migration of gas bubbles among the subarachnoid space, the subretinal space, and the vitreous cavity.[5] To our knowledge, this is the first *in vivo* report of a cystic lesion on the bottom of an optic disc pit. This finding could suggest a communication between the subarachnoid and subretinal space. In conclusion, it is conceivable that the peculiar pillars outside the optic pit area could be dysplastic retinal tissue mechanically stretched by subretinal fluid. This mechanism could explain seepage of subretinal fluid.

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Sir,

Persistent retinal macrocyst following pars plana vitrectomy for rhegmatogenous retinal detachment

A 45-year-old Caucasian male presented with sudden painless loss of vision in his left eye 2 years after undergoing uneventful cataract surgery. Original refraction was −6.50 D in both eyes. On examination, visual acuity was 0.8 RE and HM LE. There was a dense vitreous haemorrhage. A pars plana vitrectomy was performed and revealed superonasal and inferotemporal rhegmatogenous retinal detachments (RRD). A break was identified in the superior but not in the inferior detachment. The retina flattened under perfluorooctane (PFO). Tamponnade was achieved with 20% SF_6 after PFO-air exchange. Postoperative acuity LE was 0.8 at 1 month and the retina remained attached. A large postoral inferotemporal retinal cyst was noted. The wall was uncorrugated and vascularized (Figure 1). A small subretinal PFO residue was noted posterior to the cyst. B-scan ultrasonography confirmed the hypoechogeneity of the lesion (Figure 2). No prophylactic retinopexy was applied and the lesion remained stable over 24 months.

Figure 1 Large postoral cyst in the inferotemporal retina. Note smooth vascularized surface. Small subretinal perfluorooctane cyst posterior to the large lesion.

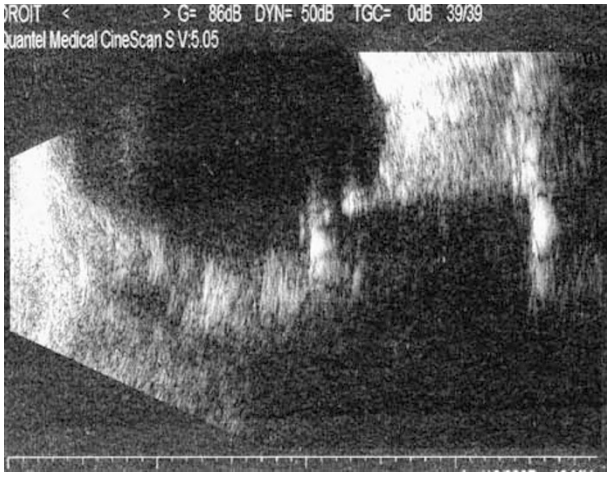

Figure 2 Standard B-scan echography showing the inferior macrocyst as an elevated, hypoechogenic lesion. Note long orbital shadow.

The differential diagnosis of a large postoral cyst includes pars plana cysts (PPCs) and retinal cysts. PPCs are physiological and can become quite large.[1] Less common causes include haemorrhagic retinal macrocysts,[2] cysts of chronic RRD,[3] subretinal hydatid cysts,[4] and retained subretinal perfluorocarbon.[5] Known risk factors for perfluorocarbon retention include peroperative retinotomy or retinectomy. In this case no break was identified in the inferotemporal detachment, but PFO could have entered the subretinal space through the superior horsehoe tear and tracked inferiorly postorally.

The postoral cyst did not change in size, shape or pigmentation over 24-month follow-up. As it was not seen peroperatively, it is likely to represent a PFO

retention cyst. The echographic characteristics of retained PFO have been described by Hasenfratz *et al*.[5] Delayed display of the echo signal results in a hypoechogenic image due to slower sound conduction in PFO. The lesion presented here is unusually large. On the basis of clinical history and echographic findings, we believe this cyst is likely to contain PFO. It has had no functional effect on vision and no attempt should be made to remove it.

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Sir,

Uveitis associated with concurrent administration of rifabutin and lopinavir/ritonavir (Kaletra)

Case report

A 41-year-old HIV-infected man received lamivudine/zidovudine and nevirapine therapy. He also took isoniazid/rifampicin/pyrazinamide and ethambutol for pulmonary tuberculosis. Because skin rash appeared, nevirapine was replaced by lopinavir/ritonavir 400/100 mg twice daily. The antituberculosis regimens were replaced by rifabutin 300 mg/day and methaniazide 600 mg/day because of known interactions between lopinavir/ritonavir and rifampicin.[1]

Panuveitis was found in his left eye after receiving rifabutin for 86 days (Figure 1). The uveitis resolved following the discontinuation of rifabutin with the administration of topical steroids and cycloplegics. The final visual acuity recovered from hand motion to 1.0.

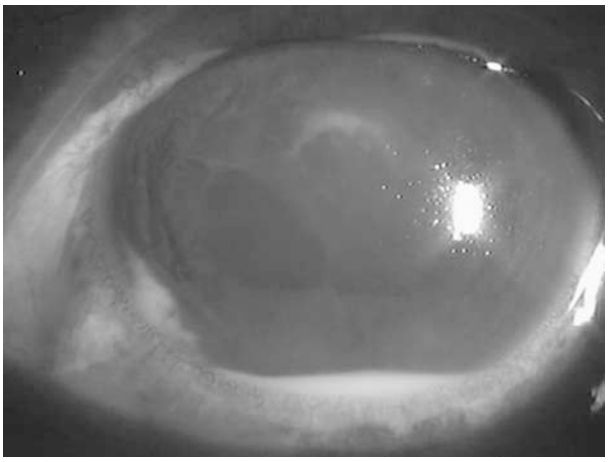

Figure 1 A slit-lamp photograph shows severe cell reaction, fibrin, and a hypopyon in the anterior chamber.

Comment

Conditions associated with uveitis in HIV-positive patients include opportunistic infection, neoplasms, inflammation due to HIV infection itself, and drug toxicities.[2] The patient was negative for HLA-B27 and syphilis. Uveitis did not recur after discontinuing rifabutin for one year. Refabutin was suspected as the cause of uveitis by evidences of timing condition, lack of other causes, and resolution after stopping the implicated drug.

Rifabutin associated uveitis has been described in AIDS patients and identified as a dosage-dependent side effect.[3] Adverse effects are unusual at the recommended dose of 300 mg/day.[4] Clarithromycon or fluconazole was known to increase concentration of rifabutin and the incidence of rifabutin toxicities, including uveitis.[5] Neither clarithromycon nor fluconazole was given to our case.

Ritonavir is a potent inhibitor of CYP3A4 and has been shown to substantially increase rifabutin concentration. Co-administration of rifabutin with ritonavir increased area under the concentration–time curve (AUC) of rifabutin and its 25-*O*-desacetyl metabolite by four times and 35 times, compared with administration of rifabutin alone.[6] Patients receiving rifabutin and ritonavir without the reduction of dosages increased the risk of developing leucopenia, arthralgia, joint disorder, uveitis, and skin discoloration.[6,7] Because of the increased likelihood of rifabutin toxicities, the dosage of rifabutin should be reduced by at least 75% of usual dosage (300 mg once daily) or 150 mg 2–3 times a week when given with lopinavir/ritonavir.[8,9]

To our knowledge, this is the first report of uveitis associated with concurrent administration of rifabutin and lopinavir/ritonavir. Our finding suggests that the dosage of rifabutin should be reduced when it is administered with lopinavir/ritonavir.

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