

Massive retinal gliosis: A case report and literature review

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Massive retinal gliosis is a rare, benign disorder, extreme form of reactive glial cell proliferation and can cause difficulties in the differential diagnosis of intraocular tumors. A 65-year-old female patient who had a blind left eye since birth was admitted to the hospital. Ophthalmologic examination showed dense nuclear cataract with large, smooth, white-pink colored peripapillary tumor in the left eye. Histologically the intraocular tumor proved to be a massive retinal gliosis. Immunohistological reactions were positive for GFAP. Clinically, distinguishing of massive retinal gliosis from the other intraocular tumor is very important.

Keywords: Intraocular tumors, massive gliosis, retina

Introduction

Massive Retinal Gliosis is a benign non-invasive proliferation of well-differentiated glial cells.¹⁻¹⁰ In 1918, von Hippel defined two such examples and called as benign growth of retina. The term of massive gliosis of retina was used in 1926 by Friedenwald.²

The sources of cellular constituents of fibrous and cellophane membranes formed in massive periretinal proliferation have been unclear.² Yanoff et al⁵ reevaluated 38 histologically diagnosed cases, and suggested that the lesion is a non-neoplastic proliferation of retinal glia in response to diverse pathologic states initiated by a variety of causative factors included trauma, congenital malformations, chronic inflammation, glaucoma, phthisis bulbi and intraocular neoplasms. Some of this condition may also occur as an idiopathic entity.¹⁻⁴ We present herein a rare case of massive retinal gliosis diagnosed by light microscopy and immunohistochemical findings.

Case report

A 65-year-old woman had had blind left eye since birth. On presentation, she had dense nuclear sclerotic cataract without history of glaucoma and any

predisposing disorder such as chronic inflammation, vascular disorders, glaucoma, trauma, or congenital abnormalities. So the lesion was considered clinically as primary. Ophthalmoscopic examination showed a large, smooth, white-pink colored peripapillary tumor in the left eye. Scan ultrasonography revealed high and low internal reflectivity and dense opacity.

The left eye was subsequently enucleated. For light microscopic evaluation, the whole eye was fixed in 10% formaldehyde for 24 hours, after 0,1 ml of fixative was injected into the anterior chamber for preserving the shape of the eye. After fixation, the cornea was removed. The tumor mass was seen as the large elevated scar occurred near the disc as a salmon-colored single nodule. The sample was embedded in paraffin and 5 µm-thick sections were stained with hematoxylin-eosin, Periodic Acid Schiff (PAS), and rabbit antihuman glial fibrillary acidic protein (GFAP) by the peroxidase method.

Histologically, the retina was replaced by interweaving bundles of spindle shaped glial cells with uniform oval nuclei and abundant fibrillary cytoplasm (Figure 1 and 2). There were thin-walled vessels surrounded by thick hyaline layer. GFAP was positive in large numbers of astrocytes (Figure 3).

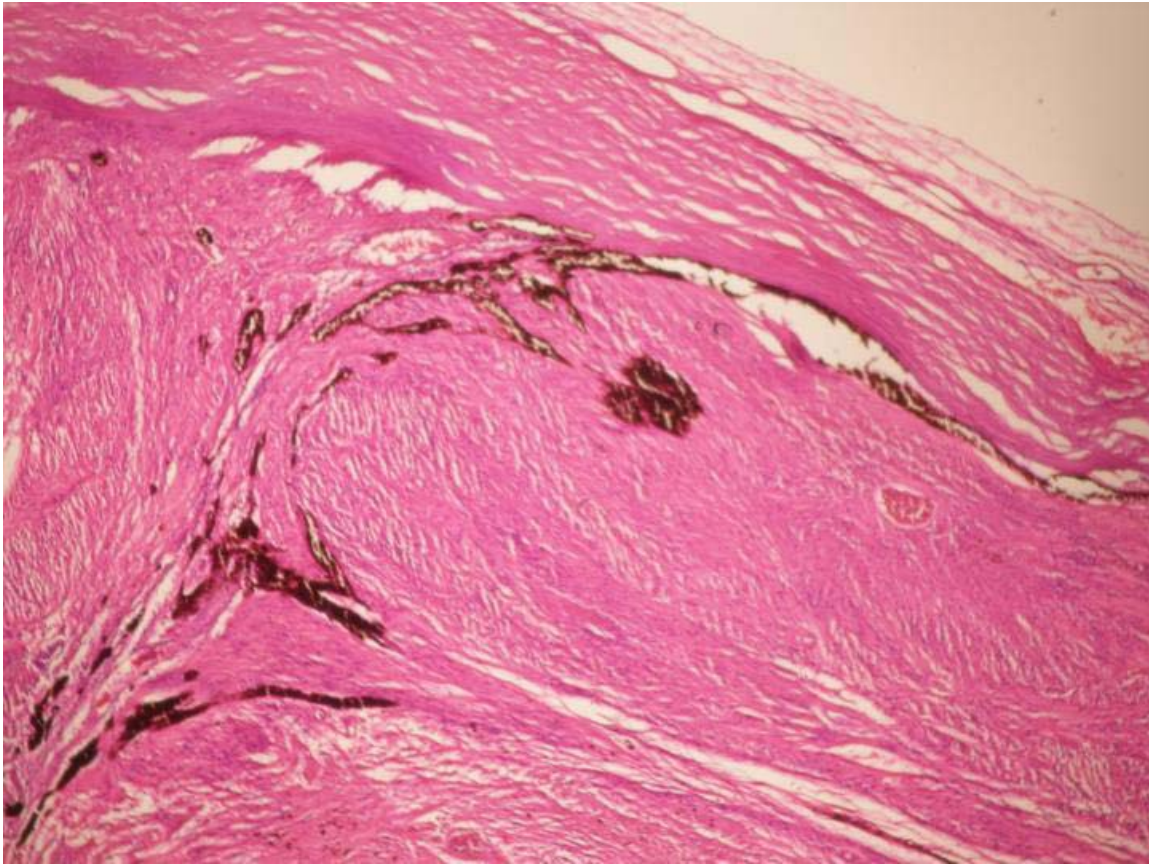


Figure 1. Interweaving groups of spindle shaped glial cells with uniform oval nuclei and abundant fibrillary cytoplasm (H&E x 40)

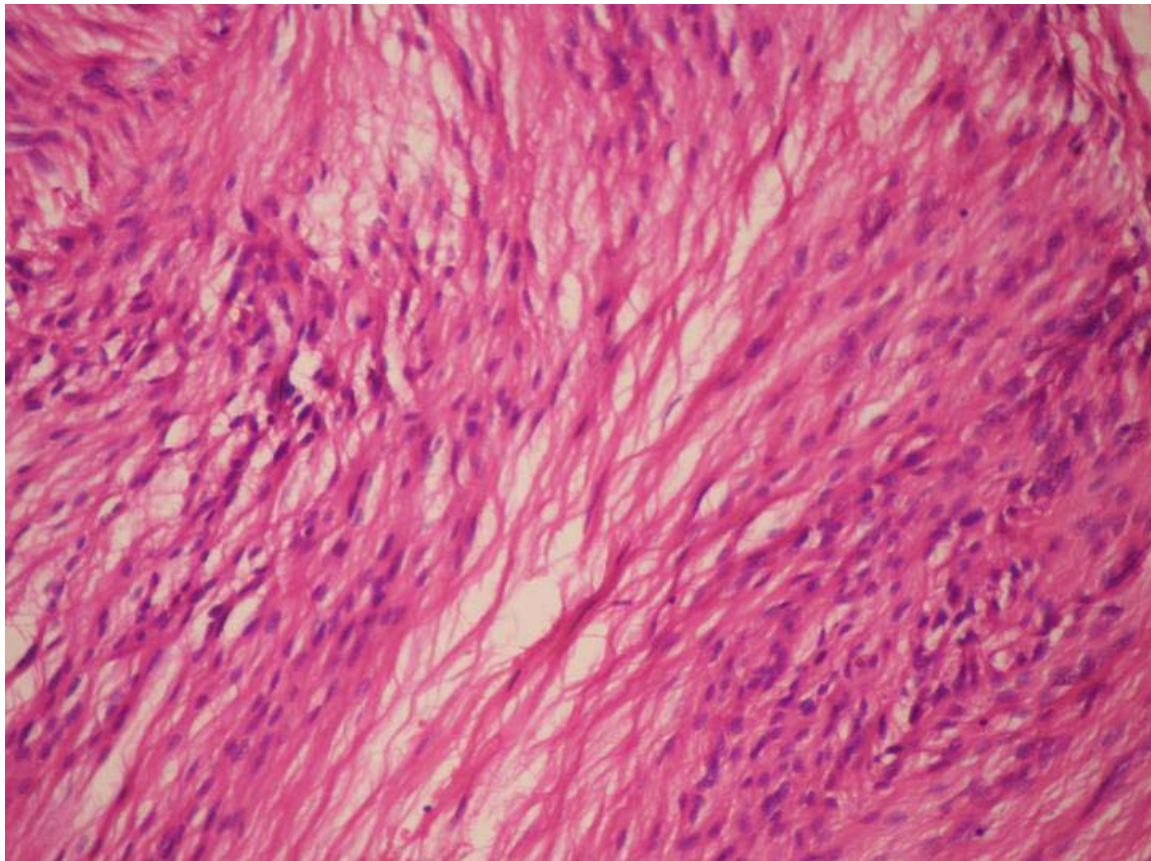


Figure 2. Interweaving groups of spindle shaped glial cells with uniform oval nuclei and abundant fibrillary cytoplasm (H&E x 200)

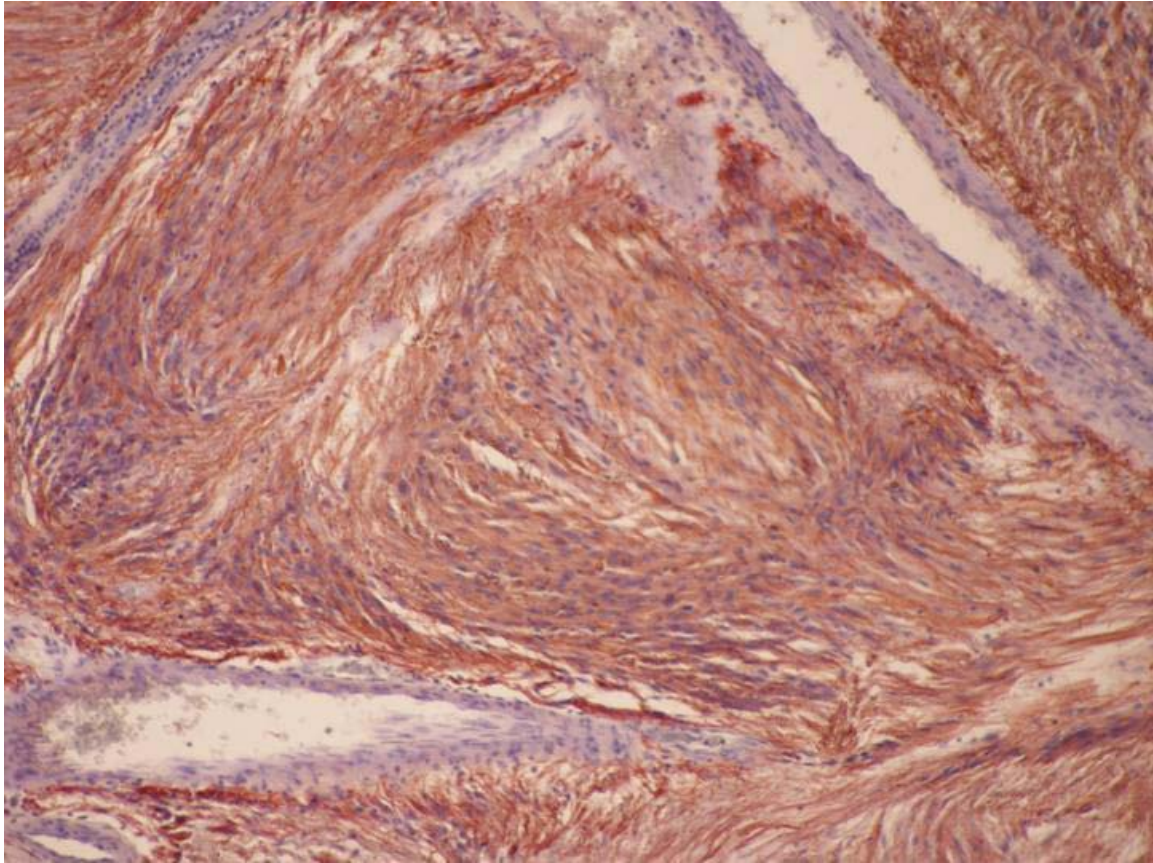


Figure 3. GFAP positivity with AEC chromogene in glial cells (GFAP with AEC chromogene x 100)

Discussion

Massive retinal gliosis represents non-neoplastic proliferation of the retinal glia.⁵ Its onset is often occurring ten or more years after predisposing disorder such as chronic inflammation, vascular disorders, glaucoma, trauma, or congenital abnormalities.¹⁰ In massive retinal gliosis, both sexes and all ages may be affected nearly in equal frequency.²

Distinguishing massive retinal gliosis clinically from intraocular neoplasm may be difficult.⁴ This lesion often appears as a single or multiple well-vascularized nodules, which has a predilection for the peripheral retina but may occur anywhere.¹⁻⁵ In our case the tumor mass was seen as a single, well-vascularized nodule diagnosed as an intraorbital tumor (particularly melanoma) by ophthalmologists preoperatively.

Newsome et al.³ investigated the cellular constituents present in massive periretinal proliferation. By phase contrast and ultrastructural morphological criteria, they identified four major

constituents: 1- Macrophages, 2- Pigmented epitheloid cells, 3- Glial cells, and 4- Fibroblastic cells.

Machamer and Norton⁶ and Horn et al.⁷ had studied for massive retinal gliosis on experimental models and suggested that the experimental models had similar features with the human disease. Also proliferation and fibrous metaplasia of retinal pigment epithelium and retinal glial cells might play role in the pathogenesis of this lesion.⁸ Laqua et al.⁹ showed that proliferation of glial cells occurred in preretinal and subretinal tissues. Besides these, by electron microscopy, the cells were identified of glial origin growing out of the retina.^{7,8} In the present case, on light microscopy, the nodules composed of interweaving bundles of glial cells stained positively for GFAP.

The differential diagnosis of our case includes non-melanotic melanoma of the choroid, angiomatosis retina, peripheral disciform lesion, secondary tumors with a late complication of chronic retinal detachment, subretinal drainage during the retinal detachment surgery, and vasoproliferative tumors of the fundus.¹⁰

In conclusion, massive retinal gliosis is a very rare clinically important disorder. Distinguishing of massive retinal gliosis from intraocular neoplasm, especially choroidal melanoma may be difficult clinically.

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