Science Publications

Global Journal of Medical Case Reports, 2022, 2, 481
www.scipublications.org/journal/index.php/gjmcr
DOI: 10.31586/gjmcr.2022.481

Case Report

Branch Retinal Vein Occlusion: A Case Report

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Abstract: To report a case of a major branch retinal vein occlusion (BRVO) in the superotemporal region with superior macular involvement and a review of its natural history. This is a retrospective case presentation of a 49-year-old African Caribbean female patient presented to Accident and Emergency with unilateral persistent acute history of reduced central vision of left eye for 10 days with no other associated symptoms. Treatment options included using scatter (pan-retinal) laser photocoagulation followed by intravitreal injections as the area of non-perfusion is >5-disc area. It is warranted to understand the risk factors associated with BRVO and to develop appropriate treatment and follow-up plans.

Keywords: Central retinal vein occlusion, Branch retinal vein occlusion, Macular oedema, Anti-vascular endothelial growth factor

How to cite this paper:
Soliman, N. (2022). Branch Retinal Vein Occlusion: A Case Report. *Global Journal of Medical Case Reports*, *2*(1), 9–15. Retrieved from https://www.scipublications.com/journal/index.php/gjmcr/article/view/481

Received: September 12, 2022
Accepted: October 26, 2022
Published: October 28, 2022

1. Introduction

Branch retinal vein occlusion (BRVO) is the second most common retinal vascular abnormality after diabetic retinopathy.[1] BRVO can be classified into two clinical entities: major BRVO and macular BRVO.[2] Major BRVO occurs because of occlusion of one of the four main branch retinal veins. It affects the whole quadrant of the retina drained by that vein to the peripheral retina.[2] In majority of eyes, BRVO appears more in the temporal than the nasal quadrant of the retina due to the high number of arteriovenous crossing spots.[1] The quadrant of the retina that is most affected is the superotemporal quadrant.[3] Macular BRVO on the other hand is caused by an occlusion to a venule in the macular region[2], with the superior macular area being affected more than the inferior macular area.[4] BRVO can be additionally categorised as ischemic BRVO or nonischaemic BRVO.[5] Ischemic BRVO is defined as ≥5 disc areas (DA) of retinal capillary dropout on standard fundus fluorescein angiography (FFA).[6] In this case report, we present a case of major BRVO in the superotemporal region with superior macular involvement and a review of its natural history.

2. Case Report

A 49-year-old African Caribbean female presented to Accident and Emergency department with persistent acute history of reduced central vision of left eft eye for 10 days. There were no other associated symptoms. She had no visual changes or symptoms in the right eye. Her past medical history included hypertension, diabetes type, and obesity. Her diabetes and hypertension are not well controlled. In terms of ocular history, she has been using reading glasses for 5 years and has been following-up with the retinal screening service annually for the past 6 years. She worked as a pastry chef and was the sole carer for her family. She is a heavy smoker but was trying to cut down her smoking consumption. She had 3 siblings all of whom had a history of hypertension and are diabetes. Her father passed away at the age of 50 due to a heart attack. Her mother, who

had a history of glaucoma and suffered from loss of vision bilaterally in the last 5 years of her life, died at the age of 60 due to a stroke.

Full ocular examination was completed upon presentation: visual acuity (VA) with glasses was 6/6 in the right eye and 6/36 in the left eye. The best corrected visual acuity (BCVA) for the right eye was 6/6, and 6/24 for the left eye. Relative afferent pupillary defect (RAPD) and Ishihara were normal. Lids, conjunctiva, sclera, cornea, anterior segment and lens were normal. Intraocular pressure (IOP) was 13 in the right eye and 17mmHg in the left eye. Following mydriasis, fundoscopy of the left eye (Figure 1) revealed dilatation and tortuosity of the superotemporal quadrant veins with flame-shaped and dot/ blot haemorrhages at the same region. Arteriovenous crossing points, cotton wool spots, collateral vessels and exudates were present. The size of the affected area was >5 DA. Fundoscopy of the right eye is otherwise normal.

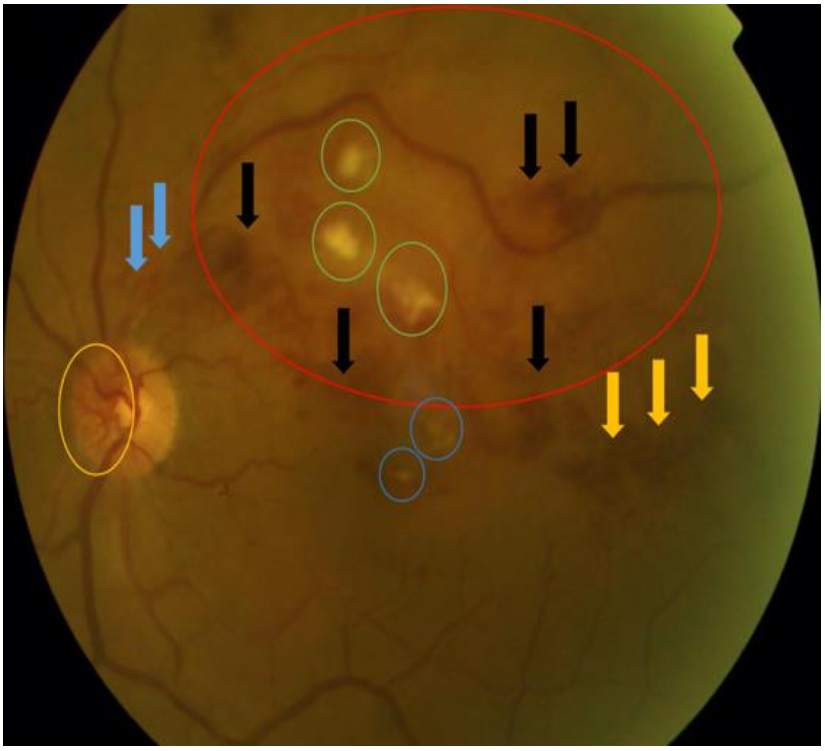

Figure 1. Fundoscopy of the left eye revealed dilatation and tortuosity of the superotemporal quadrant veins (red circle) with flame-shaped and dot/ blot (black arrows) haemorrhages in the same region. Arteriovenous crossing points (blue arrow), cotton wool spots (green circles), collateral vessels (yellow arrows) and exudates (blue circles) were present. The size of the affected area was >5 DA. There were collateral vessels on the nasal side of the optic disc (yellow circle). The central macula appeared oedematous.

FFA (Figure 2) revealed dilatation and tortuosity of the superotemporal veins. Early phase FFA (Figure 2A) staining demonstrated delayed venous filling. There were hypofluorescent massive areas of capillary non-perfusion (>5 DA) of the superotemporal quadrant and the superior region of the central region. Mid phase FFA (Figure 2B) clearly demonstrated vessel wall staining and hypofluorescence representing capillaries dropout. Collaterals formation could be spotted as well. There was hyperfluorescence on the nasal side of the optic disc. In late stages (Figure 2C-F), prominent hypofluorescence capillary remodelling of the peripheral vasculature superotemporally.

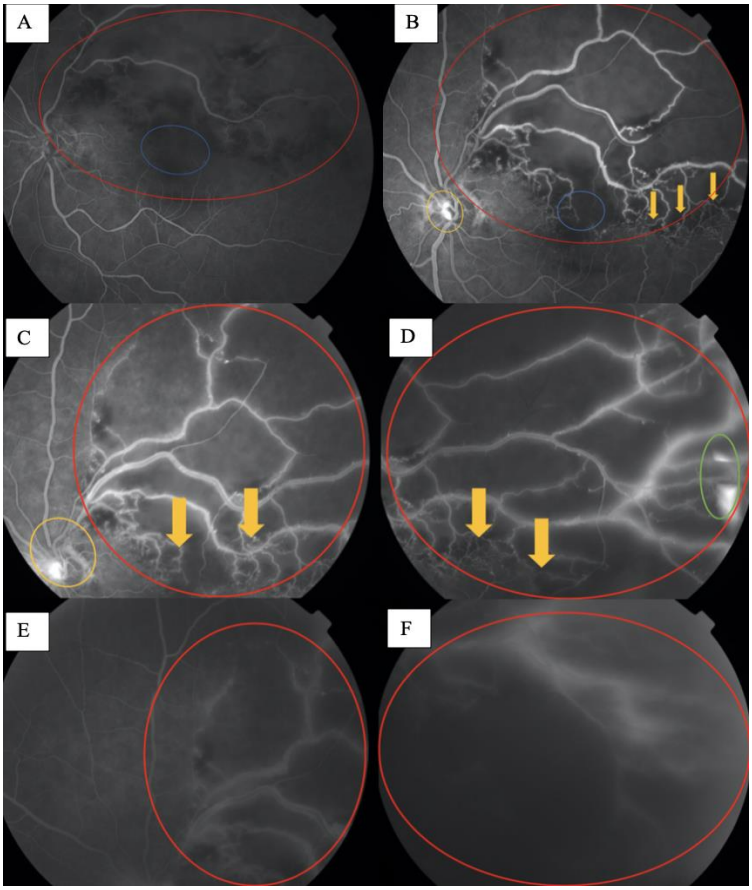

Figure 2. Fundus Fluorescein angiography (FFA) for left eye. A) Early phase FFA staining demonstrates delayed venous filling. There were massive areas of hypofluorescent areas of capillary non-perfusion of the superotemporal quadrant (red circle) and the superior central region (blue circle). B) Mid phase FFA clearly demonstrated vessel wall staining and hypofluorescence representing capillaries non-perfusion/ dropout (red circle). Collaterals formation could be spotted as well (yellow arrows). There was hyperfluorescence on the nasal side of the optic disc (yellow circle). C-F) Late phase FFA stages, illustrated prominent hypofluorescence and capillary remodelling of the peripheral vasculature superotemporally (red circle). Collaterals formation could be spotted as well (yellow arrows). There is hyperfluorescence superotemporally (green circle). The non-perfusion and blockage superotemporally on FFA corresponded to the intraretinal haemorrhage on the fundus photography in Figure 1.

In terms of her physical examinations, the patient was found to have mild hypertension (BP was 140/90 mmHg), normal pulse rate (78bpm) and normal postprandial blood sugar level. Baseline blood lab requested previously by her GP showed low Hb, microcytic anaemia, high ESR, high urea, high creatinine, high HbA1c, normal CRP, normal TFTs and normal LFTs. A clinical diagnosis of left eye superotemporal ischemic branch retinal vein occlusion with superior macular involvement was made on the basis of our findings.

3. Epidemiology

BRVO is the second most common acquired retinal vascular abnormality following diabetic retinopathy[1] with 0.5-1.2% incidence overall.[7] There is no data from England or Wales regarding the prevalence or incidence of BRVO.[8] In 2008, US data stated a 15 year incidence of 1800 BRVO cases per 100,000 population.[9]

4. Pathogenesis

The chief cause of BRVO is thought to be arterial compression of the vein at the intersection. This results in compression of the vein, leading to turbulent flow. This in addition to retinal endothelial injury due to various conditions such as atherosclerosis, hypertension, inflammation, hypercoagulopathy disorders which create a local environment for the formation of intravascular thrombus.[3] Due to interrupted venous flow, retinal ischemia proceeded downstream from the occlusion site.[4] Retinal non-perfusion/ ischemia is the most crucial aspect for vascular endothelial growth factor (VEGF) expression and new vessels formation.[4]

5. History and Clinical Examination

Major BRVO is often asymptomatic yet it can present with painless sudden onset monocular loss of vision or peripheral field loss corresponding to the perfusion region of obstructed vessels.[3] The average VA reduction in ischemic BRVO is 6/15, and nonischaemic BRVO is 6/18.[5] Macular BRVO symptoms are similar to major BRVO, however, macular BRVO presents with central visual defect,[5] and monocular visual distortions on grey vision.[4] Some patients may present with floaters because of vitreous haemorrhage (VH).[4] The degree of macular involvement defines the visual impairment level, which may result from ischemia and/ or secondary macular oedema.[10] It is vital to document a history of the risk factors associated with BRVO. This patient had multiple risk factors for developing BRVO, including a history of systemic hypertension,[3, 8, 9, 11] diabetes,[3, 8, 9, 11] smoking,[3] renal failure[12] and high BMI.[13] Hyperlipidaemia is another risk factor for BRVO that needs to be investigated.[14] Both the patient's parents passed away due to atherosclerotic conditions; her father passed away due to a heart attack and her mother passed away due to stroke. BRVO is associated with increasing risk of atherosclerosis, thus patient should be evaluated further regarding her risk of developing other atherosclerotic conditions besides hypertension.[8] She also had a maternal family history of glaucoma. Glaucoma[3, 8] is another risk factor that needs to be carefully addressed when following up the patient and in the management plan. Our patient was ≤50 years old, which warranted further evaluation for other risk factors related to BRVO.[11] These include thrombophilia[11] (such as hyperhomocysteinemia[8, 11] and antiphospholipid antibody syndrome, especially if there is a history of miscarriage[11]) and other blood coagulation disorders such as leukaemia, macroglobulinaemia, Waldenstrom's, myeloma, myelofibrosis and Factor V Leiden.[8] Systemic inflammatory disorders[8] (such as Behçets disease, sarcoidoisis, Goodpasture's Syndrome, polyarteritis nodosa and Wegener's Granulomatosis), connective tissue diseases,[11] systemic vasculitis[13] (such as systemic lupus erythematosus, sarcoidosis, and syphilis) and drug history (oral contraceptive use[11] and diuretics[13]) should be investigated. Besides random blood sugar, and haemoglobin A1C, FBC with differential and platelets, ESR, RFT and TFT, the patient should have her lipid profile and coagulation profile with PT/PTT checked.[3] Furthermore, this patient should undergo a thorough physical and systemic examination, including measuring blood pressure, ECG, carotid doppler and transoesophageal echocardiogram to rule out cardiovascular causes of vision loss.[3]

Typical acute fundoscopic examination findings include flame haemorrhages, dot and blot haemorrhages, hard exudates, cotton wool spots, dilated tortuous veins and retinal oedema.[5] Bonnet sign-haemorrhage at an AV crossing can be present.[5] Chronic BRVO findings include sclerosed veins, hard exudates, telangiectatic vessels, microaneurysms, collateral vascular shunt at the optic disc, tractional retinal detachment, vascular sheathing, VH, retinal neovascularization, iris neovascularization and optic disc neovascularisation.[3] An indirect ophthalmoscopy exam should be

performed to check for neovascularisation, which was not done for this patient.[3] Careful undilated iris and angle examinations for early rubeosis and neovascular glaucoma are also warranted.[3] Ultra-wide field (UWF) is crucial for measuring areas of ischemic retina, particularly in the periphery.[15] BRVO is diagnosed clinically using optical coherence tomography (OCT) and FFA. OCT (Figure 3) is used to quantify the macular oedema and measure central macular thickness to assess for cystoid macular oedema.[5] Information from OCT would have been helpful if it was available form this patient. Optical coherence angiography (OCTA) can be used to provide quantitative information of vascular alterations in BRVO. In BRVO, the foveal avascular zone (FAZ) revealed significant morphological changes and reduction of vessel density in the surrounding region.[17] FFA is used to assess peripheral and central ischemia, vascular leakage and neovascularisation.[18] It will also help to differentiate between new vessels and collateral vessels.[16] The characteristic FA for BRVO is late staining of vessels wall, delayed arteriovenous transit time and dye extravasation due to intraretinal haemorrhages.[16]

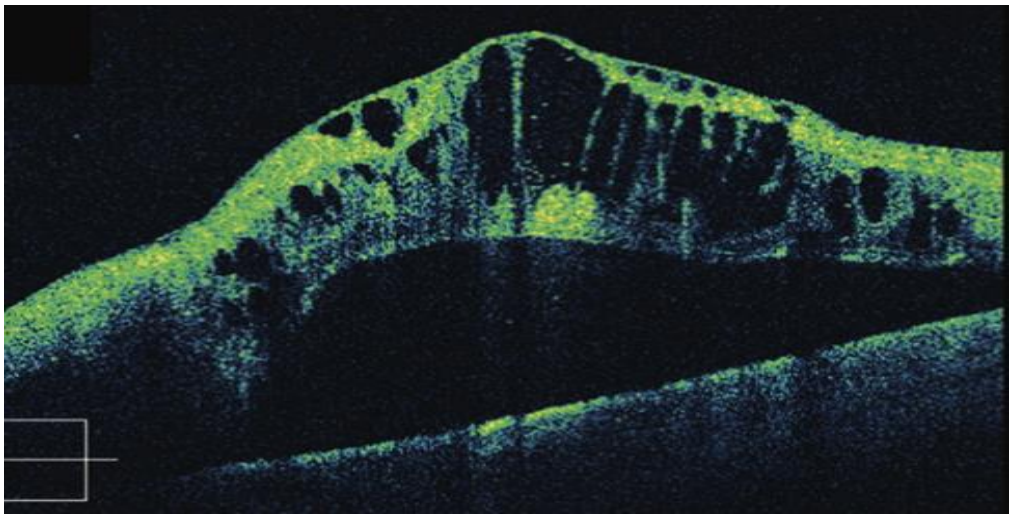

Figure 3. OCT of the macula shows cystoid maculopathy with neurosensory detachment.[16]

6. Differential Diagnosis

Differential diagnosis for this case includes diabetic retinopathy and hypertensive retinopathy. Both of these conditions may present with dot/blot haemorrhages and microaneurysms like BRVO.[3] Unlike BRVO that occurs unilaterally and respects the horizontal raphe, they usually occur bilaterally and extend across the horizontal raphe.[3]

7. Management

The purpose of BRVO treatment is to prevent complications that cause loss of vision such as macular oedema, macular ischemia, NV, VH and RD and management of those complications.[3, 5] Systemic risk factors should be addressed with appropriate special referral when warranted as mentioned previously.[3] For our patient, according to the Branch Vein Occlusion Study (BVOS) study, scatter (pan-retinal) laser photocoagulation must be considered as a first management option as the area of non-perfusion is >5 disc area.[19] It will also reduce the risk of VH from 60% to 30% and lower the risk of developing neovascular glaucoma in case of iris NV.[19]

Adjunctive pharmacological management such as intravitreal injections of anti-VEGF therapy ranibizumab (Lucentis)[20] and aflibercept (Eylea)[21] and/or corticosteroids[22] can be administered if OCT confirms the presence of CMO.[3] Intra-

ocular implant releasing dexamethasone (Ozurdex) is another effective and safe treatment option in RVO-related CMO.[22] As our patient had a maternal family history of glaucoma, intravitreal corticosteroids should not be used as they will elevate the intraocular pressure.[22] Their use can also increase the risk of cataract formation.[22] This treatment option is advisable if patients are pseudophakic and anti-VEGF cannot be used, for example in the case of pregnant patients.[22] Ozurdex should be avoided as it elevates her risk of developing glaucoma.[22] Pars plana vitrectomy can be indicated if she developed non-clearing VH or RD.[3] She should be followed up at 3 month intervals for 24 months.[8]

8. Prognosis

BRVO often has a good prognosis: 50% to 60% of eyes have a final VA of 20/40 or better even without management. Crucial prognostic factors for final VA are initial VA and level of foveal and macular involvement. However, as this patient presented with ischemic BRVO with >5-disc area, she has a 36% chance of developing NVE/NVD and a 60%-90% chance of developing VH if laser photocoagulation is not performed.[3]

Consent: Informed consent was obtained from the patient to publish their anonymised data.

Interest of conflict: Author declares no interest of conflict.

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