Magnetic resonance imaging characteristics of posterior scleritis mimicking choroidal mass

A. Osman Saatci a, Isil Saatci b,*, Nilüfer Kocak a, Ismet Durak a

a Department of Ophthalmology, Dokuz Eylül University, İzmir, Turkey
b Department of Radiology, Hacettepe University Hospital, Sihhiye, 06100 Ankara, Turkey

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Abstract

We present imaging findings in a case of posterior scleritis, which may mimic tumoral mass lesion resulting in unnecessary enucleation. Magnetic resonance imaging was remarkable for a subretinal mass hypointense on T2 and hyperintense on T1 weighted images. A peripheral rim of hypointensity was noteworthy, suggestive of sclerouveal thickening. There was an ill-defined area of increased T2 signal intensity adjacent to globe at the site of nodular lesion implying an inflammatory process. A linear contrast enhancement was seen within the bulbus oculi which may represent detached retina by exudation or displaced retina due to thickened sclera and choroidal layers. The CSF space around the optic nerve was enlarged. © 2001 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

Scleral inflammatory disease is demonstrable on sectional imaging, computed tomography or magnetic resonance imaging (MRI), in majority of which involvement is unilateral [1,2]. When the inflammation is nodular or focal in distribution, it may be mistaken for tumour, mostly malignant disease, such as malignant melanoma or metastasis [3,4]. In this case report, MRI findings of posterior scleritis are described with emphasis on its differential diagnosis with ocular tumours which may cause unnecessary enucleation [4].

2. Case report

A 36-year-old man was examined due to painless, severe visual loss of 5 days duration in his right eye. His past medical and family histories were unremarkable. His left eye was normal. Visual acuity was counting fingers in the right eye. There was episcleral vessel engorgement temporally and the ocular media was clear. On ophthalmoscopy, a subretinal mass with surrounding retinal detachment was detected (Fig. 1(A)). Ultrasonography depicted a mass with moderate to high internal reflectivity and an adjacent echolucent area. No evidence of any systemic or ocular disorder was detected despite a thorough systemic evaluation.

Orbital MRI, performed with and without contrast administration, revealed a subretinal mass lesion hypointense on T2 and hyperintense on T1 weighted images (Fig. 2). A thickened rim of hypointensity, with ill-defined margins at the vicinity of the mass, was noted around the right bulbus oculi, circumferentially, on T1 weighted images with no enhancement which may represent detached retina with minimal exudation and/or thickening of the sclerouveal coat. The thin layer with contrast enhancement deep to this which was displaced inside vitreus at the medial aspect may represent detached retina with minimal exudation and/or the displaced retina by the thickened sclera and choroidal layers. Also noted was an ill-defined region hypointense on T1 and hyperintense on T2 weighted images around the bulbus oculi laterally which suggested the presence of an inflammatory process (Fig. 2(A,B,D)). On T2
weighted images, the cerebrospinal fluid (CSF) space around the optic nerve was prominent (Fig. 2(B)).

A diagnosis of posterior scleritis was made and oral 80 mg daily prednisolon was started. A dramatic increase in visual acuity and clearing up of mass on ophthalmoscopic examination (Fig. 1(B)) were noted. Two weeks later, oral steroid was tapered slowly. Control MRI at 6 months showed complete resolution of the imaging findings. No systemic or ophthalmologic problem occurred during the follow-up of 1 year.

3. Discussion

Posterior scleritis, a not well-known entity for radiologists, is a severe form of scleral inflammation, which has often eluded diagnosis because of its varied clinical presentations. It most frequently occurs in middle aged women, when it is often associated with connective tissue disorder, or in young patients who are rarely found to have systemic disease. It may clinically present with pain, decreased vision, impaired visual field and double vision. Due to systemic associations a meticulous systemic investigation is mandatory [2,5].

Posterior scleritis may manifest as a nodular mass, as it did in our case, which may be mistaken for a neoplasm, most likely choroidal malignant melanoma or metastatic carcinoma to the uvea, choroidal hemangioma, benign lymphoid hyperplasia [2–4,6,7]. The MRI appearance in our case may have suggested tumor, choroidal melanoma in particular, based on the findings of a nodular mass hypointense on T2 and hyperintense on T1 weighted series and the enhancing layer suggestive of detached retina (Fig. 2(A,C,E)). Hypointensity on T2 and hyperintensity on T1 weighted images may represent paramagnetic nature of the mass, also a frequent feature of melanoma [8]. T1 hyperintensity may have resulted from paramagnetic substances, e.g. subacute blood products, melanin pigment or inflammatory cells with free radicals, collagen and calcification. The thickened circumferential rim at the periphery of the globe which may be attributed to edematous Tenon capsule and/or the thickened scleral coat [1,2,9] and associated adjacent inflammatory reaction rather implied an inflammatory process (Fig. 2(B,D)). Rarely, choroidal melanoma may be associated with scleritis [10]. On the other hand ultrasonographic findings were inconsistent with malignant melanoma in our case.

Retinal detachment may be associated with subretinal mass as well as resulting from exudation due to scleritis [4,5,7,11]. Therefore, linear contrast enhancement within the globe in our case may represent detached retina. However, another postulation for this appearance might be this linear enhancement representing displaced retina by the thickened scleral and choroidal layers toward the retinal surface, not necessarily causing retinal detachment [3,9], which corresponds to the ophthalmoscopic finding of choroidal folds. In our case, subretinal fluid was clinically apparent, rather supporting the former explanation.

In posterior scleritis retrobulbar edema and papillitis may be present. In our patient papill edema was present on fundoscopy examination. Enlargement of the CSF space around the optic nerve was noted on MRI in our patient. The causes resulting in optic nerve sheath enlargement include dural ectasia or increased intracranial pressure from any source such as dural sinus thrombosis, pseudotumor cerebri or intracranial mass [12], which may be presented with choroidal folds in the ophthalmoscopic examination, that is also a finding of posterior scleritis.

Orbital pseudotumor and infections [13] may involve sclera, however, no other finding which may be attributed to pseudotumor was present and no evidence of orbital cellulitis, which accompany infectious involvement of the sclera was detected [1].

In conclusion, posterior scleritis should be included in the list of entities presenting with a subretinal mass, whether subretinal fluid is present or not, especially

![Fig. 1. Color fundus pictures of the right eye: (A) at initial presentation depicting the subretinal mass (arrowheads); and (B) following steroid treatment demonstrating the dramatic resolution of the mass.](image-url)
Fig. 2. MRI examination (A–B): T2 weighted (TR 4112, TE 120 ms) consecutive images showing a subretinal hypointense mass (black arrowhead), posterolaterally in the right globe. Note the adjacent ill-defined region of hyperintensity around the globe representing the inflammatory reaction (large white arrows). CSF space around the optic nerve is enlarged (small white arrows) (B). (C–D): Precontrast T1 weighted (TR 673, TE 25 ms) images demonstrating the mass being markedly hyperintense (white arrow). A thickened rim of hypointensity is seen at the periphery of the globe (small black arrowheads) with irregular margins (large black arrowheads) in the vicinity of the mass. (E): Postcontrast T1 weighted (TR 673, TE 25 ms) image almost at the same level with Fig. 2(C), showing contrast enhancement (arrow).
when there is adjacent inflammatory reaction. Correct diagnosis avoids unfortunate enucleation and promotes the recognition of underlying systemic disorder. An outer rim of hypointensity at the periphery of the globe may warn the radiologist in favor of an inflammatory process.

References