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CASE REPORT

The Therapeutic Predicament of Branch Retinal Vein Occlusion in uncontrolled Hypertension in Pakistan

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SUMMARY

Around the globe, hypertension is a silent pandemic. After diabetic retinopathies, hypertensive retinal vascular diseases are one of the most prevalent. In Pakistan, diagnostic investigations due to their cost are not done on regular screening basis as the financial burden of management is a point at issue. Thus, such conditions can often go undetected or therapeutically compromised.

Here is a case of unilateral ischemic branch retinal vein occlusion in a 48-year-old male with a history of uncontrolled hypertension and diabetes. Additional laboratory investigations were carried out including complete lipid profile, serum urea and creatinine levels. Fundus fluorescein angiography and optical coherence tomography was done. The patient was managed on anti-VEGF injections and Argon Laser. Follow-up along with a multi-disciplinary management approach is essential for the best possible prognosis.

Keywords: hypertension, central retinal vein occlusion (CRVO), branch retinal vein occlusion (BRVO)

INTRODUCTION

Blockages in the retinal venous vessels when occurring in the main retinal vein is referred to as a central retinal vein occlusion (CRVO), while a blockage in the smaller vein is called a branch retinal vein occlusion (BRVO).[1] The first case of BRVO was reported in 1877 by the German ophthalmologist Theodor von Leber. The Beaver Dam Eye Study found a 15-year cumulative risk of BRVO to be 1.8%, three times more than CRVO at 0.5%.[2] A pooled data from population studies from the United States, Europe, Asia and Australia found BRVO to have a higher prevalence in Asians and Hispanics and lower in Caucasians, though not carrying a statistical significance. [3]

BRVO is characterized with sudden, painless vision loss. If the affected area is not ocularly central, it will be unnoticed as it will go asymptomatic. In rare undetected cases, visual floaters from a vitreous haemorrhage can be the main presenting symptom due to retinal neovascularization. Arterial compression of the vein at arteriovenous crossings, alongside pre-existing systemic cardiovascular risk factors, subsequently leading to thrombus formation is currently thought to be the main pathological event initiating BRVO.[1-3] Other prevalent etiological risk factors, include uncontrolled high blood pressure, increased body mass index, hyper-coagulability, diabetes and glaucoma. After diabetic retinopathy, amongst vascular retinal diseases, it is commonly seen in middle aged and the elderly, but remains a challenge in the field of ophthalmology, due to limited treatment options.

CASE PRESENTATION

A 48-year-old married male, driver by profession, presented with a history of sudden profound deterioration of vision in left eye for 48 hours, which was not improving. He is a known diabetic for the last 7 years and hypertensive for the past 16 years with no strict adherence to the medications prescribed for it. The patient has a history of chronic smoking for 27 years. The patient was afebrile and his blood pressure measurement was 160/95 mmHg upon presentation. Other vital signs, included a pulse of 68bpm and respiratory rate of 18 breaths per minute. Laboratory investigations, including 14-hour fasting lipid profile were completed and showed: random blood sugar 231 mg/dL, HDL 47 mg/dL, LDL 160mg/dL, triglycerides 125mg/dL, and serum urea 38mg/dL.

Ophthalmologic exam showed best corrected visual acuity of counting fingers at a distance of 2 feet in the left eye and 6/9 in the right eye. Pupillary exam showed sluggish left pupil with relative afferent pupillary defect of grade 1, and a round, reactive pupil on the right. Intraocular pressures were measured to be 14 mmHg in both eyes. Fundus examination of left eye revealed flame shaped haemorrhages, cotton wool spots, macular oedema along with dilated and tortuous vessels of the superotemporal arcade *(see **Figure 1a**)*. Fundus examination of the right eye was within normal limits. Fundus fluorescein angiography of the left eye revealed delayed filling of the occluded retinal vein alongside areas of capillary non-perfusion *(see **Figure 1b**)*. Optical coherence tomography (OCT) of the left eye demonstrated cystoid macular oedema *(see **Figure 1c**)*.

Differential diagnoses for the left eye, included BRVO, CRVO, diabetic retinopathy and acute hypertensive retinopathy.

Ocular management planned out for the patient included monthly intravitreal injections of anti-VEGF and Argon laser to treat ischemic retina. Patient was also advised to follow-up closely during the next six months, to monitor any possible complications. According to the multi-disciplinary approach, the patient was referred to the medicine unit for the proper control and management of hypertension.

DISCUSSION

BRVO is classified into 2 main types; major and macular. Major BRVO is the occlusion of a vein that drains one of the retinal quadrants, while the occlusion of a vein involving the macula is the Macular BRVO. Although the exact mechanism of BRVO has not been completely understood, it is known to start with narrowing of the venous lumen at the sites of arteriovenous crossing and follows the principle of Virchow's triad to incite thrombus formation. [4] These crossings are an intersection between retinal artery and vein. They share a common sheath of connective tissue. So, when the artery loses its flexibility, as with atherosclerosis (hardening of the arteries), the vein is compressed. The narrowed vein experiences turbulent blood flow that promotes clotting, leading to a blockage or occlusion. Thus, there is thrombus formation secondary to underlying clotting.[4] This curtails blood drainage and may lead to fluid leakage in the center of vision and poor perfusion flow to the blood vessels, causing macular oedema and ischemia.

Furthermore, disorders such as deficiency of Protein C and S, deficiency of antithrombin III, or a genetic mutation in prothrombin gene, antiphospholipid antibodies and hyperhomocysteinemia are other possible pathophysiologic mechanisms of BRVO. [5]

A multitude of risk factors have been considered in BRVOs, including diabetes mellitus, atherosclerotic disease, hypertension, open angle glaucoma, connective tissue pathologies,

thrombophilia and other causes of hyper-coagulability. In a study carried out to determine the relation between RVO and systemic hypertension, diabetes and hyperlipidemia, the percentage of cases with any form of RVO attributed to hypertension and hyperlipidemia was 47.9% and 20.1% respectively as compared to diabetes contributing only 4.9%, concluding that the association of diabetes alone with any form of RVO was not significant.[6]

Currently, there are three anti-VEGF drugs available to treat BRVO and randomized controlled trials have also reported significant results with them. However, nearly 50 patients require repeated injections for up to 4 years to sustain visual improvements.[7] One of these drugs is Bevacizumab, which is a recombinant humanized monoclonal antibody that inhibits the Vascular Endothelial Growth Factor A (VEGF-A). It is also found effective in controlling macular oedema secondary to BRVO. Another agent is Ranibizumab, which is a monoclonal Fab-fragment which binds to all forms of VEGF. A study showed that 55-61% of patients receiving 0.3 mg and 0.5 mg of Ranibizumab for the treatment of macular oedema secondary to BRVO, experienced a three-line improvement in vision.[8]

The third drug is a triple action drug; Aflibercept. It is a recombinant fusion protein which acts by inhibiting the subtypes VEGF-A, VEGF-B as well as placental growth factor (PGF). In a prospective study carried out to assess the efficacy of Aflibercept for macular oedema due to BRVO, it was concluded that the Intravitreal Aflibercept (IVA) therapy significantly improved best corrected visual acuity (BCVA) and effectively reduced the number of injections required along with hospital visits.[9]

The use of these drugs requires frequent visits to the attending consultant with injection schedules varying from case to case. Laser treatment may be considered alongside anti-VEGF therapy in cases presenting with macular oedema which involves applying light laser pulses to the macula in a grid pattern. A multi-center clinical trial demonstrated vision improvement in approximately two-thirds of patients following this treatment. Intraocular steroid injections with dexamethasone also show satisfactory outcomes in patients not responding to anti-VEGF therapy with clinical trials showing that approximately 30% of BRVO patients enjoyed significant visual improvements following steroid therapy using a slow-release steroid implant in the eye.

While intra-ocular steroids do generally carry side effects such as an increase in intra-ocular pressure and/or cataract formation, in most cases, these can be managed effectively. Overall, BRVO serves a favourable prognosis with over 60% of patients, maintaining a vision of more than 20/40 after 1 year of treatment.

CONCLUSION

This case presentation of unilateral branch retinal vein occlusion was deduced to have a multi-factorial etiology. This creates the therapeutic dilemma of treating underlying causes. However, hypertension was the most probable triggering factor and one that needs to be controlled. Monthly intravitreal injections of anti-VEGF along with Argon laser therapy were planned out to treat the ischemic retina and lifelong anti-hypertensive management was ensured for effective control. Close follow-up was emphasized to prevent further complications. Patient's health results as well as disease prognosis can be improved with awareness regarding lifestyle changes such as regular aerobic exercise, dietary modifications including salt restriction, reducing alcohol intake and/or smoking and highlighting the importance of adherence to medication.

Conflict of interest: None

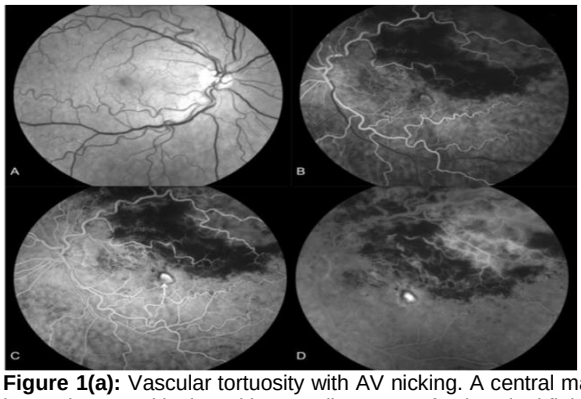

Figure 1(a): Vascular tortuosity with AV nicking. A central macular hypo-pigmented lesion with a small amount of sub-retinal fluid OS

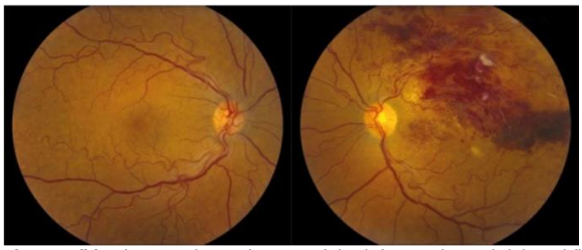

Figure 1(b): Fluorescein Angiogram of the left eye showed delayed filling of occluded vein and areas of ischemic peripheral retina.

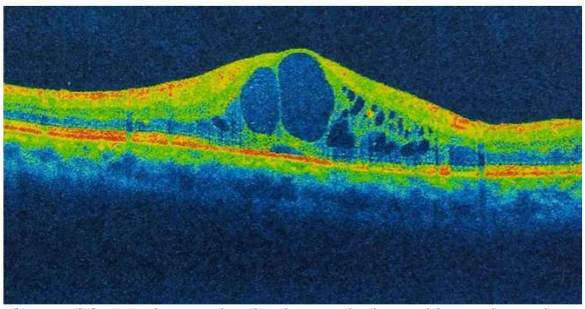

Figure 1(c): OCT image showing intra-retinal cystoid macular oedema

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