

Profound Presentation of Retinopathy in a Patient with Sickle Cell Trait and Diabetes Mellitus

Gautam Vangipuarm¹, MD; Steven S. Saraf¹, MD; Qinqin Zhang², PhD; Ruikang Wang^{1,2}, PhD; Kasra A Rezaei¹, MD

¹Department of Ophthalmology, University of Washington, Seattle, WA, USA ²Department of Bioengineering, University of Washington, Seattle, WA, USA

ORCID:

Kasra A Rezaei: https://orcid.org/0000-0003-4287-3187

J Ophthalmic Vis Res 2020; 15 (1): 116–117

Correspondence to:

Kasra A Rezaei, MD. University of Washington, Department of Ophthalmology, Seattle, WA, 908 Jefferson St, Seattle, WA 98104. E-mail: krezaei@uw.edu

Received: 01-01-2019 Accepted: 01-05-2019

Access this article online				
Website: https://knepublishing.com/index.php/JOVR				
DOI: 10.18502/jovr.v15i1.5962				

PRESENTATION

A 43-year-old functionally monocular African American woman with longstanding type 2 diabetes mellitus presented for care of her better-seeing left eye. Originally suspected of having proliferative diabetic retinopathy (PDR) as the cause of her bilateral visual impairment, fluorescein angiography and optical coherence tomography angiography revealed a marked peripheral nonperfusion which was out of proportion for a typical diabetic retinopathy (Figure 1). A comprehensive uveitic and vasculopathic workup was therefore initiated. The workup was largely negative except for hemoglobin electrophoresis, which was consistent with the sickle cell trait (or hemoglobinopathy) (Table 1). The patient was counseled on her diagnosis and continues to be treated with laser photocoagulation for her peripheral neovascularization.

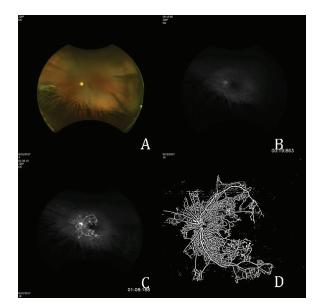


Figure 1. Color fundus photo, left eye (A) early (B) and late (C) fluorescein angiography of the left eye showing marked peripheral ischemia and posterior pole neovascularization. OCT angiography (D) showing severely decreased vascular density.

DISCUSSION

This report strengthens the hypothesis that diabetic retinopathy and coexisting vasculopathic

How to cite this article: Vangipuarm G, Saraf SS, Zhang Q, Wang R, Rezaei KA. Profound Presentation of Retinopathy in a Patient with Sickle Cell Trait and Diabetes Mellitus. J Ophthalmic Vis Res 2020;15:116–117.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Table 1. Laboratory	assessment of othe	r etiologies for	extensive	peripheral	non-perfusion	including	pro-thrombotic	and
vasculitic causes								

Test ordered	Result (normal range)
Angiotensin converting enzyme (U/L)	26U/L (8–53 U/L)
Anti-nuclear antibody	Negative
Cryoglobulin	Negative
Erythrocyte sedimentation rate (mm/H)	60 mm/H high (0–20 mm/H)
HIV Ag and Ab	Nonreactive
Anti-myeloperoxidase	Negative
Anti PR3	Negative
Rheumatoid factor	< 10
Serologic syphilis panel	Negative
Anti-thrombin activity	123% (normal)
C-reactive protein (mg/L)	24.9 mg/L high (0–10 mg/L)
Activated protein S (%)	113% (65–150%)
Activated protein C (%)	121% (55–150%)
Factor V Leiden	Negative
Homocysteine	Negative
Prothrombin time (s)	14.1 s (10.7–15.6 s)
INR (s)	1.1 s (0.8–1.3 s)
CBC	Normal
СМР	Glucose 353 mg/dL (62–125 mg/dL
Herpes type 1&2 serology	Positive for HSV-1 and HSV-2
CMV (serum antibody)	Positive
Hemoglobin electrophoresis	Consistent with HbS trait
Quantiferon-TB Gold	Negative

CBC, complete blood count; CMP, comprehensive metabolic panel; CMV, antibodies to cytomegalovirus; HIV, human immunodeficiency virus; INR, international normalized ratio; mg/dL, milligrams per deciliter; mm/H, millimeter per hour U/L, Units Per Liter

diseases, even sickle cell trait, may have a synergistic effect on the overall disease burden. A broad differential must be maintained in patients with presumed diabetic retinopathy, especially those with uncharacteristic imaging findings.^[1–5]

Financial Support and Sponsorship

This study was supported by the Department of Ophthalmology, University of Washington.

Conflicts of Interest

There are no conflicts of interest.

REFERENCES

- Tsaras G, Owusu-Anash A, Boateng FO, Amoateng-Adjepong Y. Complications associated with SCT: A brief narrative review. *Am J Med* 2009;122:507–512.
- Downes S, Hambleton I, Chuang EL, Lois N, Serjeant GR, Bird AC, et al. Incidence and natural history of proliferative sickle cell retinopathy. *Ophthalmology* 2005;112:1869– 1875
- 3. Jampol L, Goldbaum M. Peripheral proliferative retinopathies. *Surv Ophthalmol* 1980;25:1–14
- 4. Jackson H, Bentley CR, Hingorani M, Atkinson, P Aclimandos WA, Thompson GM. Sickle retinopathy in patients with sickle trait. *Eye* 1995;9:589–593.
- Nagpal KC, Asdourian GK, Patrianakos D, Goldberg MF, Rabb MF, Goldbaum M. Proliferative retinopathy in SCT. Report of seven cases. *Arch Intern Med* 1977;137:325–328.