culture of both aqueous and vitreous aspirates revealed *A. faecalis*, sensitive to chloramphenicol, tetracycline, cefotaxime, amikacin, and ciprofloxacin. Cefotaxime eye drops (5% concentrated) and 2% fortified amikacin eye drops in addition to 1% prednisolone acetate eyedrops were started. The patient improved symptomatically as well as clinically (Figure 1b). The second set of cultures taken from the vitreous was found to be negative. With negative cultures and reduction in vitreous exudates by ultrasonography, the patient was started on oral prednisolone 60 mg daily. At the end of 2 months the eye was quiet (Figure 1c). Posterior capsule opacification was noted. Visual acuity did not improve beyond hand movements. Fundus examination after Nd:YAG capsulotomy (Figure 1d) revealed consecutive optic atrophy and macular infarction.

Organisms of the *Alcaligenes* genus are a group of nonfermenting Gram-negative bacilli found in soil and water. Most isolates of *A. faecalis* from blood or respiratory secretions are related to the contamination of hospital equipment or fluids with the organism, with resulting human colonization or infection.[2] It has also been recovered from corneal ulcers, ear discharges, wound drainage, and faeces.[3,4] Identification of *Alcaligenes* species is made by oxidase-positive, indole-negative, and urease-negative organisms with flat, spreading edges on blood-agar plates.[5] *A. faecalis* has been associated with infections in immunocompromised patients, but our patient's medical history was unremarkable. The pathogenic role and virulence of this organism is not clear. The virulence has been attributed to various factors including histamine sensitizing factor, adherence and cytotoxicity, and exocellular 'o' antigen. Although most of the studies conducted were based on susceptibility of avian and mammalian cells to the above factors, the same can hold true for humans.[6]

The patient developed endophthalmitis on the second day after surgery. *A. faecalis* endophthalmitis developing on the fourth day following penetrating keratoplasty was reported.[7] An epidemiological search for the organism revealed the conjunctival sac as the source of infection. The organism must have gained entry into the eye as surface fluid refluxes through the wound during surgery. Another explanation could be the intraocular lens getting contaminated as it touches the ocular surface. There was good anatomic outcome and good response to intravitreal and intravenous antibiotics. Perhaps a better visual outcome could have been possible if the macula and optic nerve events did not take place. However, no conclusion can be drawn regarding its virulence based on this case report.

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Sir,

**Choroidal neovascularization in retinochoroidal coloboma: thermal laser treatment achieves long-term stabilization of visual acuity**

Retinochoroidal coloboma is a congenital abnormality caused by faulty closure of the embryonic fissure. Choroidal neovascularization (CNV) secondary to retinochoroidal coloboma is an uncommon complication.

The treatment option in vision-threatening cases may be laser photocoagulation.[1–4] However, no reports about long term outcome of this therapy has been reported so far. This may be of interest in the era of new therapeutic

options such as photodynamic therapy or subretinal surgery. Herein we present a case of CNV associated with retinochoroidal coloboma with a follow-up of 12 years after thermal laser treatment.

### *Case report*

A 28-year-old woman presented complaining of decreased vision and metamorphopsia in the left eye in October 1993. The left eye reportedly had had poor central vision since childhood. The best-corrected visual acuity was 20/20 OD and 20/400 OS. Refractive errors were $-0.75\ -1.0 \times 160^{\circ}$ OD, and $-0.25\ -2.0 \times 175^{\circ}$ OS. Slit-lamp biomicroscopy showed bilateral iris coloboma. Funduscopic examination of the right eye revealed an inferior retinochoroidal coloboma that did not involve macula or disc. Indirect ophthalmoscopy of the left eye revealed a large inferior retinochoroidal coloboma involving macula and optic disc. Subretinal and intraretinal haemorrhage, and a subretinal, greyish, partly pigmented lesion at the border between coloboma and normal retina (Figure 1a) was noted. Fluorescein angiography of the left eye showed subretinal leakage at the foveal edge of the retinochoroidal coloboma, consistent with an extrafoveal CNV (Figure 1b and c).

After obtaining written informed consent, the CNV was treated with argon green laser photocoagulation. The visual acuity of the left eye improved to 20/160. In November 1994, the patient again complained of blurred vision, which started a few days ago. A fluorescein angiogram performed at this time showed recurrent extrafoveal CNV. The patient subsequently underwent a

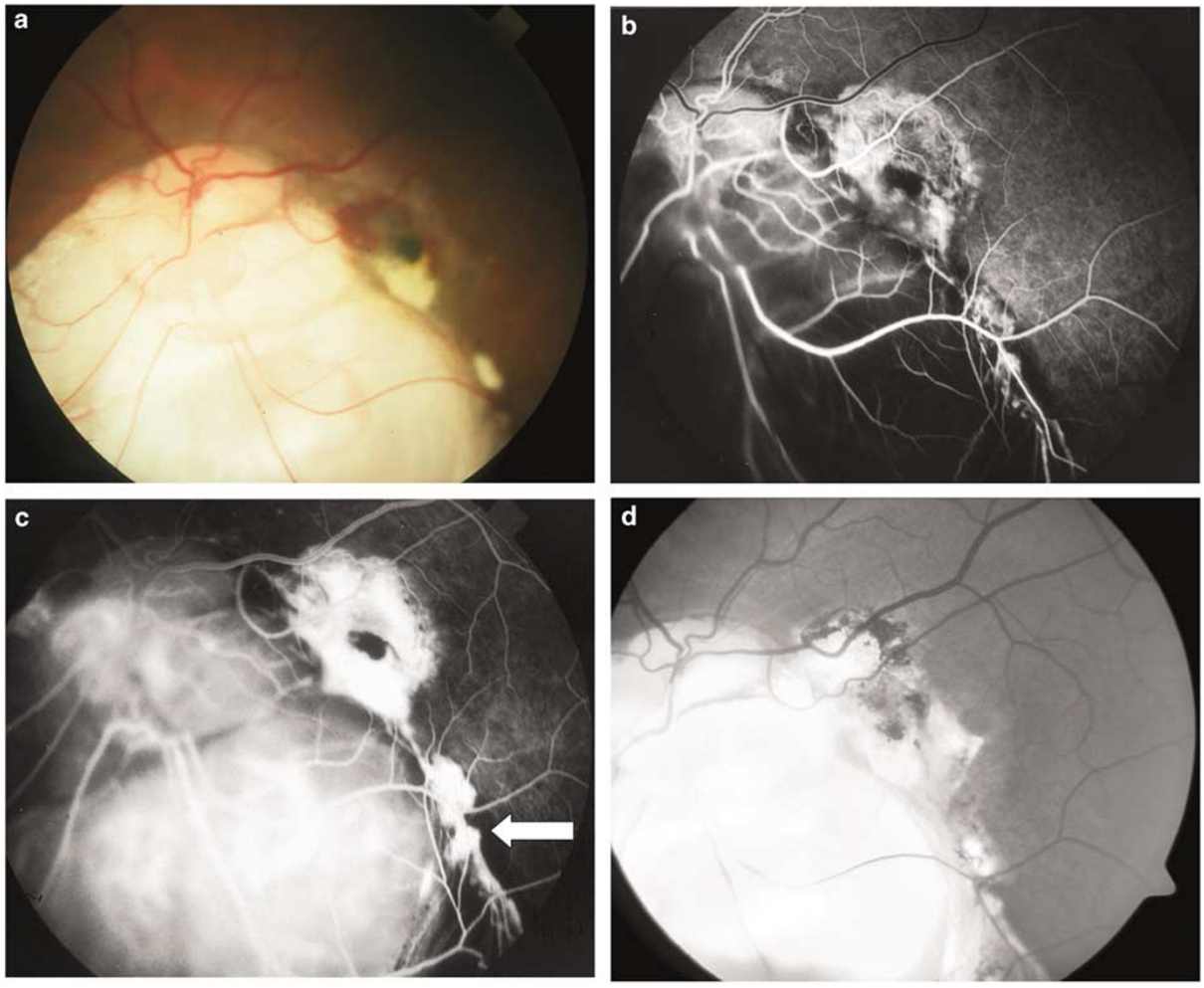


**Figure 1** (a) Fundus photograph of the left eye shows subretinal and intraretinal haemorrhage and subretinal fluid at the border of the coloboma at the time of first presentation (November 1993). (b) Fluorescein angiogram (early phase) of the left eye demonstrates hyperfluorescence at the margin of the coloboma (November 1993). (c) Fluorescein angiogram (late phase) of the left eye showing increased hyperfluorescence consistent with choroidal neovascularisation (November 1993). The inferior part of the CNV was not considered for the laser treatment (arrow). (d) Fundus photograph of the left eye 12 years after the last photocoagulation shows a dry macula and subretinal fibrosis.

**Table 1** Patients with retinochoroidal coloboma associated with choroidal neovascularization—review of the literature

| *Author* | *Age at onset of symptoms (years)* | *Sex* | *Treatment* | *Follow-up/outcome* |
|---|---|---|---|---|
| Brodsky *et al*[1] | 1 | F | LC | 3 months/stable VA |
| Gupta *et al*[5] | 20 | M | None | Not reported |
| Leff *et al*[6] | 65 | F | None | Not reported |
| Leff *et al*[6] | 70 | F | None | Not reported |
| Maberly *et al*[2] | 57 | M | LC | Not specified/stable VA |
| Rouland *et al*[7] | 26 | F | None | 10 months/stable VA |
| Shaika *et al*[3] | 1 | F | LC | 10 months/VA not reported |
| Steahly[4] | 29 | M | LC | 33 months/improved VA |
| Spitzer *et al* (this study) | 28 | F | LC | 12 years/improved VA |

LC: laser photocoagulation, M: male, F: female, VA: visual acuity.

second laser photocoagulation. Follow-up angiographies obtained during the following years showed subretinal staining and a dry macula. At the last follow-up visit, 12 years after the initial laser treatment, atrophy and fibrosis were noted at the border of the coloboma (Figure 1d). The macula was dry. The visual acuity of the left eye remained stable at 20/160.

## Comment

Retinochoroidal coloboma is a developmental abnormality of the eye. At the border of a coloboma there is a disruption of the normal anatomy. The marginal retina splits into the intercalary membrane, which bridges the base of the coloboma, and a retinal duplication, which represents the eversion of the optic cup that originally caused the coloboma.[8] Complications of this malformation are retinal detachment, cataract, microphthalmia with cyst, and rarely CNV. To the author's knowledge, the case presented is the ninth case of CNV associated with retinochoroidal coloboma in the literature.[1–7] In all of these cases, the CNV developed at the margin between the coloboma and the normal retina. Discontinuities of Bruch's membrane are present at the border of a coloboma where Bruch's membrane abruptly terminates and the retinal pigment epithelium is laterally displaced. Subpigmentepithelial and choroidal vessels may enter the subretinal space at this junction (the locus minoris resistentiae).

Two of the previously reported patients were 65 and 70-years-old. Age related degenerative changes may have contributed to the development of CNV in these cases. One of these patients reportedly had scattered macular drusen in both the eyes.[6]

Five of the nine cases reported in the literature received thermal laser treatment. In two cases no treatment was considered because the membrane did not threaten visual acuity,[6] one patient refused to undergo photocoagulation,[7] and in another case the lesion was located subfoveally and observational management was chosen.[5] Four previously reported patients who underwent photocoagulation retained stable or improved visual acuity (follow-up 0–33 months) (Table 1).

CNV secondary to retinochoroidal coloboma can be a vision threatening disorder. The visual acuity of the presented patient remained stable for 12 years after laser treatment. Patients with retinochoroidal coloboma may be at risk for developing CNV throughout life. Thus, regular monitoring of such patients is advisable as early detection and laser photocoagulation may permanently prevent visual loss.

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Sir,

**Bilateral lacrimal gland enlargement due to post-transplant lymphoproliferative disorder**

Over the years, post-transplant lymphoproliferative disorder (PTLD) has become increasingly reported as organ transplantation becomes more common. To our knowledge this is the first report of bilateral lacrimal gland enlargement secondary to PTLD.

### *Case report*

A 49-year-old male, on oral cyclosporine A following renal transplantation performed 10 years previously for polyarteritis nodosa associated renal vasculitis, presented with a 2-week history of bilateral upper lid swelling secondary to bilateral nontender lacrimal gland enlargement. Examination was otherwise unremarkable. Significantly, he had been diagnosed with biopsy-proven cervical lymph node PTLD 6 weeks earlier.

Bilateral lacrimal gland incisional biopsy was performed. Histology of both lacrimal glands revealed atrophic acini and diffuse infiltration by a polymorphous population of small lymphocytes, plasma cells, and larger centroblast-like cells (Figure 1). Immunohistochemistry demonstrated that the majority of the cells, including the larger blasts, were B-cells, many aberrantly positive for the Epstein–Barr virus (EBV) upregulated T-cell antigen, CD43 (Figure 2). In situ hybridisation with the EBER mRNA probe (Figure 3) confirmed EBV infection and confirmed the diagnosis of EBV-positive polymorphic B-cell PTLD. Identical histology, immunohistochemistry, and in situ hybridisation appearances were seen in the cervical lymph node excised 6 weeks previously.

Under oncology supervision, the cyclosporin A therapy was withdrawn. However, due to the minimal response, treatment with monoclonal anti-CD20 (Rituximab, Roche) was started. Dramatic improvement ensued with shrinkage of both lacrimal glands and cervical lymphadenopathy. Unfortunately, the transplanted kidney underwent rejection, necessitating its removal. The patient remains well on dialysis with no recurrence of PTLD.

### Discussion

PTLD, first described over 30 years ago,[1] represents a spectrum of lymphoproliferative disease, ranging from early polyclonal proliferations, often presenting with an infectious mononucleosis-like syndrome, to frank lymphoma, usually of the B-cell type.[2]

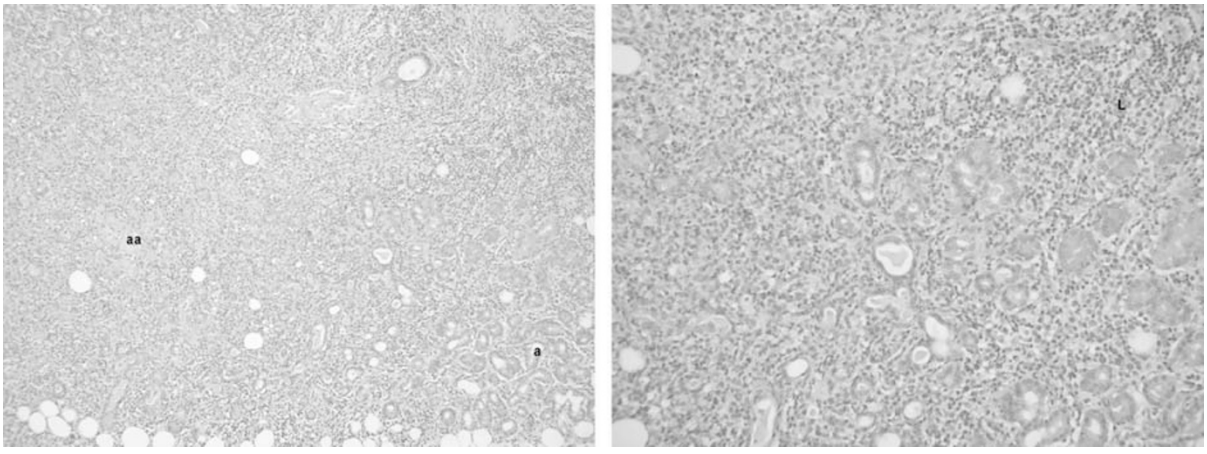


**Figure 1** (Left) Histology of lacrimal gland incisional biopsy showing marked acinar atrophy associated with predominantly diffuse infiltrates of mature lymphocytes (aa—acinar atrophy, a—lacrimal gland acinus; H&E ×10). (Right) Higher power view showing diffuse infiltration of lymphocytes (L) among lacrimal gland acini (H&E ×20).