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Case report

Retinal findings in membranoproliferative glomerulonephritis

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ABSTRACT

Purpose: To assess the evolution of retinal findings in patients with membranoproliferative glomerulonephritis (MPGN) by funduscopy, intravenous fluorescein angiography and optical coherence tomography.

Observations: Three women and one man were followed for a period of 1.5–37 years. Four patients (8 eyes) had drusen detected at first fundus exam at age 24, 29, 50 and 55. Three patients (6 eyes) had diffuse thickening of Bruch's membrane, and two patients (3 eyes) had detachment of the retinal pigment epithelium with serous retinal detachment. Drusen tended to widen over a period of 10-year follow-up in one case.

Conclusion and importance: Drusen remain the ocular stigmata for MPGN occuring at an early age. The retinal disease is progressive with gradual thickening of Bruch's membrane and occurrence of retinal pigment epithelium detachment.

1. Introduction

Membranoproliferative glomerulonephritis (MPGN) is a rare progressive glomerular disease with poor prognosis. It was previously classified ultrastructurally into 3 patterns based on location of electron dense deposits: subendothelial deposits for Type I, diffuse dense deposits in the glomerular basement membrane for Type II, and both subepithelial and subendothelial deposits for Type III. More recently and based on the role of complement in its pathogenesis,[1] many researchers proposed reclassifying MPGN into immunoglobulin-mediated disease (activation of classical complement pathway) and non-immunoglobulin-mediated disease (activation of the alternative complement pathway).[2–4] The vast majority of MPGN cases represent immune-complex disease while a minority (around 5%) represent complement-mediated C3 glomerulonephritis.[2]

We present the findings of a case series of MPGN collected through soliciting cases to members of the Pan American Retina & Vitreous Society (Table 1).

2. Findings

Case 1. This 53-year-old Caucasian woman with MPGN II was asymptomatic with best corrected visual acuity of 20/25 (right eye) and 20/20 (left eye). Funduscopy revealed bilateral diffuse drusen (Fig. 1) involving uniquely the posterior pole (Fig. 2) with retinal pigment epithelium (RPE) alterations in the right eye on fluorescein angiography (Fig. 2) and diffusely thickened Bruch's membrane (Fig. 3).

Case 2. This 50-year-old Caucasian woman, product of first cousin parents, was followed since age 13 in the Motility clinic for blurred

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Table 1
Clinical summary of cases with membranoproliferative glomerulonephritis with positive eye exam.

	Case 1	Case 2	Case 3	Case 4
Age at first presentation	52	14	24	52
Gender	Female	Female	Male	Female
Race	Caucasian	Caucasian	Caucasian	Caucasian
Systemic disease	Diabetes mellitus; HTN	Diabetes mellitus; HTN	HTN	Monoclonal gammopathy
Type of MPGN	II*	Probably Primary**	II*	Probably Secondary*
Duplication of basal lamina of glomerulus	Yes	Yes	Yes	Yes
Antibodies present (IgG, IgM)	No	Yes	Yes	Yes
Complement present (C3)	Yes	Yes	Yes	Yes
Smoker	No	No	No	No
Age at dx of MPGN	50	28	24	55
HTN duration (years)	2	26	3	No HTN
Hemoglobin level serum	11.0	13.0	11.1	NA
Serum creatinine mg/dl	2.2	1.6	1.5	5.0
Oral corticosteroid intake	Yes	Yes	No	Yes
Mycophenolate mofetil intake	Yes	Yes	No	Yes
Age at first diagnosis of drusen	50	29	24	55
Initial vision	20/25 OD 20/20 OS	20/20 OD 20/30 OS	20/20 OD 20/20 OS	20/60 OD 20/40 OS
Final vision	20/25 OD 20/20 OS	20/30 OD 20/30 OS	20/20 OD 20/20 OS	20/200 OD 20/40 OS
Length of follow-up (years)	1.5	37	2.5	2.0
Drusen type (laminar)	Yes	Yes	Yes	Yes
Drusen location	Diffuse till equator	Diffuse till equator	Macula	Posterior pole
Drusen size (microns)	50	200	25	50
Fundus Autofluorescence	Yes	Yes	No	Yes
Diffuse thickening of Bruch's membrane	Yes	Yes	No	Yes
Serous retinal detachment	No	Yes	No	Yes
RPE detachment	No	Yes	No	Yes
Choroidal new vessel	No	No	No	No

Abbreviations: HTN, systemic hypertension; MPGN, membranoproliferative glomerulonephritis; NA, not assessed; OD, right eye; OS, left eye, RPE, retinal pigment epithelium (one asterisk refers to documentation by transmission electron microscopy, and 2 asterisks refers to negative workup for secondary causes of MPGN).

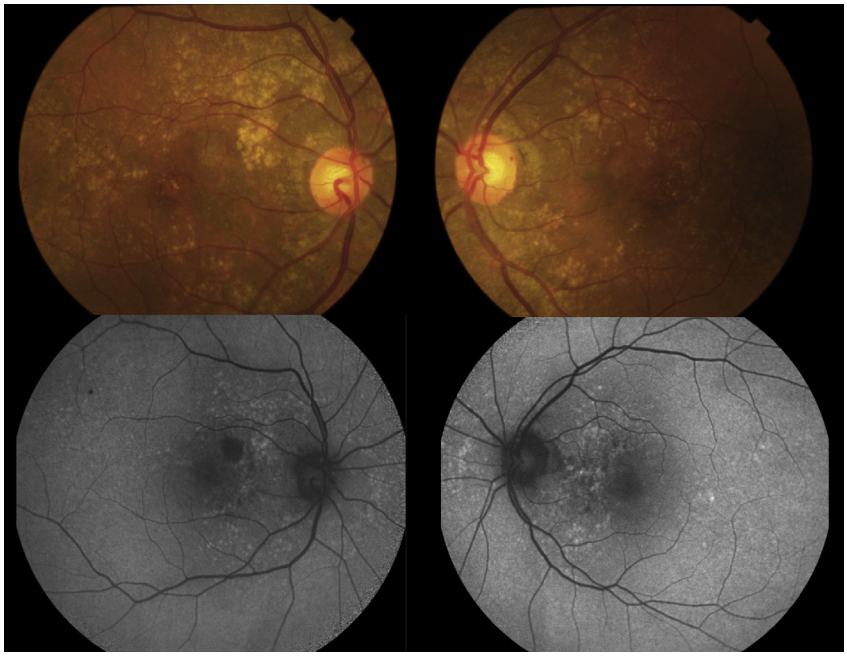

Fig. 1. Case 1-Color and autofluorescence of Case 1 showing posterior pole drusen in both eyes. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

vision of 20/40 attributed to esophoria and hyperopia. At age 24, she was started on oral corticosteroid and various immunosuppressive regimens to treat the glomerulonephritis. At age 27, renal biopsy revealed 30% loss of glomeruli with linear deposit at the glomerular basement membrane but negative immunofluorescence for C3 and immunoglobulins. Drusen were first detected at age 29 concomitant with a second kidney biopsy compatible with MPGN (positive immunofluorescence for complement C3 and IgM). At age 30, visual loss in the left eye was attributed to central serous chorioretinopathy. There was subfoveal RPE detachment that resolved after 6 months. Ten years later, there was progression of drusen along with diffuse marked thickening of Bruch's membrane (Figs. 4–7) with preservation of vision at 20/30 level in both eyes.