Sir,

Myopic tractional maculopathy associated with rhegmatogenous retinal detachment

The increasing application of optical coherence tomography (OCT) has led to identification of a new cause for visual loss in degenerative myopia—myopic tractional maculopathy (MTM).[1–3] MTM typically manifests as foveal schisis; rarely, it is severe enough to cause a tractional retinal detachment (TRD).[4] Most reports describe MTM as an isolated phenomenon.[1–4] We report a rare case of MTM associated with rhegmatogenous retinal detachment (RRD).

Case report

A 38-year-old lady presented to us with defective vision OS for a week. The right eye was blind since childhood. Best-corrected visual acuity (BCVA) was hand motions OD and 3/60 OS. On examination, anterior segment was unremarkable; posterior segment showed myopic chorioretinal degeneration OU. Additionally, OD had extensive pigmentary mottling and subretinal gliosis suggestive of a spontaneously settled RRD. OS showed a fresh subtotal RRD with multiple peripheral breaks; however, macular contours appeared to be concave (Figure 1a). OCT (version3, Carl Zeiss Meditec, Dublin, CA, USA) revealed vitreomacular traction causing focal macular detachment (Figure 1b). She underwent belt-buckling and pars plana vitrectomy; posterior hyaloid was detached and epimacular membranes were removed. Silicone oil was used for tamponade due to the patient's inability to maintain prone position. One month postoperatively, retina was attached OS. OCT confirmed the relief of vitreomacular traction with a shallow lamellar hole. Silicone oil was removed 2 months later. The patient was last reviewed 6 months thereafter: status quo was maintained (Figure 1c and d); final BCVA was 6/18.

Comment

Myopic foveoschisis is characterized by an intraretinal splitting in a myopic posterior staphyloma, caused by anteroposterior and/or tangential traction, sometimes combined with foveal detachment, culminating in a lamellar or full-thickness macular hole.[1–3] Our case was unusual, because the macular TRD was surrounded by convex RRD on all sides, indicating the two pathologies as discrete but synchronous phenomena. It is probable that an acute posterior vitreous detachment simultaneously opened the peripheral retinal tears, as well as pulled at the tenacious vitreomacular adhesions into a TRD. It has been suggested that when the area of adhesion is small ($\leqslant 500\,\mu$), a macular hole results; a broader adhesion ($\sim 1500\,\mu$) causes a retinal schisis/detachment.[5] Removal of posterior vitreous cortex and epimacular membranes relieved the traction, while possibly deroofing an inner macular schisis into a lamellar hole. It is advisable to include OCT in the preoperative evaluation of a myopic RRD associated with macular staphyloma to reveal such undetected pathologies, which have implications for surgical management.

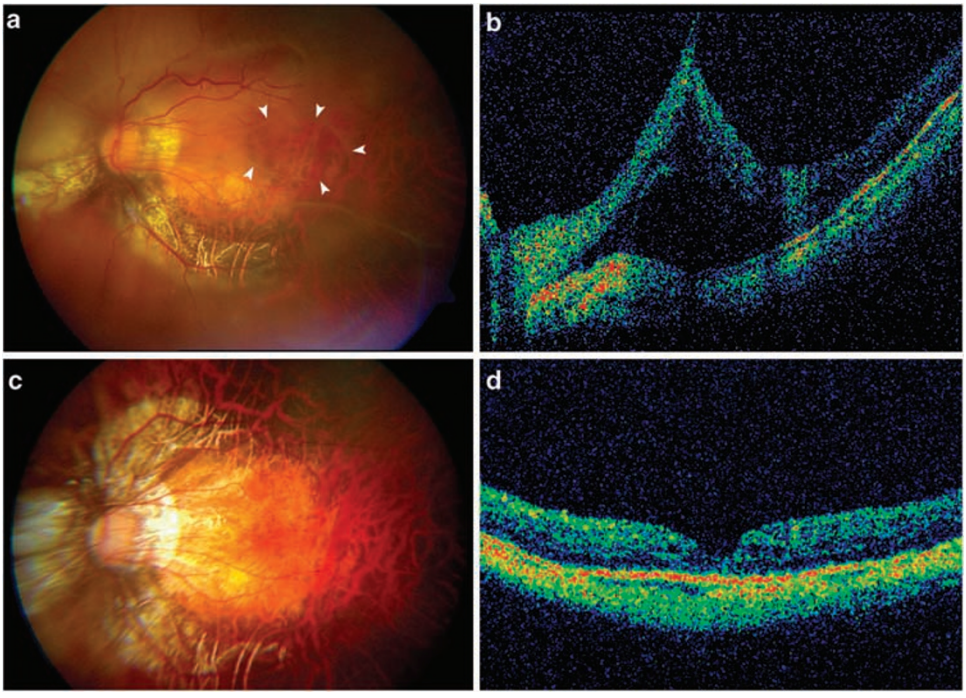

Figure 1 (a) Fundus view of the left eye reveals suspected areas of vitreomacular traction (arrowheads) with surrounding convex rhegmatogenous retinal detachment. Myopic chorioretinal degeneration and posterior staphyloma are also evident. (b) Horizontal optical coherence tomography (OCT) scan clearly shows tractional elevation of macula. Note that retina temporal to the macula appears attached. (c) Fundus view 6 months after silicone oil removal shows globally attached retina. (d) An inner lamellar macular hole is evident on horizontal OCT scan.

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Sir,

Should we anticipate intraoperative floppy iris syndrome (IFIS) even with very short history of tamsulosin?

Intraoperative floppy iris syndrome (IFIS) is characterised by a triad of intraoperative features including a flaccid and undulating iris stroma, a propensity to prolapse towards corneal incisions and progressive intraoperative pupillary constriction associated with oral tamsulosin intake.[1] Tamsulosin, used most commonly for benign prostate hypertrophy has high bioavailability and a long half-life. It has selectivity for α-1A type adrenoreceptor, predominant in base of bladder and prostate smooth muscle.[2] Studies have found occurrence of IFIS in patients started on tamsulosin for 9 months to 3 years.[3] A minimum duration of 3 months was noted in one study.[3] We report a case of probable IFIS following a minimum of 2 days intake of tamsulosin.

Case report

A 70-year-old male patient was due for routine cataract surgery. There was no past history of ocular trauma/surgery or any significant refractive error. He had complete preoperative assessment 4 weeks before operation. He had a systemic history of peptic ulcer and chronic obstructive pulmonary disease and the only other drug history was oral lansaprazole and beclomethasone inhaler. On operative day, topical mydriatic were instilled twice in operative eye (1% cyclopentolate and 2.5% phenylepinephrine) 20 min apart. After 45 min inadequate pupillary dilation was observed. Sub-Tenon's block with 2% lignocaine (3 ml) was given. No preanaesthetic medication was used. Intraoperative bellowing and flaccid nature of iris and iris prolapse through the corneal section were noted suggesting probable IFIS. However, standard phacoemulsification and intraocular lens implantation were completed without any significant complication. Postoperatively, on direct questioning the patient mentioned that he was started on FLOMAX (tamsulosin), for the first time, only 2 days before the day of surgery.

Comment

Tamsulosin is a uroselective drug, targeting α-1A-adrenoreceptor.[4] Human dilator iris muscle also has α-1 adrenoreceptor. It is hypothesised that tamsulosin blocks this receptor by constant blockade to cause disuse atrophy.[1] IFIS has been noted even in patients who had stopped tamsulosin 2 years before cataract surgery.[5] However, none of the studies have established a minimum duration of intake of tamsulosin leading to IFIS. Our case report suggests a possibility of the IFIS occurrence after 2 days of tamsulosin intake. Given the short onset of action of 6–7 h, high bioavailability, and the long half-life of tamsulosin, there is a strong possibility in our case that the iris dilator muscle had only suffered a recent receptor blockade without muscular atrophy, resulting in IFIS. Hence, according to the current belief, IFIS is caused by the disuse atrophy of dilator iris muscle due to constant receptor blockade; there is also a possibility that IFIS can result from the receptor blockade alone. However, a larger case series/study is warranted to establish the fact.

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