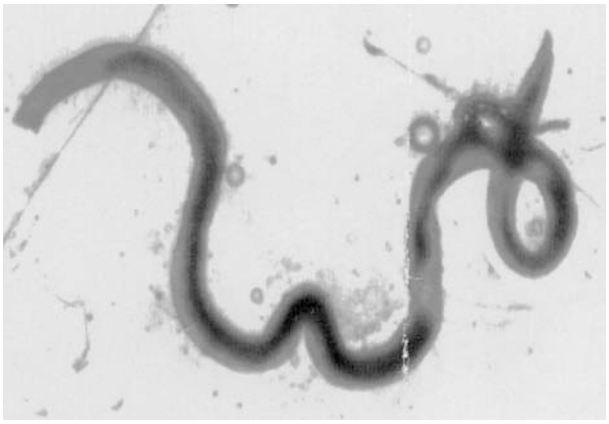

Figure 2 Light micrograph of *Angiostrongylus cantonensis* following removal from the eye (magnification x25).

literature, 14 previously reported cases were found.[2–5] Clinical findings included anterior uveitis, episcleritis, raised intraocular pressure, macular oedema, pigment dispersion, and retinal detachment.

Angiostrongylus cantonensis, a lung fluke of rodents, was first described by Chen in 1935 from the bronchial tree of *Rattus norvegicus* and *R.rattus rattus*, caught in Canton, China.[6] It is a delicate filiform worm, and has a length of 16-25 mm and a maximum diameter of 0.26-0.36 mm; the female of the species is larger. The rodent is the definite host. It is thought that humans are infected by eating inadequately cooked intermediate hosts (slugs, snails, crabs), or vegetables contaminated by larvae. The most likely routes of entry into the orbit are between the optic nerve and sheath, through the cribriform plate, and into the anterior chamber through the limbus.

It is difficult to know where our patient came into contact with the infected intermediate molluscum host. Although he is resident in the United Kingdom, he had recently visited South Africa. Medline search, however, revealed no previous reports of ocular Angiostrongyliasis from United Kingdom or South Africa.

Acknowledgements

We are grateful to Dr William Newsholme and Dr Peter Chiodini (Hospital of Tropical Diseases, London) for their help with the identification of the parasite.

References

1 Sabrosa NA, Sajdenweber M. Nematode infections of the eye: toxocariasis, onchocerciasis, diffuse unilateral subacute neuroretinitis and cysticercosis. *Ophthalmol Clin North Am* 2002; **15**(3): 351–356.

2 Toma H, Matsumura S, Oshiro C, Hidaka T, Sato Y. Ocular angiostrongyliasis without meningitis in Okinawa, Japan. *J Parasitol* 2002; **88**(1): 211–213.

3 Dissanaike AS, Ihalamulla RL, Naotunne TS, Senarathna T, Withana DS. Third report of ocular parastrongyliasis (angiostrongyliasis) from Sri Lanka. *Parassitologia* 2001; **43**(3): 95–97.

4 Kanchanaranya C, Prechanond A, Punyagupta S. Removal of living worm in retinal Angiostrongylus cantonensis. *Am J Ophthalmol* 1972; **74**: 456–458.

5 Kunchanaranya C, Punyagupta S. Case of ocular angiostrongyliasis associated with eosinophilic meningitis. *Am J Ophthalmol* 1971; **71**: 931–934.

6 Makerras MJ, Sandras DF. The life history of the rat lungworm Angiostrongylus cantonmensis (Chen). *Aust J Zool* 1995; **3**: 1–25.

V Kumar[1], I Kyprianou[1] and JM Keenan[2]

[1]Birmingham and Midland Eye Centre
Dudley Road,
Birmingham, UK

[2]The Eye Unit,
The Royal Surrey County Hospital
Guildford, B18 7QH,
UK

Correspondence:
V Kumar
Tel: +44 121 554 3801
Fax: +44 121 507 6791
E-mail: vkumar@rcsed.ac.uk

Eye (2005) **19,** 229–230. doi:10.1038/sj.eye.6701442
Published online 2 July 2004

Sir,

Acquired Brown's syndrome secondary to Ahmed valve implant for neovascular glaucoma

Aqueous drainage implants are successfully used in the treatment of severe cases of glaucoma that are refractory to standard antiglaucomatous surgery. However, complications following these implants can occur.[1] In this paper, we report a case of acquired Brown's syndrome (ABS) that occurred 3 weeks following the surgical implantation of an Ahmed valve implant in the superior nasal quadrant of an eye with neovascular glaucoma. Unlike a previous report[2] in which an Ahmed valve caused ABS because the implant was placed in a patient with a small orbit, in the present case there was no

implant–orbital disproportion, and the ABS occurred presumably secondary to fibrosis and consequent shortening of the superior oblique tendon of the affected eye.

Case report

A 74-year-old male patient was admitted to our Glaucoma Service complaining of pain in the right eye (RE) during the last 3 months. He had been submitted to trabeculectomy in this eye in another hospital, 6 months previously. When first examined by our staff, his best-corrected visual acuity was 20/400 in the RE and 20/25 in the left eye (LE). Slit-lamp examination of the RE revealed a diffuse conjunctival hyperaemia, fibrosis in the superior temporal quadrant (area of previous trabeculectomy), mild corneal odema, hyphaema (1 mm), iris neovascularization, ectropion uveae, and discrete nuclear opacification of the lens; the LE was unremarkable. The intraocular pressure (IOP) was 62 mmHg in RE and 14 mmHg in LE. Gonioscopy revealed peripheral anterior synechia in 360° of the angle; the LE angle was open. Ophthalmoscopy revealed a pale optic disc with a cup/disc ratio of 0.9 in RE. In LE the cup/disc ratio was 0.3. Ocular motility was normal.

Fluorescein angiograms of the RE taken 1 year before were available and indicated that the patient had been previously affected by a central retinal arterial occlusion before developing neovascular glaucoma. The patient then received retinal pan-photocoagulation therapy as well as clinical treatment to decrease the IOP. Despite the retinal laser therapy and the maximally tolerated medication the IOP was 42 mmHg. Drainage implant (Ahmed valve, model AGV-S2) surgery was planned. The superior nasal quadrant was chosen as the location for the implant due to the fact that the superior temporal region was extremely fibrotic due to a previous trabeculectomy. Within the first few days postsurgery, the IOP was 15 mmHg. However, within 3 weeks of surgery the patient presented a hypotropic RE in the primary position, with limitation of elevation in all superior gazes, but greater in adducted position (Figures 1 and 2). The forced duction test of this eye showed a mechanical restriction of elevation in adduction, which characterizes an ABS. As the IOP was under control and the patient did not complain of the ocular deviation, we opted for observation alone with regular follow-up.

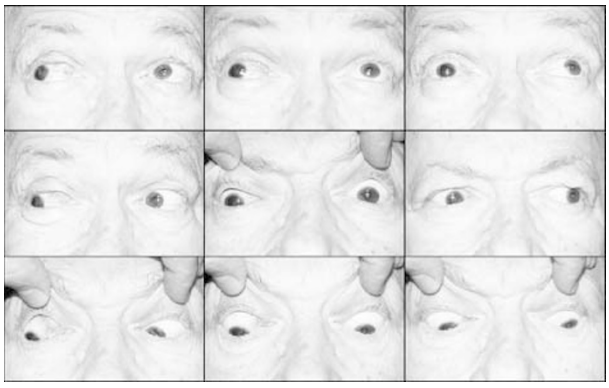

Figure 1 Acquired Brown's syndrome 3 weeks after superior nasal placement of the Ahmed implant in the RE. Note the inability to elevate the RE in the adducted position.

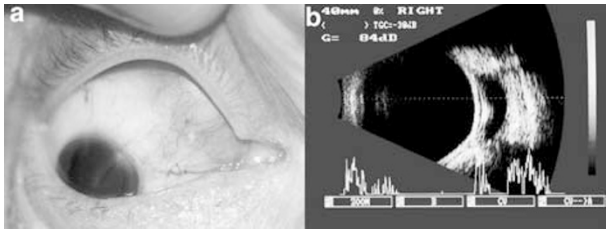

Figure 2 (a) Observe the large fibrotic bleb in the superior nasal region of the RE. (b) Ultrasound of the same eye revealing that there was no implant–orbital disproportion.

Comments

Aqueous drainage implants are currently used in the treatment of cases of glaucoma refractory to standard antiglaucomatous surgery. Despite the satisfactory results in the control of the IOP, multiple complications are associated with these implants, including hypotony, cataracts, choroidal effusion, and haemorrhage, corneal decompensation, flat anterior chamber, retinal detachment, endophthalmitis, tube or plate erosion, and hyphaema.[1] Extraocular motility disturbances are not rare following surgery with these drainage devices. In fact, muscle palsies, ABS syndrome, and generalized restrictions can occur.[1]

Ball *et al*[3] described the first case of ABS secondary to a drainage valve implant, in this case a Baerveldt type, which was placed in the superior nasal quadrant of a glaucomatous eye of a patient with Cogan–Reese syndrome. The authors thought that the ABS resulted from expansion of the fibrous capsule around the bleb, causing a mechanical restriction and capture of the superior oblique tendon. Later, Dobler *et al*[4] and Dobler-Dixon *et al*[1] described four cases of ABS following a double-plate Molteno implant. ABS has also been reported secondary to a Krupin valve.[5] In these previously mentioned papers, the patients had the drainage device implanted in the superior nasal quadrant of the affected eyes. Prata Jr *et al*[5] suggested that the restriction of ocular motility was due to the presence of large filtering blebs or increased

inflammation and scar formation in the superior nasal quadrant. These authors also hypothesized that the size of the implant was a predisposing factor for the development of ocular motility disturbances. Nevertheless, even with smaller implants, like Ahmed's type, these problems can occur. Indeed, Coats *et al*[2] described a case in which the primary cause of ABS was the disproportion between the orbital size and the Ahmed implant, since ocular deviation began immediately after surgery.

In the present case, there was no disproportion between the size of the orbit and the implant, which demonstrated that even with the use of an implant of a regular size (Ahmed), limitation of ocular motility could occur due to fibrosis. This fibrotic reaction presumably caused a shortening of the superior oblique tendon, causing limited elevation in the adduction of the affected eye of our patient.

In selected cases in which the superior nasal region is the only possible place to implant the device, one must choose those with small areas of drainage and the procedure must be performed with the least inflammatory stimuli. Moreover, patients must be informed about the possibility of postoperative complications such as ocular motility abnormalities and diplopia.

References

1 Dobler-Dixon AA, Cantor LB, Sondhi N, Ku WS, Hoop J. Prospective evaluation of extraocular motility following double-plate Molteno implantation. *Arch Ophthalmol* 1999; **117**: 1155–1160.
2 Coats DK, Paysse EA, Orenga-Nania S. Acquired pseudo-Brown's syndrome immediately following Ahmed valve glaucoma implant. *Ophthalmic Surg Lasers* 1999; **30**: 396–397.
3 Ball SF, Elis Jr GS, Herrington RG, Liang K. Brown's superior oblique tendon syndrome after Baerveldt glaucoma implant. *Arch Ophthalmol* 1992; **110**: 1368.
4 Dobler AA, Sondhi N, Cantor LB, Ku S. Acquired Brown's syndrome after a double-plate Molteno implant. *Am J Ophthalmol* 1993; **116**: 641–642.
5 Prata Jr JA, Minckler DS, Green RL. Pseudo-Brown's syndrome as a complication of glaucoma drainage implant surgery. *Ophthalmic Surg* 1993; **24**: 608–611.

Marcelo Palis Ventura[1], Raul NG Vianna[1,2], João Pessoa Souza Filho[2], Helena Parente Solari[1] and Renato LN Curi[1]

[1]Department of Ophthalmology
Fluminense Federal University
Niteroi, Rio de Janeiro, Brazil

[2]Department of Ophthalmology
McGill University Health Center
Montreal, Quebec, Canada

Correspondence: Raul NG Vianna
Av. Ary Parreiras, 438, Icaraí-Niterói-RJ
CEP: 24230-322, Brazil
Tel: +21 2610 1452
Fax: +21 2610 1051
E-mail: raulngvianna@ig.com.br

The authors do not have any commercial or proprietary interest in the products used in this study.

Eye (2005) **19,** 230–232. doi:10.1038/sj.eye.6701439
Published online 21 May 2004

Sir,

Vertical Gaze Palsy in a case with Growing Skull Fracture and Porencephalic Cyst

Linear or nonlinear skull fractures that enlarge with time are termed growing skull fractures.[1–3] These are commonly seen in children, possibly because of the greater malleability of the infant skull and the tighter adherence of the dura to the bone.[1] This growth of the fracture may cause atrophy of the underlying cerebral tissue along with the formation of a porencephalic cyst, which may extend through the skull defect into the subgaleal space, all of which may result in progressive neurological deficits.[1–3]

We report an unusual case of a young boy harbouring a large porencephalic cyst, who was found to have vertical gaze palsy on ocular examination in the absence of other neurological signs and symptoms.

Case report

A practitioner referred a 10-year-old boy with no systemic abnormalities to our strabismus clinic with the diagnosis of alternate convergent squint, present for the past 9 years. Ocular examination revealed a visual acuity of 6/6 (Snellen's alphabetical chart) bilaterally, with no significant refractive errors. Right suppression was present. Ocular motility examination revealed mild restriction of right abduction associated with nominal contracture of the right medial rectus muscle on passive ductions. Gross restriction of upgaze and downgaze was present in both eyes but the forced ductions were normal (Figure 1). The Bell's phenomena were intact (Figure 1). Fundus examination revealed mild pallor of both the optic discs. The rest of the anterior and posterior segment evaluation was unremarkable bilaterally. A diagnosis of