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Case report

Drusen Formation in Type II Membranoproliferative Glomerulonephritis after Renal Transplantation

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Abstract

Type II membranoproliferative glomerulonephritis (MPGN) is a systemic disease that almost invariably recurs in renal allografts. This is a case of a 45-year-old woman with biopsy proven type II MPGN that led to renal failure 10 years after diagnosis. During the fifth month after cadaveric transplantation she was treated with pulse doses of methylprednisolone owing to acute T-cell mediated rejection without pathohistological signs of type II MPGN recurrence. One month later the patient was hospitalized due to acute bilateral vision deterioration. Ophthalmoscopy showed bilateral, multifocal drusen, concentrating in the posterior pole, and exudative ablation of the retinal pigment epithelium. Ocular coherence tomography (OCT) revealed focal retinal pigment epithelial elevation and detachments. The patient was treated with methylprednisolone (1 mg/kg) for 3 days. Therapy led to regression of exudation and flattening of the pigment epithelial detachments with discrete subjective visual improvement. Type II MPGN almost invariably recurs and leads to graft failure in 50% of cases. Our patient had evident chronic eye changes due to type II MPGN leaving allograft function intact during the first year of follow-up. Considering these potentially devastating effects of the disease, type II MPGN patients should be observed carefully from both the renal and eye point of view, because the severity of ocular changes, like in our case, is not always in line with allograft function.

Key words: type II membranoproliferative glomerulonephritis, drusen

Introduction

Type II membranoproliferative glomerulonephritis (MPGN) is a systemic disease of insufficiently understood origin [1]. The pathognomonic feature of type II MPGN is the presence of white-yellowish spots or drusen, extracellular dense depositions within the glomerular basement membrane (GBM), the choriocapillaris-Bruch's membrane-retinal pigment epithelial interface and the sinusoidal basement membranes of the spleen [2]. The first clinical report of fundus changes with "drusen-like" deposits was made by Duvall-Young in 1989 and since then drusen and retinal pigment epithelium damage have been recognized as features of type II MPGN [3]. Recent reports describe similar changes in type I MPGN [4]. Type II MPGN almost invariably recurs morphologically in renal allografts and, although progression to end-stage renal disease is not inevitable, half of the allografts ultimately fail [5].

Case report

We report a case of a 45-year-old woman with biopsy proven type II MPGN that led to renal failure 10 years after being diagnosed. After a year of hemodialysis she received a renal allograft from a cadaveric donor. Induction therapy consisted of antithymocyte globulin (9 mg/kg) followed by maintenance with tacrolimus (0.15 mg/kg), mycophenolate sodium and prednisone. During the fifth month after transplantation she was treated with pulse doses of methylprednisolone due to an episode of biopsy proven acute T-cell mediated rejection, without pathohistological signs of type II MPGN recurrence. At discharge allograft function was stable (Table 1).

Table 1. Allograft function during ocular drusen formation

	At the onset of visual deterioration	At discharge	One year later
scr (µmol/l)*	97	88	86
ccr (ml/min)**	89	98	79
Proteinuria (g/day)	0.2	0.12	0.04

*scr=serum creatinine, **ccr=creatinine clearance

One month later the patient was hospitalized due to acute bilateral vision deterioration. Blood tests revealed normal allograft function without any evidence of sys-

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temic inflammation (Table 1). There were no signs of vasculitis or recent history of accelerated hypertension. The NMR excluded expansive processes in the endocranium. Ophthalmoscopy showed bilateral, multifocal drusen, concentrating in the posterior pole and exudative ablation of the retinal pigment epithelium (Figure 1). In addition, ocular coherence tomography (OCT) revealed focal retinal pigment epithelial elevation and detachments (Figure 2). The patient was treated with methylprednisolone (1 mg/kg) for 3 days, with graduated dose reduction. The control OCT 4 months later showed

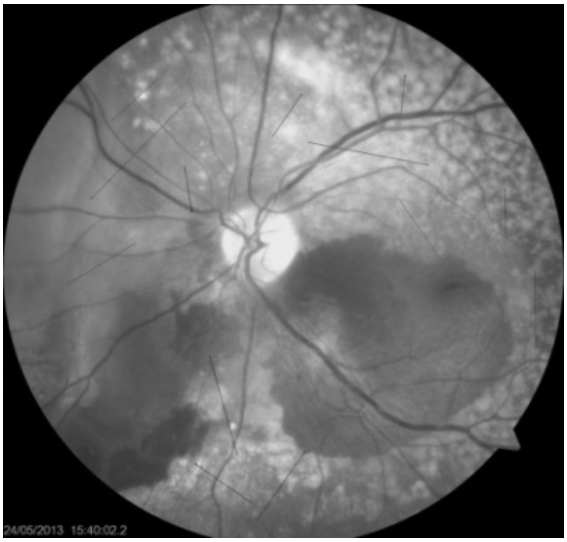

Fig. 1. Ophthalmoscopy: drusen and subretinal hemorrhage of the left eye

regression of exudation and flattening of the pigment epithelial detachments followed by discrete subjective visual improvement. During the follow-up of 1 year there were no reported signs of allograft function deterioration or proteinuria (Table 1).

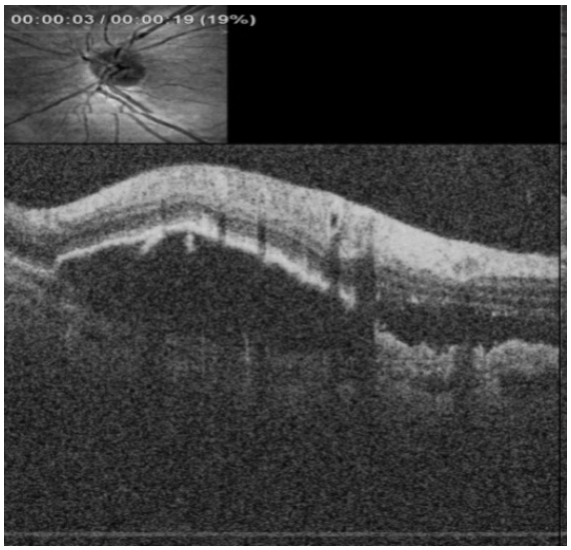

Fig. 2. Ocular coherence tomography: retinal pigment epithelial elevation of the left eye

Discussion

Type II MPGN is an uncommon renal condition leading to end-stage renal failure in 6 to 10 years [6)]. Its systemic nature lies in the fact that the electron dense deposits may also be found in the spleen, choriocapillaris and Bruchs membrane of the eye and because the disease has a tendency of recurring in allografts [7]. Unlike ocular drusen in age-related macular degeneration, typically seen in patients over 50 years old, those in type II MPGN occur at an early age and correlate with the duration of the renal disease [7,8]. These deposits initially have little impact on visual acuity. However, long-term visual deterioration caused by subretinal neovascularisation, macular detachments and central serous retinopathy occurs in approximately 10% of patients with type II MPGN [1].

There is no correlation between disease severity in the kidney and in the eye, so an ophthalmologic examination at the time of diagnosis including periodical fundoscopic assessments as part of the patient's treatment is suggested [9)]. Our patient had posterior pole drusen deposition with hemorrhagic ablation of the retinal pigment epithelium and acute vision deterioration suggesting bleeding from subretinal choroidal neovascular membranes and a chronic nature of the eye deposition. There is no universally effective treatment for type II MPGN, including the eye changes [1]. There are data suggesting spontaneous and significant improvement in the anatomical and clinical picture in the retina of patients suffering from type II MPGN [10]. We treated our patient with pulse doses of corticosteroids for 3 days and subsequent low doses of prednisone. Therapy led to relative improvement of vision acuity and OCT findings. Type II MPGN almost invariably recurs morphologically in renal allografts leading to graft failure in 50% of cases [1]. In our case, the patient had only evident chronic eye changes due to type II MPGN, leaving allograft function intact during the first year of follow-up. Segmental areas of retinal pigment epithelium detachments associated with secondary exudation following organ transplantation were first described in 1992 and it was suggested that local intravascular coagulation induced by subclinical graft rejection may have some impact on the ocular symptoms [11].

However, some results of long-term monitoring indicated that renal transplantation did not appear to increase the risk of progression of drusen and choroidal neovascularisation in patients with type II MPGN [7, 9].

Conclusions

Considering the potentially devastating effects of the disease, type II MPGN patients should be observed carefully from both renal and eye points of view, although the severity of ocular changes, like in our case, is not always in line with allograft function.

Conflict of interest statement. None declared.

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