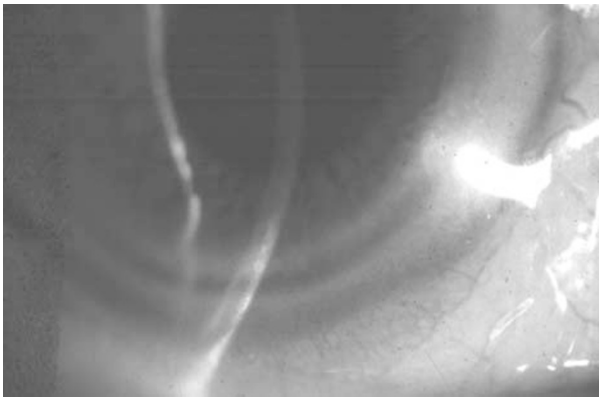

Figure 2 (a) higher magnification ($\times 25$) showing a more detailed contour of the double arcus cornealis.

interval of Vogt.[1] It develops as a result of lipid deposition in the deep corneal stroma and the limbal sclera, and its prevalence increases with age. In children, it may be a manifestation of hyperlipidemia, but in elderly people it may be an isolated finding. Arcus cornealis should be differentiated from other lipid metabolisms affecting the cornea, such as LCAT deficiency, 'fish eye' disease, or Tangier disease where lipid deposition extends into the centre of the cornea.

Double corneal arcus were recently described in two patients from India.[2] However, this report had no follow-up on the patients, lipid profile was not reported, and no theory regarding the pathophysiology of the findings was suggested.

In our case, the innermost ring was narrower and less distinct than the peripheral one. It was more distinct in the upper and lower quadrants of the cornea, as usually seen in typical single arcus cornealis. The patient had hypercholesterolaemia.

The development of a second ring of lipids may suggest that lipids diffused from perilimbal vessels may migrate through the stroma according to properties such as their size and polarity. Similar phenomena can be seen when diffusion zones are created in separation of lipids[3] or antigens and antibodies seen as Mancini radial rings[4] and Ouchterlony immudifussion rings.[5] In these laboratory techniques, the agents are separated based on their structural properties and create sediments where they are bound to other molecules through a complementary binding site. Differences in the lipid compositions of the two rings in the arcus may support this theory, if a biopsy is obtained and analysis of lipids is performed. Another explanation is that the formation of the arcus is inherent and that they are formed *in situ* either as single or double ring. The classic theory concerning the appearance of arcus cornealis is that lipids migrate from the limbal vascular arcade to the peripheral cornea and the clear interval of Vogt is formed due to absorption of the lipids that are closer to the vascular arcade.[1] The appearance of two rings makes the second part of the classic theory less plausible.

References

1 Barchiesi BJ, Eckel RH, Ellis PP. The cornea and disorders of lipid metabolism. *Surv Ophthalmol* 1991; **36**: 1–22.
2 Agrawal S, Agrawal J, Agrawal TP. Double-ring corneal arcus. *J Cataract Refract Surg* 2002; **28**: 1885–1886.
3 Christie WW. Lipid Analysis: Isolation, Separation, Identification and Structural Analysis of Lipids. *Pergamon Press: Oxford* 1973, pp 43–72.
4 Mancini G, Carabonara AO, Heremans JF. Immunochemical quantitation of antigens by single radial immunodiffusion. *Immunochemistry* 1965; **2**: 235–254.
5 Moor GW, Goodman M. A set theoretical approach to immunotaxonomy: analysis of species comparisons in modified Ouchterlony plates. *Bull Math Byophys* 1968; **30**: 279–289.

S Rumelt and I Rumelt-Blitstein

Department of Ophthalmology, Western Galilee — Nahariya Medical Center, Nahariya and 'Leumit' Health Care, Jerusalem, Israel

Correspondence: S Rumelt, MD
Department of Ophthalmology, Western Galilee — Nahariya Medical Center
P.O. Box 21, 22100 Nahariya, Israel
Tel: +11 972 4 9107635
Fax: +11 972 4 9107611

Eye (2004) **18,** 1020–1021. doi:10.1038/sj.eye.6701393
Published online 2 April 2004

Sir,

Late presentation of severe thyroid eye disease

The presentation of thyroid eye disease is usually closely temporally related to the diagnosis of thyrotoxicosis. A very small percentage of patients with thyroid eye disease develop sight-threatening disease due to compressive optic neuropathy. We report a case of severe thyroid eye disease with sight loss presenting 14 years after the diagnosis of thyrotoxicosis.

Case report

A 57-year-old Caucasian female, who smoked five cigarettes a day, presented with a severe reduction in right visual acuity associated with proptosis. Fourteen years previously, she had been treated with carbimazole

and radioactive iodine for thyrotoxicosis. Subsequent hypothyroidism was treated with thyroxine. She had controlled hypertension and seasonal episodes of bronchitis. There was a strong family history of thyroid problems with her mother and sister being on replacement thyroxine treatment, although the underlying thyroid condition was unknown. Her ophthalmological symptoms had started approximately 9 months previously, shortly after the death of her father, initially with right lower lid swelling followed 3 months later by horizontal diplopia and conjunctival injection. She then developed blurred right vision and she was referred to the local Eye Department where it was felt unlikely that she had thyroid eye disease in view of the long interval between her thyrotoxicosis and the onset of her eye problems. Three months later, she developed right proptosis with reduced vision and following a CT scan was started on 100 μg prednisolone reducing to 10 μg over 3 weeks. The left vision deteriorated, and the steroid dosage was increased to a maintenance dose of 25 μg. She was referred to our service for further opinion, and presented with a corrected Snellen visual acuity of 2/60 in her right and 6/12 in her left eye. She was unable to identify any of the Ishihara test plates with her right eye and scored only 10 out of 17 with the left eye. There was right proptosis with upper and lower lid retraction and restricted extraocular movements worse on the right side (Figure 1). The anterior segments were normal. Intraocular pressures increased significantly on upgaze. She had a right afferent pupillary defect, nasal swelling of the right optic disc, absent spontaneous venous pulsation of both optic discs, and extensive arteriovenous nipping.

Electrodiagnostic tests were reported as consistent with a diagnosis of a right optic neuropathy. The MRI scan showed compression of the right optic nerve at the orbital apex, with bilateral proptosis more marked on the right side due to asymmetrical enlargement of the extraocular muscles (Figure 2). Blood results showed a neutrophilia, normal haemoglobin and platelet concentrations with normal blood chemistry. Thyroid function tests had recently shown an elevated free thyroxine concentration with an associated low TSH level and her thyroxine had been reduced to 75 μg.

The patient was started on a 2-day course of intravenous methylprednisolone and a three-wall orbital decompression was advised.

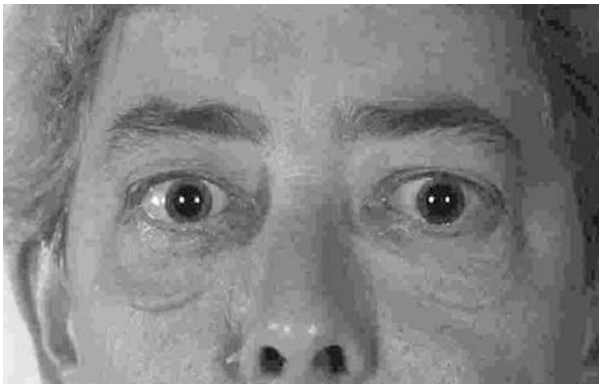

Figure 1 Ocular appearance of the patient in the primary position.

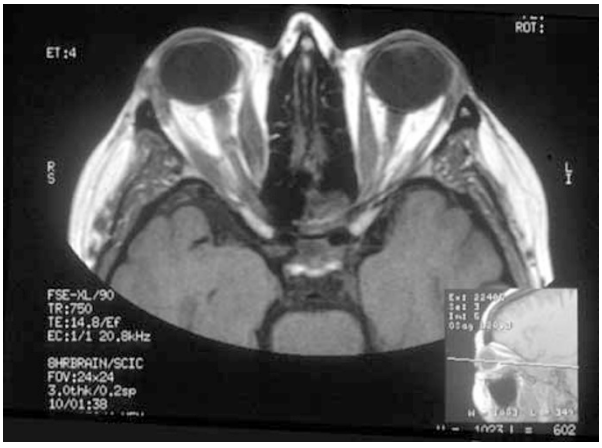

Figure 2 MRI scan showing compression of the right optic nerve at the orbital apex.

Comment

Although the majority of patients with thyroid eye disease usually present very soon after the diagnosis of thyrotoxicosis, it is important to remember that, rarely, late presentation can occur. Anecdotally, it has been suggested that these patients can often identify a stressful and identifiable precipitating factor. Such an event was noted in our patient. Only 5% of patients have the complete spectrum of clinical features with eyelid retraction, exophthalmos, extraocular muscle involvement, optic nerve dysfunction and hyperthyroidism.[1] The close temporal relationship of the diagnosis of Graves' disease and thyroid eye disease is well known and it has been found in 81% of patients to develop in the 18 months prior to and after the diagnosis of thyroid dysfunction.[2] It has been reported that 69.5% of patients requiring decompression surgery develop thyroid eye disease in the year before or after diagnosis.[3] Ophthalmopathy is rarely diagnosed more than 6 months before the diagnosis of thyrotoxicosis.[4] In one report no patient developed eye problems more than 4 years after the diagnosis of hyperthyroidism.[4] It has been suggested that thyroid eye disease develops at a time when thyroid autoimmunity also exists, thus strongly suggesting a common factor in the pathogenesis of thyroidal and ocular expressions of Graves' disease.[5]

This patient is a rare case of severe sight-threatening thyroid eye disease presenting 14 years after diagnosis of thyrotoxicosis. While the vast majority of patients who develop thyroid eye disease will do so in an 18-month period before or after the time of systemic diagnosis, it is important to be aware that sight-threatening thyroid eye disease may present many years after the initial diagnosis and treatment of thyrotoxicosis. Prompt diagnosis and treatment of this condition is likely to preserve sight.

References

1 Bartley GB, Fatourechi B, Kadrmas EF, Jacobsen SJ, Ilstrup DM, Garrity JA *et al*. Clinical features of Graves' ophthalmopathy in an incidence cohort. *Am J Ophthalmopathy* 1996; **121**(3): 284–290.
2 Gorman CA. Temporal relationship between onset of Graves' ophthalmopathy and diagnosis of thyrotoxicosis. *Mayo Clin Proc* 1983; **58**: 515–519.
3 Garrity JA, Fatourechi V, Bergstralh EJ, Bartley GB, Beatty CW, DeSanto LW *et al*. Results of transantral orbital decompression in 428 patients with severe Graves' ophthalmopathy. *Am J Ophthalmol* 1993; **116**: 533–547.
4 Bartley GB, Fatourechi V, Kadrmas EF, Jacobsen SJ, Ilstrup DM, Garrity JA *et al*. The chronology of Graves' ophthalmopathy in an incidence cohort. *Am J Ophthalmol* 1996; **121**: 426–434.
5 Wiersinga WM, Smit T, van der Gaag R, Koornneef L. Temporal relationship between onset of Graves' ophthalmopathy and onset of thyroidal Graves' disease. *J Endocrinol Invest* 1988; **11**(8): 615–619.

B Manzouri, A Bates, GE Rose and GGW Adams

Moorfields Eye Hospital, City Road
London EC1V 2PD, UK

Correspondence: B Manzouri
Tel: +2075662013
Fax: +2075662972
E-mail: b.manzouri@ntlworld.com

Eye (2004) **18,** 1021–1023. doi:10.1038/sj.eye.6701396
Published online 16 April 2004

Sir,

Leuconostoc mesenteroids as a cause of post-operative endophthalmitis—a case report

Postoperative endophthalmitis is a dreadful complication after uncomplicated cataract surgery. Acute postoperative endophthalmitis has been reported with various organisms; particularly Gram-positive cocci are known to cause endophthalmitis in the first 48 h after surgery.

Leuconostoc mesenteroides has been reported as a cause of nosocomial urinary tract infection, infection in patients with neutropenia and cancer, prosthetic valve endocarditis, liver transplantation, severe burn injuries, and AIDS.[1–9] Intrinsically, vancomycin-resistant Gram positives are usually opportunistic pathogens. To our knowledge, this is the first case report of *L. mesenteroides* endophthalmitis after phacoemulsification.

Case report

A healthy 76-year-old gentleman has a routine uncomplicated phacoemulsification with posterior chamber implantation. After 48 h, he presented to the casualty department with very poor vision and pain in the operated eye even though he could see well after 24 h after surgery. He had noticed some floaters prior to complete loss of vision. He did not have any ocular or systemic disease, that could predispose for a postoperative infection. He presented with only 'perception of light', hypopyon, slightly elevated (23 mmHg) intraocular pressure and fundus could not be seen due to vitreous opacification. A diagnosis of endophthalmitis was made and vitreous biopsy was carried out urgently, which included intravitreal injection of vancomycin and amikacin at the end of the procedure. Initial microbiologic examination showed Gram-positive cocci and he was treated with systemic ciprofloxacin and prednisolone. He also received topical gentamicin, ciprofloxacin, and cycloplegics. After 48 h, culture showed *L. Mesenteroides* that was sensitive to gentamicin, erythromycin, and chloromphenicol, but resistant to vancomycin, penicillin, ceftazidime, and amikacin. His symptoms improved in 3 days, but vision remains 'hand movements' after 3 months.

This report highlights the importance of *Leuconostoc* spp as an emerging pathogen, even though the modes of transmission and reservoirs are yet unknown.

Comment

L. Mesenteroides are Gram-positive cocci in pairs or short chains. They are microaerophilic; catalase-negative, ferment sugars, and produce gas. Species of this genus are normally present in plant materials, dairy products, and other fermented food stuffs as well as spoiled foods so that they are likely to be encountered in public health laboratories; such isolates are usually vancomycin-resistant. They have been isolated from the blood cultures and, as with pediococci, some were resistant to vancomycin.[4] In the laboratory, detran-forming cultures may be confused initially with *Streptococcus bovis* biotype, because both yield watery colonies on sucrose agar and share other biochemical characteristics. The *Leuconostoc*