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Nil Perception of Light Due to Delayed Presentation of an Inflammed Orbit

KEYWORDS: Orbital cellulitis, hemi retinal artery occlusion, ischemic optic neuropathy

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ABSTRACT *We report a case of 64 year old female who presented with an acute onset of right sided painful abaxial proptosis, superficially ruptured inferonasal orbital abscess with nil perception of light.*

On further evaluation the cause for the visual loss was attributed to hemi retinal artery occlusion with ischemic optic neuropathy . Orbital abscess has serious vision threatening and life threatening consequences. CT scan delineates the source and extent of tissue involvement.

Prompt diagnosis and appropriate management is imperative.

CASE PRESENTATION

A 64 year old female was admitted to our hospital with an acute onset of right sided painful abaxial proptosis and a superficially ruptured inferonasal orbital abscess below the right medial canthus. There was a three-day history of fever, eye pain and right upper and lower lid swelling.

Visual acuity at presentation was nil perception of light with afferent pupillary defect in right eye and BCVA of 6/24 in left with direct light reflex being present. Examination of her right eye revealed severe ptosis, 7mm of proptosis, a tender, fluctuant mass over medial canthus and hemi retinal artery occlusion. Her white blood cell count of 10,700 cells/cumm with a left shift. Sinus x-raydemonstrated normal sinuses. Non contrast CT scan of orbit showed extensive soft tissue thickening in the region of lacrimal fossa,inferonasal orbital space, adjacentretroorbital fat and overlying lower lidresulting in proptosis (Fig 1a,b), displacing the globe superotemporally

(Fig 2). There was a chronic history of epiphora, suggestive of chronic dacryocystitis. A fundus fluorescein angiography performed after subsidence of fever confirmed hemi retinal artery occlusion. Immediate treatment consisted ofabscess drainage and culture which grew Staphylococcus epidermidis sensitive topenicillins and cephalosporins. Patient was put on IntravenousCeftazidime1 gm q 12h and Metronidazole 500mg q 8h. Following this, postoperatively same antibiotics were continued after which she showed a rapid symptomatic improvement but with no visual recovery.

Fig 1

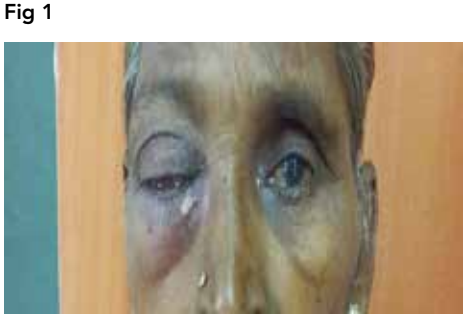


Fig 2a

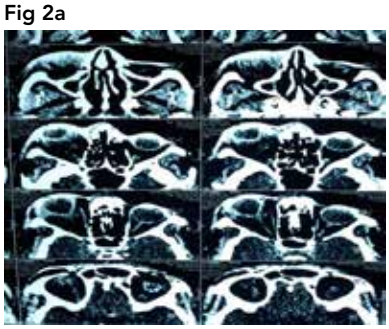


Fig 2b

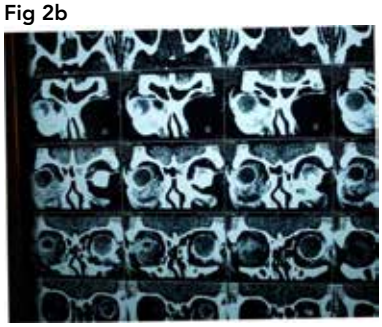
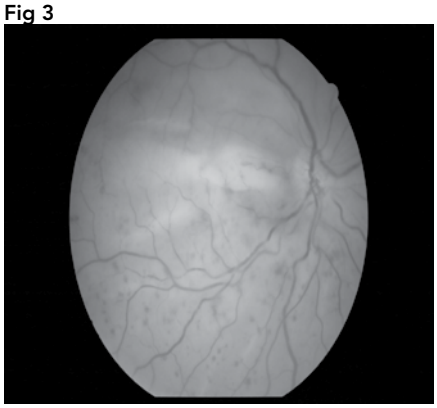


Fig 3



Volume : 3 | Issue : 11 | Nov 2013 | ISSN - 2249-555X

Fig 4

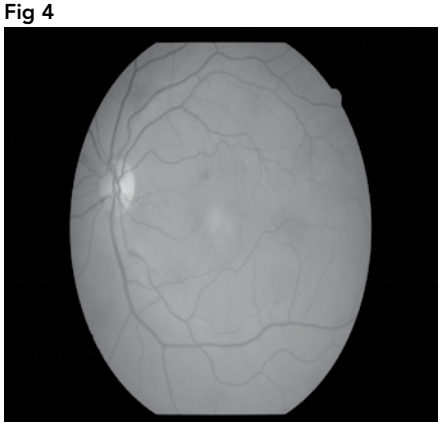


Fig 5

Fig 6

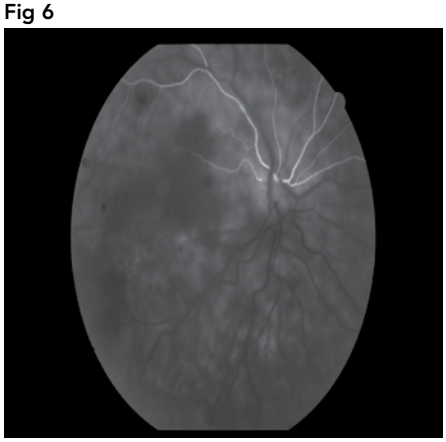


Fig 7

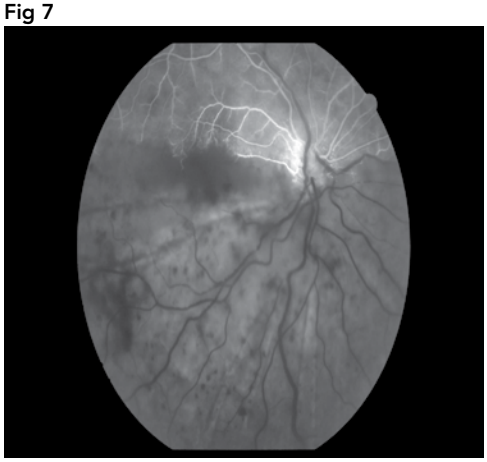


Fig 8

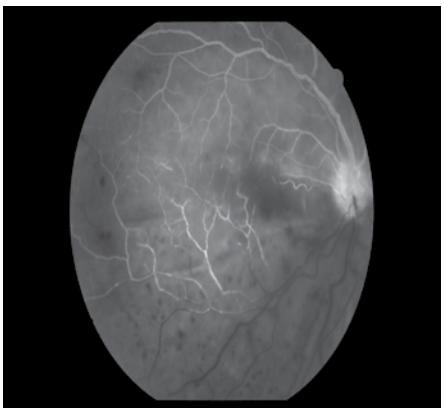


Fig 9

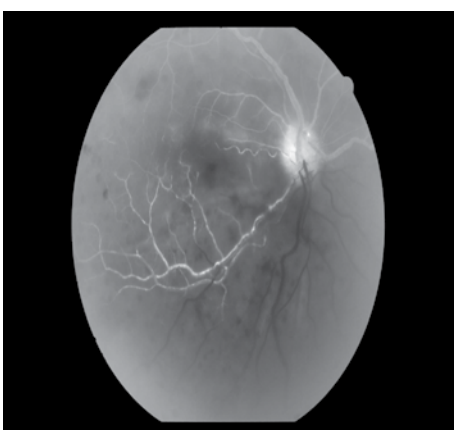


Fig 10

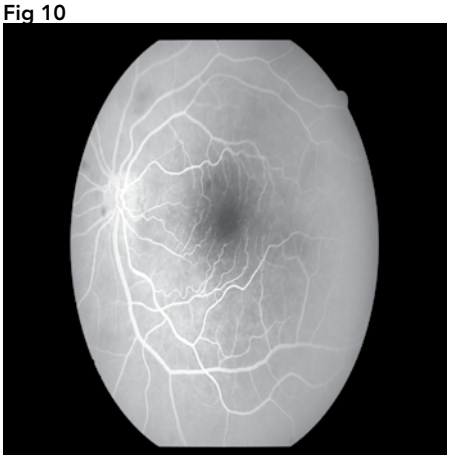


FIGURE CAPTIONS:

1) Clinical picture of right eye showing proptosis, ptosis and a superficially ruptured infraorbital abscess.
2a) Orbital axial CT scan showing right sided proptosis, supero lateral globe displacement and optic nerve stretching.
2b) Orbital coronal CT scan showing an inferonasal orbital abscess.

Red free fundus picture of:

3) Right eye showing retinal thickening only in the inferior retina.
4) Left eye which is unremarkable.
5) Fundus picture of the right eye showing inferior and posterior pole thickening, disc edema,hemorrhages in inferotemporal retina.

Volume : 3 | Issue : 11 | Nov 2013 | ISSN - 2249-555X

FFA pictures of right eye at:

6) 15 sec arterial phase showing non-filling of inferior retina.
7) 24 sec showing normal arteriovenous phase in superior retina and non-perfusion of inferior retina.
8) 2min 40 sec ,showing slow filling of inferior capillaries demonstrating retinal water shed zone.
9) 5 min 12 sec demonstrating filling of inferior vein.

FFA picture of left eye at :

10) 7 min 03 sec which is unremarkable.

DIAGNOSIS

Right sided orbital abscess with hemi retinal artery occlusion and ischemic optic neuropathy .

DISCUSSION

Infection of the orbit rarely occurs spontaneously. Organisms gain access to the orbital space mainly by implantation with foreign bodies, through septicemia or direct extension from adjacent tissues(sinuses,lacrimal sac,lids or teeth). An estimated 60% – 84% of orbital cellulitis results from paranasal sinus infections. [1,2]

Visual loss in a case of orbital abscess may be due to optic atrophy, central retinal artery occlusion or exposure keratopathy with ulcer formation. Other hypothesized mechanisms of visual loss include septic optic neuritis, embolic or thrombotic lesions in the vascular supply to the retina, choroid or optic nerve, or rapid elevations of intraocular pressure. Delayed surgical intervention is likely to produce a poor visual result.[3-8]

Our patient presented to us with a visual acuity of nil perception of light. An undue delayed presentation leading to hemiretinal artery occlusion with compressive ischemic optic neuropathy resulted in a grave visual outcome in this case. FFA demonstrates retinal water shed zone through filling of inferior hemi retinal vein in late phase. Prompt diagnosis and treatment is necessary to prevent severe visual loss and even death.

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