Takayasu, an ophthalmologist, was the first to describe the disease in 1908.⁶

Takayasu's retinopathy is the most common ophthalmic manifestation. It reflects ocular hypoperfusion and is manifested by micro-aneurysms, arterio-venous anastomosis and neovascular complications. Hypertensive retinopathy is less frequently encountered. Retinal arterial occlusion was recently reported as well.⁷ Smith and Rosenbaum¹ suggested a real association between TA and scleritis because of the strong temporal relationship between the two conditions in their patient. We herein provide the first histopathological evidence of a necrotising inflammatory process in the sclera as part of a generalised necrotising granulomatous aortitis in TA.

Administration of corticosteroids is recognised as the first-line management of TA. Our patient received high-dose parenteral steroid therapy over 3 days. Thereafter, he manifested a remarkable improvement both clinically and echographically. He was then put on a tapering dose of prednisolone. We speculate that an occult systemic inflammation would also respond to this therapy, but unfortunately TA here proved fatal with ischaemic colitis being the direct cause of death. This again highlights an unusual feature of TA, as Mwipatayi *et al*⁸ reported that cardiac and renal failure accounted for 57% of deaths in 57 TA patients.

Disease patterns are influenced by many factors that are either inborn or acquired. Examples include genetic background, gender, ethnicity and environmental hazards. Moriwaki *et al*⁹ reviewed the clinical manifestations and angiographic findings of 102 Indian and 80 Japanese patients with TA. In Japanese patients, TA involved the ascending aorta, the aortic arch and/or its branches, whereas in Indian patients, vasculitis occurred in the abdominal aorta and involved renal arteries, subsequently extending into the thoracic aorta within one or two decades. Disease pattern is not well defined in Caucasians due to its rarity. Our patient showed evidence of granulomatous aortitis affecting both the thoracic and abdominal aorta, with the inflammatory changes in the thoracic aorta more granulomatous and older than those in the abdominal aorta. This pathology was clinically occult until the development of mesenteric arteritis, which proved refractory to treatment and resulted in fatal ischaemic colitis.

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References

- 1 Smith JR, Rosenbaum JT. Case reports and small case series: scleritis occurring in association with Takayasu disease. *Arch Ophthalmol* 2001;119(5):778-9.
- 2 Jain R, Ionides A, Pavesio C, *et al*. Scleritis as a presenting feature of Takayasu's disease [letter]. *Br J Ophthalmol* 2000;84:801.
- 3 Arya SK, Nahar R, Narang S, *et al*. Sclerokeratitis in Takayasu's arteritis: a case report. *Jpn J Ophthalmol* 2005;49(6):548-50.
- 4 Arend WP, Michel BA, Bloch DA, *et al*. The American College of Rheumatology 1990 criteria for the classification of Takayasu arteritis. *Arthritis Rheum* 1990;33(8):1129-34.
- 5 Pepys MB. The acute phase response and C-reactive protein. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford Textbook of Medicine*, 3rd edn, Vol 2. Oxford: Oxford University Press, 1996:1527-33.
- 6 Takayasu M. A case with peculiar changes of the central retinal vessels. *Acta Soc Ophthalmol Jpn* 1908;12:55.
- 7 Kiyosawa M, Baba T. Ophthalmological findings in patients with Takayasu disease. *Int J Cardiol* 1998;66(Suppl 1):S141-7.
- 8 Mwipatayi BP, Jeffery PC, Beningfield SJ, *et al*. Takayasu arteritis: clinical features and management: report of 272 cases. *ANZ J Surg* 2005;75(3):110-7.
- 9 Moriwaki R, Noda M, Yajima M, *et al*. Clinical manifestations of Takayasu arteritis in India and Japan--new classification of angiographic findings. *Angiology* 1997;48(5):369-79.

Cyclical orbital eosinophilic myositis

Eosinophilic myositis is a rare inflammatory condition of skeletal muscle associated with peripheral eosinophilia.¹ Orbital eosinophilic angiocentric fibrosis² and idiopathic orbital myositis³ have been reported. We report the first case of cyclical orbital eosinophilic myositis (OEM).

Case report

A 38-year-old woman presented to the ocular motility service in January 2006 with a one-week history of left-sided ptosis and diplopia. In the past four years, she had reported alternating ptosis and retro-ocular pain that recurred at six-week intervals. She was systemically well. Cranial computed tomography, magnetic resonance imaging, tensilon testing, neurophysiology and anti-acetylcholine receptor antibody had been normal.

Ocular examination revealed left partial ptosis, left hypotropia (20 prism dioptres (PD)) ipsilateral limitation of elevation, and ipsilateral temporal conjunctival hyperaemia (fig 1A, B). Visual acuity was normal bilaterally. Full blood count, inflammatory markers, serum angiotensin-converting enzyme, autoimmune screen, thyroid antibody, Lyme and worm serology were normal. Chest X-ray, thorax and abdominal computed tomography did not demonstrate regional lymphadenopathy or metastases. Magnetic resonance imaging scan showed increased signal in all rectus muscles (fig 1C). Presumed bilateral orbital myositis was managed with intravenous methylprednisolone 1 g a day for two days.

The hypotropia and ptosis improved rapidly (2-PD esophoria). She developed two recurrences over a 10-week period, one affecting the right side (fig 2A). Incisional biopsy of the left lateral rectus muscle demonstrated extensive disruption of the skeletal muscle by lymphoid aggregates with abundant eosinophils (fig 2B). This was reviewed by the national centre for tropical medicine and the regional reference laboratory. There were no histological features to suggest parasitic infection, and a diagnosis of OEM was made. Maintenance steroid therapy was started; however, the condition has remained unresponsive (fig 2C). Topical

Figure 1 (A) A 38-year-old woman with a left partial ptosis (left upper lid is kept open during testing) and limitation of elevation. There is a 20-PD left hypotropia. (B) Severe engorgement of the conjunctival and episcleral vessels overlying the left lateral rectus muscle. (C) Magnetic resonance imaging scan performed on presentation in 2006. Coronal short τ inversion recovery sequence of the orbits. Increased signal is present within all recti muscles bilaterally, particularly the left lateral rectus and superior rectus muscles bilaterally. Informed consent was obtained for publication of this figure.

Figure 2 (A) Second episode two months after presentation. An almost complete right-sided ptosis, a 35-PD right hypotropia, and limitation of right elevation. (B) Biopsy specimen of left lateral rectus muscle. Striated skeletal muscle is extensively disrupted by lymphoid aggregate. The infiltrate consists of numerous macrophages together with abundant eosinophils, in a sparse background population of small lymphocytes. There is no evidence of lymphoid neoplasia or other malignancy. (C) Six months after presentation on maintenance steroid treatment. Right hypertropia (patient often preferentially fixed with paretic eye), left upper lid retraction, limitation of left elevation and left episcleritis. Informed consent was obtained for publication of this figure.

cyclosporin A has been tried without effect, and the patient, who remains disabled by these episodes, has declined any further intervention.

Comment

Cyclical OEM is previously unreported. It is a benign inflammation with a cyclical frequency and chronic course. Important clinical features include alternating ptosis, large-angle vertical ocular deviations up to 50PD, and episcleral inflammation. A review by the regional clinical immunology unit confirmed a non-infectious, immune-mediated OEM. The pathogenesis of OEM may represent an abnormal activation of ocular allergic inflammatory mechanisms.⁴ Allergy-inducing T-helper type 2 cytokines such as IL-5 and eotaxin promote the activation and degranulation of eosinophils⁵ within ocular skeletal muscle. Focal necrosis of extra-ocular

skeletal muscle fibres leads to a self-limiting ocular myopathy. Eosinophils may persist in tissue, and recurrent reactivation may account for the cyclical frequency in our patient. Muscle is recognised to be resistant to apoptosis, and in inflammatory myopathies muscle fibres do not die.⁶ In our patient, a complete recovery of extra-ocular muscle function occurred between attacks. Our biopsy did not reveal any signs of myofibrosis. High-dose intravenous steroids appeared to be effective early on; however, OEM becomes steroid-resistant over time. Newer immunomodulatory therapy may play a role in reducing the duration of episodes and delay recurrence.⁷ Cyclical OEM is an unusual condition that is diagnosed by exclusion and early ocular muscle biopsy is advisable.

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References

- 1 Kaufman LD, Kephart GM, Seidman RJ, *et al.* The spectrum of eosinophilic myositis. Clinical and immunopathogenic studies of three patients, and review of the literature. *Arthritis Rheum* 1993;**36**:1014–24.
- 2 Leibovitch I, James CL, Wormald PJ, *et al.* Orbital eosinophilic angiocentric fibrosis. *Ophthalmology* 2006;**113**:148–52.
- 3 Gordon UK. Orbital inflammatory disease: a diagnostic and therapeutic challenge. *Eye* 2006;**20**:1196–206.
- 4 Solomon A, Shmilowich R, Shasha D, *et al.* Conjunctival fibroblasts enhance the survival and functional activity of peripheral blood eosinophils in vitro. *Invest Ophthalmol Vis Sci* 2000;**41**:1038–44.
- 5 Walsh GM, Hartnell A, Wardlaw AJ, *et al.* IL-5 enhances the in vitro adhesion of human eosinophils, but not neutrophils, in a leucocyte integrin (CD11/18)-dependent manner. *Immunology* 1990;**71**:258–65.
- 6 Schneider C, Gold R, Dalakas MC, *et al.* MHC class I-mediated cytotoxicity does not induce apoptosis in muscle fibres nor in inflammatory T cells: studies in patients with polymyositis, dermatomyositis, and inclusion body myositis. *J Neuropathol Exp Neurol* 1996;**55**:1205–9.
- 7 Powell N, Till S, Bungre J, *et al.* The immunomodulatory drugs cyclosporine A, mycophenolate mofetil, and sirolimus (rapamycin) inhibit allergen-induced proliferation and IL-5 production by PBMCs from atopic asthmatic patients. *J Allergy Clin Immunol* 2001;**108**:915–17.

Visual acuity standards for beach lifeguards

We have undertaken the first study to determine, in an operational scenario, the visual acuity required by beach lifeguards in order to identify a human head at the outer edge of a patrolled area of 300 m out to sea. The required visual acuity as determined by the angle subtended by a human head at this distance would be 6/17, but as a result of location and detection factors we hypothesised that it would have to be better than this.

The experiment received ethical approval and 21 beach lifeguards (16 men, 15 women, all under 35 years) gave their consent to participate. After eye tests to ensure normal vision (mean [range] visual acuity 6/4.8 [6/3.8–6/5]), they undertook a series of tests on beaches at Bournemouth (n = 9) and Westward Ho! (n = 12), during which their vision was blurred (using spherical lenses placed within a trial frame) to a visual acuity at which they could not identify the targets presented to them (approximately 6/70); these were human heads or equivalent-sized buoys. The subjects looked out to sea or across a wet beach and their visual acuity was improved every minute by reducing the refractive blur in 0.25 dioptre increments until they could identify the target to the point at which they would investigate it further using binoculars. The tests were performed on the same day, in good weather, uniform lighting conditions and a sea state of 0–1 (calm).

Results are presented in table 1, data were normally distributed (Anderson–Darling normality test).

Table 1 Results from Bournemouth and Westward Ho!

Condition	LogMAR mean (SD)	Snellen
(i) B: locate and identify a human head within a 300 m radius out to sea (volunteer immersed to neck)	0.11 (0.06)	6/8
(ii) W: locate and identify a human head within a 300 m radius out to sea (volunteer immersed to neck)	0.06 (0.17)	6/7
(iii) B: identify an arm waving at 300 m in the sea (volunteer immersed to neck)	0.33 (0.16)	6/13
(iii) B: identify a human immersed to the waist at 300 m in the sea	0.74 (0.15)	6/33
(iv) B: locate and identify a human head within a 100 m radius out to sea (volunteer immersed to neck)	0.86 (0.23)	6/43
(ii) W: identify a buoy at 200 m out to sea (n = 8)	0.28 (0.18)	6/11
(iii) W: identify a buoy at 100 m out to sea (n = 8)	0.78 (0.25)	6/36
(iv) W: identify a buoy on the wet beach at 300 m	0.23 (0.19)	6/10
(v) W: identify a buoy on the wet beach at 200 m	0.48 (0.13)	6/18
(vi) W: identify a buoy on the wet beach at 100 m	0.87 (0.15)	6/44

B, Bournemouth; logMAR, logarithm of the minimum angle of resolution; W, Westward Ho!