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.

Table 1. *Median pulsatile ocular blood flow, pulse amplitude and intraocular pressure for the 24 eyes with reproducible data*

ROP stage	No. of eyes	POBF (μl/min)	Pulse amplitude (mmHg)	Intraocular pressure (mmHg)
None	10	1603	2.7	14.0
Stage 1 zone 3	12	1452	3.1	15.6
Threshold	2	1057	2.9	22.4
All infants	22	1603	2.9	15.5

POBF, pulsatile ocular blood flow.

Twenty infants were recruited into the study and reproducible data were obtained for 24 eyes from 13 infants at the time of initial or follow-up screening. The median post-conceptional age at the time of examination of these 13 infants was 34 weeks and there were 8 female and 5 male infants. Ten eyes had no ROP, 12 had stage 1 zone 3 disease and 2 eyes had threshold disease. For the 24 eyes the following median values were obtained: IOP was 15.5 mmHg (CI = 13.5–18.2), PA was 2.9 mmHg (CI = 2.6–4.0) and POBF was 1603 μl/min (CI = 1400–1790). It was not possible to demonstrate a correlation between POBF values and either increasing post-conceptional age or the stage of ROP (Table 1).

Comment

The aim of this study was to provide baseline POBF parameters for premature infants and to investigate the use of POBF measurement as a potential method of predicting those infants at particular risk of blinding ROP. Although reproducible baseline data were obtained, the small sample size prevented any useful evaluation of POBF measurement as a predictive feature of disease severity.

The median IOP of 15.5 mmHg for the 24 eyes in this study is higher than that found in previous studies.[6,7] This may be the result of a difference in either the post-conceptional ages or, more importantly, the recording method.[8] Topical mydriatics and the stress response to conventional ROP screening may affect pulse rate and blood pressure and therefore may also affect PA and POBF.[9] To our knowledge, however, data for PA and POBF does not exist for premature infants. We were unable to show a change in any parameter with post-conceptional age or with the stage of ROP. This may in part be the result of the small sample size and in particular the fact that only one infant had ROP more advanced than stage 1 zone 3 disease.

Of the many techniques available to monitor ocular blood flow, we felt that the Ocular Blood Flow Tonograph was most likely to be useful in studying blood flow in neonates. The device provides a fast, accurate and repeatable measurement of POBF and has been used to study it in several conditions in which the ocular blood flow is known or believed to be impaired.[10–12] However, specific difficulties were encountered with the measurement when infants had become agitated during or after conventional screening. This was a particular problem with infants of increasing post-conceptional age. These difficulties are such that we believe that while reproducible IOP, PA and POBF measurements are possible, widespread use of this technique in premature infants is not practical.

References

1. Working Party of the British Association of Perinatal Medicine and the College of Ophthalmologists. Screening for retinopathy of prematurity. London, 1990.
2. Maly E. Frequency and natural history of retinopathy of prematurity: a prospective study in a Swedish city 1986–1990. Acta Ophthalmol (Copenh) 1993;210(Suppl):52–5.
3. Landers MB, Toth CA, Semole C, Morse LS. Treatment of retinopathy of prematurity with argon laser photocoagulation. Arch Ophthalmol 1992;110:44–7.
4. Ng YK, Fielder AR, Shaw DE, Levene MI. Epidemiology of retinopathy of prematurity. Lancet 1988;II:1235–8.
5. The Royal College of Ophthalmologists and the British Association of Perinatal Medicine. Retinopathy of prematurity: guidelines for screening and management. London, 1995.
6. Tucker SM, Enzenauer RW, Levin AV, Morin JD, Hellmann J. Corneal diameter, axial length and intra-ocular pressure in premature infants. Ophthalmology 1992;99:1296–300.
7. Spicer A, Huna R, Hirsh A, Chetrit A. Normal intraocular pressure in premature infants. Am J Ophthalmol 1994;117:810–3.
8. Eisenberg DL, Sherman BG, McKeown CA, Schuman JS. Tonometry in adults and children. A manometric evaluation of pneumotonometry, applanation and Tonopen in vitro and in vivo. Ophthalmology 1998;105:1173–81.
9. Laws DE, Morton C, Weindling M, Clark D. Systemic effects of screening for retinopathy of prematurity. Br J Ophthalmol 1996;80:425–8.
10. Silver DM, Farrell RA. Validity of pulsatile ocular blood flow measurements. Surv Ophthalmol 1994;38(Suppl):72–80.
11. Claridge KG, James CB. Ocular pulse measurements to assess pulsatile blood flow in carotid artery disease. Br J Ophthalmol 1994;78:321–3.
12. James CB, Smith SE. Pulsatile ocular blood flow in patients with low tension glaucoma. Br J Ophthalmol 1991;75:466–70.

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