Bee Sting Injury to Eye

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A 63 year old male presented to our hospital with a history of bee sting injury to right eye 1 day back. On examination his BCVA was perception of light with inaccurate projection OD and 6/6 OS. The IOP was unrecordably low. There was no history of any systemic disease. On slit lamp examination the right eye revealed a diffuse stromal corneal edema with coarse descemet’s folds and the anterior chamber was totally flat. [Figure 1] B scan ultrasound revealed multiple point echoes of moderate intensity in the vitreous cavity suggestive of vitritis. [Figure 2] Ultrasound biomicroscopy was performed which revealed ciliary body effusion. [Figure 3]. Laboratory investigations revealed: Hb 13.4 g%, TC 16100/dl, DC P65, ESR 5, RBS 133, B.Urea 55mg%, S.Creatinine 1.3mg%, Cholesterol 133mg%, SGOT 80 u/l, SGPT 224, ALP 85, Total protein 7.1 g%, Albumin 4.8mg%, Globulin 2.3 g%, Sodium 139 meq/l, Potassium 3.5meq/l. The patient was then started on Systemic steroids initially IV methyl Prednisolone with systemic antibiotic followed by topical antibiotics, steroids and cycloplegic. On the next review after 1 week, on examination the corneal edema had partially resolved residual descemet’s membrane folds were seen. The anterior chamber was shallow. The pupil was oval, mid dilated and fixed. The anterior lens capsule showed pigments. The lens showed nuclear sclerosis. [Figure 4]. B scan ultrasound OD revealed multiple dome shaped elevations with a moderately reflective membrane which depicted a double spike at the corresponding location on the A scan and the interior of the elevations showing low intensity hyperechoic signals with corresponding low intensity spikes on the A scan suggestive of multiple serous ciliochoroidal detachments (Fig 5).
Discussion

The bee sting is a modified ovipositer in female bee and wasp. It has toothed lancets and poison glands. The venom acts as a toxic agent, and the sting itself has a mechanically damaging effect on the structures of the eye (Grant, 1962; Bucherl and Buckley, 1971). Toxic reaction is due to biological amines which are protoplasmic poisons. It contains neurotoxin, hemolitin, mellitin, hyaluronodase, phospholipase A and histamine (Gilboa et al; BJO 1977, 61, 662-664).

Dose of venom and adverse events are unknown and these could be toxin related or immunologically mediated. Case reports show VEP to be prolonged but pattern ERG is usually normal. The retina may not be affected. In our case it was a direct sting to globe. Various reported clinical presentations in cases of bee sting include Cilio choroidal detachment (Pal et al Eye 2005), Optic neuritis (Choi et al Korea 2003), Mimics viral keratitis (Jain et al Cornea 2007), Cataract and corneal decompensation, (Arcieri et al Cornea 2002), Cataract (Agarwal et al IJO 1995), Uveitis, glaucoma and optic neuropathy (Teoh et al CJO 2005), Disc oedema, Scotoma (Maltzman et al Ophthalmology 2000) and bilateral optic neuritis (AJO 1994) Papilloedema of 1 dioptre and much retinal venous engorgement with normal visual acuity have been observed 9 days after bee stings (Goldstein and Rucker, 1964). Optic atrophy and papillitis secondary to bee sting have been described (Walsh and Hoyt, 1969; Goldstein and Woltman, 1960).

In this case there is cataract, low IOP and choroidal detachment with no perception of light. This is probably due to the optic neuritis element which was underlying. The devastating effect in this case may be due to direct inoculum of toxin into the eye unlike in many published data where there is bite in the periorbital region.

References:

