


Case Report

Increased resistive index-induced unilateral venous stasis-like retinopathy in a patient with anemia

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ABSTRACT

A rare presentation of unilateral venous stasis-like retinopathy is reported in a 55-year-old female. Unilateral, acute onset of diminution of vision was associated with multiple superficial retinal hemorrhages and Roth spots over the posterior pole of the retina. An increase in the resistive index (RI) of the central retinal artery and vein, along with iron deficiency anemia, was observed. The cardiac workup was unremarkable. Therapeutic doses of oral iron and folic acid were administered along with an antiplatelet agent. At 6 weeks, there was resolution of venous stasis-like retinopathy with improvement in retinal hemorrhages and visual acuity.

Key words: Iron deficiency anemia, Resistive index of central retinal artery, Roth spots, Venous stasis-like retinopathy

INTRODUCTION

According to some experts, venous stasis retinopathy is considered an early form of ocular ischemia, while some consider it a non-ischemic central retinal vein occlusion. Retinal hemorrhages, constricted retinal arteries, central retinal artery blockage, neovascularization, and glaucoma are some of the fundus features. Venous stasis retinopathy is an early form of ocular ischemia, categorized by some experts as a non-ischemic central retinal vein occlusion.^{1,2} Posterior segment features include retinal hemorrhages, narrowed retinal arteries, central retinal artery occlusion, neovascularization, and glaucoma. The chronic form of ocular ischemia is explained by persistently low perfusion pressure that leads to diffuse retinal ischemia. This is manifested as increased circulation time, which first causes the retinal veins to dilate, become irregularly calibrated, and become tortuous.

Anemia is a widespread disease in both developed and developing countries, affecting nearly 1.6 billion people worldwide. Factors leading to anemia include nutritional deficiencies, worm infestations, chronic infections, and genetics.³ Ocular manifestation is not a common finding in most patients with hematological disorders.⁴ The pathophysiology behind the fundus lesion is poorly understood; however, retinal hypoxia is supposed to be the main culprit.⁵ Patients with severe anemia can present with

fundus findings such as retinal hemorrhage, soft exudates, venous tortuosity, Roth spots, and disc edema.^{3,6} The levels of platelet and hemoglobin at which the retinal changes tend to occur are yet to be determined. However, various factors may predispose to anemic retinopathy, such as low platelet count, the severity of anemia, and the onset of anemia. Studies indicate that the risk of retinopathy is significantly higher in low hemoglobin levels (<8 gm/dL).

Resistive index (RI) reflects vascular resistance peripheral to the measuring location.⁷ It has been characterized as a marker of vascular resistance. Considering vascular compliance, the RI increases with increasing resistance.⁸

CASE REPORT

A 55-year-old female presented to the retina service of this tertiary care center with complaints of diminution of vision, which was of acute onset, rapidly progressive, and painless. No history of flashes of light or floaters was present. There was no known history of diabetes mellitus, hypertension, bleeding disorder, smoking, use of oral contraceptives, or antiplatelet drugs. The blood pressure was within normal limits.

On examination, the patient's Snellen visual acuity was 6/9 and 3/60 for the right and left eyes, respectively. Intraocular pressure was 17 mm Hg oculus dexter and 16 mm Hg oculus sinister. The pupillary reaction was brisk, with no anisocoria

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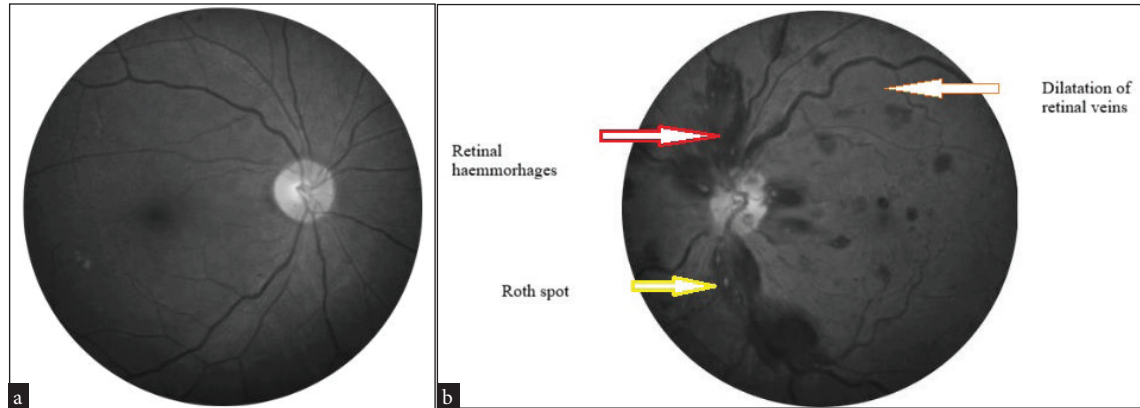


Figure 1: (a) Fundus photo of right eye at presentation, (b) Fundus photo of left eye at presentation.

or relative afferent pupillary defect. On slit lamp evaluation, the anterior segment examination was unremarkable. On fundus examination of the left eye, multiple superficial retinal hemorrhages were observed adjacent to the disc, around the macular area with associated Roth spots; however, the fovea was spared. Significant venous dilatation was observed in the left eye. Dilatation of retinal veins was also appreciated in the right eye [Figures 1a and b].

Spectral-domain optical coherence tomography angiography (Carl Zeiss Cirrus Meditec OCT 5000, Dublin, USA) was performed, which showed a foveal avascular zone area of 0.20 mm² in the right eye and 0.08 mm² in the left eye, respectively.

Ocular blood flow velocity study was performed using Color Doppler imaging (Philips Affinity 70 G, Tamba, USA) with a linear probe at 3 MHz frequency, which showed an increased RI in left ophthalmic artery (0.86), left central retinal vein (0.94), and left central retinal artery (0.80) as compared to the right ophthalmic artery (0.84), right central retinal vein (0.46), and right central retinal artery (0.69), respectively. As

the diminution of vision was unilateral with no predisposing factors for vein-occlusive disease, a clinical diagnosis of venous stasis-like retinopathy was considered.

Blood samples were collected for complete blood count (CBC), general blood picture, erythrocyte sedimentation rate, random blood glucose, serum lipid profile, and kidney function test. The CBC showed hypochromic microcytic anemia with hemoglobin levels of 7.6 g/dL. Further evaluation showed iron deficiency anemia (low transferrin levels and high total iron binding capacity). The rest of the blood picture was unremarkable. The patient also underwent a thorough cardiac workup, which was found to be unremarkable. Therapeutic doses of oral iron, folic acid, and antiplatelet agents were administered. At 6 weeks of follow-up, residual retinal hemorrhages were observed [Figures 2a and b]. Snellen visual acuity had improved to 6/12.

DISCUSSION

Ocular manifestation is known to occur in cases with decompensated hematological disorders.⁸ A review by

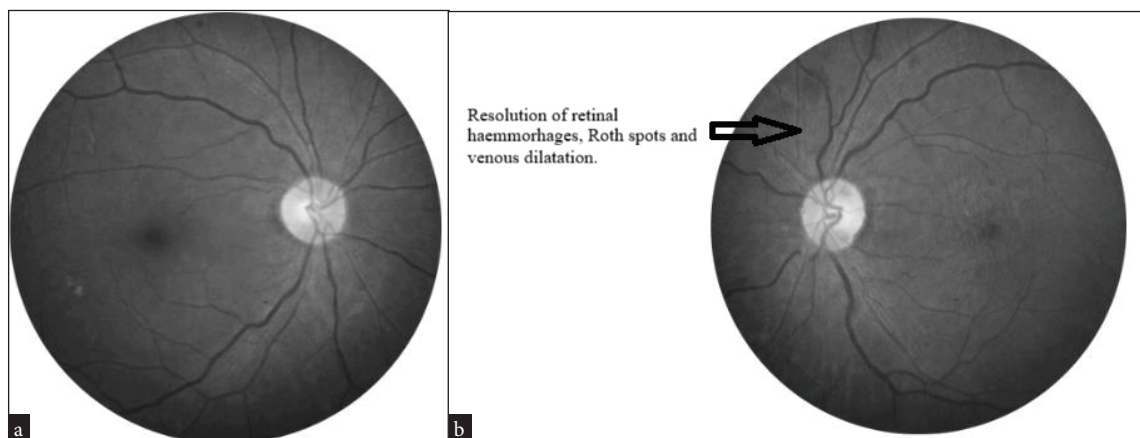


Figure 2: (a) Fundus photo of right eye after 6 weeks of anemia treatment, (b) Fundus photo of left eye after 6 weeks of anemia treatment.

Lowenstein *et al.* has stated that a precise level of anemia at which retinal abnormalities will occur cannot be given, as there are conflicting reports from various case series.⁷

Our patient presented with unilateral ocular findings of venous stasis-like retinopathy. Chronically low perfusion pressure causes diffuse retinal ischemia, reflected by increased circulation time, which initially results in dilatation, irregularity of caliber, and tortuosity of retinal veins. This in itself is a rare presentation. An earlier case report by Toh *et al.* (2022) in a patient with colorectal carcinoma had evidence of unilateral retinopathy with hemoglobin levels of 6.1 g/dL.⁶ The hemoglobin level in our patient was 7.6 g/dL with the absence of any associated risk factors such as thrombocytopenia, bleeding disorder, and hypertension, which can predispose the patient to retinopathy.

On color Doppler imaging, a novel finding of an increased RI in the central retinal artery and central retinal vein of the left eye was observed, which was a predisposing factor for the above clinical presentation. To the best of our knowledge, this is the first such report in the literature.

In the present case, an increase in the RI of retinal vessels led to an increase in shear stress in the vessel wall.⁹ An increase in shear stress occurred since the vessel diameter was unable to change in caliber, leading to mechanical injury to the vascular endothelium. Vessel walls of larger vessels of the retinal circulation suffer more circumferential stress damage, as the circumferential stress that is responsible for mechanical damage to the endothelium of the vessel wall is directly proportional to the perfusion pressure and radius and inversely proportional to the thickness of the vessel wall. Such an intravascular phenomenon led to extravasation of blood into the retinal tissue.¹⁰

Unilateral transient loss of vision can be a feature of carotid artery stenosis, which is fully recovered within 60-90 minutes. However, in this patient diminution of vision lasted more than 24 hours, which excludes a transient ischemic attack. Anemia is also known to cause transient retinopathy, especially with coexisting thrombocytopenia. As the severity of anemia increases, the risk of retinopathy increases, particularly when the hemoglobin level is below 6 g/dL. As iron deficiency is the most common type of anemia, most patients with anemic retinopathy have iron deficiency anemia. resolution of the fundus abnormalities occurs following treatment. However, in the present case, hemoglobin was 7.6 g/dL, and platelet count was within the normal limit (1.7 lac/cu mm)

CONCLUSION:

Furthermore, large sample size case control studies are warranted to establish a therapeutic model and to study the varied complication of iron deficiency on various systems of the body.

A novel case of increased resistive index-induced venous stasis-like retinopathy is documented.

Authors' contributions: DB: Concept and design of the study, acquisition of data, or analysis and interpretation of data; SS: Drafting the article or revising it critically for important intellectual content; SS: Accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved; LA: Final approval of the version to be published.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given consent for clinical information to be reported in the journal. The patient understands that the patient's names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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