

MESANGIOCAPILLARY GLOMERULONEPHRITIS TYPE II AND MACULAR DEGENERATION

GLOMERULONEFRITIS MESANGIOCAPILAR TIPO II Y DEGENERACIÓN MACULAR

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ABSTRACT

Case report: We report a 34-year-old woman with biopsy proven type II mesangiocapillary glomerulonephritis (MCG II) who had an ophthalmic fundal appearance similar to that seen in patients with age-related macular degeneration (ARMD).

Discussion: All patients with MCG II should be reviewed regularly by an ophthalmologist to assess and treat any retinal complications (*Arch Soc Esp Oftalmol 2007; 82: 43-46*).

Key words: Mesangiocapillary glomerulonephritis type II, macular degeneration, drusen, lipodystrophy, ARMD.

RESUMEN

Caso clínico: Se describe una mujer de 34 años diagnosticada por biopsia de una glomerulonefritis mesangiocapilar tipo II (GMC II) y con un fondo de ojo similar al que se puede describir en un paciente con degeneración macular asociada a la edad (DMAE).

Discusión: Todos los pacientes con GMC II deben ser evaluados periódicamente por un oftalmólogo para controlar las complicaciones retinianas.

Palabras clave: Glomerulonefritis mesangiocapilar tipo II, degeneración macular, drusas, lipodistrofia, DMAE.

INTRODUCTION

Mesangiocapillary glomerulonephritis represent 10% of all diagnosed cases of glomerular disease, with mesangiocapillary glomerulonephritis type II (GMC type II) accounting for 20% thereof (1,2). The diagnosis of GMC type II is histological, demonstrating in the capillary basal membrane the presence of «dense deposits» (1,2) which histologi-

cally are similar to retinal drusen (2,3). Most patients with GMC type II develop severe visual loss as the disease progresses. This loss is sometimes irreversible. However, retinopathy and its association with GMC type II is generally not established by the specialists who treat these patients. We describe the case of a young woman with GMC type II which developed all the typical elements of the disease.

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CASE REPORTS

A 26 year-old woman referred to the ophthalmology practice in Sept. 2005 by the nephrology service due to bilateral painless reduction of main eyesight in the left eye (LE) with several weeks of evolution. In May 2001 she was diagnosed after a kidney biopsy with mesangiocapillary glomerulonephritis type II (proliferative membrane) associated to partial lipodystrophia. The physical assessment showed an aged woman with increased nasogenian lines due to loss of fat, although when she

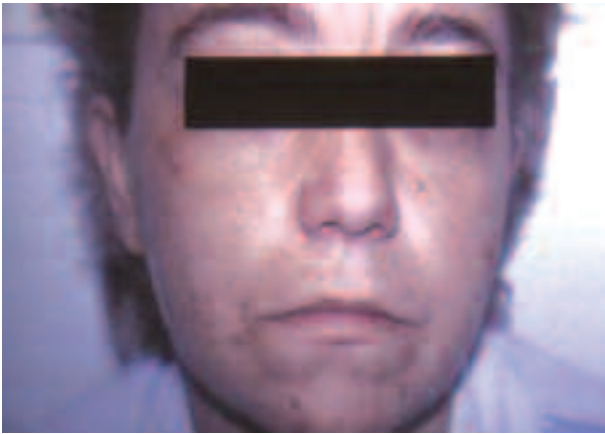


Fig. 1: Typical face of patients with mesangiocapillary glomerulonephritis type II. Lipodystrophy is due to loss of facial fat. This case has skin coloring and final renal insufficiency edema.

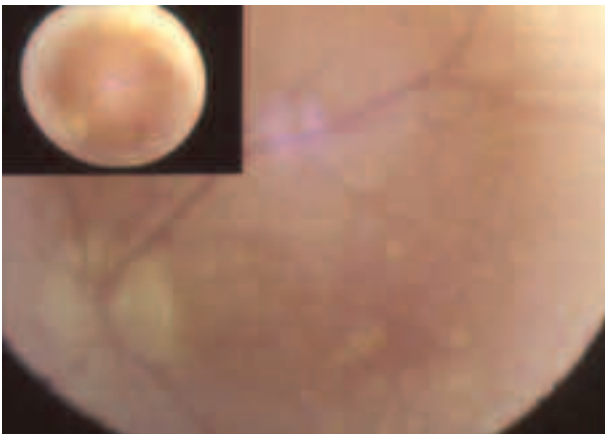


Fig. 2: LE retinography: enlargement of posterior pole in patient with mesangiocapillary glomerulonephritis type II undistinguishable from ARMD eye fundus. In the top left insert, complete retinography.

was explored in the practice she exhibited a general edema due to terminal kidney insufficiency (fig. 1). The maximum visual acuity in the right eye (RE) was of 0.5 and in the LE of 0.15 difficult. Anterior biomicroscopy did not present relevant data. The retinal study showed central, uniform-looking deposits in the central area, similar to hard drusen which, around the macula, were grouped and in larger sizes with semi-solid appearance (fig. 2). The angiogram showed hyperfluorescence of choroidal transmission in the drusen area and in late times no loss of contrast, but macular pigmentary epithelium alterations were evidenced (fig. 3). In Nov. 2005 a kidney transplant operation was performed but she died 15 days later due to multiorganic failure.

DISCUSSION

In general, glomerular diseases constitute the original of terminal kidney insufficiency in one third of patients treated with dialysis or kidney transplant (1). Mesangiocapillary glomerulonephritis type II (proliferative membrane) is part of a group of processes which have in common an excess of cells (proliferation) in the glomerule. The type we are referring to is characterized by a thickening of the capillary wall secondary to a dense linear deposit inside the basal membrane, made up mainly by C3 (1). GMC II mainly affects children in school age and young adults and is slightly more frequent in females than in males (1). Clinical presentation (which influences prognosis) can be non-

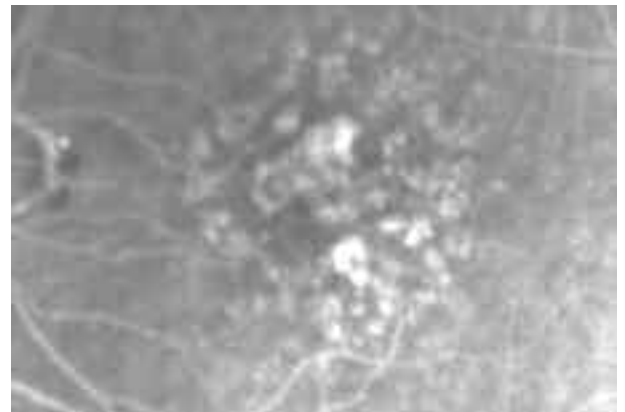


Fig. 3: LE Angiography: accumulation of contrast in late times as if there was a pigmentary epithelium detachment.

symptomatic proteinuria, recurrent hematuria, nephrotic syndrome or acute nephritis simulation. Prognosis is usually negative, and not die due to uremia, high pressure or problems derived from transplant within a 10-year term (1-3). Patients with GMC II usually have partial lypodystrophia and, as a characteristic of the disease, a persistent reduction of C3 (1-3). The majority of these patients also have subretinal deposits clinically similar to drusen, histopathologically identical to glomerular basal membrane deposits (2,3). They usually have good eyesight in the beginning of the disease but night vision gradually diminishes together with VA. In time, the presence of drusen is evident and one third of patients develop neovascularization, particularly in the macular region, with an evolution similar to that of ARMD membranes (2,3). The patient we describe exhibited clinical and angiographic characteristics which cannot be differentiated from ARMD were it not for her age. Pickering MC et al (4) demonstrated that uncontrolled activation of C3 originates GMC in mice with factor H deficiency. Hageman et al (5) published that a variant in the factor H gene (HF1/CFH) greatly increased the development of ARMD and GMC type II. Factor H is a key component which regulates the alternative pathway of the supplement, and all the above mentioned research consider the factor H gene only in the description of type II and ARMD. This work is

focused not on an infrequent pathology (GMC II) but on raising the attention of ophthalmologists and nephrologists about the fact that these patients need regular ophthalmological studies to avoid losing their eyesight and can benefit from the new antiangiogenic therapies.

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