Case Report

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All that Glitters is not Gold: A Misdiagnosed Case of Retinopathy

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Abstract

Central retinal vein occlusion (CRVO) and diabetic retinopathy are the commonest causes of retinopathies, among several others. The fundus findings alone will sometimes lead to a misdiagnosis. The attending doctor who sees hemorrhages, exudates, venous changes, neovascularization, and edema should not be in a hurry to jump to the diagnosis. All that glitters is not gold. As a clinician - It is very important to take a good history, examine the patient systemically and investigate as necessary, before arriving at a diagnosis, and then managing the patient appropriately. Here, a case is described, where a female in the seventh decade of life, who was visually handicapped at presentation, had undergone cataract extraction with intraocular lenses implanted. She denied having any medical problems in the past. Post-operative fundus evaluation showed features that were diagnosed to be diabetic retinopathy bilaterally. She underwent complete pan retinal photocoagulation in the right eye for neovascularization and macula edema. It was only a few visits later that the attending doctor repeated asking a detailed history and examination and ordered a few investigations and reversed the diagnosis to CRVO secondary to hypercholesterolemia and hypertension.

Key words: Diabetes, Diagnosis, Retinopathy

INTRODUCTION

Retinopathy is a presentation seen in a number of conditions and is more often than not, seen in diabetics. However, retinopathy is fairly common in adults without diabetes.[1-4] The fundus shows dot haemorrhages (DH), blot haemorrhages (BH) and flame hemorrhages, hard exudates (HE), cotton wool spots, neovascularization at the disc or elsewhere, venous tortuosity and dilatation. Fundus examination alone will not be able to diagnose the condition that the patient has, and the treating physician will have to be armed with adequate knowledge and a high index of suspicion in order to arrive at the correct diagnosis and management.

In central retinal vein occlusion (CRVO), thrombosis of the central retinal vein sets a cascade of events which will impede capillary perfusion and cause ischemia. In diabetic retinopathy - The retinal capillaries are affected. In both diabetic retinopathy and CRVO - elevated vitreous levels of vascular endothelial growth factor (VEGF) increases vascular permeability causing macula edema, capillary damage and retinal ischemia. VEGF promotes angiogenesis causing break down in the blood-retinal barrier, which stimulates vascular permeability in the ischemic retina. Several other factors such as insulin-like growth factor, hemodynamic changes, oxidative stress, and activation of the renin-angiotensin-aldosterone system have also been postulated in the pathogenesis of diabetic retinopathy.

Retinal vein occlusion (RVO) and diabetic retinopathy are both major causes of vision loss. Risk factors for retinopathy include systemic conditions like hypertension, arteriosclerosis, diabetes mellitus, hyperlipidemia, vascular cerebral stroke, blood hyperviscosity, and thrombophilia. A strong risk factor for RVO is the metabolic syndrome (hypertension, diabetes mellitus, and hyperlipidemia).[5]

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CASE REPORT

FD, a 76-year-old Malay woman, first presented to the Eye Clinic of Hospital Melaka on 23rd March 2012 with a complain of bilateral progressive blurring of vision since

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the past 2 years. She had been relatively well in the past with no ocular or systemic disorders. Her visual acuities were hand movement bilaterally. There was a brunescent cataract in the right eye and a white mature cataract in the left eye. The intraocular pressures and the rest of the anterior segments were unremarkable. The fundi could not be assessed due to the density of cataracts. B-scan ultrasonography showed flat retinas and clear vitreous cavities bilaterally.

Left phacoemulsification converted to extracapsular cataract extraction (ECCE) with sponge vitrectomy and sulcus posterior chamber intraocular lens (PCIOL) was done on April 9th, 2012. Intraoperatively, the surgery was complicated by zonular dehiscence and vitreous loss. Therefore, the phaco was converted to ECCE. Right ECCE with anterior vitrectomy and implantation of anterior chamber intraocular lens (ACIOL) was done on May 31st, 2012. Intraoperatively, there was a complication of posterior capsule rupture and vitreous loss.

The pupils were dilated, and the fundi were assessed on July 19th, 2012. The pupils were not dilated earlier as one eye had an ACIOL and the other had a PCIOL placed in the sulcus. Both eyes showed extensive DH and BH and HE. The right eye showed neovascularization between the superior and inferior temporal arcades as well as edema of the macula (Figure 1). No neovascularization was seen in the left eye (Figure 2). A diagnosis of right proliferative diabetic retinopathy (PDR) and left moderate non-PDR (NPDR) was made. The patient refused fundus fluorescein angiogram. Complete panretinal photocoagulation (PRP) was given for the right eye.

Almost 6 months later (on December 5th, 2012), the attending ophthalmologist decided to do a systemic review. There was no pallor, skin changes or peripheral neuropathy seen in poorly controlled diabetics. On further questioning, the patient denied ever having had diabetes mellitus. However, the patient admitted to having had high blood pressure 7 years earlier but had defaulted treatment. The blood pressure on that visit was controlled at 120/80 mmHg, but it was 170/110 mmHg on the next visit. The serum cholesterol was elevated (6.59) with an increase in low-density lipoprotein (4.26). The blood sugars were within normal limits each time; it was tested. Carotid Doppler ultrasound was normal bilaterally.

The diagnosis of the patient was reversed to bilateral CRVO secondary to uncontrolled hypertension and hyperlipidemia. The patient was referred to the physician for appropriate management of the medical conditions.

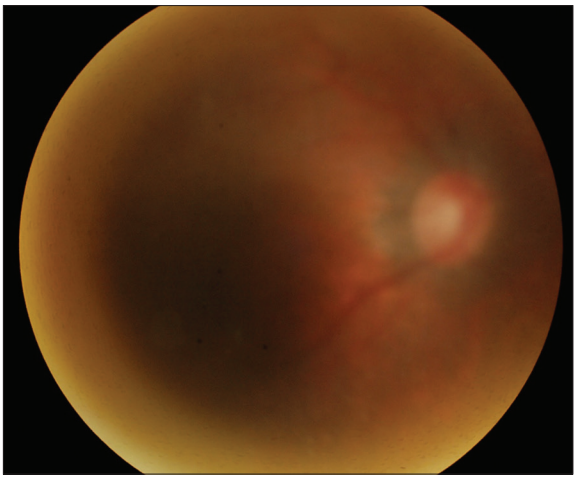

Figure 1: Right fundus

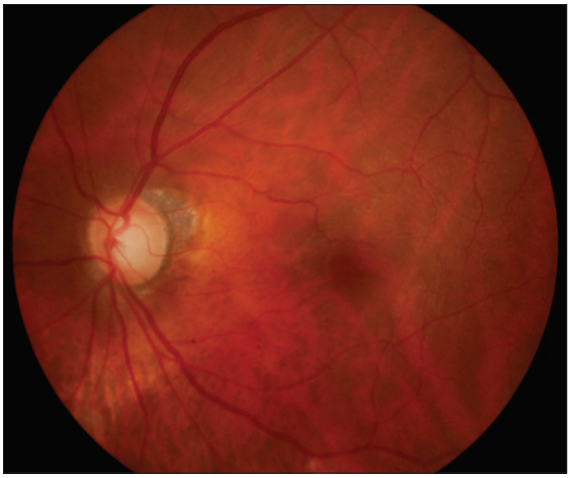

Figure 2: Left fundus photo

DISCUSSION

Fundus examination alone will not be able to diagnose the condition that the patient has, and the treating physician will have to be armed with adequate knowledge and a high index of suspicion in order to arrive at the correct diagnosis and management.

Ophthalmologically, both types of retinopathies are treated in the same manner.

Laser remains the therapy of choice when neovascularization secondary to CRVO is detected. Adjunctive anti-VEGF could be considered in managing neovascularization secondary to RVO in cases of vitreous hemorrhage.[6] PDR is also treated with laser and anti-VEGF. According to the CRVO study Macular grid photocoagulation was effective in reducing angiographic evidence of macular edema, but it did not improve visual acuity in eyes with reduced vision due to macular edema from CRVO. At present, the results of this study do not support a recommendation for macular

grid photocoagulation for macular edema.[7] Some anti-VEGF therapies, including bevacizumab, ranibizumab, and aflibercept have been shown to be effective in short-term studies for the treatment of patients with macular edema and CRVO.[8] CRVO study found that prophylactic PRP did not prevent the development of iris neovascularization and recommended to wait for the development of early iris neovascularization and then apply PRP.[9]

Royle *et al.*[10] recommend a trial of PRP for severe NPDR and early PDR compared with deferring PRP till the high risk-PDR stage. ETDRS Report No. 9 recommends that when retinopathy is more severe, scatter photocoagulation should be considered and usually should not be delayed if the eye has reached the high-risk proliferative stage.[11]

Using these guidelines, had we diagnosed the right eye of the patient to have CRVO and not low risk PDR with macula edema, we would have observed and not done the laser.

However, medically, the systemic disorders are treated based on the etiology of the disease. Hence, the disease must be diagnosed correctly in order to treat the underlying cause and to prevent further complications from occurring.

A diabetic may have symptoms of polyuria and polydypsia which are not seen in other conditions causing retinopathy. A simple dextrostix test done will show whether the patient has diabetes. A more sophisticated test would be to do hemoglobin A1c. Diabetics are treated with either oral hypoglycemic agents or insulin. The aim of treatment apart from arresting the disease would be to prevent complications such as nephropathy, dermatopathy and neuropathy apart from ocular complications such as neovascular glaucoma. Adequate blood sugar control and lifestyle modification including a low sugar diet will achieve this.

A patient who has high cholesterol will usually be asymptomatic. The treating doctor whether a family physician or general practitioner or ophthalmologist - Should do a blood lipid profile when a patient presents with retinopathy. Hypercholesterolemia will need life style modification of low fat diet in addition to statins specific in lowering cholesterol. The aim of treatment is to prevent complications such as cerebrovascular accidents and myocardial infarction apart from neovascular glaucoma.

Hypertension patients can be asymptomatic – As seen in our patient or can present with headaches and dizziness. Again, the treating doctor must be inquisitive enough to find out the cause of hemorrhages in the fundus and use the sphygmomanometer which can be done by the nurse. Again apart from lifestyle modification - The specific treatment to prevent complications is by using anti-hypertensive drugs.

CONCLUSION

All that glitters is not gold. The presence of hemorrhages in the fundus can be due to a wide variety of diseases, apart from diabetic retinopathy. The attending doctor should take a history, do a systemic examination and order relevant blood/radiological investigations to diagnose the condition correctly and treat it to prevent further complications from occurring. Prevention is better than cure.

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