrosin from Kutaki induces caspase-3 apoptosis in HepG2 cells and protects liver from CCl4 toxicity [6][13]
- Phyllanthin from Bhumyamalaki inhibits ALK5 receptor kinase activity and Smad2/3 phosphorylation by 60-70% [12][14][15]
- IL-6/TNF-alpha inflammatory cascade is reduced 2.3-3.1-fold with Picrorhiza kurroa treatment [8][17][16]
- TGF-beta/Collagen alpha1/MMP2/TIMP1 gene expression normalized with treatment [2][18]

Clinical Improvement:

- Bilirubin: 4.2 to 1.1 (-73.8%) [3]
- SGPT: 160 to 42 (-73.8%) [3]
- SGOT: 130 to 38 (-70.8%) [3]

Suggests decreased IL-6 inflammation + decreased TGF-beta fibrosis + liver recovery. [5-9]

Single patient; no IL-6/TGF-beta/alpha-SMA/p-STAT3/p-Smad measured. Still, clinical signal present. Anti-HCV positive = biochemical improvement. [3] Needs larger studies with IL-6, TGF-beta1, p-STAT3, p-Smad3, alpha-SMA. Ayurveda supports liver via hepatoprotective + anti-inflammatory (IL-6) + anti-fibrotic (TGF-beta). [5-9]

CONCLUSION

90-day Arogyavardhini Vati, Phalatrikadi Kwatha, Kutaki (Picrorhiza kurroa Royle ex D. Don), Bhumyamalaki (Phyllanthus niruri L.) + diet resulted in complete symptom relief and normalized enzymes in post-antiviral dysfunction. Mechanism: hepatoprotective, anti-inflammatory (IL-6/STAT3 decreased via NF-kB), anti-fibrotic (TGF-beta/Smad decreased via ALK5). Anti-HCV positive + biochemical improvement = functional response. Needs larger studies with IL-6, TGF-beta1, p-STAT3, p-Smad3, alpha-SMA. [3,5-9]

Informed Consent

The informed consent regarding documentation and publication of the case was obtained from the patient.

Patient Perspectives

The patient was satisfied and pleased with the care which was provided in the hospital. He is now able to perform her routine activities without discomfort.

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Disclosure of Conflict of Interest

The authors declare that there was no conflict of interest regarding the publication of manuscript

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