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Hydrated scleral buckle: a late complication of MAI explants

Several long term complications have been reported with MAI scleral buckles,[1–3] a synthetic hydrophilic scleral buckling element, first introduced in the 1970s.[4] We present a case of an extruding, hydrated MAI scleral buckle that presented as an orbital lesion. The magnetic resonance imaging (MRI) characteristics of the hydrated MAI buckle are also described.

Case report

An 81 year old woman was referred to the orbital service for evaluation of a freely mobile non-painful subconjunctival lesion in the left eye present for at least a year. The extraocular movement was full but with a small left exotropia in primary gaze. Anterior segment examination revealed a firm, nodular subconjunctival lesion in the superomedial quadrant. The diagnosis of extruding scleral buckle versus a large orbital conjunctival inclusion cyst was entertained. She, however, denied a history of retinal detachment repair.

The patient underwent MRI of the orbits with and without gadolinium infusion which revealed a 2.5×0.7 cm^2 elongated, elliptical mass in the medial intraconal space. The hyperintensity of the mass on T2 weighted images, indicative of high water content, led to the mistaken radiological diagnosis of a cystic, fluid filled lesion (fig 1). Intraoperatively, a swollen intact segmental buckle was encountered that disintegrated into small and large pieces when grasped with a forceps. Complete meticulous removal of these small fragments was performed (fig 2).

Comment

MAI hydrogel explant is made of polymethyl acrylate-co-2-hydroxyethyl acrylate crossed linked with ethylene diacrylate with 15% water[2] and was considered an ideal scleral buckling material since it was as effective as solid silicone rubber buckle and sponge but with lower incidence of erosion.[1] However, long term complications are being more frequently recognised with the MAI buckle.[1] Complications include hydration of the buckle with erosion and extrusion of the explant.[2] Owing to their hydrophilic nature, buckles tend to swell, become bulky, and displace from their intended position. These explants change from an opaque, soft, spongy, whitish appearance to a translucent, gel-like, cream coloured material with hydration.[2] They become extremely friable and fragment easily with slight traction upon removal.[1 3] Scanning electron microscopy of these hydrated buckles has revealed distortion of their micropore architecture.[1 2]

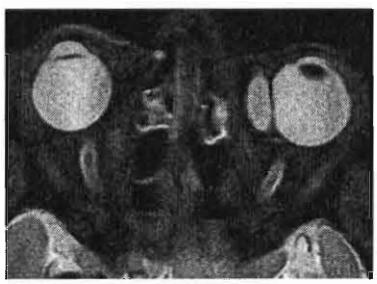

Figure 1 T2 weighted MRI image reveals a hyperintense mass exhibiting a mass effect on the left globe.

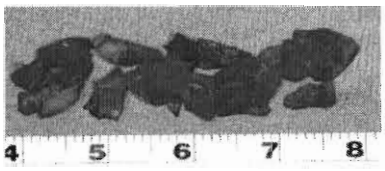

Figure 2 Hydrated, friable buckle removed in multiple small fragments.

The presentations of hydrated MAI buckles are varied. They can become extremely loose and migrate anteriorly, or enlarge in situ and present as orbital masses. Since complications do not occur until many years after the orbital surgery, the history of a retinal detachment repair may not be elicited. In addition, these loose buckles usually do not indent the globe and may not be recognised as being present on indirect ophthalmoscopy.

Radiological imaging may be helpful. MRI T1 weighted images of a hydrated buckle reveal a well defined, hypointense mass, while T2 weighted images reveal a hyperintense mass as a result of high water content. (In contrast, a silicone element will be black on MRI images.)

Removal of hydrated MAI buckles can be very difficult. Careful sub-Tenon dissection should be carried out since any pressure on the buckle can lead to its fragmentation. The procedure is often complicated because of the extremely friable nature of these buckles. Cryoprobe has been used successfully in some cases for removal of the buckles.[5] Le Rouic *et al* demonstrated that removal of MAI buckles with a cryoprobe is a safe and effective technique with lower fragmentation rate compared to the use of forceps.[5] The cryoprobe allows the water in the swollen explant material to freeze, which helps to reduce fragmentation.

In conclusion, ophthalmologists should be aware of the possible complications of the MAI hydrogel buckles. Knowledge of the MRI characteristics of hydrated MAI buckles may be helpful in identifying them in the event they present as a space occupying orbital mass.

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Complex I respiratory defect in LHON plus dystonia with no mitochondrial DNA mutation

Leber hereditary optic neuropathy (LHON) sometimes occurs with dystonia[1] in association with mitochondrial DNA (mtDNA) mutations in complex I.[2] We describe a patient with LHON plus dystonia who had a severe complex I respiratory defect with no pathological mtDNA mutation, implying a mitochondrial abnormality of nuclear origin.

Case report

The proband was healthy until age 17 years when she developed progressive right sided weakness followed 5 years later by a left hemiparesis with involuntary posturing of the left arm and leg. Intelligence was average, and she finished grade 6 at school. Her parents were unrelated, and she had five healthy siblings.

Neuro-ophthalmological examination at age 23 documented excellent afferent and efferent visual functioning with no Kaiser-Fleischer ring. Optic discs were hyperaemic and slightly elevated with peripapillary telangiectasias in both eyes (fig 1A and B). She had modest right sided weakness, diffuse hyper-reflexia greater on the right, bilateral Babinski signs, and dystonic posturing on the right while walking.

On return 21 months later, she reported that the vision of both eyes had declined 9 months previously. Visual acuity was 20/100 in both eyes with poor colour vision in both eyes, a mild left afferent pupillary defect, and bilateral optic atrophy with no residual hyperaemia or peripapillary telangiectasias (fig 1C). Gait was somewhat worse, and she was modestly dysarthric. Vision did not improve during 18 months of follow up.

Normal laboratory studies included haemogram, liver and renal function, serum lactate, pyruvate, and 24 hour urine copper excretion. Cerebral spinal fluid (CSF) lactic acid was slightly elevated (2.8 mm/l with normal range 0.6–2.2 mm/l), and brain MRI revealed abnormalities in both basal ganglia (fig 1D).

After signing informed consent, the proband, her mother and father, and four siblings had blood drawn for DNA extraction, polymerase chain reaction amplification, and sequencing of the entire mitochondrial genomic coding region as previously described.[3] Sequence results were compared to Mitomap (www.mitomap.org/mitomap/mitoseq.html), the human mitochondrial genome database (www.genpat.uu.se/mtDB), GenBank (www.ncbi.nlm.nih.gov/Genbank/index.html), and

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New onset diplopia: 14 years after retinal detachment surgery with a hydrogel scleral buckle

In 1979, the hydrogel explant (Miragel, Waltham, MA, USA) was introduced as a scleral buckling material in the surgical management of retinal detachment.[1] It was widely used in the 1980s and early 1990s as it was initially believed to be well tolerated, less prone to infection, and easy to manipulate.[2] However, long term complications related to swelling and fragmentation of the explant have been reported over recent years,[3–6] resulting in discontinuation of its use in 1995.

Case report

A 36 year old healthy man presented on 2003 with symptoms of mild right ocular discomfort. Past ocular history included a right retinal detachment repair 14 years previously, using a 907 (3×5 mm) Miragel scleral buckle (Miragel, Medical Instruments Research Associates, Waltham, MA, USA), sutured to the inferior sclera. On examination, visual acuity was 20/120 right and 20/20 left. There was no diplopia or limitation of eye movements. What was thought to be a small conjunctival cyst was noted inferiorly but, otherwise, the ocular examination was unremarkable and the retina was secure.

A year later (2004), he presented with increasing marked right ocular discomfort and diplopia in all fields. His visual acuity was unchanged, but there was marked restriction of elevation and reduction in adduction of the right eye and binocular diplopia in all fields of gaze. A tense swelling of the inferior conjunctiva was noted (fig 1, top), intraocular pressure was normal, and the retina was flat with a moderate anterior buckle effect. Computed tomography (CT) (fig 1, bottom) demonstrated a right orbital circumferential soft tissue mass surrounding the lower half of the globe with a small area of calcification. The initial diagnosis was a postoperative giant conjunctival cyst and the patient underwent surgery.

Intraoperatively, exploration revealed no conjunctival cyst, but a large encapsulated scleral buckle. The explant was friable, gel-like, and translucent, but could be removed in one piece (fig 2). A 3 mm diameter area of scleral thinning associated with calcification was found underlying the buckle inferotemporally. At 1 month follow up the patient was asymptomatic with no diplopia, unrestricted extraocular movements, and the retina was flat.

Comment

Hydrogel explants are composed of a low molecular weight hydrophilic material that is water permeable. These explants have a tendency to absorb water over the years and increase dramatically in size. The resulting complications range from a non-tender subconjunctival mass to intraocular or external extrusion.[3–6] The long time lapse from buckle surgery may result in a high misdiagnosis rate. Kearney *et al*[6] reported 17 eyes of

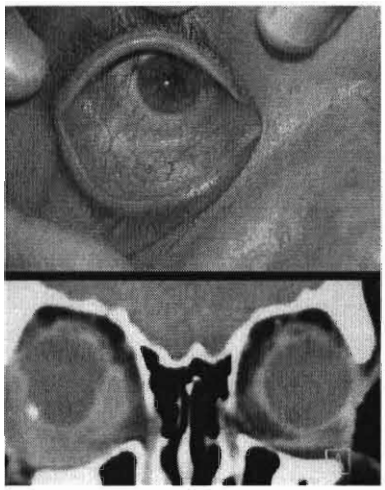

Figure 1 Top: Clinical photograph of the right eye showing bulging of the inferior fornix. Bottom: Coronal computed tomographic scan showing a circumferential soft tissue mass surrounding lower half of the globe and a small area of calcification on the inferotemporal sclera.

patients with complications related to hydrogel explant swelling. In nine cases the initial diagnosis was incorrect, being mainly Graves' disease, idiopathic orbital fibrosis, and a subconjunctival inclusion cyst.

In our case, there was a profound increase in the explant volume during a 14 year period. The resulting diplopia and restriction of extraocular movement as well as the clinical evaluation mimicked a giant orbital inclusion cyst. The correct diagnosis was only made intraoperatively. Scleral thinning and necrosis as seen in our case has been reported previously,[7] resulting in intraoperative vitreous leak after removal of the expanded explant.[8] In our patient, there was an area of thinned sclera, but the surrounding calcification and the early removal of the explant prevented vitreous leak.

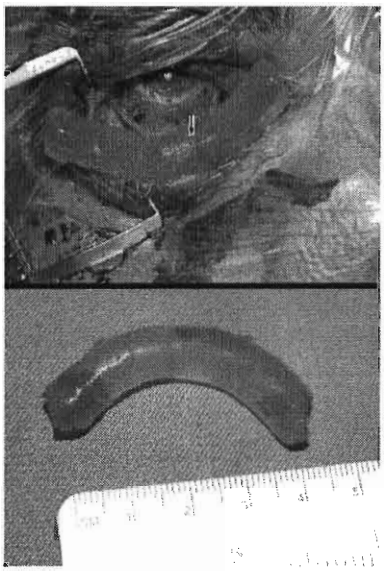

Figure 2 Top: Intraoperative photograph showing the swollen hydrogel buckle under the inferior rectus muscle (arrow). Bottom: The hydrogel buckle after removal.

It is important to note that patients who have undergone scleral buckling with hydrogel explants before 1995 are at risk of developing this complication. Symptoms of progressive diplopia, pain, and restriction of extraocular muscle movement in these patients should also raise the possibility of explant expansion. The assistance of a retinal surgeon may sometimes be required because of the increased risk of scleral thinning and leakage of liquid vitreous intraoperatively.

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Inverse globe retraction syndrome complicating recurrent pterygium

Often larger and more aggressive than the original lesion, recurrent pterygia can cause visual symptoms that are most often secondary to their mechanical effects on the cornea.[1] We report a case of inverse globe retraction syndrome (that is, retraction during abduction) due to the restrictive effect of a recurrent pterygium and the management of this complication.

Case report

A 28 year old man without a medical history or ocular symptoms underwent pterygium excision in his left eye with a superotemporal conjunctival autograft and intraoperative mitomycin C. Three weeks postoperatively, he noted a feeling of pressure in the left eye