Photoreceptor Damage in Terson Syndrome

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INTRODUCTION

Terson syndrome (TS) is defined as intraocular haemorrhage secondary to acute rise in intracranial pressure, commonly due to subarachnoid haemorrhage (SAH). Here, we demonstrate photoreceptor damage in Terson syndrome patients with poor spatial correlation to areas of inner retinal haemorrhage.

METHODS

Retrospective case series of 6 patients with TS at Oxford University Hospitals. For each patient, spectral domain optical coherence tomography (OCT) and confocal near-infrared (NIR) reflectance images (Spectralis HRA-OCT, Heidelberg Engineering, Heidelberg, Germany) were obtained through routine clinical care.

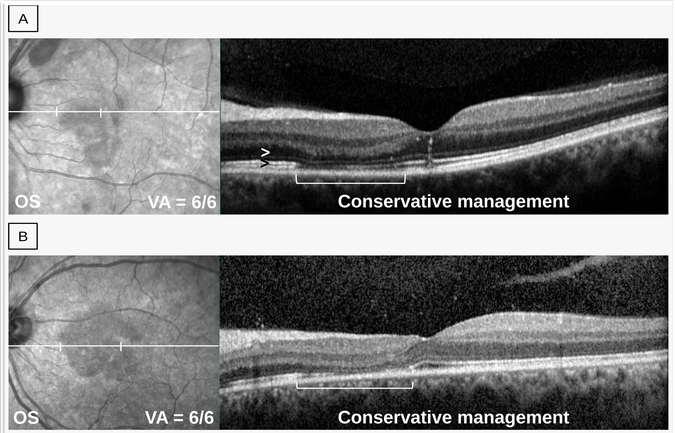


Figure 1. Outer retinal damage in patients with Terson Syndrome. (A) 53-year-old female with TS secondary to SAH. Images taken 3 months post-SAH. **(B)** 34-year-old male with TS secondary to superior sagittal sinus thrombosis. Images taken 2 months post-admission. In both patients well-defined areas of hypo-reflectance can be seen on NIR reflectance images (left panels), and correspond to areas of outer nuclear layer thinning (ONL, white arrow) and ellipsoid zone disruption (EZ, black arrow) visible on OCT scans (right panels). These are marked with white brackets and indicate photoreceptor damage. The lesions localise to the central macula but with sparing of the foveola which may explain good final visual acuity.

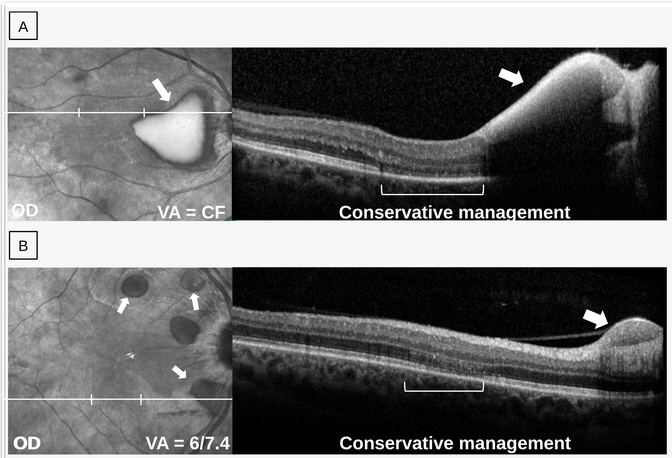


Figure 2. Poor spatial correlation between outer retinal damage and sub-ILM haemorrhage in TS. (A) 54-year-old female with TS secondary to SAH. **(B)** 56-year-old female with TS secondary to SAH. Images for both patients taken 6 weeks post-SAH. In both patients, regions of photoreceptor damage can be seen in areas not affected by sub-ILM haemorrhage (white arrows), thus indicating that photoreceptor damage is not caused by direct toxic effects of retinal blood. Areas directly under the haemorrhages cannot be assessed due to the optical effects of blood.

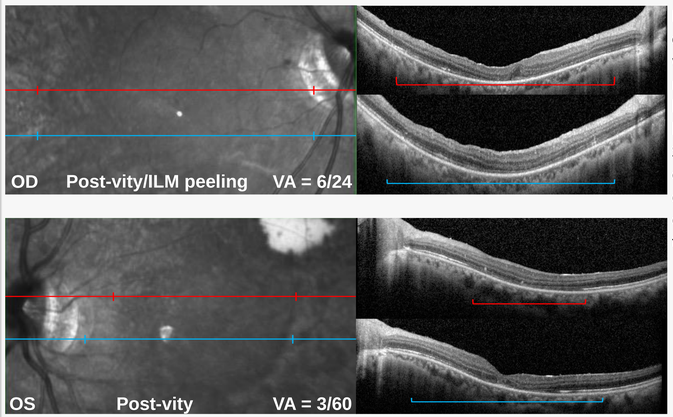


Figure 3. Bilateral outer retinal damage in TS despite only unilateral sub-ILM haemorrhage. 35-year-old male with TS secondary to SAH. Presented with bilaterally reduced vision 15-days post-SAH. Bilateral vitreous haemorrhages were noted, but only right-sided sub-ILM haemorrhage. Images taken 2.5 months post-SAH. Surgery was performed 3 weeks post-SAH in the right eye, and 5 weeks in the left eye. Extensive outer retinal damage was observed in both eyes (coloured brackets) despite surgical management. Low final VA may reflect the extent of photoreceptor damage in this patient.

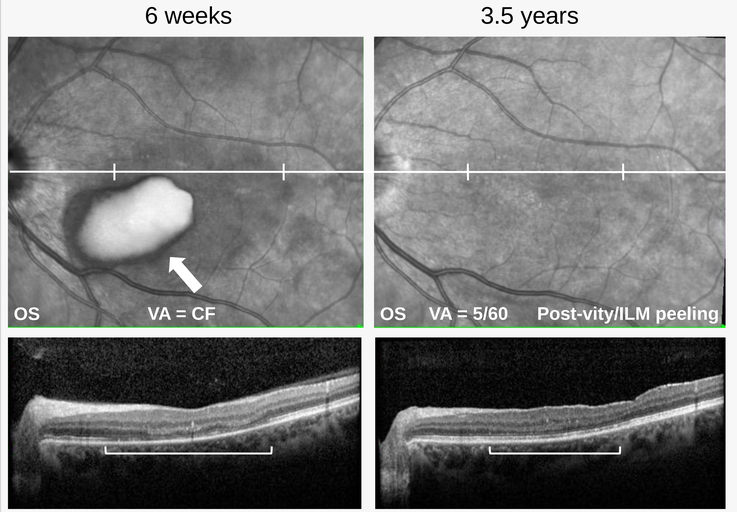


Figure 4. Incomplete recovery of photoreceptor damage in TS. Images taken from the same patient as in Figure 2 (A). There is incomplete recovery of outer retinal damage (white brackets) 3.5 years after presentation despite surgery performed 6 weeks post-SAH. Low final visual acuity in this patient was deemed likely to stem from cortical impairment.

CONCLUSIONS

1. TS can be associated with areas of photoreceptor damage localised primarily to the central macula, with variable impact on visual function. This calls for wider use of multimodal retinal imaging in TS patients.
2. There is poor spatial correlation between photoreceptor damage and sub-ILM haemorrhage, indicating their independence. Photoreceptor damage could be caused by disturbed choroidal perfusion due to raised ICP in early stages of SAH.
3. Surgical intervention does not address photoreceptor damage in TS, hence final visual acuity may be limited despite satisfactory evacuation of blood.

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