BRIDGING AYURVEDA AND HEMATOLOGY
Adjunctive Use of *Raktamrut* Vati in a 14-Year-Old Boy with Severe Hemophilia A
A CARE-Aligned Case Report with Clinical Outcome Analysis

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Abstract

Background: Hemophilia A is an inherited deficiency of coagulation factor VIII that predisposes affected individuals to prolonged bleeding, spontaneous hemorrhage, recurrent hemarthrosis, chronic pain, and progressive joint damage. Severe disease is defined by factor VIII activity below 1% of normal and generally requires lifelong specialist-directed prophylactic or episodic hemostatic treatment. This report describes the clinical course of a 14-year-old boy with severe Hemophilia A who received Raktamrut Vati as an adjunct to standard factor replacement and supportive care.

Case presentation: The patient had recurrent ankle and knee pain and swelling, muscle hematomas, hemarthrosis, epistaxis, bruising, prolonged bleeding after minor wounds, and a family history consistent with X-linked inheritance. Baseline activated partial thromboplastin time was 135 seconds, while factor VIII activity remained below 1%. Raktamrut Vati was administered at a dose of two tablets twice daily for six months without discontinuing access to factor VIII concentrate or other medically indicated treatment.

Outcomes: During follow-up, most recorded bleeding categories decreased, including muscle bleeding, hemarthrosis, spontaneous bleeding, and bruising. Hospital admissions, factor administrations, analgesic use, tranexamic acid use, pain score, movement restriction, fatigue, and school absenteeism also declined, while self-rated muscle strength and quality of life improved. One external-orifice bleeding episode was recorded at the final assessment despite a baseline value of zero, and this data point requires source verification. Factor VIII activity remained below 1%, and aPTT remained markedly prolonged despite decreasing from 135 to 120 seconds.

Conclusion: The observed temporal association suggests that Raktamrut Vati may warrant further investigation as a supportive adjunct in carefully monitored patients receiving standard hemophilia care. A single uncontrolled case cannot establish efficacy, causality, or statistical significance. Raktamrut Vati must not be considered a replacement for factor VIII concentrate, non-factor prophylaxis, emergency hemostatic treatment, or specialist follow-up.

Keywords: Hemophilia A; Factor VIII deficiency; Raktamrut Vati; hemarthrosis; adjunctive Ayurveda; bleeding episodes; quality of life; case report.

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1. Introduction

Hemophilia A is a congenital bleeding disorder caused by quantitative or qualitative deficiency of coagulation factor VIII.[4] The disorder is usually inherited in an X-linked pattern and therefore predominantly affects males, although symptomatic females can occur because of lyonization, homozygosity, Turner syndrome, or other genetic mechanisms.[1] Factor VIII functions as a cofactor in the intrinsic tenase complex, and severe deficiency impairs thrombin generation and stable fibrin clot formation.[6]

The clinical phenotype ranges from bleeding only after major trauma or surgery in mild disease to recurrent spontaneous joint and muscle bleeding in severe disease.[5] Repeated hemarthrosis can initiate synovial inflammation, iron deposition, cartilage injury, muscle weakness, restricted movement, and progressive hemophilic arthropathy.[8] These complications can interfere with education, employment, physical activity, family functioning, and psychosocial well-being.[1]

Contemporary management prioritizes prevention of bleeding through regular prophylaxis with factor

concentrates or approved non-factor therapies.[1] Prompt treatment of breakthrough bleeding remains essential because delayed hemostasis increases the risk of tissue damage and disability.[1] Multidisciplinary care generally includes hematology, nursing, physiotherapy, dental care, pain management, laboratory monitoring, and psychosocial support.[1]

The present case concerns a 14-year-old boy with severe Hemophilia A and recurrent bleeding manifestations who received Raktamrut Vati for six months as an adjunct to standard care. The report focuses on longitudinal changes in bleeding frequency, hospital utilization, factor requirement, supportive medication use, pain, joint movement, muscle strength, fatigue, school attendance, and quality of life. The purpose is descriptive rather than confirmatory, because a single case cannot distinguish treatment effect from natural variation, changes in activity, concurrent medical care, reporting effects, or regression to the mean.

2. Epidemiology and Public-Health Burden

A registry-based meta-analysis estimated the prevalence at birth of Hemophilia A at approximately 24.6 cases per 100,000 males, with severe Hemophilia A accounting for a substantial proportion of affected individuals.[2] Reported prevalence is lower in many resource-limited settings because of underdiagnosis, early mortality, limited access to diagnostic laboratories, and incomplete national registration.[2] The number of identified patients therefore does not necessarily represent the true disease burden.[2]

Indian data have documented substantial direct and indirect costs related to factor use, hospitalization, travel, disability, school absence, and loss of family income.[3] The burden is amplified when patients receive treatment only after bleeding begins rather than through sustained prophylaxis.[3] Geographic inequalities in access to comprehensive hemophilia treatment remain clinically important in India.[3]

Severe Hemophilia A commonly becomes apparent in early childhood through bruising, prolonged bleeding after procedures, or spontaneous musculoskeletal bleeding.[1] Without effective prevention, recurrent hemarthrosis may produce irreversible joint damage during childhood or adolescence.[7] The Joint Outcome Study demonstrated that prophylaxis initiated in young boys preserved joint structure more effectively than episodic factor treatment.[7]

The case described here illustrates several aspects of the burden reported in the literature. At baseline, the patient experienced frequent muscle bleeding and hemarthrosis, required repeated factor administration, missed approximately 15 school days per month, and had substantial pain, fatigue, movement restriction, and anxiety about injury. These outcomes are relevant because successful hemophilia care should be assessed not only by laboratory measures but also by bleed prevention, joint preservation, participation, and quality of life.

3. Molecular Basis and Pathophysiology

The F8 gene is located on the X chromosome and encodes coagulation factor VIII.[4] Pathogenic F8 variants may reduce synthesis, secretion, stability, or functional activity of the factor VIII protein.[4] Because males usually have one X chromosome, a pathogenic variant can produce the full disease phenotype.[4]

In normal coagulation, activated factor VIII forms the intrinsic tenase complex with activated factor IX on phospholipid surfaces.[6] This complex accelerates factor X activation and supports the thrombin burst required for a stable fibrin clot.[6] Severe factor VIII deficiency therefore causes inefficient thrombin generation even when platelet number and the extrinsic pathway are otherwise intact.[6]

The laboratory pattern typically includes a prolonged aPTT with a normal PT, although the degree of aPTT prolongation depends on reagents and assay conditions.[1] Definitive classification requires a specific factor VIII activity assay and evaluation for inhibitors when clinically indicated.[1] Severe Hemophilia A is conventionally defined by factor VIII activity below 1 IU/dL or below 1% of normal.[5]

Blood within a joint triggers synovial iron deposition, cytokine release, angiogenesis, cartilage degradation, and recurrent susceptibility to bleeding.[8] This self-perpetuating process explains why a target joint may continue to bleed even when individual episodes are treated.[8] Preventing joint bleeding is therefore a central therapeutic objective rather than merely controlling pain after bleeding has occurred.[1]

In the present case, factor VIII activity remained below 1% before and after the six-month observation period. The aPTT decreased from 135 seconds to 120 seconds but remained markedly prolonged relative to the laboratory reference range. These findings indicate that the congenital coagulation deficiency persisted. Any clinical improvement should therefore be interpreted as symptomatic or supportive rather than as correction of the underlying factor VIII defect.

4. Clinical Manifestations and Natural History

Typical manifestations of severe Hemophilia A include spontaneous hemarthrosis, deep muscle hematoma, prolonged bleeding after trauma, and excessive bleeding after surgery or dental procedures.[1] Mucosal bleeding can occur, but recurrent deep-tissue and joint bleeding is more characteristic of coagulation-factor deficiency than of primary platelet disorders.[6] Life-threatening sites include the intracranial, neck, throat, retroperitoneal, and gastrointestinal compartments.[1]

Hemarthrosis most frequently affects large synovial joints such as the knees, ankles, and elbows.[1] Acute joint bleeding may produce warmth, pain, swelling, guarded posture, and reduced range of motion.[12] Repeated episodes can lead to chronic synovitis, fixed flexion deformity, muscle wasting, and functional limitation.[13]

Pain in hemophilia can be acute during a bleed or chronic because of established arthropathy.[14] Analgesic selection requires caution because aspirin and many non-selective non-steroidal anti-inflammatory drugs can impair platelet function or increase gastrointestinal bleeding risk.[1] Physiotherapy should be coordinated with hemostatic treatment to protect the bleeding joint while minimizing deconditioning.[1]

The supplied history included recurrent painful swelling of the knees and ankles from approximately five years of age, muscle hematomas, hemarthrosis, epistaxis, bruising, prolonged bleeding after minor wounds, and gastrointestinal bleeding. The patient had previously required surgical hemostasis for molar extraction. Participation in outdoor activities was restricted, and mild social withdrawal and anxiety about injury were reported. This pattern is compatible with the functional and psychosocial burden expected in severe disease, although each manifestation should be confirmed against original medical records.

5. Diagnosis and Severity Classification

The diagnostic evaluation of suspected hemophilia begins with a detailed personal and family bleeding history, physical examination, complete blood count, PT, aPTT, and specific coagulation-factor assays.[1] A prolonged isolated aPTT suggests a deficiency or inhibitor affecting the intrinsic pathway, but it is not specific for Hemophilia A.[1] Mixing studies, lupus anticoagulant testing, von Willebrand factor studies, and inhibitor assays may be needed according to the clinical context.[1]

Factor VIII activity below 1% defines severe disease, activity from 1% to 5% defines moderate disease, and activity above 5% to below 40% defines mild disease.[5] Severity classification predicts bleeding tendency at a population level but does not fully explain individual variation in bleeding phenotype.[5] Genetic testing can confirm the causative F8 variant and facilitate carrier detection, counseling, and prenatal or preimplantation options when appropriate.[1]

In this case, the diagnosis of severe Hemophilia A was supported by a markedly prolonged aPTT, normal-range PT, factor VIII activity below 1%, recurrent spontaneous musculoskeletal bleeding, and a positive maternal family history. The maternal uncle reportedly had hemophilia, which is consistent with X-linked transmission. The original document did not provide inhibitor testing, von Willebrand factor results, genetic analysis, blood group, platelet count, or complete mixing-study data. These omissions should be acknowledged rather than assumed to be normal.

Table 1. Diagnostic synthesis

Diagnostic domain	Case finding	Interpretation
Clinical phenotype	Recurrent hemarthrosis, muscle hematoma, bruising, epistaxis, prolonged bleeding	Compatible with severe inherited bleeding disorder
Family history	Maternal uncle with hemophilia	Supports X-linked inheritance
aPTT	135 seconds at baseline	Markedly prolonged
PT	13.8 seconds at baseline	Within supplied reference range
Factor VIII activity	<1% before and after treatment	Severe Hemophilia A; no documented correction
Inhibitor status	Not provided	Must be documented if tested
Genetic testing	Not provided	Recommended for definitive variant characterization and counseling

6. Evidence-Based Standard Management

The standard of care for severe Hemophilia A is regular prophylaxis that prevents spontaneous bleeding and preserves musculoskeletal health.[1] Prophylaxis may use conventional half-life factor VIII, extended half-life factor VIII, or an approved non-factor therapy selected according to age, inhibitor status, venous access, availability, and patient preference.[1] Episodic factor treatment alone is less effective than prophylaxis for preventing long-term joint damage.[7]

Factor VIII dosing is individualized according to body weight, desired factor increment, bleeding site, pharmacokinetics, and clinical response.[1] Patients should be trained to recognize early symptoms and administer prescribed treatment promptly when home therapy is available.[1] Inhibitor screening is required at clinically appropriate intervals and whenever the response to factor therapy becomes unexpectedly poor.[1]

Emicizumab provides subcutaneous prophylaxis for Hemophilia A with or without inhibitors and has reduced treated bleeding rates in phase 3 trials.[10] Management of breakthrough bleeding during emicizumab prophylaxis requires product-specific planning because some bypassing-agent combinations can cause serious thrombotic complications.[1] Choice of therapy must remain under the direction of a hemophilia treatment center or qualified hematologist.[1]

The source record described recombinant or plasma-derived factor VIII concentrate at 500 IU as needed, with a baseline frequency of three to four administrations per month. The case did not document a regular prophylaxis schedule, inhibitor status, pharmacokinetic assessment, or a formal home-treatment plan. The observed reduction in factor administrations should therefore be reported as a utilization outcome during the observation period, not as evidence that factor VIII was no longer medically required.

7. Musculoskeletal, Pain, Rehabilitation, and Psychosocial Care

Comprehensive hemophilia care includes regular assessment of target joints, range of motion, muscle strength, gait, physical activity, and chronic pain.[1] Early physiotherapy after adequate hemostasis helps restore movement and reduce the cycle of pain, guarding, and weakness.[1] Long-term exercise programs should emphasize safe strength, balance, flexibility, cardiovascular fitness, and injury prevention.[1]

Pain assessment should distinguish acute bleeding pain from chronic arthropathy because management priorities differ.[14] Acute pain with swelling and reduced motion should be treated as a possible bleed until clinically assessed.[1] Analgesic plans should consider bleeding risk,

gastrointestinal risk, renal function, age, and concurrent medicines.[1]

Children and adolescents with hemophilia may experience fear of injury, reduced peer participation, school absence, dependence on caregivers, and uncertainty about future health.[1] Psychosocial support and school coordination are therefore integral components of comprehensive care.[1] Patient-reported outcomes complement bleed counts because they capture pain, fatigue, confidence, participation, and perceived well-being.[16]

In the present record, pain severity fell from 7 to 1 on a 0-10 VAS, movement restriction fell from 7 to 0, self-rated muscle strength rose from 4 to 8, fatigue fell from 7 to 1, and missed school days fell from 15 to 0 per month. These changes are clinically relevant to the patient and family, but the scales were self-rated and no validated hemophilia-specific quality-of-life instrument, standardized joint score, physiotherapy examination, or blinded assessor was documented. Future studies should use validated measures such as a hemophilia joint health score, functional independence assessment, and age-appropriate quality-of-life questionnaire.

8. Ayurvedic Conceptual Framework and Integrative Rationale

Classical Ayurvedic texts describe disorders of excessive bleeding within conceptual frameworks such as Raktapitta and discuss measures intended to restrain bleeding and support tissue balance.[28] These traditional concepts are historically important but are not biologically equivalent to the modern diagnosis of factor VIII deficiency.[28] Hemophilia A should therefore retain its hematologic diagnosis and evidence-based management even when an Ayurvedic adjunct is used.[1]

Polyherbal formulations are commonly used in Ayurveda, where combinations are selected according to traditional pharmacologic attributes and therapeutic objectives.[27] A traditional rationale does not by itself establish clinical efficacy, dose standardization, pharmacokinetics, or safety in congenital bleeding disorders.[25] Clinical claims require reproducible product characterization and appropriately designed human studies.[25]

Herbal products can vary in botanical identity, extraction method, excipients, contaminants, and batch-to-batch constituent levels.[24] Quality control should therefore document authenticated ingredients, manufacturing standards, batch number, microbial limits, heavy metals, pesticides, and relevant chemical markers.[24] Published analyses of some Ayurvedic products have identified potentially toxic metals, which reinforces the need for verified sourcing and testing rather than implying that all products are unsafe.[26]

The supplied case file did not include the complete composition of Raktamrut Vati, the manufacturer, batch number, tablet weight, manufacturing license, certificate of analysis, quality-control results, or pharmacognostic standardization. Consequently, this manuscript does not attribute the observed outcomes to any specific herb or mechanism. The proposed role is limited to an adjunctive, hypothesis-generating observation. Before publication or further study, the intervention must be described in sufficient detail to permit reproducibility and independent safety assessment.

9. Case-Report Method and Ethical Framework

This manuscript was structured according to the CARE framework for transparent reporting of clinical case reports.[20] CARE recommends clear reporting of patient information, clinical findings, timeline, diagnostic assessment, intervention, follow-up, outcomes, patient perspective, and informed consent.[20] Detailed intervention reporting is also consistent with the TIDieR principle that a treatment should be described sufficiently for replication.[22]

Design: Single-patient descriptive case report based on the clinical information, laboratory values, treatment details, and outcome table supplied by the investigator. No control patient, randomization, masking, inferential statistical analysis, or formal causality algorithm was used.

Observation period: Six months after initiation of Raktamrut Vati. The source document used the labels baseline, follow-up 1, follow-up 3, and final assessment, but exact visit dates were not consistently provided. The graphs in this expanded report therefore use visit labels rather than calendar dates.

Outcomes: Monthly bleeding categories, hospital admissions, factor use, average factor units per episode, analgesic and tranexamic acid use, bleeding duration, pain VAS, movement restriction, muscle strength, fatigue, school absenteeism, and self-rated quality of life.

Safety: Treatment adherence, tolerability, reported adverse events, complete blood count, liver-function indices, kidney-function indices, lipid profile, and glucose values were reviewed from the supplied table. Abnormal results were not reclassified as treatment-related because temporal association alone is insufficient for causality.

Ethics and consent Written informed consent for publication was obtained from the patient's parent/legal guardian, and age-appropriate assent was obtained from the patient. This study was reviewed and approved by the Institutional Ethics Committee (IEC), Government Ayurveda College & Hospital, Dharashiv, Maharashtra, India, vide Approval Letter No. GACD/SS/6138/2024 dated 11 November 2024, before commencement of the study.Institutional ethics approval or waiver status should be inserted with committee name and approval number.

10. Patient Information and History

Table 2. Patient information

Domain	Information from supplied record
Age and sex	14-year-old male
Occupation	Student
Residence	Anonymized as XYZ
Primary diagnosis	Severe Hemophilia A; factor VIII activity <1%
Main complaints	Bilateral ankle pain and swelling; recurrent knee swelling; muscle hematomas; hemarthrosis; epistaxis; prolonged bleeding from minor wounds;

	bruising; reported gastrointestinal bleeding
Onset and course	Painful knee and ankle swelling since approximately 5 years of age; episodes every 2-3 months in the narrative history
Past procedure	Molar extraction requiring surgical hemostasis
Family history	Maternal uncle with hemophilia; consanguinity reported; paternal grandfather with lung cancer
Psychosocial context	Supportive family; restricted outdoor activity; mild social withdrawal and injury-related anxiety
Previous treatment	Factor VIII concentrate 500 IU as needed; tramadol/paracetamol combination tablet as needed; tranexamic acid 500 mg as needed

The narrative history and the quantitative outcome table are not fully concordant. The history states that painful joint episodes occurred every two to three months, whereas the baseline outcome table records four hemarthroses, four muscle bleeds, four hospital admissions, and four factor requirements per month. This difference may reflect a change in disease activity, different recall windows, or transcription error. The original diaries, prescriptions, factor issue records, and hospital documents should be reconciled before publication.

The record states that pallor, icterus, and edema were present on general examination, while cyanosis was absent. These findings require clinical context because the hemoglobin was within the supplied normal range and bilirubin was mildly elevated. The anatomical site and severity of edema, the basis for the icterus finding, and any related investigations were not documented.

11. Clinical Findings and Timeline

Table 3. Examination findings

Clinical parameter	Finding
Pulse	75 beats/min
Blood pressure	100/80 mmHg
Respiratory rate	18 breaths/min
Temperature	97.02 degrees F
Pallor	Present
Icterus	Present
Cyanosis	Absent
Edema	Present
Musculoskeletal examination	Right and left ankle pain and swelling
Dermatological examination	Bruises over the body

Table 4. Case timeline

Time point	Event or finding
Childhood	First bleeding manifestations reported
Approximately age 5 years onward	Recurrent painful knee and ankle swelling with difficulty walking
Diagnostic phase	Prolonged aPTT and factor VIII activity <1% supported severe Hemophilia A
Before adjunctive treatment	Factor VIII concentrate, analgesic, and tranexamic acid used as needed
Treatment start	Raktamrut Vati initiated at two tablets twice daily
Early follow-up	Reduction in recorded bleeding and pain reported
Intermediate follow-up	Further reduction in medication use, movement restriction, fatigue, and school absence reported
Final follow-up at six months	Most bleeding categories were zero; VAS pain was 1; quality-of-life score was 9; one external-orifice bleeding episode was recorded

Exact dates for treatment initiation, individual bleeding episodes, factor infusions, hospital admissions, and laboratory sampling were not consistently available in the source document. A journal-ready timeline should include calendar dates or clearly defined study weeks. Where retrospective recall was used, the recall period should be stated.

12. Diagnostic Assessment and Laboratory Findings

Table 5. Hematology and liver-function results

Investigation	Baseline	Final	Supplied reference range
Hemoglobin	13.2 g/dL	13.6 g/dL	13-17 g/dL
aPTT	135 s	120 s	25-35 s
PT	13.8 s	14.1 s	11.7-15.1 s
Factor VIII activity	<1%	<1%	50-150%
Total bilirubin	1.25 mg/dL	1.31 mg/dL	0.2-1.0 mg/dL
Direct bilirubin	0.50 mg/dL	0.48 mg/dL	Up to 0.2 mg/dL
Indirect bilirubin	0.75 mg/dL	0.83 mg/dL	Up to 0.8 mg/dL
AST/SGOT	9.4 IU/L	15.7 IU/L	Up to 40 IU/L
ALT/SGPT	14.3 IU/L	14.3 IU/L	Up to 35 IU/L
Total protein	8.35 g/dL	8.61 g/dL	6.0-8.0 g/dL
Albumin	4.59 g/dL	4.78 g/dL	3.5-5.0 g/dL
Globulin	3.76 g/dL	3.83 g/dL	2.5-3.0 g/dL

Table 6. Kidney, lipid, and glucose results

Investigation	Baseline	Final	Supplied reference range
Urea	21.3 mg/dL	20.7 mg/dL	14-40 mg/dL
Creatinine	0.79 mg/dL	0.75 mg/dL	0.7-1.2 mg/dL
Uric acid	6.90 mg/dL	7.01 mg/dL	2.5-6.5 mg/dL
Total cholesterol	180.2 mg/dL	133.0 mg/dL	150-220 mg/dL

Triglycerides	58.6 mg/dL	96.0 mg/dL	25-150 mg/dL
HDL-C	26.7 mg/dL	46.2 mg/dL	Male 35-55 mg/dL
LDL-C	76.2 mg/dL	75.5 mg/dL	Up to 150 mg/dL
VLDL-C	30.68 mg/dL	19.2 mg/dL	20-40 mg/dL
Fasting glucose	96.8 mg/dL	75.0 mg/dL	70-110 mg/dL
Laboratory interpretation: aPTT remained markedly prolonged and factor VIII activity remained <1%. Mild bilirubin and uric acid elevations require clinical review and repeat testing, while HDL-C improved into the supplied reference range. These findings should not be dismissed solely because no subjective adverse reaction was reported.			

13. Therapeutic Intervention

Table 7. Intervention details

Component	Details
Standard hemostatic treatment	Recombinant or plasma-derived factor VIII concentrate, 500 IU as needed; baseline use reported as 3-4 times per month
Supportive conventional medicines	Tranexamic acid 500 mg as needed; tramadol/paracetamol combination as needed
Adjunctive Ayurvedic intervention	Raktamrut Vati
Dose	Two tablets per administration
Frequency	Twice daily
Duration	Six months
Other Ayurvedic medicines	None reported
Dietary regulation	Mentioned in the source narrative, but specific dietary instructions were not provided
Treatment adherence	Reported as good
Tolerability	Reported as well tolerated
Reported adverse drug reactions	None reported by the patient during the observation period

The exact composition and pharmaceutical characteristics of Raktamrut Vati were not provided. For reproducible reporting, the final manuscript should state the botanical identity and plant part of every ingredient, quantity per tablet, extraction or processing method, excipients, tablet weight, manufacturer, batch number, manufacturing license, storage conditions, expiry date, and analytical quality-control results. The time of administration in relation to meals and the procedure used to assess adherence should also be documented.

Clear description of an intervention is necessary so that clinicians and researchers can understand what was actually administered.[22] Herbal-product safety surveillance should include suspected adverse reactions, interactions, contamination, and clinically important laboratory changes.[23] Concurrent standard therapy should be recorded in enough detail to prevent false attribution of outcomes to the adjunct alone.[20]

14. Follow-Up and Outcomes

Table 8. Longitudinal outcome summary

Outcome	Baseline	FU 1	FU 3	Final	Baseline-to-final
Muscle bleeding episodes/month	4	0	0	0	-100%
Hemarthrosis/month	4	0	0	0	-100%
Spontaneous bleeding episodes/month	2	0	0	0	-100%
Bruising episodes/month	2	1	0	0	-100%
External-orifice bleeding/month	0	0	0	1	Not calculable; final increase
Hospital admissions/month	4	0	0	0	-100%
Factor administrations/month	4	0	0	0	-100%
Average factor units/episode	500 IU	0	0	0	No treated episode recorded
Analgesic tablets/month	8	4	2	0	-100%
Tranexamic acid tablets/month	4	2	0	0	-100%
Average bleed duration	7 days	2 days	2 days	0	No final bleed duration recorded
Pain severity, VAS	7	2	2	1	-85.7%
Movement restriction	7	2	1	0	-100%
Muscle strength	4	7	7	8	+100%
Fatigue/tiredness	7	4	3	1	-85.7%
School days missed/month	15	8	4	0	-100%
Self-rated QoL	4	7	7	9	+125%

The data show a broad temporal improvement across most bleeding, healthcare-use, medication-use, pain, functional, and participation outcomes. However, the final

external-orifice bleeding value was one episode per month compared with zero at baseline. This contradicts a statement that every bleeding category decreased and should be checked against the original case record. The complete absence of hospital admissions and factor administrations after baseline is striking and should be corroborated with independent records.

15. Graphical Outcome Analysis I

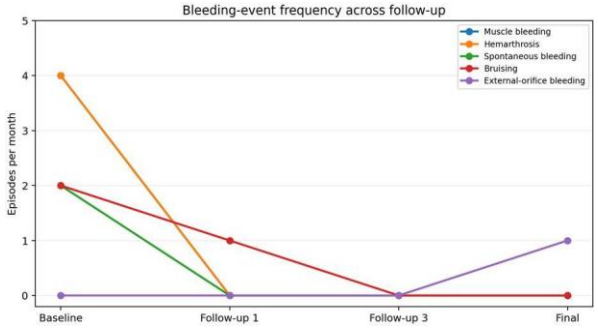

Figure 1. Monthly bleeding-event frequency across recorded visits.

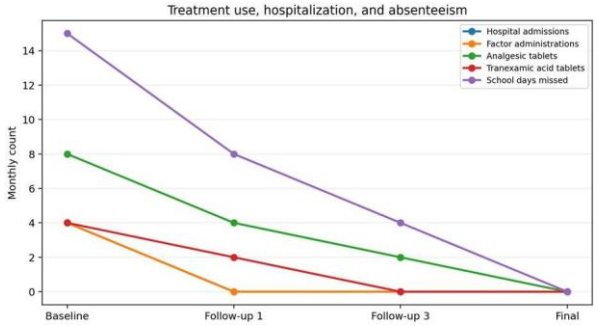

Figure 2. Monthly hospitalization, treatment use, and school absenteeism.

16. Graphical Outcome Analysis II and Study Observations

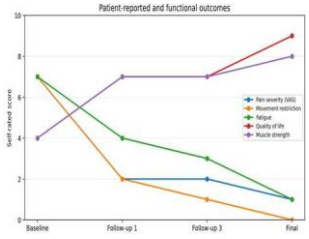

Figure 3. Patient-reported and functional outcomes.

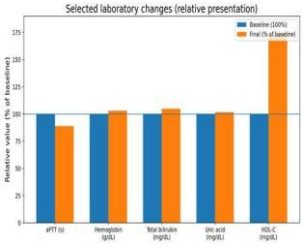

Figure 4. Selected laboratory changes relative to baseline.

Consolidated observations

- Most recorded bleeding categories decreased during follow-up, particularly joint, muscle, spontaneous, and bruising episodes.
- Bleeding-related hospital admissions, factor administrations, analgesic use, and tranexamic acid use decreased in the supplied records.
- Average bleeding duration and swelling-related movement restriction declined over time.
- Pain severity decreased progressively, while self-rated muscle strength and quality of life increased.
- Fatigue, injury-related anxiety, school absenteeism, and caregiver burden were reported to improve.
- One external-orifice bleeding episode was recorded at the final visit and requires verification because the baseline value was zero.
- Factor VIII activity remained <1%, showing persistence of severe Hemophilia A despite clinical improvement.
- No subjective adverse reaction was reported; mild bilirubin and uric acid elevations require clinical follow-up, while HDL-C improved into the supplied reference range.

17. Discussion: Bleeding and Treatment Utilization

The central observation was a marked reduction in recorded musculoskeletal and spontaneous bleeding after initiation of the adjunctive regimen. Muscle bleeds and hemarthroses decreased from four per month at baseline to zero at subsequent recorded visits, while spontaneous bleeds decreased from two per month to zero. Bruising declined more gradually, from two episodes to one and then zero. These trends are clinically important because recurrent joint and muscle bleeding drives pain, loss of motion, hospitalization, and long-term disability.

Prevention of bleeding is the primary objective of severe hemophilia management because recurrent bleeds can produce irreversible musculoskeletal damage.[1] Randomized evidence supports prophylaxis over episodic treatment for preserving joint structure in young children with severe Hemophilia A.[7] The present case does not establish that the adjunctive formulation can reproduce the effect of prophylaxis or replace it.[7]

The recorded frequency of hospital admission and factor administration fell from four per month to zero. The average factor amount per treated episode was 500 IU at baseline, with no treated episodes recorded later. This reduction could represent fewer bleeding events, but it may also reflect altered healthcare access, undocumented home treatment, changes in activity, recall error, or incomplete capture of factor use. Verification against factor-distribution records and hospital documentation is necessary.

Factor VIII remains the direct replacement treatment for Hemophilia A and must be administered promptly for clinically important breakthrough bleeding.[1] Failure to use prescribed hemostatic therapy because symptoms appear improved can expose a patient to preventable morbidity.[1] Any future controlled study should predefine rescue-treatment rules and record every factor infusion prospectively.[20]

The source observations originally stated that all categories of bleeding decreased. The final data table, however, recorded one external-orifice bleeding episode when the baseline value was zero. Scientifically accurate reporting requires preservation of this discordant finding rather than selective emphasis on favorable outcomes. The value should be rechecked, and the type, severity, site, duration, and treatment of the episode should be described.

18. Discussion: Musculoskeletal and Patient-Reported Outcomes

Pain decreased from 7 to 1 on the VAS, movement restriction decreased from 7 to 0, and the average bleeding duration decreased from seven days to no recorded duration

at the final visit. These changes were accompanied by a rise in self-rated muscle strength from 4 to 8. The pattern is internally coherent with fewer joint bleeds and reduced swelling, although no objective joint examination score, imaging, goniometry, dynamometry, or standardized physiotherapy assessment was provided.

Acute joint bleeding causes pain and loss of movement, while recurrent bleeding can produce chronic synovitis and arthropathy.[12] Muscle weakness and altered movement may persist after repeated bleeds even when acute pain has resolved.[13] Standardized musculoskeletal assessment improves comparability across visits and studies.[1]

Fatigue decreased from 7 to 1, school absence declined from 15 days per month to zero, and self-rated quality of life increased from 4 to 9. The patient and family also reported reduced disease-related anxiety and caregiver burden. These outcomes are directly relevant to adolescent development because school participation, autonomy, peer interaction, and confidence can be affected by recurrent bleeding and fear of injury.

Patient-reported outcomes provide information that cannot be inferred from factor levels or bleed counts alone.[16] Quality-of-life instruments should be age appropriate, validated, and administered consistently across time points.[16] A single unvalidated global score is useful descriptively but cannot be compared reliably with published cohorts.[16]

The reduction in analgesic and tranexamic acid use is consistent with reduced symptom burden, but medication counts can be influenced by access, adherence, prescribing changes, and family preference. Future prospective work should use treatment diaries, pill counts, electronic factor logs, and structured adverse-event interviews. Objective joint outcomes should be linked with patient-reported pain and participation measures rather than interpreted in isolation.

19. Safety, Causality, and Data Quality

The patient reportedly tolerated the formulation well and no adverse drug reaction was recorded. Hemoglobin remained stable, creatinine decreased slightly, and AST and ALT remained within the supplied reference ranges. These findings are reassuring but do not establish safety because a single patient and six months of follow-up cannot detect uncommon, delayed, dose-related, or interaction-related adverse effects.

Herbal medicines can cause adverse reactions through intrinsic constituents, contamination, adulteration, variable dosing, or interactions with conventional medicines.[25] Pharmacovigilance should consider symptoms, laboratory abnormalities, product quality, temporal sequence, dechallenge, rechallenge, and alternative explanations.[23] Absence of a reported complaint is not equivalent to absence of a clinically important safety signal.[23]

Selected final laboratory results merit review: total bilirubin was 1.31 mg/dL, direct bilirubin was 0.48 mg/dL, and uric acid was 7.01 mg/dL; HDL-C improved to 46.2 mg/dL. The record also stated that icterus and edema were present on examination. The bilirubin and uric acid findings should be repeated and clinically evaluated. The manuscript should not state unqualified safety until the treating clinician confirms their relevance and excludes laboratory error or unrelated disease.

Causality for benefit is also uncertain. The patient continued to have access to factor VIII, supportive medication, dietary regulation, family care, and medical follow-up. Potential confounders include changes in physical activity, seasonal variation, improved adherence to standard care, avoidance of injury, recall bias, regression to the mean, and selective recording. No challenge-dechallenge-rechallenge sequence, objective biomarker of formulation action, or comparator was available.

Data-quality concerns include mismatch between the narrative episode frequency and the baseline table, lack of exact visit dates, absence of source verification, an unexplained final external-orifice bleed, unvalidated self-rated scales, and incomplete product characterization. These issues do not invalidate the case, but they limit the strength and generalizability of any conclusion.

20. Limitations, Clinical Implications, and Conclusion

Limitations

- Single-patient, uncontrolled design with no comparator and no inferential statistics.
- Concurrent standard treatment and supportive care prevent attribution of benefit to Raktamrut Vati alone.
- Reliance on self-report for several outcomes and absence of validated hemophilia-specific scales.
- Incomplete visit dates, source-document verification, inhibitor status, genetic testing, and objective joint assessment.
- Incomplete description and quality-control documentation of the Ayurvedic formulation.
- Potential recall bias, regression to the mean, behavior change, and natural fluctuation in bleeding frequency.
- Laboratory abnormalities and examination findings require follow-up before an unqualified safety conclusion.
- Findings from one adolescent cannot be generalized to other ages, severities, inhibitor states, or treatment regimens.

Clinical implications

The case supports further systematic investigation of a fully characterized Raktamrut Vati formulation as a possible adjunct to comprehensive hemophilia care. A prospective pilot study should use verified diagnosis, standardized prophylaxis documentation, predefined rescue therapy, daily bleed and medication diaries, validated joint and quality-of-life tools, laboratory safety monitoring, product batch testing, and independent outcome assessment. The study should be designed to protect access to standard treatment and should not use reduced factor availability as an intervention.

Revised conclusion

During six months of adjunctive Raktamrut Vati administration, the patient showed marked reported reductions in most bleeding episodes, hospitalizations, factor administrations, analgesic use, tranexamic acid use, pain, movement restriction, fatigue, and school absence. Self-rated muscle strength and quality of life improved. The therapy was reported as well tolerated, although selected

laboratory abnormalities require clinical follow-up. Factor VIII activity remained below 1% and aPTT remained markedly prolonged, confirming persistence of severe Hemophilia A. These observations are promising but cannot establish efficacy or causality. Raktamrut Vati should be regarded only as a research-stage supportive adjunct and never as a substitute for factor VIII, non-factor prophylaxis, emergency treatment, or specialist hematology care.

21. Patient Perspective, Consent, Declarations, and CARE Checklist

Patient perspective

The patient described severe pain, swelling, repeated bleeding, and dependence on factor therapy as major burdens for him and his family. After beginning the adjunctive intervention, he reported fewer bleeding episodes, reduced pain and swelling, greater confidence in daily activities, renewed hope, and reduced emotional and financial strain on the family. This perspective is reproduced in condensed form to preserve meaning while reducing potentially promotional language and protecting anonymity.

Consent and declarations

- Written informed consent and Ethics approval: This study was reviewed and approved by the Institutional Ethics Committee (IEC), Government Ayurveda College & Hospital, Dharashiv, Maharashtra, India, vide Approval Letter No. GACD/SS/6138/2024 dated 11 November 2024, before commencement of the study.Institutional ethics
- Clinical-trial registration: CTRI/2025/07/090910.
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- Data availability: De-identified source data may be made available subject to consent, ethics approval, and applicable privacy requirements.

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