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Sir,
Choroidal thickness alterations in obstructive sleep apnea–hypopnea syndrome (OSAS)

We would like to congratulate Xin *et al*[1] on their prospective, observational case–control study of retinal and choroidal thickness (CT) evaluation by spectral domain optical coherence tomography in patients with obstructive sleep apnea–hypopnea syndrome (OSAS). The study presents, for the first time, CT alterations in OSAS patients; however, we think that some important issues need more thorough discussion.

The choroid of the eye is one of the most highly vascularized tissues of the body, and it is the major blood supply to the retina.[2] It is known that the choroid is prone to suffer from microvascular atherosclerotic changes and changes inherent to other microvascular systems.[3] However, the authors did not exclude the subjects having systemic diseases except diabetes, and about 50% of the participants were indicated to have hypertension. Also, hyperlipidemic subjects and smokers were not excluded, although both of them were shown to affect CT.[4,5] As body mass index of OSAS group in their study is significantly higher than that of controls and systemic diseases are often comorbid with OSAS, expecting hypercholesterolemia more in OSAS group is reasonable. It is obvious that including patients having hypertension and other systemic diseases may affect the conclusion of the CT studies. Although the number of the patients and controls having the hypertension are matched, severity and duration of the systemic diseases could not be standardized. Therefore, it should be kept in mind that, CT alterations seen in Xin *et al*'s study might be due to the underlying systemic diseases and concomitant treatment in patients.

Not only systemic diseases, but also ocular conditions including axial length and refractive error are known to influence CT.[6] The authors stated in their study that age and diopter were corrected using covariance analysis before comparing CT between the groups. However, spherical equivalent refraction is not stable throughout life. Myopic shifts can occur especially in elderly patients because of nuclear cataract progression. In this study, OSAS patients tended to be older than controls, although not significantly. So, considering axial length measurements instead of refractive status would be more accurate particularly in old population. Thus, we also wonder that whether the authors paid attention to the concomitant nuclear sclerosis in their patients that might influence the refractive status and consequently the results of their study.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Transient visual loss due to reversible 'pending' central retinal artery occlusion in occult giant cell arteritis

Giant cell arteritis (GCA) or temporal arteritis can cause profound and irreversible visual loss through anterior ischemic optic neuropathy (AION), posterior ischemic optic neuropathy, central retinal artery occlusion (CRAO), branch retinal artery occlusion (BRAO), choroidal infarction, and central nervous system stroke.[1] We present a case where permanent vision loss was prevented by prompt recognition of the condition seen on fluorescein angiography (FA). To our knowledge, this is the first report of fluorescein angiographic evidence of reversible retinal circulatory abnormalities associated with GCA.

Case report

A 62-year-old woman presented with complaints of complete loss of vision in the right eye on waking in the

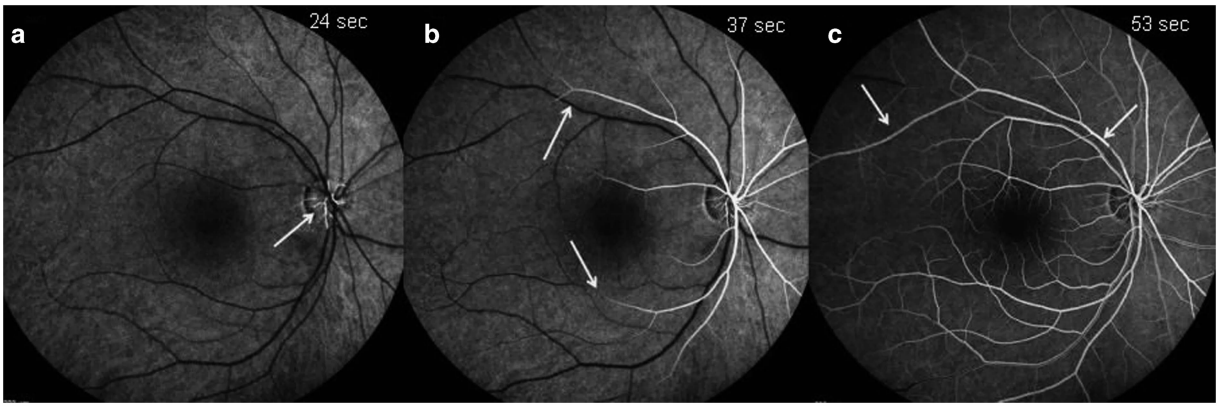
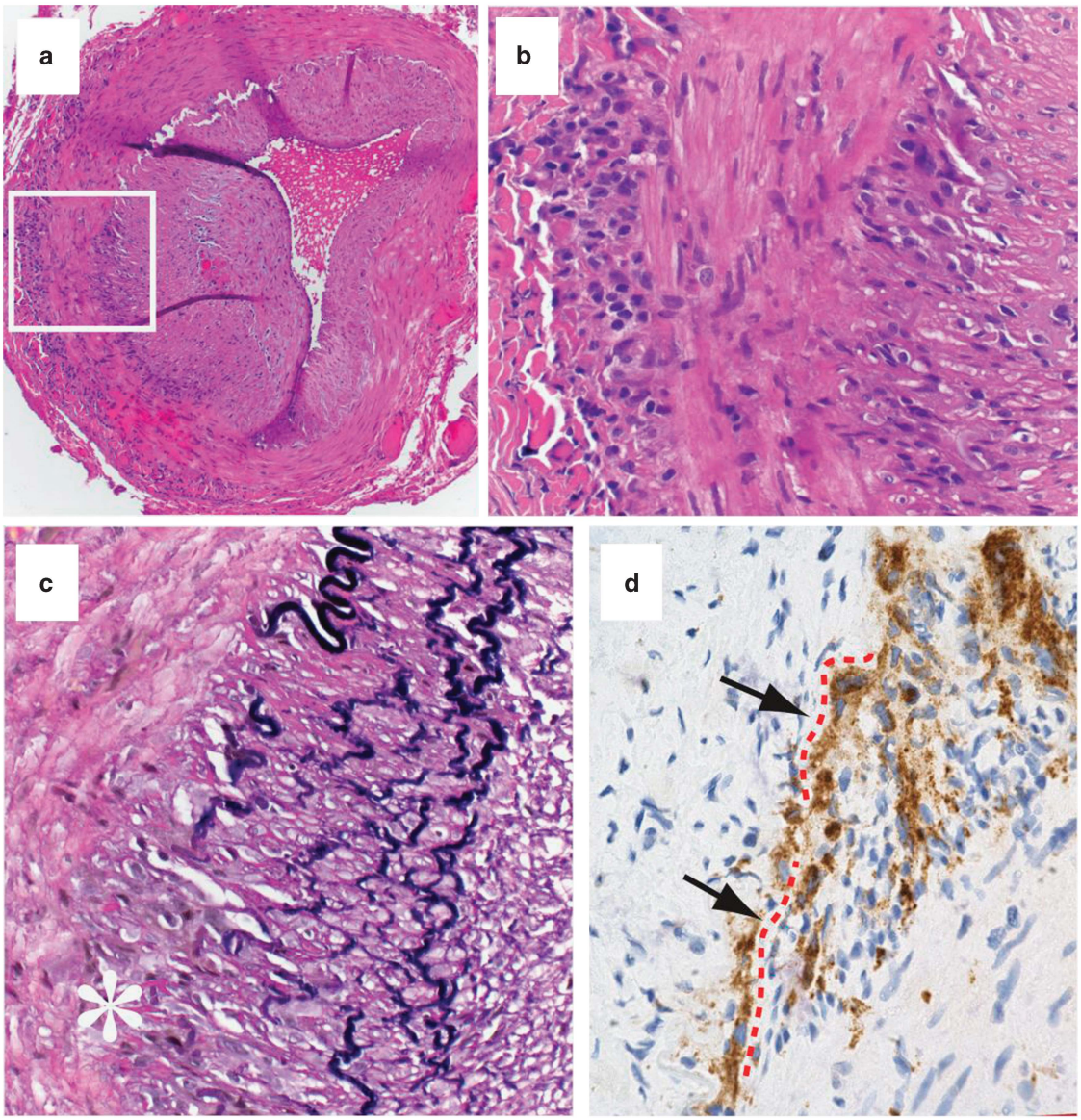


Figure 1 Fluorescein angiogram of the right eye at initial presentation. (a) Arrow points to the initial appearance of dye in the retinal arteries at 24 s. (b) Slow filling of retinal arteries at 37 s. Arrows point to the leading end of the fluorescein column in the retinal arteries around the macula. (c) Arrows point to the irregular filling and sludging of dye in the retinal arteries. Note the normal choroidal and papillary perfusion.



morning, followed by gradual improvement over few hours 3 days before presentation. She continued to have transient episodes of blurry and distorted vision associated with a dull ache in the right eye for the next 3 days. She denied any systemic symptoms often seen in GCA including scalp tenderness, headache, jaw claudication, proximal muscle weakness, myalgia, weight loss, or fatigue. She had hypercholesterolemia, but no history of diabetes or hypertension. BCVA was 20/25 in the right eye (OD) and 20/20 in the left eye (OS). The anterior segment and funduscopy were normal in both eyes. FA of the OD revealed normal choroidal circulation, but delayed and sluggish filling of retinal arterioles (Figures 1a–c). Choroidal and retinal perfusion was normal in the fellow eye. Westergren erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels were elevated to 86 and 2.5 (normal <0.8 mg/dl), respectively. Ultrasound carotid duplex revealed 60–79% stenosis of the internal carotid arteries (ICA) bilaterally, with a mild heterogenous plaque in the right ICA and a moderate plaque in the left ICA. The patient was immediately treated with intravenous methylprednisone 1 g daily for 3 days, followed by a tapering course of oral prednisone. A biopsy of the right superficial temporal artery confirmed the diagnosis of temporal arteritis (Figure 2). Two weeks following treatment, the BCVA OD was 20/20. ESR and CRP returned to normal levels of 6 and 0.1 mg/dl respectively. FA repeated 2 weeks following treatment revealed normalization of retinal arterial circulation (Figure 3). There was no recurrence of the disease at 6 months following treatment.

Comment

The incidence of occult GCA, ie, patients having GCA and visual loss but no systemic symptoms, is 21.2%.[2] Of those, 94.4% present with AION and CRAO.[2] Typically, findings on FA include absent or markedly delayed filling of the choroidal, papillary, and peripapillary circulation.[3–6] In contrast, the retinal circulation generally remains normal except in arterial occlusion.[3] In a prospective study by Hayreh *et al*,[7] FA of all seven patients with CRAO and GCA revealed occlusion or poor filling of the retinal circulation along with the posterior ciliary artery circulation abnormalities. These circulatory changes were attributed to thrombotic occlusion of either the posterior ciliary arteries, or the common trunk from the ophthalmic artery that gives off the central retinal artery and the posterior ciliary arteries. However, in our patient, presence of sluggish and stagnant retinal circulation, and normal choroidal perfusion was most likely secondary to partial and reversible thrombotic occlusion or inflammatory vasospasm of the central retinal artery.

Doppler studies in GCA patients have shown significant reduction in the mean flow velocity in the central retinal artery and short posterior ciliary arteries, along with changes in the ophthalmic artery flow.[8] A trend toward normalization of blood-flow velocities is seen following treatment with systemic steroid in patients without clinical progression of the disease.

In our patient, FA was performed due to high suspicion of GCA despite lack of obvious fundus findings. It revealed retinal perfusion abnormalities before permanent visual loss. It also demonstrated normalization of retinal circulation with systemic steroid

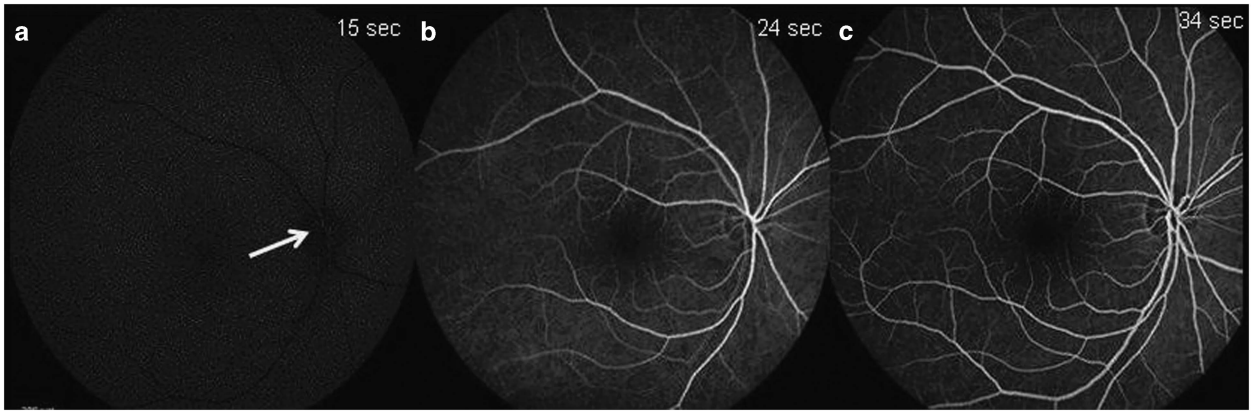


Figure 3 Fluorescein angiogram of the right eye 2 weeks following treatment. (a) Appearance of dye in the retinal arteries 15 s after injection (note the dye in arteries near the disc shown by the arrow. (b) Complete filling of the retinal arteries at 24 s. (c) Complete filling of retinal veins at 34 s.

Figure 2 Temporal artery histopathology. (a) Complete cross-section showing intimal hyperplasia, luminal narrowing, and inflammation of the external and internal elastic lamina regions (hematoxylin and eosin stain, $\times 10$ magnification). (b) Magnified photomicrograph of the boxed in region shows histiocytes admixed with lymphocytes along the elastic lamina (hematoxylin and eosin stain, $\times 40$ magnification). (c) Disruption of the elastic lamina at asterisk (Verhoeff–Van Gieson elastin stain, $\times 40$ magnification). (d) CD68+ macrophages are abundant in the inflammatory infiltrate. Portions of the elastic lamina visible under differential interference contrast (not illustrated) are marked with red dashed lines (arrows) (avidin–biotin complex peroxidase immunohistochemistry using diaminobenzidine as the chromagen responsible for the brown color, $\times 40$ magnification).

treatment. This case highlights the possibility that GCA may have an occult presentation, with disturbances in retinal artery filling being the sole demonstrable abnormality. It also emphasizes the value of fluorescein angiographic imaging in evaluating a patient with transient visual loss and a normal funduscopic appearance.

Conflict of interest

The authors declare no conflict of interest.

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Sir,
Treating maculopathy at the expense of proliferative disease: an emerging problem in 'macular treatment centres'

Approval of ranibizumab for diabetic macular oedema (DMO) has resulted in a growing number of patients with diabetic retinopathy (DR) attending so-called 'macular clinics' for regular follow-up and intravitreal treatment. These one-stop clinics were originally established to cater for patients with neovascular age-related macular degeneration.

Case report

We present the case of a 48-year-old type 1 diabetic who was referred to our macular treatment centre in early 2012 for diffuse DMO that had not responded to macular laser. Over a 12-month period, he received multiple bilateral injections of ranibizumab. His DMO settled completely in both eyes and his vision improved to 6/9 bilaterally. In mid-2013, it was decided that further intravitreal treatment was no longer necessary given that both maculae were dry. Follow-up was arranged but at a longer interval of 4 months. When seen in late 2013, bilateral florid neovascularisation with high-risk characteristics was evident. Urgent bilateral, complete panretinal photocoagulation (PRP) was undertaken.

Comment

Ischaemia of the peripheral retina has long been hypothesised to have a role in the development of DMO.[1] In DR, ischaemia leads to the release of vascular endothelial growth factor (VEGF) that causes breakdown of the blood–retina barrier.[2] This, in turn, leads to increased vessel permeability that may be the cause of DMO.[3] We believe that our patient probably developed bilateral macular oedema on account of co-existing peripheral ischaemia, which was clinically evident as severe non-proliferative disease. This diagnosis had already been made at the time of referral for intravitreal treatment. Early PRP, administered during or before intravitreal anti-VEGF treatment, could have prevented the development of sight-threatening high-risk proliferative disease.

We also believe that there may be many more patients like ours within fast-track macular pathways across the country who are at risk of suddenly developing proliferative disease upon cessation of intravitreal anti-VEGF therapy. Patients with DMO who are