

Simultaneous Central Retinal Vein Occlusion and Branch Retinal Artery Occlusion in a Young Patient with Gout

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Abstract

Purpose: To report a patient with combined, unilateral BRAO and CRVO that appeared after taking oral etoricoxib and diclofenac for 2 weeks for the first gouty attack.

Methods: Observational case report

Patient: A 34-year-old Chinese man

Results: The BRAO resolved angiographically one day after high dose oral steroids were given. The best corrected visual acuity in the right eye was 20/20 with complete resolution of retinal hemorrhages two months later

Conclusion: Simultaneous central retinal vein occlusion and branch retinal artery occlusion may be associated with oral non-steroidal anti-inflammatory drugs and gout.

Keywords: BRAO; CRVO; Gout; Simultaneous

Summary Statement

A 34 year old Chinese man without cardiovascular risks suffered from simultaneous central retinal vein occlusion (CRVO) and branch retinal artery occlusion (BRAO) after taking non-steroid anti-inflammatory drugs for two weeks for the first gouty attack. The BRAO resolved angiographically one day after high dose oral steroids were given. The systemic medical history is important in young patients who present with simultaneous CRVO and BRAO.

Simultaneous central retinal vein occlusion (CRVO) and branch retinal artery occlusion (BRAO) is rare. It has been associated with inherited plasminogen deficiency and high lipoprotein(a) levels, [1] as well as drugs like pegylated interferon plus ribavirin combination therapy [2]. To the best of our knowledge this is the first report of combined CRVO and BRAO in a young man taking oral non-steroidal anti-inflammatory drugs (NSAIDs) for his first gout attack.

Case Report

A 34 year old Chinese man presented with blurred vision in the right eye vision with a progressive right inferonasal field defect after taking oral non-steroid anti-inflammatory drugs. His past health was good except for a recent attack of right big toe pain that was diagnosed as gout by his family doctor. His serum uric acid level was 0.48 mmol/l (normal range: 0.24-0.49 mmol/l). He was started on Arcoxia (Etoricoxib) 120 mg QD for five days, followed by Voltaren

(Diclofenac) 75 mg BID for 5 days, and then switched back to Arcoxia 120 mg QD for another 4 days. The vision in both eyes remained good through 2 weeks of the treatment so the oral treatment was discontinued. On examination, best correct visual acuity was 20/25 OD, 20/15-1 OS. The pupil reactions, ocular motility, anterior segments, and intraocular pressures were normal bilaterally.



Figure 1: Right eye. Fundus photo on the day of presentation showing a combined CRVO and superotemporal BRAO with diffuse scattered retinal hemorrhages and tortuous dilated retinal veins in all four quadrants, pre-retinal blood over inferior optic disc, and retinal whitening along superotemporal arcade over superior macula.

The fundus examination of the right eye revealed a combined CRVO and superotemporal BRAO with diffuse scattered retinal hemorrhages and tortuous, dilated retinal veins in all four quadrants, pre-retinal blood over the inferior optic disc, and retinal whitening along the superotemporal arcade and superior macula (Figure 1). Retinal hemorrhages were not greater in the sector of the BRAO.

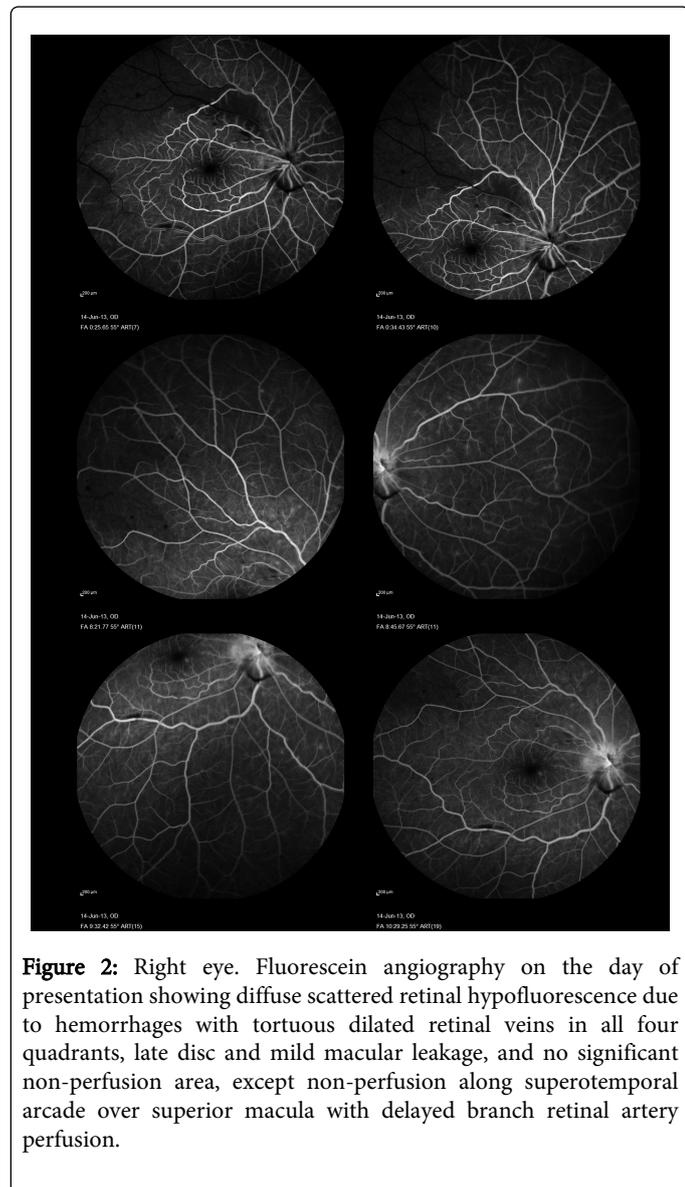


Figure 2: Right eye. Fluorescein angiography on the day of presentation showing diffuse scattered retinal hypofluorescence due to hemorrhages with tortuous dilated retinal veins in all four quadrants, late disc and mild macular leakage, and no significant non-perfusion area, except non-perfusion along superotemporal arcade over superior macula with delayed branch retinal artery perfusion.

No embolus was found. The left fundus was unremarkable. A fluorescein angiogram revealed diffuse, scattered retinal hypofluorescence due to hemorrhages with tortuous, dilated retinal veins in all four quadrants, late disc and mild macular leakage, and no significant areas of non-perfusion except along the superotemporal arcade and over the superior macula associated with delayed and pulsating branch retinal artery perfusion (Video, Figure 2). Several blood pressure measurements were normal and the patient was afebrile. Rheumatology and cardiology consultations were obtained. Blood tests including a complete blood count, erythrocyte sediment rate, fasting glucose, lipid profile, liver and renal function tests, VDRL, rheumatoid factor, ANA, anti-cardiolipin antibody, ANCA, factors II,

V, VIII-XII, von Willebrand factor antigen, fibrinogen, fibrinogen degradation product, D-dimer, antithrombin III, lupus anticoagulant ratio, protein C, protein S, and activated protein C resistance ratio were all normal. Chest X-ray and urinalysis were normal. Echocardiogram and electrocardiogram were normal. The patient refused imaging of the brain and orbit. Embolic, and systemic inflammatory, and infectious causes were deemed unlikely. One day later, oral prednisolone 30 mg BID, zantac 150 mg BID and slow K 600 mg BID were started. One day later, the patient reported decreasing right visual field loss. A repeat fluorescein angiogram revealed a resolved BRAO with a with stable CRVO. The steroid was continued for 3 days and gradually tapered and discontinued. Two months later, the best corrected visual acuity in the right eye was 20/20 with resolved retinal whitening, but thinning of the retinal nerve fiber layer along the superotemporal arcade. The retinal hemorrhages had resolved.

Comment

Duker et al first reported seven patients with simultaneous CRVO and BRAO [3]. Three of them were young (ages: 20, 24 and 42 years; 1 female, 2 male) and without systemic diseases. One of them received intravenous corticosteroids, and the other two received no treatment. All three cases had good final vision, ranging from 20/20 to 20/30 at 2-7 months after the onset of disease. The authors suggested that an underlying inflammatory condition of the optic nerve (optic neuritis) caused secondary impairment of the retinal vasculature. This was supported by the presence of afferent pupillary defect in all three patients. In our patient, however, there was no afferent pupillary defect on presentation. Furthermore, he had already received oral NSAIDs for 2 weeks before the occlusions. We speculated that the simultaneous CRVO and BRAO in our patient might be related to gout and/or oral NSAIDs.

Ocular complications of gout are rarely reported. A case with bilateral uveitis, increased intraocular pressure, and blurred disc margins occurred with an gout attack was published [4]. Hyperuricemia is not commonly considered a risk factor for cardiovascular disease. However, recent studies suggest that uric acid is biologically active and can stimulate oxidative stress, endothelial dysfunction, inflammation, and vasoconstriction. Epidemiologic studies have found that uric acid can independently predict the development of hypertension, as well as stroke and heart failure [5]. High serum levels of uric acid also predict severity of coronary artery disease in nondiabetic, nonhypertensive patients with acute coronary syndrome [6].

A thrombophilic effect of oral NSAIDs, due to an imbalance of prothrombotic and antithrombotic factors, has been reported [7]. The cardiovascular risk of thrombosis among patients who were treated on a long-term basis with a coxib (etoricoxib) was shown to be similar as the risk observed in patients receiving a conventional NSAID (diclofenac) [8].

When a BRAO is combined with a CRVO, it is usually due to a compression of the artery because of the markedly swollen optic nerve and/or central retinal vein. This might explain why the BRAO subsided shortly after high dose oral corticosteroids were given to our patient. Our case report highlights the importance of the medical and drug history of patients who present with simultaneous CRVO and BRAO.

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