ejpmr, 2022, 9(11), 497-498

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Study

ISSN 2394-3211

EJPMR

CASE OF DIABETIC RETINOPATHY WITH MARKED RETINAL ARTERIES SHEATHING

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Article Received on 03/09/2022 **Article Revised on 24/09/2022** **Article Accepted on 14/10/2022**

INTRODUCTION

Diabetic vascular disease is characterized by vascular calcification, and coronary artery calcification. Vascular calcification has traditionally been regarded as a process involving degeneration and necrosis of arterial walls, alongwith transformation of vascular wall cells due to hypoxia and inflammation that ultimately leads to calcification.[1] Reportedly, cartilage-like tissue has been found in vascular calcified lesions, suggesting that the process of vascular calcification may be quite similar to osteogenesis.[2] Here, we report a patient with proliferative diabetic retinopathy (PDR) who showed marked sheathing, thought to be caused by retinal artery calcification, after retinal redetachment following vitrectomy.

CASE REPORT

Case involved a 65-year-old female with highly active, untreated PDR in both eyes. Upon initial examination, visual acuity was 20/100 (uncorrected) in her right eye and 20/80 (uncorrected) in her left eye, and intraocular pressure was 15 mm Hg in her right eye and 13 mm Hg in her left eye. Examination of the anterior eye segment and the optic media showed no abnormalities. Funduscopy showed neovascularization of the optic disc in both eyes and preretinal hemorrhages around the optic disc in her right eye. In her left eye, fibrovascular membranes of the optic disc extended inferiorly, and partial traction retinal detachment (TRD) was evident. No retinal artery sheathing was observed at this time. Vitrectomy and cataract extraction was later performed for her left eye, and the fundus visualization was improved and the retina was reattached. However, after 1 month following the initial surgery, reRD due to reproliferation occurred from the posterior pole toward the midperiphery of 4 quadrants. At the time of this reRD, some well-demarcated sheathing of the retinal arterioles was intermittently observed, primarily at the posterior pole, yet no retinal vein abnormalities were seen. Silicone oil tamponade was performed at reoperation, but the sheathing persisted for more than 1 year after surgery. Fluorescein fundus angiography (FA) before the reoperation showed that blood flow was maintained at the sites of sheathing, with no dye leakage. Optical coherence tomography (OCT) after the reoperation showed high reflectance and remarkable acoustic shadows of the vessel walls corresponding to the areas of sheathing. Approximately 1 year later, TRD of the patient's right eye had also progressed, so vitrectomy was performed. However, after 2 months following the initial surgery, reRD due to reproliferation occurred from the posterior pole toward the midperiphery of 4 quadrants. Findings similar to those of her left eye were observed postoperatively.

DISCUSSION

The pathogenesis of the sheathing of retinal arterioles may involve retinal arteritis, arterial occlusion, and lipemia retinalis. However, our patients had no underlying diseases, and the FA findings revealed no leakage from blood vessels, thus ruling out vasculitis. Retinal artery occlusion was also ruled out because FA showed that blood flow was maintained. Lipemia retinalis was not likely because of normal lipid levels. A common feature in our patients was highly active PDR in which marked retinal artery sheathing had not been seen previously but was observed after RD. Blood flow was maintained in both patients, and OCT showed high reflectance and strong acoustic shadows of the vessel walls. Moreover, the lumens of the blood vessel were consistently hypointense. These findings suggest that the sheathing was due to vascular calcification. Vascular calcification is thought to be preceded by degeneration and apoptosis of VSMCs due to oxidative stress.[3] RAGE expression, which promotes ossification, is upregulated in the SMCs of retinal arterioles in PDR. Increases in the RAGE ligands of advanced glycosylation end-products (AGE), HMGB1, and S100 protein have also been reported in PDR.[4] Hyperglycemia also promotes ossification of VSMCs, thus leading to calcification. Histopathological analysis is necessary to confirm that the vessel sheathing in our patients was due to vessel calcification, but this is difficult. However, OCT is thought to be useful for examining retinal pathology. OCT of calcified coronary

arterioles shows well-defined areas of high intensity and heterogeneous low intensity within vessels.[5]

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