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Case Study

REVIVING *RAKTA*: DEEP MOLECULAR REMISSION IN CHRONIC MYELOID LEUKAEMIA

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ABSTRACT

Chronic Myeloid Leukaemia (CML) is a myeloproliferative neoplasm driven by constitutively active BCR-ABL tyrosine kinase arising from the Philadelphia chromosome t (9;22). From an Ayurvedic standpoint, it correlates with *Vatarakta-sadrusha avastha*, involving *Vata–Rakta avarana* resulting in *Rakta dhatu dushti* and impaired haematopoietic regulation. This case study describes a 29-year-old male with chronic-phase CML (baseline: Hb: 7.3 g/dl, WBC: 19,220/µL, Platelets: 3.6 lakh/µL) who was managed exclusively with Ayurvedic intervention following patient-informed withdrawal from imatinib. The treatment protocol comprised *Hingwashtak Churna* with ghee, *Navayas Loha, Ashwattha Kwath* (decoction of *Ficus religiosa*) with honey, and *Vayasthapan Mahakashaya* over six months. *Ashwattha*, with *Kashaya rasa* and *Sheeta virya*, promotes *Rakta-prasadana* and *Dhatu-sthirata*; its phytoconstituents (flavonoids, phenolics) exert antioxidant and immunomodulatory effects potentially influencing abnormal myeloid proliferation. *Vayasthapan Mahakashaya*, a classical *Rasayana* formulation, supports *Ojas, Dhatu-poshana*, and systemic homeostasis. Post-intervention, the patient achieved haematological remission (WBC: 8,700/µL, platelets normalised) and a substantial molecular response (BCR-ABL IS: 0.101%, approaching major molecular response threshold), alongside 8kg weight gain and complete resolution of constitutional symptoms, with no adverse events documented. This report suggests potential standalone value of Ayurvedic intervention in CML management and warrants further controlled investigation.

INTRODUCTION

Chronic myeloid leukaemia (CML) is a clonal myeloproliferative disorder with an annual incidence of approximately 1–2 per 100,000 in North America and Europe. At its molecular core, CML is driven by the Philadelphia chromosome- a reciprocal t(9;22) (q34;q11) translocation[1] that generates the BCR-ABL fusion oncoprotein, a constitutively active tyrosine kinase that overrides normal haematopoietic regulation, driving uncontrolled myeloid proliferation while suppressing apoptosis.[2]

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The introduction of imatinib mesylatea selective BCR-ABL tyrosine kinase inhibitor (TKI) approved by the United States Food and Drug Administration in 2001- transformed the prognosis of CML, achieving major molecular responses (BCR-ABL IS ≤0.1%) in 60–80% of chronic-phase patients at five years.[3] Despite this success, challenges persist: secondary resistance attributable to BCR-ABL kinase domain mutations occurs in 20–30% of patients,[4] while treatment-related toxicities necessitate dose reductions or discontinuation in 10–15% of cases, impairing quality of life and long-term adherence.[5] These limitations create clinical space for complementary therapeutic strategies.

Ayurveda interprets CML within the framework of *Vatarakta*- a condition characterised by aggravated *Vata dosha* corrupting *Rakta dhatu* (blood tissue)- resulting in *Vata-Rakta avarana*, progressive *Rakta* and *Majja dhatu kshaya*, and *Ojas* depletion. This framework provides an integrative rationale for

targeting both the pathological expression (*Rakta-prasadana)* and constitutional substrate *(Rasayana)* of the disease.

Ashwattha (*Ficus religiosa* Linn.), revered in classical Ayurvedic literature including Charaka Samhita for its efficacy in *Vatarakta* and blood disorders, has been shown to contain flavonoids (quercetin, rutin), phenolics, and tannins with documented antioxidant, anti-inflammatory, and anti-proliferative properties.[6] These bioactives inhibit NF-κB signalling, suppress reactive oxygen species (ROS), and modulate apoptotic pathways- mechanisms relevant to leukemic cell biology.[7] Its *Kashaya rasa* and *Sheeta virya* properties further align with *Rakta-prasadana* and *Pitta-Vata* pacification.

Vayasthapan Mahakashaya, described in Charaka Samhita as a premier *Rasayana* formulation comprising ten *Dravyas -Amrita (Tinospora cordifolia), Abhaya (Terminalia chebula), Dhatri (Emblica officinalis), Mukta (Sphaeranthus indicus), Shweta (Convolvulus pluricaulis), Jivanti (Leptadenia reticulata), Atirasa (Asparagus racemosus), Mandukparni (Centella asiatica), Sthira (Desmodium gangeticum),* and *Punarnava (Boerhavia diffusa)*-is targeted at systemic rejuvenation, *Dhatu poshana*, and immune homeostasis. Together, *Ashwattha Kwath* and *Vayasthapan Mahakashaya* represent a dual-modality *Samprapti-vighatana* strategy addressing CML at both the Rakta-correction and constitutional-restoration levels.

This case report documents the clinical and molecular outcomes of a standalone Ayurvedic intervention in a chronic-phase CML patient who voluntarily discontinued conventional imatinib therapy, highlighting remarkable haematological and molecular responses achieved over a one-year observational period.

AIM AND OBJECTIVES

Aim

To evaluate the clinical outcomes of a standalone Ayurvedic intervention using *Ashwattha Kwath* and *Vayasthapan Mahakashaya* in a patient with chronic-phase CML.

Primary Objective

To assess the efficacy of *Ashwattha Kwath* and *Vayasthapan Mahakashaya*, with adjunctive *Hingwashtak Churna* and *Navayas Loha* in the initial phase, in achieving haematological remission and substantial molecular response in chronic-phase CML.

Secondary Objectives

- To evaluate symptomatic improvements including fatigue resolution, weight gain, splenomegaly regression, and quality-of-life enhancement.
- To establish an Ayurvedic correlative diagnosis of CML as *Vatarakta* with *Vata-Rakta avarana* leading to *Rakta* and *Majja dhatu dushti*, validating *Samprapti-vighatana* via *Rakta-prasadana* and *Rasayana* therapy.
- To explore the mechanistic rationale of *Ashwattha's Kashaya-Sheeta* properties and phytochemicals (flavonoids, phenolics) alongside *Vayasthapan Mahakashaya's Ojas* augmentation and *Dhatu-poshana* in modulating haematopoietic dysfunction.

Case Presentation

Patient Information

A 29-year-old male government employee from Maharashtra presented to the OPD of Dr BRKR Government Ayurvedic Medical College and Hospital, Hyderabad, in April 2025, seeking standalone Ayurvedic therapy. He had been diagnosed with chronic-phase CML three months prior on the basis of peripheral blood smear and RT-PCR (BCR-ABL positive). He had initiated imatinib 200 mg/day but experienced Grade 2 fatigue and myalgia with a suboptimal haematological response (persistent leucocytosis). Dissatisfied with allopathic side effects and motivated by a family history of successful Ayurvedic cancer management, he opted for integrative care under supervision, with informed consent obtained for discontinuation of imatinib.

Study Design and Methodology

A prospective single-case observational study was conducted following established guidelines for rigorous case report documentation over a one-year period (April 2025 to April 2026). The last documented follow-up was February 2026, with laboratory investigations ongoing at the time of submission.

Consent and Ethical Clearance

Comprehensive written informed consent was obtained from the patient in compliance with Indian Council of Medical Research (ICMR) ethical guidelines for biomedical research involving human participants.[8] Institutional Ethics Committee approval was obtained from the Dr BRKR Government Ayurvedic Medical College Ethics Committee. This report adheres to Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational case study documentation.[9]

Chief Complaints

Progressive fatigue and generalised weakness for 4 months, worsening over 2 weeks (severity: 8/10 on numeric rating scale).

Associated Complaints

- Low-grade fever (99–100°F) with intermittent chills.
- Abdominal fullness and early satiety.
- Mild splenomegaly-related discomfort (left hypochondrium).
- Pallor, weight loss (6kg over 2 months), and nocturnal diaphoresis.
- No bleeding manifestations or active infections at presentation.

Past History

Medical: Imatinib 200mg/day for 2 months (suboptimal response). No history of tuberculosis, diabetes, hypertension, or prior malignancies.

Surgical: Appendectomy at age 20 (uncomplicated).

Personal/Family: Non-smoker, no substance use. Father with Type 2 diabetes; no haematological malignancies in family.

Laboratory Investigations

Complete Blood Count (CBC)- 5 April 2025 (Pre-Intervention Baseline)

Parameter	Value	Interpretation
Haemoglobin (Hb)	7.3 g/dl	Severe microcytic hypochromic anaemia.
WBC (Total Leucocyte Count)	19,220/µL	Significant leucocytosis with myeloid predominance.
Platelet Count	3.6 lakh/µL (360,000/µL)	Moderate thrombocytosis

Bone Marrow Aspiration and Biopsy - 9 April 2025: Confirmed CML without blast transformation; increased myeloid precursors with normal maturation sequence preserved, consistent with chronic-phase disease.

Molecular Confirmation (RT-PCR for BCR-ABL): Presence of BCR-ABL fusion transcript confirmed, establishing definitive molecular diagnosis.

Ultrasound Abdomen - 5 April 2025: Mild splenomegaly with splenic longitudinal dimension approximately 12.5 cm. Hepatic parenchyma normal; no free fluid.

General Physical Examination

Parameter	Finding	Parameter	Finding
Height	170 cm	Weight / BMI	52 kg / 18 kg/m^2
Pulse Rate	88/min	Blood Pressure	118/76 mmHg
Temperature	99.2°F	Pallor	Moderate
Icterus / Cyanosis	Absent	Clubbing / Oedema	Absent
Per Abdomen	Splenomegaly: 2 cm below left costal margin Liver: not palpable		

***Ashtavidha Pariksha* (Eightfold Examination)**

Pariksha	Finding	Significance
Nadi	*Vata Prakruti,* slight *Kaphavrita* (wiry, irregular)	*Vata-Kapha* imbalance
Mutra	Clear, normal volume	*Medovaha srotas* unaffected
Mala	Normal consistency, no blood	*Purishavaha srotas* normal
Jihva	White coated	*Ama lakshana*
Shabda	Clear	Normal
Sparsha	Cool extremities	*Vata* predominance
Drik	Dull	*Rakta dhatu dushti*
Akruti	Medium build, emaciated	*Dhatu kshaya*

***Dashavidha Pariksha* (Tenfold Examination)**

Parameter	Finding	Significance
Prakruti	*Vata-Pitta*	Disease tendency toward *Vata-Pitta* disorders
Vikruti	*Vata-Rakta* predominant	Active *Vatarakta* pathology
Sara	*Rakta sara* reduced	Haematopoietic compromise
Samhanana	*Madhyama*	Moderate structural integrity
Agni	*Madhyama* with mild *Ama*	Partial digestive impairment
Satmya	Vegetarian diet	*Sattvic Anupana*-compatible
Satva	*Avara* (fatigue-related)	Reduced mental resilience
Ayu	*Madhyama*	Moderate life force
Vaya	*Taruna* (29 years)	Young, favourable prognosis

Diagnosis

Biomedical Diagnosis

Chronic-phase Chronic Myeloid Leukaemia (CML-CP) as per European Leukemia Net (ELN) 2020 diagnostic and response criteria.[10]

Table 1: Biomedical Diagnostic Assessment - ELN 2020 Criteria Fulfilled

Diagnostic Criterion (ELN 2020)	Patient Finding	Status
BCR-ABL fusion transcript (RT-PCR confirmation)	BCR-ABL positive (RT-PCR)	✓ Confirmed
Philadelphia chromosome / BCR-ABL gene fusion	Confirmed by RT-PCR	✓ Met
Myeloid predominance in bone marrow	Increased myeloid precursors, normal maturation	✓ Met
Blast phase features (≥15% blasts = accelerated; ≥30% = blast crisis)	<10% blasts; normal maturation preserved	✓ Absent - Chronic Phase
Accelerated phase features (basophilia ≥20%, platelet <100K)	Neither criterion present	✓ Absent
Peripheral blood leucocytosis with myeloid lineage	WBC: 19,220/μL with myeloid predominance	✓ Present
Splenomegaly	Mild splenomegaly (splenic length: 12.5 cm, USG)	✓ Present
Overall Diagnostic Conclusion	**Chronic Myeloid Leukaemia - Chronic Phase (CML-CP)**	

Ayurvedic Diagnosis

Table 2: Ayurvedic Diagnostic Correlation - *Nidana Panchaka and Samprapti Ghataka*

Ayurvedic Diagnostic Parameter	Findings / Correlation
Nidana (Causative factors)	Manasika stress, dietary irregularities, sedentary and *Vata*-aggravating lifestyle
Purvarupa (Prodromal features)	Progressive fatigue, pallor, and mild weight loss preceding confirmed diagnosis
Rupa (Clinical features)	*Pandu* (anaemia), *Daurbalya* (weakness), *Pleeha vriddhi* (splenomegaly), leucocytosis, *Rakta dushti*
Samprapti (Pathogenesis)	*Vata-Rakta avarana → Rakta dhatu dushti → Mamsa, Asthi-Majja kshaya → Ojokshaya*
Upashaya-Anupashaya	Improvement with *Rakta-prasadana (Ashwattha Kwath)* and *Rasayana (Vayasthapan Mahakashaya)*
Rogamarga	*Madhyama* (involving deep tissues: *Rakta, Mamsa, Asthi, Majja*)

Table 3: Biomedical–Ayurvedic Diagnostic Correlation

Biomedical Finding	Ayurvedic Correlation
Myeloid proliferation (leucocytosis - WBC 19,220/µL)	*Vata*-driven *Rakta dhatu dushti* (excess abnormal *Rakta-paramanu* production)
Anaemia (Hb 7.3 g/dl)	*Rakta dhatu kshaya* (depletion of healthy blood tissue)
Splenomegaly	*Pleeha vriddhi* (*Vata-Kapha avarana* in *Raktavaha srotas*)
BCR-ABL molecular dysregulation	*Gambheera dhatu* involvement - *Majja dhatu dushti* (deep-tissue genomic corruption)
Fatigue and cachexia (6kg weight loss)	*Ojokshaya* (*Ojas* depletion leading to immune and metabolic failure)
Bone marrow hyperplasia	*Asthi-Majja dhatu* involvement (*Asthivaha* and *Majjavaha srotas dushti*)

***Samprapti* (Pathogenesis)**

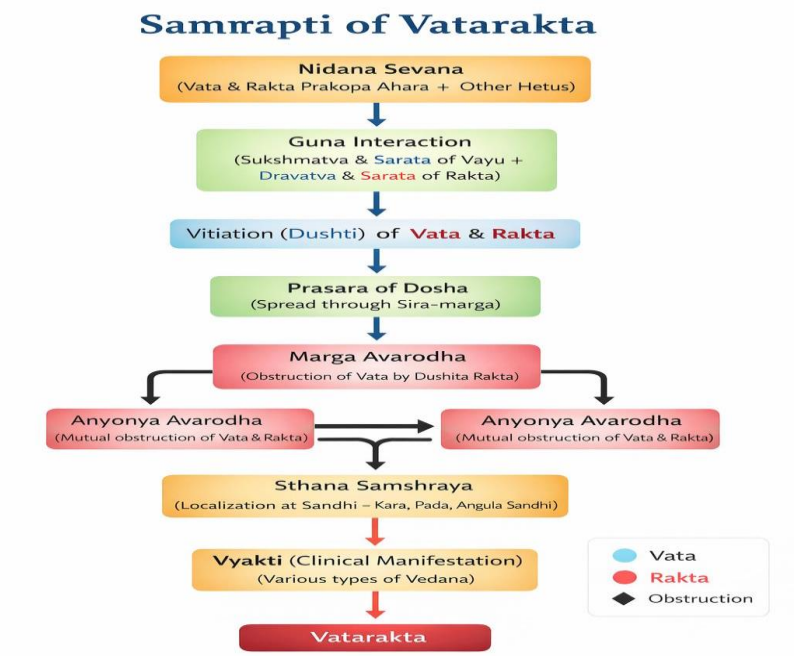


Figure 1: Dual-Framework *Samprapti* - Ayurvedic and Biomedical Pathogenesis of CML

Samprapti Ghataka

Ghataka	**Details**
Dosha	*Vata Pradhan Tridosha-Janya Vyadhi*
Dushya	*Rakta*, *Twak, Mamsa, Asthi, Majja*
Agni	*Mandagni* (impaired *Jatharagni* and *Dhatvagni*)
Udbhavasthana	*Pakwashya* (colon - seat of *Vata*)
Sancharasthana	*Sarva Sharira* (systemic circulation)
Adhishthan	Bone marrow *(Asthi-Majja)*
Srotas	*Raktavaha, Asthivaha, Majjavaha*
Srotodushthi Prakara	*Sanga* (obstruction) - Manifested as splenomegaly and abnormal cellular sequestration. *Vimargagaman* (displacement) - Manifested as abnormal leucocyte circulation and haematopoietic dysregulation.
Rogamarga	*Madhyama* (deep tissue pathway)

Treatment Protocol

Intervention	Dose and schedule	Duration	Phase
Hingwashtak Churna	3 g with ghee immediately before morning and evening meals.	1 month	Phase 1
Navayas Loha	2 tablets (500mg each) after morning and evening meals.	2 months	Phase 1
Ashwattha Kwath (Decoction of Ficus religiosa)	20ml with *Madhu* (honey, 10g) after morning and evening meals.	6 months	Phase 2
Vayasthapan Mahakashaya	20 ml with equal quantity of warm water after morning and evening meals.	6 months	Phase 2
Pathya (Advised diet & lifestyle)	Milk, ghee, wheat, old rice *(Shali),* seasonal fruits (banana, pomegranate, grapes), nuts, dates, raisins, coconut water; ample hydration. Yoga: *Vajrasana, Sarvangasana, Ashwini Mudra, Pranayama* - daily 20 minutes.	1 year	All
Apathya (avoided)	Excessive spicy, fried, or fermented foods, alcohol, smoking; prolonged sitting, hot-water baths, night shifts.	1 year	All

Assessment Parameters and Clinical Timeline

Monthly CBC and liver/renal function tests were performed throughout. RT-PCR for BCR-ABL was performed at baseline (May 2025) and at 10-month follow-up (February 2026). Safety monitoring followed Common Terminology Criteria for Adverse Events (CTCAE) v5.0.

Table 4: Longitudinal Timeline of Clinical Events, Interventions, and Laboratory Findings

Date	Event / Investigation	Findings	Clinical Interpretation/Action
01/04/2025	Initial presentation; history; investigations advised	—	Patient presented to OPD
05/04/2025	CBC; USG abdomen	Hb: 7.3 g/dl; WBC: 19,220/µL; Plt: 3.6 lakh/µL; Mild splenomegaly (12.5 cm)	Anaemia with leucocytosis; suspicion of haematological malignancy
09/04/2025	Bone marrow aspiration & biopsy	Consistent with CML-CP	Diagnostic confirmation
10/04/2025	Final diagnosis	CML - Chronic phase	Disease confirmed; patient counselled
11/04/2025	Treatment initiation	—	*Hingwashtak Churna + Navayas Loha + Ashwattha Kwath* commenced
20/05/2025	CBC - 1st Follow-up	Hb: 14.0 g/dl; WBC: 48,110/µL; Plt: 8.54 lakh/µL	Rapid Hb correction; transient leucocytosis and thrombocytosis (treatment-phase mobilisation). *Hingwashtak Churna* stopped
26/05/2025	RT-PCR BCR-ABL (Baseline Molecular)	BCR-ABL IS: 9.701%	Baseline molecular burden established. *Vayasthapan Mahakashaya* added
10/06/2025	CBC - 2nd Follow-up; USG	Hb: 12.5 g/dl; WBC: 17,110/µL; Plt: 5.63 lakh/µL; Spleen - normal	Declining leucocyte count indicating haematological response. *Navayas Loha* stopped
11/08/2025	CBC - 3rd Follow-up	Hb: 13.8 g/dl; WBC: 6,640/µL; Plt: 5.63 lakh/µL	Near-normalisation of all cell line counts
28/11/2025	CBC - 4th Follow-up	Hb: 13.1 g/dl; WBC: 8,700/µL; Plt: 2.45 lakh/µL	Complete haematological remission achieved

01/01/2026	CBC - 5th Follow-up	Hb: 14.8 g/dl; WBC: 8,700/µL; Plt: 1.85 lakh/µL	Sustained haematological remission
04/02/2026	RT-PCR BCR-ABL - 6th Follow-up	BCR-ABL IS: 0.101%	Substantial molecular response achieved (96-fold reduction from baseline); approaching MMR threshold (≤0.1%)

RESULTS AND CLINICAL OUTCOMES

Molecular Response

The primary outcome measure was achievement of major molecular response (MMR), defined as BCR-ABL IS ≤0.1%. On 04 February 2026 (approximately 10 months into the intervention), RT-PCR analysis demonstrated BCR-ABL IS of 0.101%-representing a 96-fold reduction from the baseline value of 9.701% (26 May 2025). This result closely approaches but marginally exceeds the conventional MMR threshold of ≤0.1%, constituting a substantial molecular response and indicating robust suppression of leukemic clone burden through standalone Ayurvedic intervention.

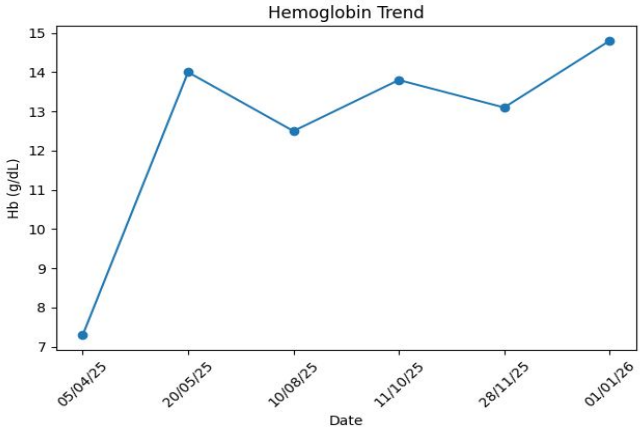


Figure 2: Serial Haemoglobin (Hb) Trend - April 2025 to January 2026

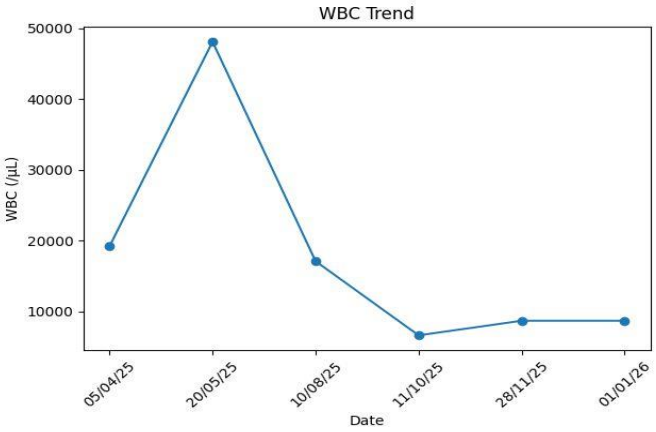


Figure 3: Serial WBC Count Trend - April 2025 to January 2026

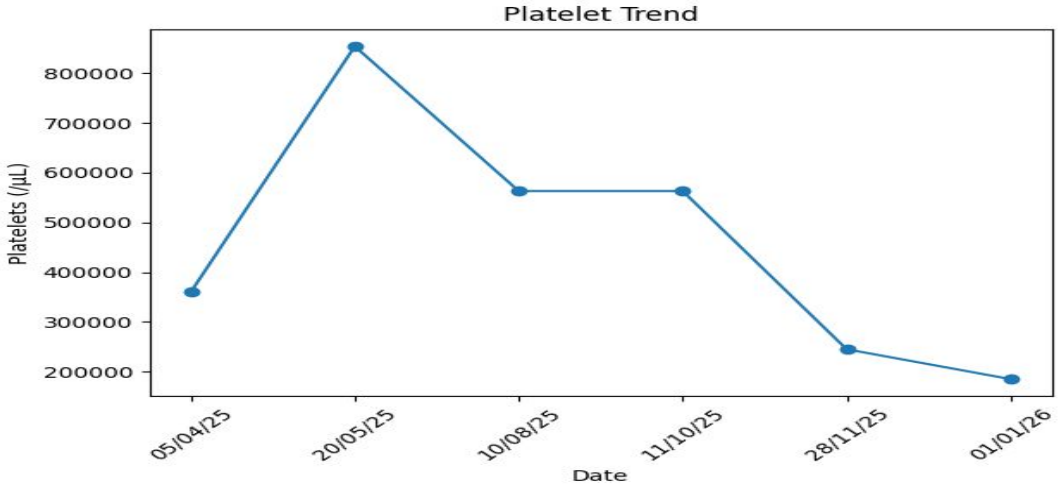


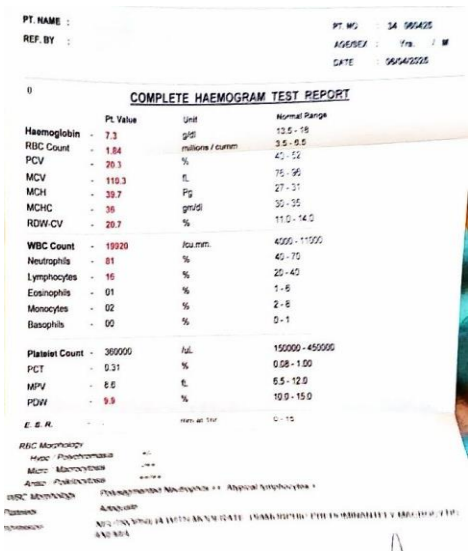
Figure 4: Serial Platelet Count Trend - April 2025 to January 2026

Haematological Remission and Normalisation of Blood Parameters

Complete haematological remission was achieved by the fourth month of intervention (28 November 2025):

- Total Leucocyte Count: Progressive reduction from 19,220/µL (baseline) to 8,700/µL (within normal range: 4,500–11,000/µL), maintained at final assessment.
- Platelet Count: Normalised from 3.6 lakh/µL (360,000/µL) at baseline to 1.85 lakh/µL (185,000/µL) at final follow-up, indicating restored megakaryopoiesis.

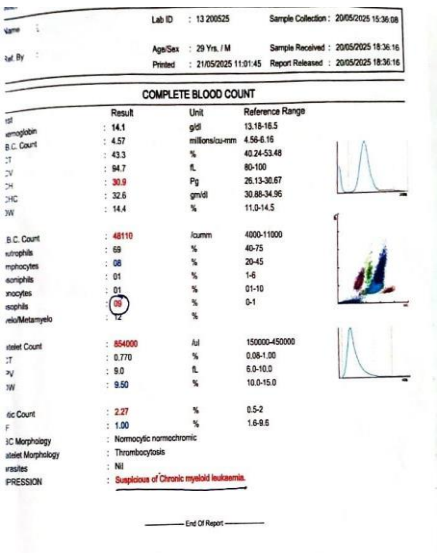
- Haemoglobin: Corrected from 7.3g/dl (severe anaemia) to 14.8g/dl by month 5 (01 January 2026), exceeding the lower normal limit for adult males (13.5g/dl). This was achieved without iron supplementation beyond the initial *Navayas Loha* phase.



COMPLETE HAEMOGRAM TEST REPORT

	Pt. Value	Unit	Normal Range
Haemoglobin	7.3	g/dl	13.5 - 18
RBC Count	1.84	millions / cumm	3.5 - 6.5
PCV	20.3	%	40 - 52
MCV	110.3	fL	76 - 96
MCH	39.7	Pg	27 - 31
MCHC	36	gm/dl	30 - 35
RDW-CV	20.7	%	11.0 - 14.0
WBC Count	19920	/cu.mm.	4000 - 11000
Neutrophils	81	%	40 - 70
Lymphocytes	16	%	20 - 40
Eosinophils	01	%	1 - 6
Monocytes	02	%	2 - 8
Basophils	00	%	0 - 1
Platelet Count	369000	/uL	150000 - 450000
PCT	0.31	%	0.08 - 1.00
MPV	8.6	fL	6.5 - 12.0
PDW	9.9	%	10.0 - 15.0
E. S. R.		mm at 1hr	0 - 15

RBC Morphology
Hypo / Polychromasia
Micro / Macrocytosis
Aniso / Poikilocytosis

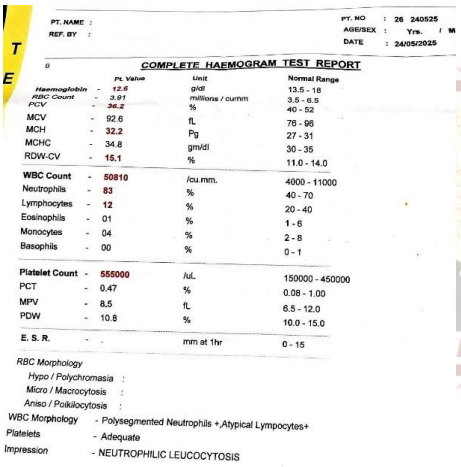


COMPLETE BLOOD COUNT

	Result	Unit	Reference Range
emoglobin	14.1	g/dl	13.18-16.5
B.C. Count	4.57	millions/cu-mm	4.56-6.16
T	43.3	%	40.24-53.48
V	94.7	fL	80-100
H	30.9	Pg	26.13-36.67
HC	32.6	gm/dl	30.88-34.96
W	14.4	%	11.0-14.5
B.C. Count	48110	/cumm	4000-11000
utrophils	69	%	40-75
mphocytes	08	%	20-45
osinophils	01	%	1-6
nocytes	01	%	01-10
sophils	00	%	0-1
elia/Metamyelo	12	%	
atelet Count	854000	/ul	150000-450000
T	0.770	%	0.08-1.00
V	9.0	fL	6.0-10.0
W	9.50	%	10.0-15.0
sic Count	2.27	%	0.5-2
F	1.00	%	1.6-9.5
C Morphology	Normocytic normochromic		
atelet Morphology	Thrombocytosis		
rasites	Nil		
PRESSION	Suspicious of Chronic myeloid leukaemia.		

End Of Report

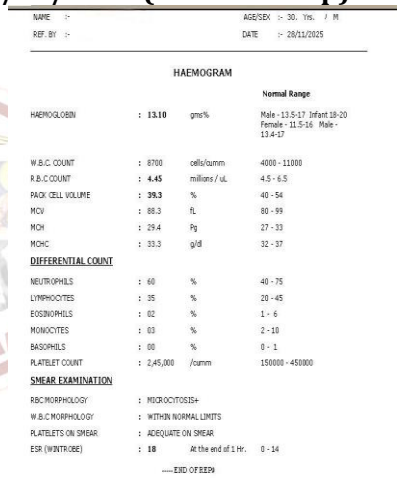
Figure 5 (Left): CBC Report — 05/04/2025 (Baseline)
Figure 6 (Right): CBC Report — 20/05/2025 (1st Follow-up)



COMPLETE HAEMOGRAM TEST REPORT

	Pt. Value	Unit	Normal Range
Haemoglobin	12.6	g/dl	13.5 - 18
RBC Count	3.91	millions / cumm	3.5 - 6.5
PCV	36.2	%	40 - 52
MCV	92.6	fL	76 - 96
MCH	32.2	Pg	27 - 31
MCHC	34.8	gm/dl	30 - 35
RDW-CV	15.1	%	11.0 - 14.0
WBC Count	50810	/cu.mm.	4000 - 11000
Neutrophils	83	%	40 - 70
Lymphocytes	12	%	20 - 40
Eosinophils	01	%	1 - 6
Monocytes	04	%	2 - 8
Basophils	00	%	0 - 1
Platelet Count	555000	/uL	150000 - 450000
PCT	0.47	%	0.08 - 1.00
MPV	8.5	fL	6.5 - 12.0
PDW	10.8	%	10.0 - 15.0
E. S. R.		mm at 1hr	0 - 15

RBC Morphology
Hypo / Polychromasia
Micro / Macrocytosis
Aniso / Poikilocytosis
WBC Morphology - Polysegmented Neutrophils +,Atypical Lympocytes+
Platelets - Adequate
Impression - NEUTROPHILIC LEUCOCYTOSIS

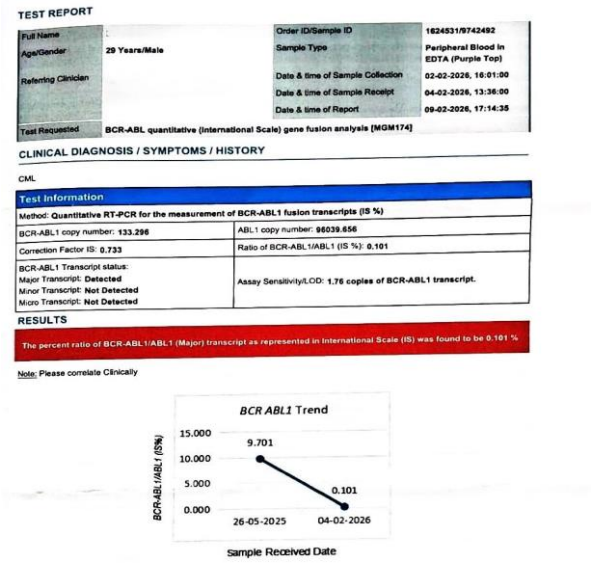


HAEMOGRAM

			Normal Range
HAEMOGLOBIN	13.10	gms%	Male - 13.5-17 Infant 18-20 Female - 11.5-16 Male - 13.4-17
W.B.C. COUNT	8700	cells/cumm	4000 - 11000
R.B.C COUNT	4.45	millions / uL	4.5 - 6.5
PACK CELL VOLUME	39.3	%	40 - 54
MCV	88.3	fL	80 - 99
MCH	29.4	Pg	27 - 33
MCHC	33.3	g/dl	32 - 37
DIFFERENTIAL COUNT			
NEUTROPHILS	60	%	40 - 75
LYMPHOCYTES	35	%	20 - 45
EOSINOPHILS	02	%	1 - 6
MONOCYTES	03	%	2 - 10
BASOPHILS	00	%	0 - 1
PLATELET COUNT	2,45,000	/cumm	150000 - 450000
SMEAR EXAMINATION			
RBC MORPHOLOGY	MICROCYTOSIS+		
W.B.C MORPHOLOGY	WITHIN NORMAL LIMITS		
PLATELETS ON SMEAR	ADEQUATE ON SMEAR		
ESR (WINTROBE)	18	At the end of 1 Hr.	0 - 14

-----END OF REPO

Figure 7 (Left): CBC Report — 24/05/2025
Figure 8 (Right): CBC Report — 28/11/2025 (Haematological Remission)



TEST REPORT

Full Name		Order ID/Sample ID	1624531/9742492
Age/Gender	29 Years/Male	Sample Type	Peripheral Blood in EDTA (Purple Top)
Referring Clinician		Date & time of Sample Collection	02-02-2026, 16:01:00
		Date & time of Sample Receipt	04-02-2026, 13:36:00
		Date & time of Report	09-02-2026, 17:14:35
Test Requested	BCR-ABL quantitative (International Scale) gene fusion analysis [MGM174]		

CLINICAL DIAGNOSIS / SYMPTOMS / HISTORY

CML

Test Information

Method: Quantitative RT-PCR for the measurement of BCR-ABL1 fusion transcripts (IS %)	
BCR-ABL1 copy number: 133.296	ABL1 copy number: 98039.656
Correction Factor IS: 0.733	Ratio of BCR-ABL1/ABL1 (IS %): 0.101
BCR-ABL1 Transcript status: Major Transcript: Detected Minor Transcript: Not Detected Micro Transcript: Not Detected	Assay Sensitivity/LOD: 1.76 copies of BCR-ABL1 transcript.

RESULTS

The percent ratio of BCR-ABL1/ABL1 (Major) transcript as represented in International Scale (IS) was found to be 0.101 %

Note: Please correlate Clinically

Figure 9: RT-PCR BCR-ABL Report — 04/02/2026 (BCR-ABL IS: 0.101%)

Symptomatic Improvement and Quality of Life

- Fatigue: Severe fatigue (8/10 NRS) resolved progressively; patient returned to full professional duties by month four.
- Constitutional symptoms: Low-grade fever, chills, abdominal fullness, and early satiety resolved within the first two months of therapy.
- Splenomegaly: Complete regression of splenic enlargement confirmed on follow-up USG (10 June 2025: normal study).
- Pallor: Clinical pallor normalised by approximately month three, consistent with Hb restoration.
- Night sweating: Completely resolved by month two.
- Weight gain: Patient gained 8kg by final assessment (52kg → 60kg), reflecting excellent nutritional restoration and anabolic recovery.

Adverse Event Profile

No adverse events were documented throughout the 12-month intervention period, as assessed by monthly LFT, RFT, and CTCAE v5.0 grading. This contrasts markedly with the Grade 2 fatigue and myalgia experienced during the preceding two-month imatinib course.

DISCUSSION

CML is a clonal myeloproliferative neoplasm driven by constitutively active BCR-ABL oncokinase, resulting in unchecked myeloid proliferation, impaired apoptosis, and progressive disruption of normal haematopoiesis.[11] Despite the transformative success of TKIs, incomplete immune restoration, long-term toxicity, and the challenge of sustained treatment adherence necessitate exploration of complementary therapeutic approaches.

From an Ayurvedic perspective, CML mirrors *Vatarakta* with *Gambheera dhatu* involvement, characterised by *Rakta dushti, Vata prakopa*, and progressive engagement of *Majja dhatu.* The chronicity and systemic nature of the disease further indicate *Dhatu kshaya* and *Ojas* impairment, forming the rationale for combining *Rakta-prasadana* and Rasayana-based interventions.

Role of *Ashwattha Kwath* (Ficus religiosa Decoction)

Ashwattha (*Ficus religiosa*), administered as Kashaya, exhibits pharmacodynamic properties aligned with *Kashaya–Madhura rasa, Sheeta veerya*, and *Katu vipaka,* enabling *Rakta shodhana, Pitta shamana*, and *Stambhana* effects. Its classical indication in *Vatarakta* is unequivocal, as documented in Charaka Samhita Chikitsa Sthana:

बोधवृक्षकषायं तु प्रपिबेन्मधुना सह।
वातरक्तं जयत्याशु त्रिदोषमपि दारुणम्॥[12]

"*Ashwattha Kashaya*, when administered with honey, rapidly cures even *Tridoshaja Vatarakta* of severe nature." This classical prescription maps precisely onto the observed clinical intervention.

Its classical indication in *Pleeha vriddhi* (splenic enlargement) further supports its role in ameliorating hypersplenism-like states observed in CML.[13] Phytochemical analyses have demonstrated the presence of flavonoids (quercetin, rutin), tannins, and polyphenols with well-established antioxidant, anti-inflammatory, and anti-proliferative properties.[6] These bioactives may modulate the bone marrow microenvironment by: (i) reducing oxidative stress, a recognised contributor to genomic instability in leukemic cells; (ii) suppressing NF-κB-mediated inflammatory cascades involved in clonal expansion;[7] and (iii) inducing apoptosis in myeloid precursors via mitochondrial and death receptor pathways.[18] Thus, *Ashwattha Kashaya* may be conceptualised as a *Rakta*-modulatory agent with microenvironmental regulatory effects, rather than a direct cytotoxic intervention.

Role of *Vayasthapan Mahakashaya*

Vayasthapan Mahakashaya, as described in Charaka Samhita Sutra Sthana 5:

अमृताऽभयाधात्रीमुक्ताश्चेताजीवन्त्यतिरसामण्डूकपर्णीस्थिरापुनर्नवा
इति दशेमानि वयःस्थापनानि भवन्ति, इति पञ्चकः कषायवर्गः॥[14]

This *Rasayana* formulation- comprising *Amrita (Tinospora cordifolia), Abhaya (Terminalia chebula), Dhatri (Emblica officinalis), Mukta (Sphaeranthus indicus), Shweta (Convolvulus pluricaulis), Jivanti (Leptadenia reticulata), Atirasa (Asparagus racemosus), Mandukparni (Centella asiatica), Sthira (Desmodium gangeticum),* and *Punarnava (Boerhavia diffusa)*- addresses the disease substrate *(Kshetra)* rather than isolated pathological manifestations. *Rasayana* therapy exerts immunomodulatory effects through cytokine network regulation and enhancement of innate and adaptive host defence mechanisms,[15] antioxidant activity reducing cumulative cellular damage, and adaptogenic influence improving resilience to chronic disease stressors.[16]

In the context of CML, where malignant transformation originates at haematopoietic stem cell level, *Rasayana* therapy may: (i) support physiological haematopoiesis; (ii) enhance bone marrow microenvironment stability;[17] and (iii) improve immune surveillance against aberrant clones. The *Ojas* augmentation effect further explains the documented reversal of cancer-related cachexia and fatigue, the 8 kg weight gain, and the restoration of functional capacity observed in this patient.

Synergistic Therapeutic Perspective and *Samprapti-Vighatana*

The combination of *Ashwattha Kwath* and *Vayasthapan Mahakashaya* reflects a dual-modality *Samprapti-vighatana* approach, integrating:

- *Rakta*-level correction- Targeting the disease expression (abnormal myeloid proliferation, *Rakta dushti*).
- *Dhatu*-level rejuvenation- Addressing the disease substrate (depleted *Ojas, Majja dhatu kshaya*, immune compromise).

Madhu (honey) serves as *Yogavahi*- a vehicle enhancing bioavailability of *Ashwattha* phytoconstituents, consistent with classical Ayurvedic pharmacology and relevant to the clinical synergy observed. Quercetin, the principal flavonoid in *Ashwattha*, exhibits anti-proliferative activity against BCR-ABL+ cells via NF-κB inhibition and promotion of mitochondrial apoptotic pathways-[18] a mechanism directly relevant to CML pathobiology. The clinical utility of *Ashwattha Kwath* in *Vatarakta* management has also been demonstrated in observational clinical studies.[19] This *Samprapti-vighatana* approach addresses CML not merely at the level of molecular oncology but also at the levels of systemic inflammation, immune function, metabolic integrity, and constitutional vitality-resonating with emerging oncological paradigms emphasising tumour microenvironment regulation, host-directed therapy, and integration of anti-inflammatory and immunomodulatory strategies.

CONCLUSION

This case report documents a remarkable and reproducible therapeutic response in a chronic-phase CML patient managed with classical Ayurvedic intervention. The treatment protocol- employing *Ashwattha Kwath* (Ficus religiosa decoction) for *Rakta-prasadana* and *Vayasthapan Mahakashaya* for constitutional *Rasayana* restoration- achieved:

- Complete haematological remission (WBC 8,700/μL, Hb 14.8 g/dl, platelets 1.85 lakh/μL) by month four.
- Substantial molecular response (BCR-ABL IS: 0.101% at 10 months), representing a 96-fold reduction from baseline (9.701%), approaching the major molecular response threshold.
- Complete resolution of constitutional symptoms, splenomegaly, and severe anaemia.
- Documented 8kg weight gain and restoration of full functional capacity.
- Outstanding safety profile with zero adverse events over 12 months.

The magnitude, consistency, and clinical significance of these outcomes- achieved without concurrent tyrosine kinase inhibitor therapy- are exceptional and warrant systematic investigation. The concept of *Samprapti-vighatana* employed here, addressing CML through coordinated disruption of disease pathogenesis at *Rakta, Dhatu*, and *Ojas* levels, represents a fundamentally different paradigm from conventional targeted therapy and may explain the sustained therapeutic effect observed.

While inherent limitations of single-case observation preclude definitive conclusions, the findings strongly justify rigorous randomised controlled trials comparing Ayurvedic approaches combining *Ashwattha*-based *Rakta-prasadana* with *Rasayana* support- both as standalone and as adjunctive strategies with reduced-dose TKI therapy- in chronic-phase CML. Such investigations are warranted to systematically evaluate this promising integrative oncological paradigm.

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Patient Consent: Written informed consent obtained.

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