rapidity of microbial keratitis onset in this patient. We therefore suggest that specific inquiry regarding recreational drug abuse, including cocaine, should be made in any patient who presents with microbial keratitis.

References

1. Rossenwasser GOD, Holland S, Pflugfelder SC, *et al.* Topical anesthetic abuse. Ophthalmology 1990;97:967–72.
2. Strominger MB, Sachs R, Hersh PS. Microbial keratitis with crack cocaine. Arch Ophthalmol 1990;108:1672.
3. Sachs R, Zagelbaum BM, Hersh PS. Corneal complications associated with the use of crack cocaine. Ophthalmology 1993;100:187–91.
4. McHenry JG, Zeiter JH, Madion MP, Cowden JW. Corneal epithelial defects after smoking crack cocaine. Am J Ophthalmol 1989;108:732.
5. Stapleton F, Dart JK, Minassian D. Risk factors with contact lens related suppurative keratitis., CLAO J 1993;19:204–10.

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Sir,

Central serous chorioretinopathy in a patient with cryoglobulinaemia

Cryoglobulinaemia has rarely been reported to cause ocular complications. Retinal vascular tortuousity and occlusions, cotton wool spots, retinal haemorrhages and uveitis have all been described in this disease.[1] However, central serous chorioretinopathy (CSC) has been described in only two cryoglobulinaemia patients. We present a case of CSC and branch retinal vein occlusion (BRVO) occurring in a patient with hepatitis-C-induced cryoglobulinaemia.

Case report

A 36-year-old man was referred to our clinic for routine ophthalmic examination. He was known to have cryoglobulinaemia secondary to chronic active hepatitis C for the past 6 years. His ocular history was unremarkable. Among the systemic complications of this condition were leucocytoclastic cutaneous vasculitis, manifesting as recurrent bouts of non-thrombocytopenic palpable purpura on the legs. He later developed nephrotic syndrome, and a renal biopsy showed mesangiocapillary glomerulonephritis. Oral prednisone, 60 mg/day, was therefore started, with significant reduction in proteinuria. Arterial hypertension was well controlled by atenolol, furosemide and hydrochlorothiazide, with values around 120/80 mmHg. He had mild normocytic normochromic anaemia. His serum glucose was 5 mmol/l, the creatinine clearance was 60 ml/min, total cholesterol was 2.6 mmol/l. Liver function tests showed mildly increased hepatocellular indices, and a cholestatic disturbance, with alkaline phosphatase levels around 200 IU/l (normal up to 126 IU/l) and gamma-GTP of 3000 IU/l (normal up to 76 IU/l). Serum albumin was 41 g/l. Total serum bilirubin was 13 μmol/l with 7 μmol/l direct fraction. Plasma immune electrophoresis showed 10% plasma cryoglobulin and an IgM kappa gammopathy. Hepatitis C virus RNA was found in his plasma by quantitative polymerase chain reaction in a titre of 186 copies/ml. Antinuclear factor and rheumatoid factor were negative; however, C3 complement level was 0.3 g/l (normal 0.71–1.56 g/l).

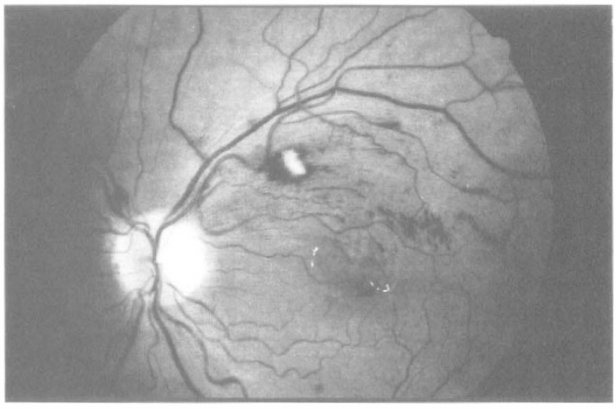

Fig. 1. *A red-free photograph of the left fundus shows a superotemporal branch retinal vein occlusion with nerve fibre layer haemorrhages and a single cotton wool spot.*

Upon examination, his visual acuity was 6/6 in both eyes. Anterior segments were normal in both eyes. The retinal vessels were slightly congested and tortuous in both eyes. There was a superotemporal BRVO in the left fundus, without macular oedema or retinal neovascularisation (Fig. 1). During the following month the patient remained asymptomatic and the retinal haemorrhages gradually cleared.

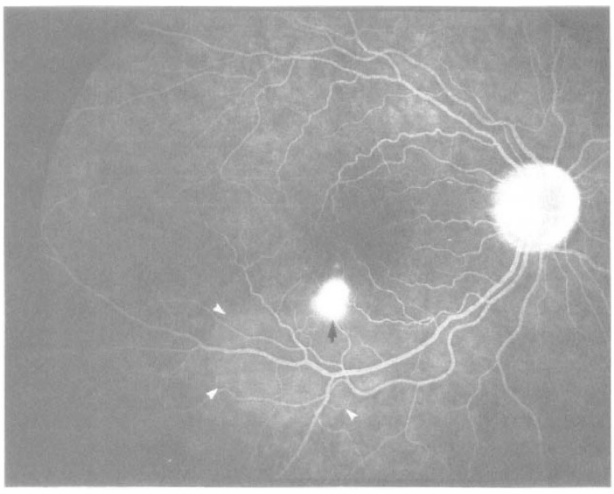

Fig. 2. *A late-phase fluorescein angiogram of the right eye shows a typical central serous chorioretinopathy with serous retinal detachment (white arrowheads) and a focal leakage point (black arrow).*

One year later the patient was still on prednisone 20 mg/day, and a routine examination revealed an asymptomatic serous retinal detachment over the inferior temporal vascular arcade of the right fundus. Fluorescein angiography showed late leakage of dye in an inkblot pattern, and confirmed the diagnosis of CSC (Fig. 2). No therapy was given, and the CSC spontaneously resolved over the following 4 months without becoming symptomatic at any time.

Comment

CSC is usually an idiopathic disorder, mostly affecting young men. It has been associated with various systemic conditions such as pregnancy[2] and systemic corticosteroid or adrenocorticotrophic hormone excess.[3] We found only two reports of CSC occurring in patients with paraproteinaemias. Altogether, four patients have been described: one[4] had Waldenström's macroglobulinaemia and developed bilateral serous retinal detachment. He was not treated with steroids. Two patients[5] had cryoglobulinaemia and were taking systemic prednisone before developing typical CSC. One patient[5] had a benign mixed IgA and IgM gammopathy and was never treated with steroids.

Although corticosteroid use may have had a pathogenic role in two of these patients, the fact that some patients were not treated with steroids may indicate an independent effect of the paraproteinaemia *per se*. Also notable in this respect is the fact that our patient recovered from the CSC despite continued prednisone therapy. Cohen *et al.*[5] attributed the accumulation of subretinal and sub-RPE fluid to increased capillary permeability from paraproteinaemia, coupled with excessive plasma protein concentration.

Our patient developed two different retinal complications of paraproteinaemia, namely BRVO and CSC. Retinal vein occlusion is a well-known complication of paraproteinaemias and other hyperviscosity states. In contrast, CSC has been described in only two other patients with cryoglobulinaemia.[5] The pathogenic mechanism of the latter is still speculative. However, we suggest that serous retinal detachment may develop in these patients, as well as the other, better-known ocular features of their disease.

References

1. Talks SJ, Shah P, Willshaw HE, Jubb RW. Cryoglobulinaemia masquerading as rheumatoid vasculitis: the retina provides the clue. Eye 1996;10:399–402.
2. Chumbley LC, Frank RN. Central serous retinopathy and pregnancy. Am J Ophthalmol 1974;77:158–60.
3. Zamir E. Central serous retinopathy associated with adrenocorticotrophic hormone therapy: a case report and a hypothesis. Graefes Arch Clin Exp Ophthalmol 1997;235:339–44.
4. Thomas EL, Olk RJ, Markman M, Braine H, Patz A. Irreversible visual loss in Waldenström's macroglobulinemia. Br J Ophthalmol 1983;67:102–6.
5. Cohen SM, Kokame GT, Gass JD. Paraproteinemias associated with serous detachments of the retinal pigment epithelium and neurosensory retina. Retina 1996;16:467–73.

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Sir,

Intraocular pressure, pulse amplitude and pulsatile ocular blood flow measurement in premature infants screened for retinopathy of prematurity

Screening for retinopathy of prematurity (ROP) in the United Kingdom is performed in accordance with the guidelines of the Working Party of the British Association for Perinatal Medicine and the College of Ophthalmologists.[1] Approximately 1% of all infants fulfil the screening criteria.[2] Although between 30% and 60% of those infants screened develop ROP of some stage, only a minority (8–10%) of these develop the advanced stages of ROP that have a poor visual outcome and that require treatment.[3–5] Examination of the peripheral retina through dilated pupils with an indirect ophthalmoscope is the only method currently used in screening. We felt that measurement of ocular blood flow might be of benefit in predicting those infants at particular risk of blinding disease. To evaluate this hypothesis we measured intraocular pressure (IOP), pulse amplitude (PA) and pulsatile ocular blood (POBF) flow on neonates being screened for ROP.

Report

Infants fulfilling the screening criteria for ROP were recruited into the study. Verbal and written consent was obtained from the parent(s) beforehand. The pupils were dilated with tropicamide 0.5% and phenylephrine 2.5%. IOP, PA and POBF were measured in the supine position under topical anaesthesia using the Ocular Blood Flow Tonograph (OBF Labs UK). The measurement was performed as soon as possible after fundal examination with an indirect ophthalmoscope. A paediatric speculum was used when necessary. No more than two separate recordings were taken to obtain a minimum of five IOP, PA and POBF values with a standard deviation of less than 15%. Full monitoring took place throughout both examinations. The birth data, IOP, PA and POBF measurements and the ROP stage for both eyes were recorded for each examination, and comparison made between those infants with and without ROP. Local ethics committee approval had been obtained for this study.