Case Report

Bilateral symmetric retinal detachment and multiple retinal pigment epithelial detachments during haemodialysis

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Introduction

In 1973 an ocular syndrome was described which had the following characteristics: (1) bilateral, exudative, bullous retinal detachment; (2) multiple detachments of the retinal pigment epithelium; (3) absence of inflammatory signs; (4) fluoroangiographic evidence of holes in the retinal pigment epithelium near the edge of the retinal pigment epithelium detachment; (5) inefficacy of the systemic administration of corticosteroids; and (6) rapid resolution of the retinal detachment after photoagulation of the detachment areas of the retinal pigment epithelium [1].

This syndrome occurs in healthy male patients as an unusual manifestation of a severe form of serous idiopathic central chorioretinopathy [2–4]. In 1992 this syndrome was also observed in two female patients undergoing haemodialysis [5]. This report describes the syndrome in a haemodialysed female patient affected by chronic renal failure.

Case

The patient was a 67-year-old white woman with ESRD due to polycystic kidney disease. Since 1988 the patient suffered from arterial hypertension treated with beta blockers (metoprolol 100 mg/die). In June 1991 aggravation of arterial hypertension and an initial normocytic, normochromic anaemia requires admission to our Renal Unit.

Renal echography and laboratory findings evidence bilateral polycystic kidney and chronic renal failure; she was dismissed after prescription of a dietetic and hypotensive therapy as follows: fosinopril 10 mg, Amlodipine 10 mg, doxasoxin 2 mg.

In January 1992 renal function deteriorated progressively, an arteriovenous fistula on the right forearm was performed and the patient started haemodialysis treatment. The dialysis schedule was 4 h three times a week using a 1.6 m² polyacrylonitrile high-permeability membrane. During haemodialysis anticoagulation was administered (heparin 1500 U in bolus + 1000 U every hour).

In May 1993 she complained of myiodesopsia (fluctuating visual hallucinations). In January 1994 she noted a progressive decrease in visual acuity which was 10/10 in both eyes with −4.00 sph + 2.00 astig in the right eye and −3.50 sph + 2.50 astig in the left eye.

Visual acuity was 10/10 in both eyes with −4.00 sph + 2.00 astig in the right eye and −3.50 sph + 2.50 astig in the left eye. In the right eye, near vision was 1 DW with −4.00 sph + 2.50 astig in the left eye and −3.50 sph + 5.50 astig in the left eye.

The anterior segment of both eyes appeared normal. A deficit of the m. superior obliquus of the left eye led to diplopia. In the fundus of the right eye a small intraretinal haemorrhage was observed (lower nasal position in respect to the optic disc) as well as an area of chorioretinal dystrophy in the periphery between the 11-o’clock and the 12-o’clock positions, with massive pigment mobilization.

In the fundus of the left eye a pre-equatorial bullous retinal detachment between the 11-o’clock and the 3-o’clock positions was observed (Figure 1), at the 7-o’clock position a retinal haemorrhage was revealed, and between the 11-o’clock and the 12-o’clock positions an area of chorioretinal pigmented atrophy was noted.

The fluoroangiographic examination showed a small retinal pigment epithelium detachment in the right eye, and in the left eye a retinal buckle containing fluid (probably serohaemorrhagic) with an important masking effect on the choroid. No therapy was administered.

During control examination (2 weeks follow-up) a progressive reduction of the retinal buckle was registered; on September 1993 only a small retinal buckle between the 11-o’clock and the 12-o’clock positions was observed.

The patient continued a monthly follow-up until
May 1994 when she presented because of myiodesopsia and phosphenes in the right eye. Examination of the fundus revealed a retinal buckle between the 11-o’clock and the 3-o’clock positions together with numerous small retinal pigment epithelium detachments. No treatment was performed and a 2-week follow-up was recommended. A progressive reduction of the retinal buckle was limited to a small area between the 1-o’clock and the 2-o’clock positions of the right eye from October of the same year.

On March 1995, the patient presented at the First Aid department complaining of a sudden visual impairment in the left eye. Case history revealed that for 3 months the dose of heparin had been increased. Ocular examination showed extensive chorioretinal haemorrhage with haemorrhagic clouding of the vitreous; a B-scan echographic examination showed that there was persisting residual retinal serous bilateral buckle (Figure 2). Once again no therapy was administered. In the following examinations the chorioretinal haemorrhage diminished progressively, while vitreal clouding and serous bilateral detachment were unchanged. In May 1995 in the right eye an initial syndrome of the vitreoretinal interface was observed. During the last examination in December 1995 the clinical picture was unchanged, except for an initial subcapsular posterior cataract; visual acuity of the right eye was 10/10 and 1 DW with corrections, while visual acuity of the left eye was 4/10 and 5 DW with the corrections.

Discussion

The clinical feature observed in this haemodialysed patient were the same as that observed in healthy patients with serous idiopathic central chorioretinopathy.

The histological alterations observed in patients with a similar clinical feature [5] induce us to suppose that the retinal detachment as well as the retinal pigment epithelium detachment in healthy subjects with serous idiopathic central chorioretinopathy and in dialysed patients are due to focal alteration of choriocapillar permeability which allow large molecules, like fibrinogen, to exit into the subretinal space.

In healthy subjects and in patients receiving haemodialysis, a decisive role in the pathogenesis of serous idiopathic central chorioretinopathy has been ascribed to a state of hyperactivity of the adrenomedullary system [6,7].

A similar picture was described in a female patient who underwent renal transplantation after a short period of haemodialysis treatment [8] and in several patients affected by nephritis due to lupus erythematosus without hypertension who never underwent haemodialysis [5,9].

Gass [5] suggested that renal failure was more important than hypertension and haemodialysis in the genesis of the retinal lesions. On the other hand the uraemic state, fluid shifts and variations of osmolarity in the different compartment before and after haemodialysis, may have contributed to altered permeability. However, the limited number of cases reported up to date does not allow us to definitely state that there is a relationship between the retinal alterations and the uraemic state or its treatment.

In conclusion, patients affected by renal failure, above all when undergoing haemodialysis have apparently a higher risk of sudden visual impairment due to
an exudative retinal detachment. This may affect both eyes at different times. Similar risk may exist in patients with a renal transplantation or with transplantation of another major organ.

There is no significant treatment for this ocular condition: in some cases laser photocoagulation of the areas of the retinal pigment epithelium detachment was followed by rapid resolution of the retinal detachment [5].

References


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