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Sir,

Streptococcus pneumoniae meningitis following postoperative endophthalmitis

Postoperative bacterial endophthalmitis is usually confined to the eye. Metastatic spread to the meninges is very rare and could have a devastating outcome.

Case report

An 87-year-old man presented with one day history of severe pain and reduced vision in his left eye 3 days following uneventful cataract surgery under topical anaesthesia. He previously had successful right cataract surgery and bilateral ptosis correction. His medical history included Hypertension and Aortic Valve replacement.

His visual acuity was 6/60 in the affected eye. Examination revealed corneal epithelial and stromal oedema, +3 cells in the anterior chamber and raised intraocular pressure (45 mm Hg). A red reflex was present but no fundus details were visible.

Intravitreal injections of Tiecoplanin (1 mg) and Ciprofloxacin (0.2 mg) were administered. Oral Ciprofloxacin and topical Tiecoplanin, Ciprofloxacin, Dexamethasone, Ketorolac, and Atropine were commenced.

The AC tap and vitreous tap grew *Streptococcus pneumoniae,* which was sensitive to Chloramphenicol but resistant to Ciprofloxacin. Hence topical Ciprofloxacin was substituted by Chloramphenicol.

Two days later, he developed tonic-clonic seizures and reduced GCS (10/15). An urgent CT of Head did not reveal any intracranial haemorrhage. His bloods showed WBC 26.5, Neutrophils 24.4, and CRP 168. A lumber puncture showed RBCs 1600×10^6/l, WBCs 2500×10^6/l (Neutrophils 95%, Lymphocytes 5%); pneumococcus was positive on PCR.

He was diagnosed with pneumococcal meningitis. Intravenous Ceftriaxone 2 gm BD given for 2 weeks resulted in resolution of meningitis. His final visual acuity dropped to 3/60.

Comment

Postoperative endophthalmitis is a dreaded complication of cataract surgery. Prompt diagnosis and treatment with intravitreal and intensive topical antibiotics is required to salvage vision.[1] Systemic antibiotics may have a role in prevention of systemic spread of infection. Systemic spread to involve meninges has been reported[2] once but is extremely rare.

Our patient developed meningitis despite receiving oral Ciprofloxacin because the offending organism was resistant to this drug. He was successfully treated under guidance of microbiology results.

Patients with endophthalmitis should be observed closely for signs or symptoms of metastatic spread. Although postoperative bacterial endophthalmitis is typically confined to the eye, this case report indicates that the infection can spread to the central nervous system. The treatment of endophthalmitis (including systemic antibiotics) should be guided by microbiology to ensure that the antibiotics administered are effective.

Conflict of interest

The authors declare no conflict of interest.

References

1 Lemley CA, Han DP. Endophthalmitis: a review of current evaluation and management. *Retina* 2007; **27**(6): 662–680.
2 Chan SM, Hodge WG, Leonard BC. Postoperative Streptococcus pneumoniae endophthalmitis complicated by meningitis. *Arch Ophthalmol* 1998; **116**(7): 951–953.

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Sir,

Longitudinal sectioning of temporal artery biopsy specimens

Despite increasing interest in the use of various imaging modalities such as ultrasound, magnetic resonance imaging, and positron-emission tomography, temporal artery biopsy remains the gold standard in the diagnosis of giant cell arteritis (GCA).[1] Given that GCA can lead to profound irreversible blindness and other devastating complications, every effort must be made to ensure a correct diagnosis is made in each and every case.

Case report

We report a case of an 88-year-old patient referred with suspected GCA. A temporal artery biopsy was performed. Macroscopically, the specimen had a segmented, earthworm-like appearance. The 22-mm-long specimen was processed *in toto* and sectioned in the longitudinal plane at six levels, $\sim 120\,\mu$m apart. This demonstrated patchy focal inflammation, predominantly in the outer media, with 'skip areas' of 3–5 mm of uninflamed artery (Figure 1). The inflammatory infiltrate consisted predominantly of mixed mononuclear

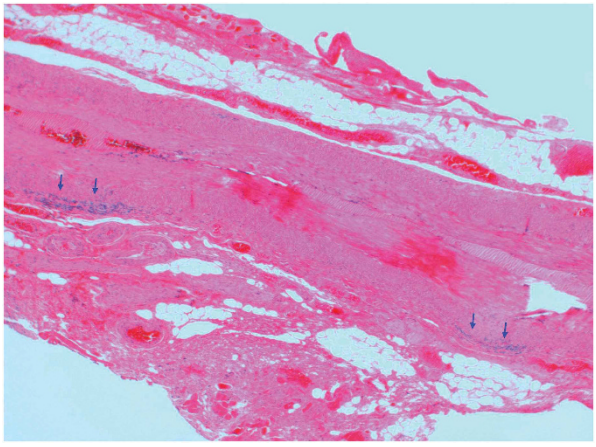

Figure 1 Low magnification of longitudinal cross-section of TAB specimen demonstrating patchy areas of inflammation (arrowheads) with skip lesions of 3–5 mm of uninflamed artery.

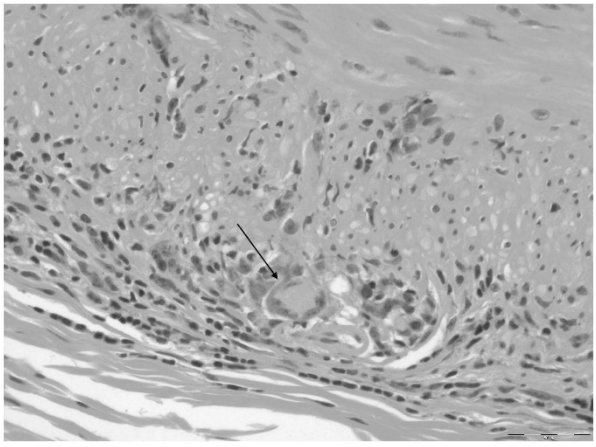

Figure 2 Higher magnification showing inflammatory foci consisting of mononuclear inflammatory cells and a multinucleated histiocyte ('giant cell', marked by arrowhead).

inflammatory cells (Figure 2). Had the artery been sectioned transversely alone, it is possible that the patchy inflammation would have been missed.

Simple geometrical calculation shows that the approximate area of a longitudinal section of artery is $2lr$ (where l is the length of the artery and r is its radius). The area of a transverse section is πr^2. Where the entire artery is bread-loafed at intervals of about 3–4 mm, as is the standard textbook recommendation,[2] then the total transverse area available for examination would be $\pi r^2 l/3$ (for transverse block length of 3 mm). Further calculation reveals that with an arterial transverse diameter of about 2.5 mm,[3,4] the area of tissue available for examination using longitudinal sections is about 50% greater than using transverse sections, and that difference doubles to the advantage of the longitudinal section if the transverse slices are 4 mm long. (The calculations are more complex in reality as we have not considered factors such as the diminishing areas obtained with deeper longitudinal sectioning and the influence of the variable size of the arterial lumen).

In the practical experience of one of the authors (AG) over several decades of practice, almost invariably, many of the transverse slices end up longer than 3–4 mm and the entire artery is not processed. Furthermore, any perceived advantage with transverse sectioning is on occasion lost owing to the difficulty of handling and orientating small pieces of tissue during processing. Despite the best intentions of the pathologist, this may result in many of the transverse pieces being actually embedded and sectioned in the longitudinal plane.

Comment

We therefore believe that longitudinal sectioning of the artery is a more appropriate strategy than transverse sectioning as it allows a greater proportion of the available specimen to be examined microscopically. That method of tissue handling is also considerably easier for laboratory staff to process.

To the best of our knowledge, there has been very little research into this area. A study presented at a conference in 2007 that examined 495 TAB specimens over a 15-year period reported that longitudinal sectioning yielded a 20% increase in positive biopsy results over transverse sectioning.[5] However, to our knowledge these results were not published in the peer-reviewed literature.

It is important that all clinicians involved in the diagnosis and management of giant cell arteritis are aware of the issues of specimen length and sectioning strategy when evaluating the TAB result. We recommend surgeons discuss with their pathologist colleagues the sectioning strategy used to yield the most accurate result.

Conflict of interest

The authors declare no conflict of interest.

References

1 Kale N, Eggenberger E. Diagnosis and management of giant cell arteritis: a review. *Curr Opin Ophthalmol* 2010; **21**: 417–422.
2 Lee WR. *Ophthalmic Histopathology*. 2nd ed. Springer: New York, USA, 2002, pp 325–326.
3 Bley TA, Wieben O, Uhl M, Thiel J, Schmidt D, Langer M. High-resolution MRI in giant cell arteritis: imaging of the wall of the superficial temporal artery. *Am J Roentgenol* 2005; **184**: 283–287.
4 Pinar YA, Govsa F. Anatomy of the superficial temporal artery and its branches: its importance for surgery. *Surg Radiol Anat* 2006; **28**: 248–253.
5 Chatelain D, Duhaut P, Loire R, Bosshard S, Pellet H, Piette J *et al*. Features of temporal artery biopsy and diagnostic of giant cell arteritis: the multicentric, prospective GRACG study. *American College of Rheumatology Annual Scientific Meeting* 2007; 1240.

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Sir,

Choroidal new vessels in type 1 myotonic dystrophy-related macular dystrophy respond to anti-VEGF therapy

Mytonic dystrophy type 1 (DM1) is an autosomal dominant disorder caused by mutation in the dystrophia myotonica protein kinase (*DMPK*) gene, located on chromosome 19 (19q13.2–q13.3). Ocular findings reported to date include colored iridescent cataract, strabismus, and limitation of extraocular muscle movement, ptosis, and rarely macular dystrophy.[1]

Case report

A 21-year-old male with DM1 presented with a 4-week history of deteriorating vision in his left eye, assumed to be cataract. Clinical examination, however, showed bilateral pattern dystrophy (Figure 1a) with best corrected visual acuity (VA) recorded at 6/6 in the right eye and 6/15 in the left eye, which had choroidal new vessels (CNVs) (Figures 1b and 2aI). Treatment followed consent of the off-label nature of intravitreal bevacizumab (IVB). After 4 weeks, symptoms had improved but due to persisting intraretinal fluid (Figure 2aII) a second IVB injection was given. After 4 weeks, VA had improved to 6/7.5 with both OCT and angiography confirming no leakage (Figures 2aIII and b). No recurrence was seen at most recent follow-up 14 months later. His father who had DM1 was also examined, but did not have macular dystrophy.

Comment

To our knowledge this is the first report of CNVs complicating DM1. The cause of the macular phenotype is still unknown. The expansion of CTG triplet repeats in DM1 arise within the 3′ untranslated region of the *DMPK* gene,[2,3] but this region also overlaps downstream with the promoter of the homeobox gene *SIX5* on chromosome 19q13.3.[4] As expression of this gene has been localized to the lens, retina, and choroid, it has been

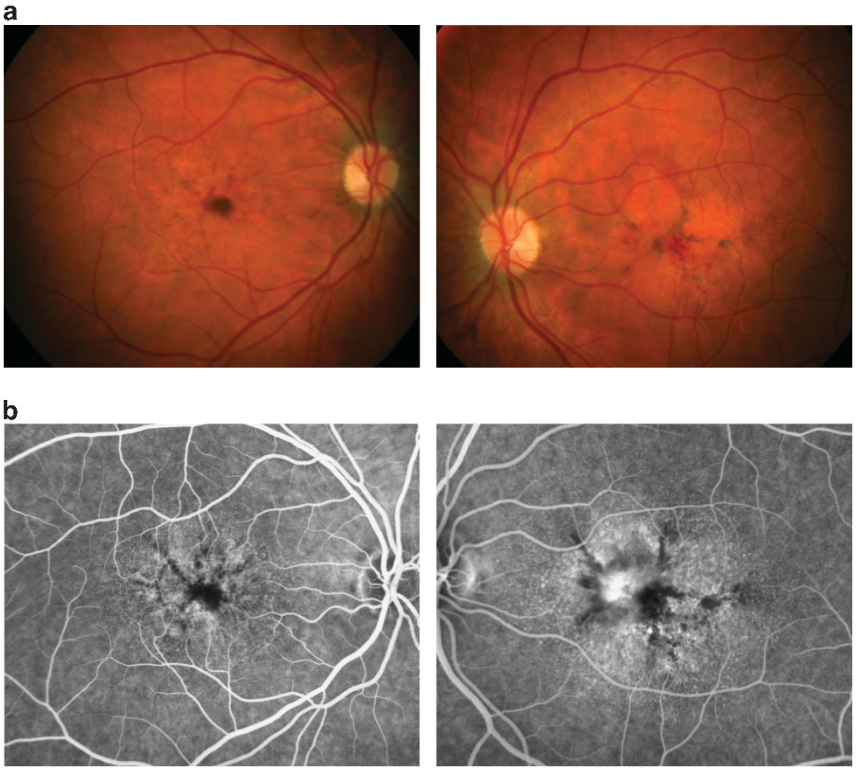

Figure 1 (a) Color images of right and left fundus with pattern retinal pigment epithelium changes. Subretinal hemorrhages are evident in the center of the left macula. (b) Late fundus fluorescein angiogram images before treatment shows RPE changes and, in the left eye, central leakage.