RESEARCH PAPER

When a Non-Healing Leg Ulcer Unveils Sickle Cell Disease: A Delayed Diagnosis in a Young Adult Male

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Received: 15th Jul, 2026 | Revised: 25th Jul, 2026 | Accepted: 5th Aug, 2026 | Available Online: 12th Aug, 2026

Abstract

Chronic leg ulcers in young adults are uncommon and warrant evaluation for underlying systemic disorders. Sickle cell disease (SCD), although predominantly diagnosed during childhood, may occasionally remain undetected until adulthood, particularly in individuals with milder clinical phenotypes. Chronic leg ulceration is a recognized but relatively infrequent manifestation of SCD and often reflects ongoing vaso-occlusion, chronic hemolysis, endothelial dysfunction, and tissue ischemia. We report the case of a 25-year-old man presenting with a chronic non-healing ulcer over the lower leg of six months' duration without diabetes, peripheral arterial disease, or venous insufficiency. Initial management elsewhere focused on repeated wound dressings and empirical antibiotic therapy with minimal improvement. Detailed evaluation revealed chronic hemolytic anemia, indirect hyperbilirubinemia, elevated lactate dehydrogenase, and peripheral smear findings suggestive of sickling. Hemoglobin electrophoresis confirmed sickle cell disease. The patient was managed with wound care, hydroxyurea therapy, folic acid supplementation, analgesia, nutritional optimization, and supportive measures, resulting in gradual ulcer healing. This case highlights the importance of considering hemoglobinopathies in young patients with chronic leg ulcers, particularly in regions where sickle cell disease is prevalent.

Keywords: Sickle cell disease, Chronic leg ulcer, Hemoglobin electrophoresis, Hemolytic anemia, Hydroxyurea, Case report

How to cite this article: N.N. Anand1 Dr. Aswathy T Menon2* Dr. Adit Mithilesh A B3 Dr. S. Nava Mallika Sreya4 Dr. Bidisha Borthakur5. When a Non-Healing Leg Ulcer Unveils Sickle Cell Disease: A Delayed Diagnosis in a Young Adult Male. Int J Drug Deliv Technol. 2026;16(78s):576-577. DOI: 10.25258/ijddt.16.78s.68

Introduction

Chronic lower-limb ulcers are commonly attributed to diabetes mellitus, chronic venous insufficiency, peripheral arterial disease, infection, or vasculitis. However, inherited hematological disorders such as sickle cell disease should also be considered, particularly in young individuals without conventional vascular risk factors.

Sickle cell disease is an inherited disorder caused by a mutation in the β-globin gene resulting in the formation of hemoglobin S. Under conditions of hypoxia, dehydration, or acidosis, erythrocytes assume a sickled configuration leading to vaso-occlusion, chronic hemolysis, endothelial injury, and microvascular ischemia. Chronic leg ulcers develop in approximately 8–10% of affected adults and are associated with severe hemolysis and recurrent vaso-occlusive episodes.

We report a young man whose chronic leg ulcer served as the initial manifestation leading to the diagnosis of sickle cell disease.

Case Report

A 25-year-old man presented with a painful non-healing ulcer over the medial aspect of the left lower leg for six months. The lesion initially appeared as a small painful wound following trivial trauma and progressively enlarged despite multiple courses of antibiotics and regular wound dressings.

The patient reported intermittent fatigue and occasional episodes of generalized body pain since adolescence but had never undergone evaluation for an underlying hematological disorder. There was no history of diabetes mellitus, hypertension, peripheral vascular disease, tuberculosis, autoimmune disease, smoking, or intravenous drug use.

On examination, he was pale and mildly icteric. A solitary ulcer measuring approximately 8 × 5 cm was present over the medial malleolar region. The ulcer had irregular margins with healthy granulation tissue interspersed with slough. Mild surrounding hyperpigmentation was noted without features suggestive of chronic venous insufficiency. Peripheral pulses were palpable and symmetrical. There was no evidence of neuropathy.

Investigations

Laboratory investigations demonstrated normocytic normochromic anemia with elevated reticulocyte count. Serum bilirubin showed indirect hyperbilirubinemia, while lactate dehydrogenase levels were slightly elevated.

Peripheral blood smear revealed anisopoikilocytosis with numerous sickle-shaped erythrocytes, target cells, and polychromasia.

Inflammatory markers were mildly elevated.

Renal and liver function tests were otherwise unremarkable.

Doppler ultrasonography of the lower limbs demonstrated normal arterial and venous circulation without evidence of thrombosis or venous reflux.

Hemoglobin electrophoresis demonstrated predominant HbS with reduced HbA and elevated HbF, confirming sickle cell disease.

Wound culture grew normal skin flora without significant pathogenic bacterial growth.

Differential Diagnosis

- Chronic venous ulcer
- Vasculitic ulcer
- Diabetic foot ulcer
- Pyoderma gangrenosum
- Arterial insufficiency ulcer
- Chronic osteomyelitis
- Sickle cell leg ulcer

Management

The patient received multidisciplinary management involving physicians, hematologists, vascular surgeons, and wound-care specialists.

Treatment included:

- Hydroxyurea
- Folic acid supplementation
- Adequate hydration
- Analgesics
- Regular sterile wound dressings
- Appropriate antibiotics only when clinically indicated
- Nutritional optimization with a high-protein diet
- Limb elevation and compression avoidance in the absence of venous disease

Blood transfusion was not required as the patient remained hemodynamically stable.

Gradual granulation tissue formation and reduction in ulcer size were observed during follow-up.

Discussion

Leg ulcers are a chronic complication of sickle cell disease and are believed to result from repeated vaso-occlusion, endothelial dysfunction, chronic hemolysis, nitric oxide depletion, tissue hypoxia, and impaired wound healing. The ulcers commonly occur around the medial or lateral malleolus where skin perfusion is relatively poor.

Although sickle cell disease is generally diagnosed during childhood, delayed presentation may occur in individuals with milder genotypes or inadequate access to healthcare. Consequently, the diagnosis may be overlooked in adults presenting with chronic ulcers.

The differential diagnosis of chronic leg ulcers is broad and includes diabetes mellitus, venous insufficiency, peripheral arterial disease, vasculitis, infection, malignancy, and neuropathic ulcers. Careful history, clinical examination, vascular assessment, and laboratory evaluation are essential to identify uncommon etiologies.

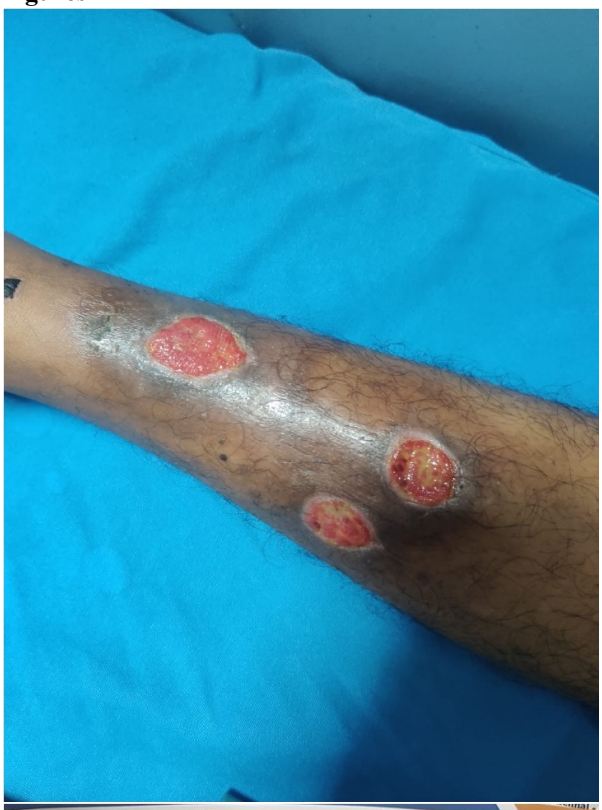
Hydroxyurea remains the cornerstone of disease-modifying therapy in sickle cell disease by increasing fetal hemoglobin levels and reducing vaso-occlusive events. Comprehensive wound care combined with optimization of the underlying hematological disorder improves ulcer healing and reduces recurrence.

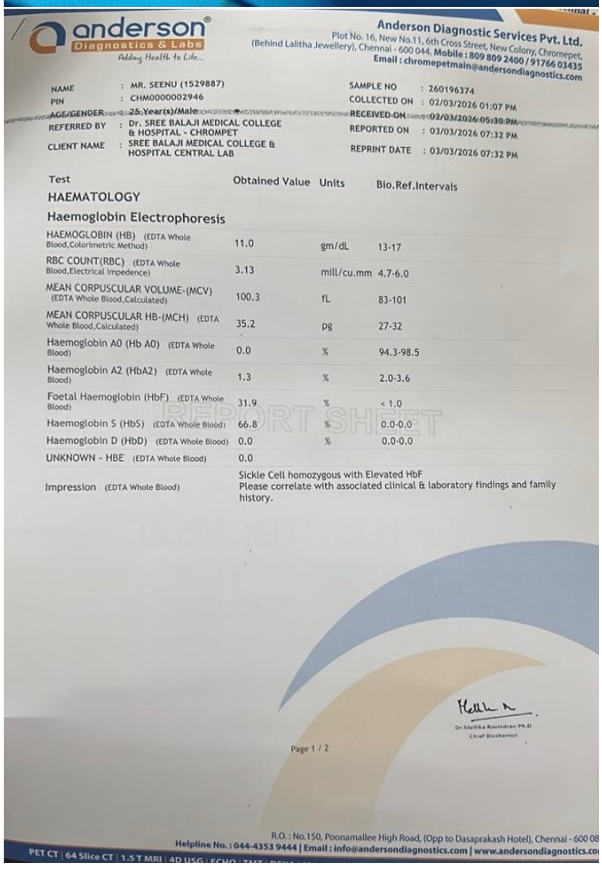
This case emphasizes that chronic leg ulcers in young adults should prompt evaluation for inherited hemoglobinopathies, especially when conventional risk factors are absent.

Conclusion

Chronic non-healing leg ulcers in young adults require a systematic search for underlying systemic disorders. Sickle cell disease, although uncommon as a first presentation in adulthood, should be considered in patients with chronic ulcers accompanied by anemia or evidence of hemolysis. Early recognition through peripheral smear and hemoglobin electrophoresis allows timely initiation of disease-specific therapy, promotes ulcer healing, and prevents future sickle cell-related complications.

Figures





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NAME	: MR. SEENU (1529887)	SAMPLE NO	: 260196374
PIN	: CHM0000002946	COLLECTED ON	: 02/03/2026 01:07 PM
AGE/GENDER	: 25 Year(s)/Male	RECEIVED ON	: 02/03/2026 05:30 PM
REFERRED BY	: Dr. SREE BALAJI MEDICAL COLLEGE & HOSPITAL - CHROMPET	REPORTED ON	: 03/03/2026 07:32 PM
CLIENT NAME	: SREE BALAJI MEDICAL COLLEGE & HOSPITAL CENTRAL LAB	REPRINT DATE	: 03/03/2026 07:32 PM

Test	Obtained Value	Units	Bio.Ref.Intervals
HAEMATOLOGY			
Haemoglobin Electrophoresis			
HAEMOGLOBIN (HB) (EDTA Whole Blood,Colorimetric Method)	11.0	gm/dL	13-17
RBC COUNT(RBC) (EDTA Whole Blood,Electrical Impedence)	3.13	mill/cu.mm	4.7-6.0
MEAN CORPUSCULAR VOLUME-(MCV) (EDTA Whole Blood,Calculated)	100.3	fL	83-101
MEAN CORPUSCULAR HB-(MCH) (EDTA Whole Blood,Calculated)	35.2	pg	27-32
Haemoglobin A0 (Hb A0) (EDTA Whole Blood)	0.0	%	94.3-98.5
Haemoglobin A2 (HbA2) (EDTA Whole Blood)	1.3	%	2.0-3.6
Foetal Haemoglobin (HbF) (EDTA Whole Blood)	31.9	%	< 1.0
Haemoglobin S (HbS) (EDTA Whole Blood)	66.8	%	0.0-0.0
Haemoglobin D (HbD) (EDTA Whole Blood)	0.0	%	0.0-0.0
UNKNOWN - HBE (EDTA Whole Blood)	0.0		
Impression (EDTA Whole Blood)	Sickle Cell homozygous with Elevated HbF Please correlate with associated clinical & laboratory findings and family history.		

REPORT SHEET

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