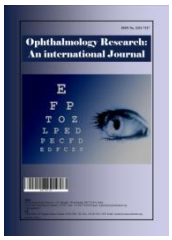

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Central Retinal Vein Occlusion as a Presenting Feature Caused by Hypertensive Crisis Secondary to IgA Nephropathy

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

We present a case of Ischemic Central retinal vein occlusion (CRVO) caused by hypertension secondary to renal failure in IgA nephropathy. A 17 year old male came with chief complaints of sudden painless diminution of vision in RE since 15 days. On examination the Right eye showed multiple superficial retinal haemorrhages in all 4 quadrents with dilated veins and cystoid macular edema suggestive of CRVO and Left eye showed superficial flame shaped haemorrhage suggestive of grade 3 hypertensive changes.

Keywords: Immunoglobulin a nephropathy; central retinal vein occlusion; iron deficiency anemia.

1. INTRODUCTION

Immunoglobulin A nephropathy (IgAN), also known as Berger's disease, is one of the most common primary glomerulopathies worldwide. This immune complex- mediated disease typically affects males, in the second and third decades of life, and may be asymptomatic or manifest with hematuria and/or proteinuria. Renal biopsy is essential for its diagnosis, taking into account that IgA deposits may be observed even in patients without evidence of kidney disease

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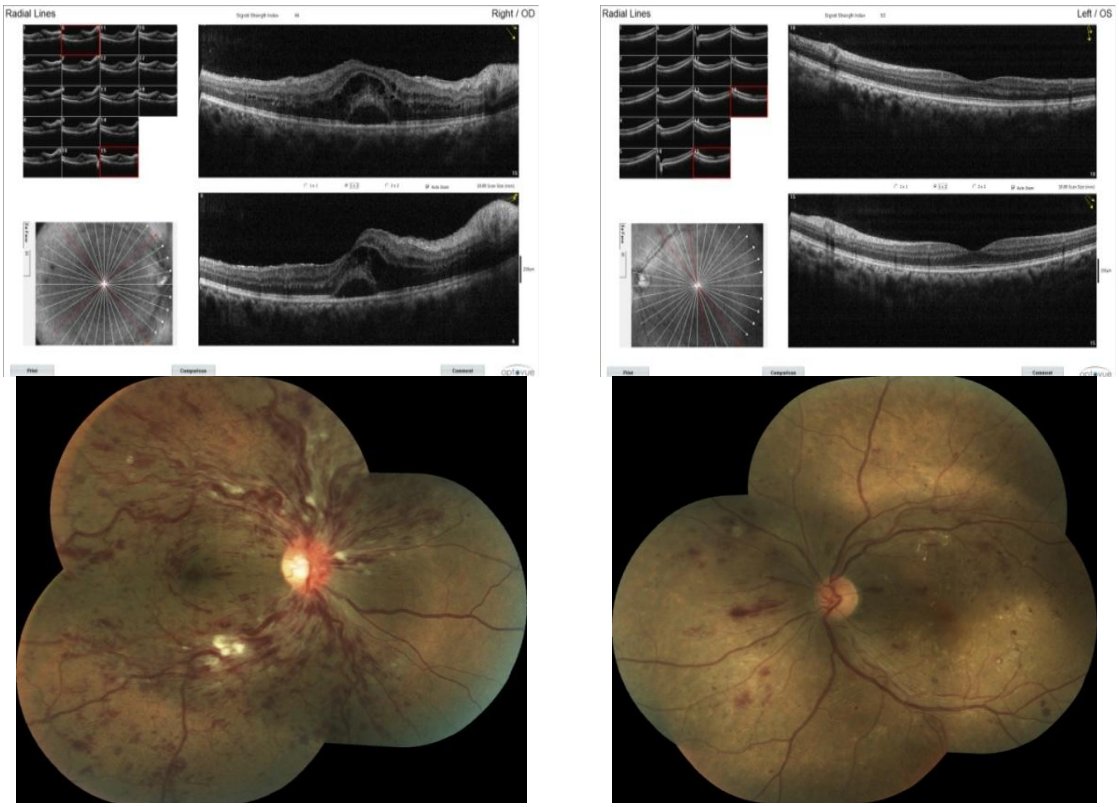
[1]. Although in most cases, “IgAN is clinically reserved to the kidneys, other conditions associated with a number of immune and inflammatory diseases, commonly rheumatic (i.e. ankylosing spondilytis, rheumatoid arthritis and Reiter syndrome), gastrointestinal (i.e. celiac disease), hepatic (i.e. alcoholic and non-alcoholic liver disease, and schistosomiasis), pulmonary (i.e. sarcoidosis), and cutaneous (i.e. dermatitis herpetiformis)” [2]. “Human immunodeficiency virus infection and hepatitis B (in endemic areas) have also been associated with IgAN” [2]. “Ocular involvement in patients with IgAN is rare and the most common ocular association are uveitis and scleritis” [3–5].

2. CASE REPORT

A 17 -year-old male presented with chief complaints of sudden painless diminution of vision in the Right eye since 15 days. On examination, his best corrected visual acuity was 6/36 N6 in the right eye and 6/6 N6 in the left eye. color vision was 25/25 and contrast sensitivity was 0.8 in both eyes intraocular pressure in the right eye was 20mmHg and in the left eye it was 21mmHg. Central corneal thickness was 0.523 and 0.527 respectively. Anterior segment examination was normal in both eyes and on swinging flash light test showed relative afferent pupillary defect in right eye and normal size reacting to light in left eye. On examination of the posterior segment right eye showed a disc edema and hyperemic disc, multiple retinal hemorrhages all over the fundus, tortuous and dilated retinal vasculature and macular edema (Fig. 1), while fundus of the other eye showed flame shaped haemorrhage with generalised arteriolar attenuation (Fig. 2). ÓCT showed increased central macular thickness with intra retinal fluid and neurosensory detachment in the right eye and oct was normal for left eye.

The workup included a normal complete hemogram (Hb- 6.6 gm percent, MCV- 95.9fl, MCH- 30.3 picogram, MCHC- 31.6 gm/dl, WBC- 5.4 1000/mul, Platelet count- 210 1000/mul), lipid profile (Total cholesterol 138 mg%, HDL Cholesterol 62.1mg%, Triglycerides 80 mg%) and The C- Reactive protein rate was normal 4.2 mg/l and Serum homocysteine level was 7.10 µmol/L, which was in the normal



Figs. 1,2. Intra retinal fluid and neurosensory detachment with irregular foveal contour

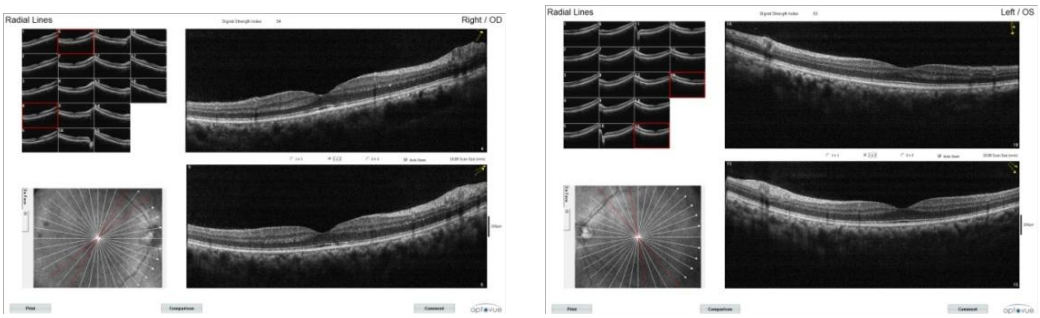


Fig. 3. Radical line

range of 6-15 µmol/L. His Montoux (tuberculin) Test and COVID antigen test was negative. His Sickling test came out to be positive, creatinine was 5.61 mg% and urea was 85.3 mg%, fasting and post prandial sugars were 73.7 mg% and 81.3 mg%. His BP was 171\81mmhg on presentation. On screening the family members his maternal uncle was postive for sickling test. Patient was referred to nephrologist and after multiple episodes of dialysis macular edema completly resolved and vision was RE 6/9 and LE 6/6.

3. DISCUSSION

We report a case of CRVO secondary to hypertensive crisis in a patient with IgA nephropathy. “Uncontrolled blood pressure is inherently associated with vascular damage. Mechanical stress caused by shearing forces causes endothelial dysfunction as measured by the reduced bioavailability of the nitric oxide, a potent vasodilatory factor. Concurrently, angiotensin II excess contributes to constriction of the vessels, which further increases the risk of hypertension development. The examination of the retinal vessels may also show characteristic changes of severe arteriolar attenuation and superficial flame shaped and dot and blot haemorrages resulting into development of CRVO”.[6]. The Vascular endothelial growth factor excess is also claimed to be responsible for the initiation and progression of the macular oedema [6,7]. “The reported patient also presented two other common retinal vein occlusion risk factors, i.e dyslipidaemia and impaired renal function”.[8]. “The other factor which possibly added the risk of the development of the RVO in our patient was iron deficiency anemia. Iron functions to regulate platelet numbers and function by inhibiting thrombopoiesis” [9]. “In a state of iron-deficiency, there is a reactive thrombocytosis, thus leading to hypercoagulability. Red cell deformability is reduced in microcytic iron-deficient cells, resulting in an increased viscosity and furthermore contributing to the hypercoagulable state” [10]. “It has been hypothesized that anemic hypoxic injury to the retino-choroidal circulation causes endothelial cell dysfunction” [11] and “a weaker anti-oxidant defence in the IDA state, results in increased platelet aggregation” [12]. Treatment includes multiple episodes of dialysis at regular intervals under a nephrologist and intravitreal antivegf injections to reduce macular edema with frequent follow ups 3 months for the fellow eye and to look for neovascularization.

4. CONCLUSION

We present this as an interesting case report of CRVO with previously undiagnosed IgA nephropathy where hypertension, dyslipedemia and iron deficiency anemia were the risk factors. The aetiology of CRVO in the presented case was multifactorial; however, hypertension was the most probable triggering factor. Fundus fluroscein angiography was not performed due to impaired renal function; Ideally, future studies might be able to correlate the extent of macular flow loss seen on OCTA with severity and/or quantification of peripheral retinal ischemia, and allow for risk stratification.

DECLARATION OF PATIENT CONSENT

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. D'Amico G. The commonest glomerulonephritis in the world: IgA nephropathy. Q J Med. 1987;64(245):709-27.
2. Feehally J, Floege J. IgA Nephropathy and Henoch–Schonlein Nephritis. In: J Feehally, J Floege, RJ Johnson (eds). Comprehensive Clinical Nephrology. 3rd edn Philadelphia, PA, USA: Mosby Elsevier. 2007;253–264.
3. Nomoto Y, Sakai H, Endoh M et al. Scleritis and IgA nephropathy. Arch Intern Med 1980;140:783–785.
4. Endoh M, Kaneshige H, Tomino Yet al. IgA nephropathy associated with myasthenia gravis and scleritis. Tokai J Exp Clin Med 1981;6:421–425.
5. Chung YM, Sheir CT, Lee SM. Scleritis in IgA nephropathy: A case report. Zhonghua Yi Xue Za Zhi 1995;56:279–282.
6. Noma H, Funatsu H, Sakata K, et al. Macular microcirculation in hypertensive patients with and without branch retinal vein occlusion. Acta Ophthalmol. 2009;87 (6):638–642, DOI:10.1111/j.17553768.2008.01318.x, indexed in Pubmed: 18631327.
7. Keilani C, Halalchi A, Wakpi Djeugue D, et al. Retinal oximetry during treatment of retinal vein occlusion by ranibizumab in patients with high blood pressure and dyslipidemia. J Fr Ophtalmol. 2016;39(10): 816–821. DOI: 10.1016/j.jfo.2016.08.007, indexed in Pubmed: 27865689.
8. Menezes VP, Cohen C, Del-Rei J, et al. Evaluation of endothelial function.
9. Keung Y, Owen J. Iron deficiency and thrombosis: literature review. Clin Appl Thromb Hemost. 2004;10(4):387–91.
10. Ogata T, Kamouchi M, Kitazono T, et al. Cerebral venous thrombosis associated with iron deficiency anemia. J Stroke Cerebrovasc Dis. 2008;17(6):426–8.
11. Kaur C. Hypoxia-ischemia and retinal ganglion cell damage. Clin Ophthalmol. 2008;2(4):879. Available:https://doi.org/10.2147/opth.s3361.
12. Tekin D, Yavuzer S, Tekin M, Akar N, Cin S. Possible effects of antioxidant status on increased platelet aggregation in childhood iron-deficiency anemia. Pediatr Int. 2001; 43(1):74–7.

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