after intravitreal injection of ranibizumab in neovascular age-related macular degeneration. *Eye* 2010; **24**: 1825.
3 Charalampidou S, Nolan J, Ormonde GO, Beatty S. Visual perceptions induced by intravitreous injections of therapeutic agents. *Eye* 2010; **25**: 494–501.

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Sir,

Recurrent intradialytic elevation of intraocular pressure in a case of neovascular glaucoma

We report a case of elevated intraocular pressure (IOP) during haemodialysis (HD), the commonest treatment for end-stage renal failure.

Case report

A 48-year-old male presented with recurrent right ocular pain and nausea during regular HD for post-kidney transplant failure. He was diagnosed with right central retinal vein occlusion 10 weeks previously. He developed rubeotic glaucoma 4 weeks earlier, with 360°-angle neovascularisation (Shaffer Grade 4 all quadrants on gonioscopy) and visual acuities (VA) of hand movements (HM), right and 6/6 left. IOPs were 39 mm Hg right and 11 mm Hg left. Treatment included pan-retinal photocoagulation, cyclodiode and topical IOP-lowering drugs. IOP reduced to 19 mm Hg and rubeosis regressed.

At presentation, 10-min post-dialysis, VAs were barely HM right and 6/6 left with IOPs of 62 and 10 mm Hg, respectively. Intravenous acetazolamide and topical agents lowered IOP to 20 mm Hg. He presented with further intradialytic symptoms and IOPs of 60 and 55 mm Hg two further times. The eye was comfortable between HD with IOP consistently 19–24 mm Hg. We initiated oral acetazolamide 250 mg SR the evening before and on the day of HD. After 5 months, he remains comfortable during HD, with IOPs immediately post-HD, consistently between 22–25 mm Hg.

Comment

The effect of HD on IOP has been widely studied, with conflicting reports. A systematic review concluded that the relationship was not clear.[1] Reports of IOP rise during HD in neovascular[2,3] and pseudo-exfoliative glaucoma are extremely limited.[4,5] Mechanisms for elevated IOP during HD are unclear; some authors propose that reduced plasma osmolality causes increased aqueous humour production and potential to elevate IOP.[3] Normal eyes may increase outflow to compensate.

In our patient, we suggest that raised IOP resulted from imbalance between aqueous outflow, due to obstruction by angle neovascularisation and aqueous production. The higher IOP in the affected eye between dialysis sessions supports a theory of outflow obstruction; the drainage system could not compensate for increased aqueous during HD.

Regular acetazolamide, an interim measure until renal transplant, has successfully prevented IOP surges without causing side effects in our patient, particularly metabolic acidosis. Other reported strategies include cyclodiode,[3] filtration surgery,[2] adjusted HD including a hyperosmotic agent[5] or eviscerating treatment-resistant cases.[2]

Clinicians must recognise that IOP may rise intolerably during HD in glaucoma patients. We suggest that acetazolamide, with close systemic monitoring, is an effective and safe strategy.

Conflict of interest

The authors declare no conflict of interest.

References

1 Levy J, Tovbin D, Lifshitz T, Zlotnik M, Tessler Z. Intraocular pressure during haemodialysis: a review. *Eye* 2005; **19**(12): 1249–1256.
2 Song WK, Ha SJ, Yeom HY, Seong GJ, Hong YJ. Recurrent intraocular pressure elevation during haemodialysis in neovascular glaucoma. *Korean J Ophthalmol* 2006; **20**(2): 109–112.
3 Choong YF, Menage MJ. Symptomatic acute raised IOP following hemeodialysis in a patient with end stage renal failure. *Br J Ophthalmol* 1998; **82**(11): 1342.
4 Masuda H, Shibuya Y, Ohira A. Markedly increased unilateral intraocular pressure during haemodialysis in a patient with ipsilateral exfoliative glaucoma. *Am J Ophthalmol* 2000; **129**(4): 534–536.
5 Ikeda N, Saito T, Hayasaka S, Hayasaka Y. Unilateral symptomatic elevation of intraocular pressure and prevention using a hyperosmotic agent during haemodialysis. *Jpn J Ophthalmol* 2001; **45**(6): 659–661.

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Sir,

Acute lymphocytic leukemia relapsing as bilateral serous retinal detachment: a case report

We report a case of bilateral serous retinal detachment (SRD) as an initial sign of relapse of acute lymphocytic leukemia. A 31-year-old woman with acute pre B-cell lymphocytic leukemia in complete remission, who presented with symptoms of visual blurring, was found to have bilateral SRD. A bone marrow aspirate revealed relapse of the disease. Her maculopathy completely resolved following systemic chemotherapy.

Case report

A 31-year-old woman with acute pre B-cell lymphocytic leukemia achieved complete remission 3 months after treatment with chemotherapy (CALGB881), brain radiation, and intrathecal methotrexate. She was designated for allogenic bone marrow transplantation (BMT) and

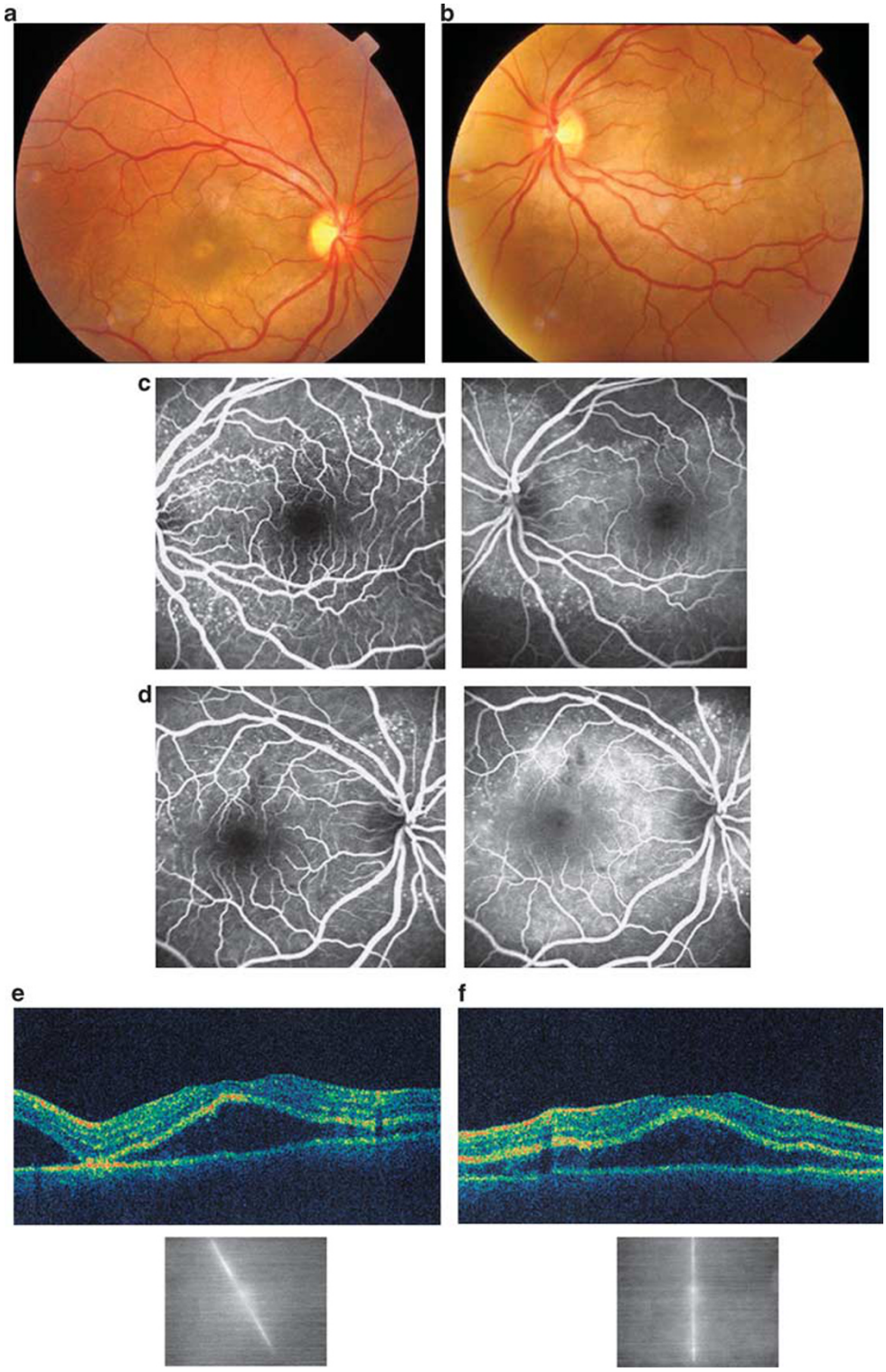

Figure 1 Fundus photograph (a) of the right eye showing SRD in the posterior pole (b) left eye showing SRD in the posterior pole. Fluorescein angiography of the left eye (c) and right eye (d) showing early multifocal hyperfluorescence foci beneath the detachment, with late subretinal diffuse leakage. Optical coherence tomography of the left eye (e) and right eye (f) showing subretinal fluids.

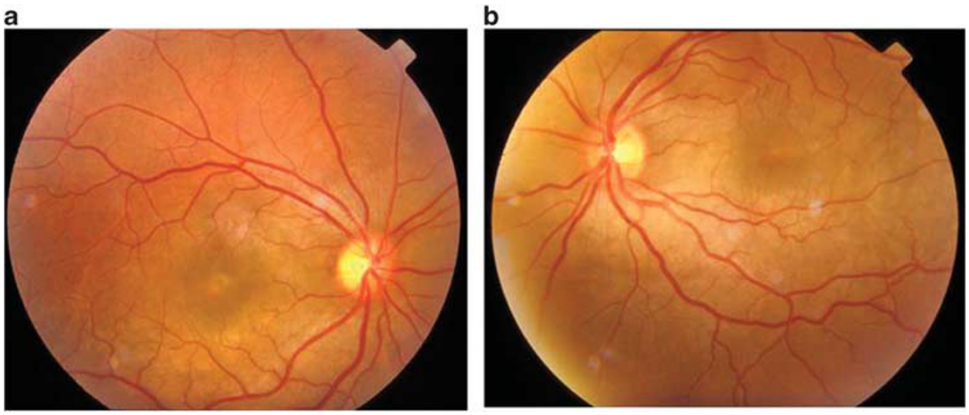

Figure 2 Fundus photograph (a, b) of both eyes showing complete resolution of the serous detachment with residual hypopigmented small spots in the RPE.

treated with Fludarabine, Cytosar, and Cytosine arabinoside as salvage therapy, and with Piperacillin, Amphotericin B, Amikacin, and Hydrocortisone for suspected septicemia. While waiting for the BMT, she complained of bilateral blurred vision.

On presentation BCVA was 20/50 in both eyes. Full ophthalmologic examination revealed clear and quite anterior segments, and on fundus examination SRD, involving the posterior pole OU, accompanied by a small retinal hemorrhage in the inferior arcade OD (Figures 1a and b).

Fluorescein angiography showed multifocal, pinpoint hyperflourescent spots beneath the detachment with diffuse late subretinal leakage (Figures 1c and d).

Optical coherence tomography disclosed large amount of subretinal fluid OU (Figures 1e and f).

As the patient was treated with hydrocortisone at the time, a possible diagnosis of central serous retinopathy (CSR) was suspected, and discontinuation of hydrocortisone treatment was advised. However, no resolution of the SRD was noticed. We then suspected that the SRD may be a sign of disease relapse and insisted on repeated systemic evaluation, which indeed revealed disease relapse. Another chemotherapy protocol (FLA-IDA) was initiated. Seven days later, BCVA improved to 20/20 OU and complete resolution of the SRD OU was seen (Figures 2a and b).

Later on, the patient underwent the planned BMT, but unfortunately suffered from severe fungal septicemia and passed away 1 month later.

Comment

Retinal manifestations are estimated to occur in up to 90% of persons with acute leukemia,[1,2] and include large spectrum of small vessel disease, ranging from mild ischemia with scattered cotton wool spots, and few retinal hemorrhages (round or flame shaped, often with a white component, which consists of leukemic cells and debris, platelet fibrin aggregates, or septic emboli) to proliferative retinopathy.[1–3]

Histopathological examination shows discrete and diffuse hemorrhages and leukemic infiltration.[3] The hemorrhages and infiltrates are found at all levels of the retina, but especially in the inner layers with focal destruction.

These retinal findings are observed commonly, but SRD in leukemia is rare[4–10] and seldom seen during complete remission.[10,11] The posterior manifestations of leukemia are probably due to leukemic infiltration of the choroid,[1] or from hematological abnormalities associated with leukemia, such as anemia, thrombocytopenia, hyperviscosity states, or opportunistic infections.[1,2]

The SRD observed in leukemia is reported to be shallow in the posterior pole[1,7,11] and could be attributed to direct invasion of lymphoblasts to the choroidal vasculature causing dysfunction of the choroid—retinal pigment epithelial (RPE) complex, resulting in ischemia of the RPE and disruption of their pumping ability and accumulation of fluid beneath the retina.

The involvement of the choroid by leukemic cells tends to be perivascular, and may be patchy or diffuse.[3] The choroid at the posterior pole may be significantly thickened. The overlying RPE may show secondary alterations, including atrophy and hypertrophy.

Fluorescein angiography demonstrates multifocal deep hyperfluorescent foci beneath the detachment in the early phase and diffuse leakage to the subretinal space in the late phase,[1,2] as seen in our case.

This angiographic finding is probably due to RPE disturbances secondary to circulatory or metabolic changes in the underlying choriocapillaris. Stewart *et al*[4] postulated that leukemic infiltration of the choroid caused decreased blood flow in the choriocapillaris, resulting in ischemia to the overlying RPE and disruption of the intercellular tight junctions.

The intraocular manifestations of leukemia are usually treated with systemic chemotherapy, and when this fails, ocular radiation is recommended.[1,2]

In our case, shortly after systemic chemotherapy was administered, the SRD resolved and BCVA improved.

This case suggested that SRD caused by leukemia may mimic CSR. However, the appearance of SRD in a patient with ALL, although on apparent remission, should alert the ophthalmologist for a relapse of the disease.

Conflict of interest

The authors declare no conflict of interest.

References

1 Kincaid MC, Green WR. Ocular and orbital involvement in leukemia. *Surv Ophthalmol* 1983; **27**: 211–232.
2 Schachat AP, Dhaliwal RS. Leukemias and lymphomas. In: Ryan SJ (ed). *Retina*, 2nd ed. CV Mosby: St Louis, 1994, pp 873–890.
3 Allen RA, Straatsma BR. Ocular involvement in leukemia and allied disorders. *Arch Ophthalmol* 1961; **66**: 490–508.
4 Tang RA, Vila-Coro AA, Wall S, Frankel LS. Case report. Acute leukemia presenting as a retinal pigment epithelium detachment. *Arch Ophthalmol* 1988; **106**: 21–22.
5 Stewart MW, Gitter KA, Cohen G. Acute leukemia presenting as a unilateral exudative retinal detachment. *Retina* 1989; **9**: 110–114.
6 Vangheluwe O, Ducasse A, Culioli B, Kwong FH, Segal A. Macular manifestations of acute lymphblastic leukemia. A case report. *Bull Suc Ophthalmol Fr* 1990; **90**: 677–682.
7 Fackler TK, Bearelly S, Odom T, Fekrat S, Conney MJ. Acute lymphoblastic leukemia presenting as bilateral serous macular detachments. *Retina* 2006; **26**(6): 710–712.
8 Malik R, Shah A, Greaney MJ, Dick AD. Bilateral serous macular detachment as a presenting feature of acute lymphoblastic leukemia. *Eur J Ophthalmol* 2005; **15**(2): 284–286.
9 Abdallah E, Hajji Z, Mellal Z, Belmekki M, Bencherifa F, Berraho A. Macular serous detachment revealing acute lymphoblastic leukemia. *J Fr Ophtalmol* 2005; **28**(1): 39–44.
10 Wu L, Calderón M, Hernández G, Marbis J, Ramírez V. Bilateral exudative retinal detachment as the first sign of relapsing acute myelogenous leukaemia. *Clin Experiment Ophthalmol* 2006; **34**(6): 623–625.
11 Miyamoto K, Kashii S, Honda Y. Serous retinal detachment caused by leukemic choroidal infiltration during complete remission. *Br J Ophthalmol* 2000; **84**(11): 1318–1319.

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Sir,

Pseudoexfoliative deposits on an intraocular lens implant

Pseudoexfoliation sydrome is the most common identifiable form of secondary open-angle glaucoma worldwide. It is characterized by white powdery deposits at the pupillary margin of the iris and throughout the inner surface of the anterior chamber.[1] We present a case of an 86-year-old gentleman, who presented with pseudoexfoliative material deposited on an intraocular lens implant, a phenomenon widely accepted as rare.[2]

Case report

An 86-year-old gentleman presented for review of a right-sided cataract. He had a left eye phacoemulsification and insertion of posterior intraocular, Akreous Adapt Bausch and Lomb lens (Bausch and Lomb, Kingston upon Thames, UK), in 2004. His post-operative course had been complicated by persistent iritis and cystoid macular oedema, which settled leaving him with a best-corrected visual acuity of 6/9 in that eye, at which point he was discharged. On re-referral for cataract in the right eye, a new finding of bilateral pseudoexfoliation was noted, with pseudoexfoliative plaques growing over the left intraocular implant (Figure 1). His best-corrected visual acuity was 6/12 in the left eye, with his intraocular pressure measuring 24 mm Hg in the left eye and 38 mm Hg in the right. The use of Bimatoprost (Allergan, Marlow, UK) daily in both eyes adequately controlled the intraocular pressures, which were measured at 20 and 16 mm Hg in the right and left eyes respectively, approximately seven weeks after commencing treatment.

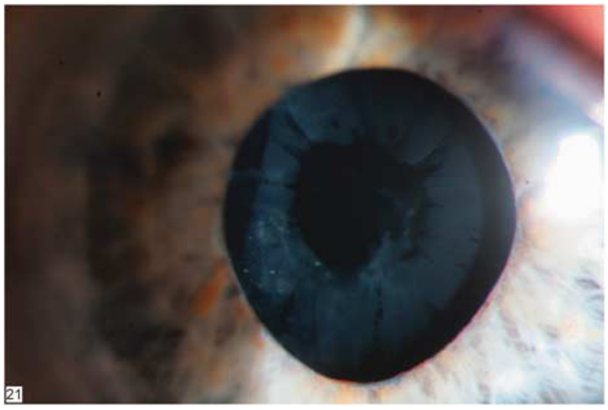

Figure 1 Colour photograph showing pseudoexfoliative deposits on intraocular lens implant.

Comment

Pseudoexfoliative deposits are known to be found on natural lenses; however, here we present a case of pseudoexfoliation deposits on an intraocular lens implant. It has been suggested that such cases are only infrequently noted as the distance between the posterior iris epithelium, and an intraocular lens is too large to allow deposition of pseudoexfoliation material resulting in these cells passing into the posterior chamber.[3] This case is of interest to raise awareness of vigilant examination for pseudoexfoliation, even in pseudophakic patients, where plaques on the lens implant may be the only sign for diagnosis of secondary glaucoma.

Conflict of interest

The authors declare no conflict of interest.

References

1 Conway RM, Schötzer-Schrehardt U, Küchle M, Nauman GO. Pseudoexfoliation syndrome: pathological manifestations relevant to intraocular surgery. *Clin Experiment Ophthalmol* 2004; **32**(2): 199–210.