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Case Report

Branch Retinal Vein Occlusion as a Clinical Manifestation of Sickle Cell Trait: A Case Report.

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Abstract

Background

Sickle hemoglobin disorders are associated with vaso-occlusive and hyperviscosity-related complications involving multiple organ systems, including the eye. Ocular manifestations usually include non-proliferative or proliferative sickle retinopathy, whereas retinal vein occlusion is less commonly emphasized, particularly in individuals with sickle cell trait. This case highlights branch retinal vein occlusion as a clinically significant ophthalmic manifestation associated with sickle cell trait.

Case presentation

A 41-year-old man from Araku Valley presented with sudden blurring of vision in the right eye for 10 days, associated with decreased vision and recurrent headache. Ocular examination showed a positive comma sign in the inferior bulbar conjunctiva of both eyes. Fundus examination of the right eye revealed diffuse superficial hemorrhages with sclerotic arterioles in the superotemporal quadrant and associated macular edema, leading to a diagnosis of superotemporal branch retinal vein occlusion. Hematological evaluation showed mild anemia with microcytic indices, fragmented red blood cells, and a positive sickling test after 24 hours. High-performance liquid chromatography showed a heterozygous sickle hemoglobin pattern, with HbS at 23.8%, consistent with sickle cell trait. Extensive systemic evaluation for alternative causes of retinal vein occlusion, including thrombophilia, autoimmune, and vasculitic profiles, was negative. The patient was treated with three intravitreal ranibizumab injections and laser photocoagulation for ischemic retina, following which macular edema resolved and visual acuity stabilized at 6/9.

Conclusions

Branch retinal vein occlusion can occur as an important ocular manifestation of sickle cell trait after exclusion of other etiologies. Early ophthalmic recognition, hematological confirmation, and timely intervention are crucial for visual recovery and prevention of recurrent retinal vascular events in susceptible individuals.

Keywords: *Branch Retinal Vein Occlusion, Clinical Manifestation, Sickle Cell*
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Introduction

Sickle cell disease is an autosomal recessive genetic disorder that affects hemoglobin. The Global Burden of Disease (GBD) study in 2021 estimated that sickle cell disease affected approximately 1.2 million people in India, and that the number of cases had risen by 29.4% since 2000.[1] Sickle cell disease is associated with a lot of complications associated with hyperviscosity and vaso-occlusion, affecting almost all organ systems, including the central nervous system, musculoskeletal system, cardiovascular system, respiratory system, genitourinary system, hepatobiliary system, and the eye.[2] This case report aims to highlight superotemporal branch retinal vein occlusion as a rare ocular manifestation of sickle cell trait and to underscore the need for early recognition and multidisciplinary management.

Case presentation

A 41-year-old man from Araku Valley, Visakhapatnam, presented to the Ophthalmology outpatient department with sudden blurring of vision for 10 days, associated with decreased vision in the right eye and recurrent headache. He was a known case of sickle hemoglobinopathy and was subsequently confirmed on hematological evaluation to have sickle cell trait. The patient belonged to a tribal population from a region with a recognized burden of sickle-cell hemoglobin disorders. His occupation was [insert occupation if available]. Details regarding smoking, alcohol

intake, hydration status, strenuous physical exertion, and other lifestyle factors that could influence sickling episodes or retinal vascular events were not documented. On presentation, he was conscious, oriented, and clinically stable. General physical examination did not reveal any major abnormality; however, detailed vital signs and systemic examination findings were not specifically recorded in the case notes.

Ocular examination revealed a positive comma sign in the inferior bulbar conjunctiva of both eyes (Fig. 1), while the remaining anterior segment findings were unremarkable. Fundus examination of the right eye showed diffuse superficial hemorrhages in the superotemporal quadrant of the retina with a few sclerotic arterioles (Fig. 2). Macular edema was present. The left eye showed a normal fundus picture. Based on these findings, a clinical diagnosis of superotemporal branch retinal vein occlusion with macular edema in the right eye, probably related to sickle cell trait, was made.

Complete blood count revealed a hemoglobin level of 11.7 g/dL, mean corpuscular volume of 66.2 fL, mean corpuscular hemoglobin of 19.8 pg, and mean corpuscular hemoglobin concentration of 30.0 g/dL. Differential leukocyte count showed 9% eosinophils. Peripheral smear examination demonstrated predominantly normocytic normochromic red blood cells, admixed with a few microcytic hypochromic forms and a few fragmented red blood cells, suggestive of a hemolytic picture. The reticulocyte count was 2%. Sickling test was positive after 24 hours (Fig. 3). High-performance liquid chromatography showed HbF <0.8%, HbA0 63.4%, HbA2 3.1%, and HbS 23.8%, consistent with a sickle cell heterozygous pattern (Fig. 4). Liver function tests were mildly deranged, with serum total bilirubin 1.4 mg/dL, direct bilirubin 0.6 mg/dL, indirect bilirubin 0.8 mg/dL, and alanine aminotransferase 63 IU/L.

An extensive workup was performed to exclude other causes of branch retinal vein occlusion. Serum homocysteine, protein C, protein S, c-ANCA, p-ANCA, and antinuclear antibody blot profile, including dsDNA, nucleosomes, histones, SmD1, PCNA, PO(RPP), SS-A/Ro52, SS-A/Ro60, SS-B/La, CENP-B, Scl-70, U1-snRNP, AMA-M2, Jo-1, PM-Scl, Mi-2, Ku, and DFS70 antibodies, were all negative. These findings supported sickle cell trait as the most likely etiological factor for branch retinal vein occlusion in this patient.

Family screening was also performed. Complete blood counts and peripheral smear of the patient's wife (34 years) were unremarkable. Her sickling test was positive after 24 hours, and high-performance liquid chromatography showed HbF 1.2%, HbA0 57.3%, HbA2 3.3%, and HbS 30.2%, suggestive of a sickle cell heterozygote pattern. The elder son had no abnormal findings on complete blood count and peripheral smear; his sickling test was negative, and high-performance liquid chromatography showed a normal pattern. The younger son had a hemoglobin level of 10.5 g/dL, mean corpuscular volume of 62.6 fL, mean corpuscular hemoglobin of 19.1 pg, and mean corpuscular hemoglobin concentration of 30.6 g/dL. Peripheral smear showed mild anisopoikilocytosis with predominantly normocytic normochromic red blood cells and some microcytic hypochromic cells. A few fragmented cells were also seen, suggestive of hemolysis. His sickling test was positive after 24 hours, and high-performance liquid chromatography showed HbF 3.1%, HbA0 56.3%, HbA2 2.7%, and HbS 19.9%, again suggestive of a sickle cell heterozygous pattern.

Follow-up and post-intervention clinical condition

The patient was treated with three doses of intravitreal ranibizumab (0.5 mg/0.05 mL) along with laser photocoagulation to the ischemic retinal areas. He was followed up for [insert follow-up duration, for example, 3 months/6 months]. At the end of follow-up, visual acuity in the right eye stabilized at 6/9, with resolution of macular edema. No adverse or unanticipated events related to intravitreal anti-vascular endothelial growth factor therapy or laser photocoagulation were reported during the treatment and follow-up period.

Figure 1: Positive Comma sign in the inferior bulbar conjunctiva of the right eye. **Figure 2:** Fundus examination of the right eye showing diffuse superficial hemorrhages and a few sclerotic arterioles in the superotemporal quadrant. **Figure 3:** Positive sickling test of a patient, showing sickle-shaped red blood cells. **Figure 4:** High-performance liquid chromatograph of the patient showing 23.8% of HbS.

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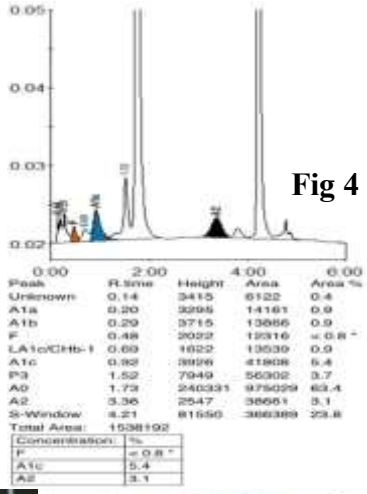

Fig 4

Peak	R.time	Height	Area	Area %
Unknown	0.14	3415	6122	0.4
A1a	0.20	3295	14161	0.9
A1b	0.29	3715	13986	0.9
F	0.48	2022	12316	< 0.8 *
LA1c/CHb-1	0.69	1622	13539	0.9
A1c	0.92	3926	41808	5.4
P3	1.52	7949	56302	3.7
A0	1.73	240331	975029	63.4
A2	3.36	2547	38661	3.1
S-Window	4.21	81550	386389	23.8
Total Area:		1538192		

Concentration:	%
F	< 0.8 *
A1c	5.4
A2	3.1

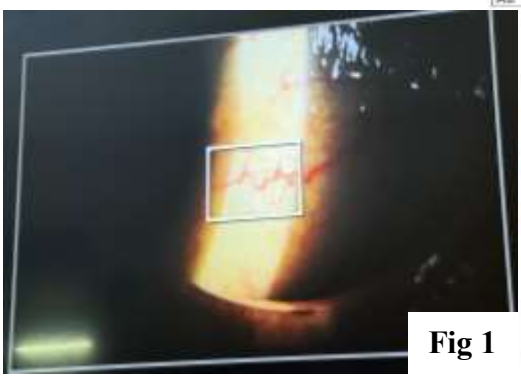

Fig 1

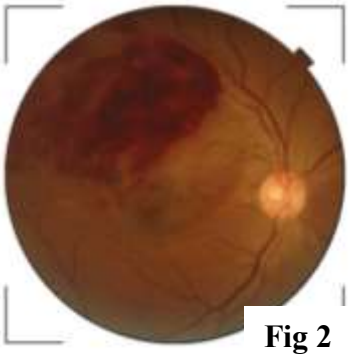

Fig 2

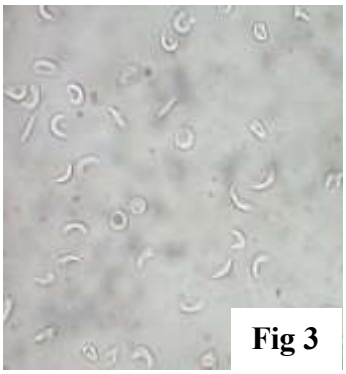

Fig 3

Discussion

Hemoglobin is a tetramer composed of two alpha globin and two beta globin chains. Sickle mutation substitutes thymine for adenine in the sixth codon of the beta globin gene (GAG to GTG), thereby encoding valine instead of glutamic acid (P.Glu6Val) in that position. The root cause of sickle cell pathobiology is polymerisation of HbS, resulting in the classically sickled erythrocyte, also known as a drepanocyte, that may be seen directly with either light or scanning electron microscopy.[2] The physiological determinants of this polymerisation are oxygen tension, concentration of HbS, temperature, pH, and the intraerythrocyte concentration of hemoglobin molecules other than HbS. HbA, HbA2, HbF, and other hemoglobin molecules have an inhibitory effect on the polymerisation of HbS molecules.[3] Sickling is initially associated with reversible red blood cell membrane changes. Following repeated cycles of sickling and unsickling, aberrations in membrane function and structure become increasingly pronounced, culminating in fixation of the membrane in a permanent sickled configuration.[3] Extravascular hemolysis is the predominant cause of fragmentation of red blood cells in these cases, with intravascular hemolysis also contributing to some extent.[2]

Sickle cell retinopathy is an ocular manifestation of the spectrum of sickle cell disease. Its prevalence increases with age. The sickle-shaped red cells get trapped in the small blood vessels in various structures of the eye, both in the anterior and posterior segments, leading to characteristic damage.[3] Sickle cell retinopathy, like the other retinopathies, is of two types: non-proliferative and proliferative. Fundus examination of non-proliferative sickle retinopathy shows retinal hemorrhages characteristically known as salmon patches, arteriolar and capillary occlusions with sunburst appearance. Proliferative sickle retinopathy shows retinal neovascularization, vitreous hemorrhages, and tractional retinal detachment.[4]

Retinal vein occlusions are divided into central, hemiretinal, and branch retinal vein occlusions. Branch retinal vein occlusion (BRVO) is a venous occlusion that occurs at any branch of the central retinal vein. It is of two types: major, which involves occlusion of a retinal vein that drains one of the retinal quadrants, and minor, which involves the occlusion of a venule supplying the macula. Superotemporal branch retinal vein occlusion is a type of major BRVO. Branch retinal vein occlusion can be perfused (nonischemic) or nonperfused (ischemic), and can involve the superotemporal (59%), inferotemporal (29%), or nasal

quadrants (12%) of the retina. The risk factors for branch retinal vein occlusion include conditions that cause hyperviscosity, such as increasing age, glaucoma, hypertension, hyperlipidemia, hyperhomocysteinemia, factor V Leiden mutation, protein C and protein S deficiencies, and systemic lupus erythematosus.[5] It can also occur as part of the retinopathy spectrum in sickle cell anemia.[6]

In our case, the patient is a known case of sickle cell trait and developed superotemporal branch retinal vein occlusion. Because there are many other etiological factors also known to be associated with this condition, we have done a comprehensive investigative workup to rule them out. All these investigations showed no abnormalities, confirming sickle cell trait as the cause.

Follow-up

Three doses of intravitreal anti-VEGF agent, injection Ranibizumab 0.5 mg in 0.05 mL, were given to the patient. Laser photocoagulation was done for ischaemic areas. The patient's visual acuity stabilised at 6/9 with resolution of macular edema at the end of treatment. However, its etiological factor, that is, sickle cell disease, must be addressed to prevent recurrent episodes of retinal vein occlusions.

Conclusion

There is an increase in the number of sickle cell disease cases in India. This disease can have a plethora of clinical manifestations, affecting every organ system and ranging from mild to severe. It is therefore important to look out for even uncommon signs and symptoms in known cases, to ease early recognition and prompt treatment of the complications associated with this condition.

Acknowledgement

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Recommendations

Patients with sickle cell trait or sickle cell disease presenting with sudden visual symptoms should undergo prompt ophthalmic evaluation, including detailed fundus examination and macular assessment. Screening for retinal vascular complications is advisable, particularly in individuals from high-prevalence regions. A structured systemic workup should be performed to exclude other causes of retinal vein occlusion. Family screening and hemoglobin analysis are useful for identifying affected relatives and enabling counseling. Early treatment with intravitreal therapy and laser, when indicated, can preserve vision. Multidisciplinary follow-up involving ophthalmologists, pathologists, and physicians is essential to prevent recurrence and improve long-term outcomes in susceptible patients overall.

Abbreviations

Alanine aminotransferase - ALT;
Antinuclear antibody - ANA;
Branch retinal vein occlusion - BRVO;
Bulbar conjunctiva - BC;
Central retinal vein - CRV;
Hemoglobin - Hb;
Hemoglobin A0 - HbA0;
Hemoglobin A2 - HbA2;
Hemoglobin F - HbF;
Hemoglobin S - HbS;
High-performance liquid chromatography - HPLC;
Mean corpuscular hemoglobin - MCH;
Mean corpuscular hemoglobin concentration - MCHC;
Mean corpuscular volume - MCV;
Ophthalmology outpatient department - OPD;
Perinuclear antineutrophil cytoplasmic antibody - p-ANCA;
Red blood cells - RBCs;
Ranibizumab - RBZ;
Superotemporal branch retinal vein occlusion - STBRVO;
Vascular endothelial growth factor - VEGF.

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This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest

The authors declare no conflict of interest.

Availability of Data

Data Available on request

Author contribution

Dr Josephine Bindu Prathipaty contributed to case documentation, data collection, literature review, and initial manuscript drafting. Dr Jyothsna Duvvada was involved in clinical ophthalmic evaluation, patient follow-up, and preparation of ophthalmology-related content. Dr Dharma Raju Bennabhaktula contributed to case supervision, clinical interpretation, and critical revision of the manuscript. Dr Bhagyalakshmi Atla contributed to hematological evaluation, laboratory interpretation, overall conceptual guidance, and final manuscript review. All authors read and approved the final version of the manuscript and agree to be accountable for the integrity of the work.

Author Biography

Dr Prathipaty Josephine Bindu, MBBS, MD Pathology, is a Senior Resident in the Department of Pathology at NRI Institute of Medical Sciences, Visakhapatnam. She completed her undergraduate training at Guntur Medical

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Dr. Atla Bhagyalakshmi, MBBS, MD Pathology, is Professor and Head of the Department of Pathology at NRI Institute of Medical Sciences, Visakhapatnam. She holds an MBBS (1982) and an MD in Pathology (1992) from Andhra Medical College, Visakhapatnam, and has over 37 years of teaching and professional experience. She previously served as Professor and Head, and Vice Principal (Admin), Andhra Medical College, Visakhapatnam, and retired from government service in 2021 after distinguished contributions to medical education and research.

She has held numerous leadership positions, including Vice Chairman of the Scientific and Research Committee and Coordinator of the PG Medical Education Committee at NRIIMS, Executive Council Member of Dr. NTR University of Health Sciences (2021–2024), Chairperson of the AMC Research Forum, and PhD guide in pathology under Dr. NTRUHS. She has also served as Chairperson of undergraduate and postgraduate examination boards, MCI assessor for UG/PG courses, and nodal officer of the Multidisciplinary Research Unit (MRU), AMC, where she was principal investigator for four funded projects.

Dr. Bhagya Lakshmi has published over 150 research papers in reputed national and international journals and has served as a reviewer and editorial board member for several indexed journals. She is a life member of the Indian Medical Association, Indian Association of Pathologists and Microbiologists, Indian Association of Cytologists, and NJBMS. She has received multiple awards, including two Certificates of Merit from the District Collector for exemplary service (1998 and 2011). Her major academic interests include oncopathology, hematopathology, and translational research in pathology.ORCID: http://orcid.org/0009-0007-0606-7301

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