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Siderosis bulbi of eye due to undetected intraocular foreign body

KEYWORDS: Siderosis bulbi, Rosette cataract,Phacoemulsification

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ABSTRACT *The Siderosis bulbi of eye is caused due to presence of intraocular iron foreign body. A male, 26 years, came with chief complains of painless gradual diminishing vision in Right eye (RE) and night blindness for past 6 month. He had a history of trauma by metallic rod 1 year back. Corneal foreign body was removed under topical anesthesia. His vision started deteriorating after 6 month of trauma. RE findings: leucomatous corneal opacity at 7O clock in midpheriphery. Rusty pigment dispersion on endothelium was present. Pupil was semi dilated. Pupil reactions were sluggish. Rosette cataract with brown pigment on the lens was present. No foreign body was present in the eyeball.RE Phacoemulsifications with foldable intraocular lens done. Postoperative RE vision was 6/6 with refraction -0.75 x 80°.*

Purpose:
The Siderosis bulbi of eye is caused due to presence of intraocular iron foreign body. It affects all ocular structures. The purpose is to report a case of siderosis bulbi due to undetected intraocular foreign body.

Methods:
26 years, M residing at Pune,India came with chief complains of painless gradual diminished of vision of Right eye (RE) since 6 month. He had night blindness since last 6 month. He had a history of trauma by metallic rod 1 year back. Corneal foreign body was removed under topical anesthesia. He developed corneal irritation, so he put ciprofloxacin eye drop twice a day with lubricating eye drop for 8 days. His vision started deteorating after 6 month of trauma. No history of pain, redness, watering.

On ocular examination his vision was RE 6/60, Left eye (LE) 6/6.

RE findings: leucomatous corneal opacity at 7 0 clock in midpheriphery. Rusty pigment dispersion on endothelium was present. Anterior were within normal limits. Intraocular pressure was 11 mm of Hg in both eyes. Extra ocular movements were normal. Both eyes sac were patent. Both eyes fundus were normal.

Laboratory investigations: Hb – 14.9 gm%, WBC – 6300, DC – 56/37/4/3/0 (N/L/E/M/B), RBC – normocytic normochromic. Platelets – adequatechamber depth was normal. Pupil was semi dilated. Pupil reactions direct and indirect were sluggish. Rosette cataract with brown pigment on the lens was present with nuclear sclerosis grade II. LE findings, parasites- not seen, ESR- 5 mm at the end of 1 hr, urine routine, and microscopy was normal, BSL F 102, BSL PP 95, ECG – within normal limit, Preanaesthesia check up was normal.USG B scan was normal. CT orbit was normal. No foreign body was present in the eyeball.

A scan reading :RE KH- 44 D,KV- 43D,Axial length – 22.74 mm ,IOL 23 D. LE KH- 44.75D, KV- 43.75D, Axial length- 23.14mm,IOL 23.5D

Surgical procedure: Temporal incision taken. Side port made at 12 o' clock and 6 o' clock. CCC completed with side port. Phacoemulsifications done by stop and chop technique. Acrylic posterior chamber intraocular lens put in the bag. Visibility was poor during the surgery due to corneal endothelial dusting. Postoperative recovery was good.

RE findings: visual acuity 6/9 anterior segment was normal. After 4 weeks his vision was 6/6 with refraction -0.75 x 80°. Close follow up with this patient with 6/6 vision till 6 month.

Results: A trauma by iron object could result in siderosis bulbi and such patients need close follow-up.

Discussion:
The pathological anatomy of siderosis was first reported by von Hippel (1894), who distinguished two types of siderosis: haematogenous, in which the iron was derived from blood, and exogenous, in which it came from an intraocular foreign body[1].

When metal ions remained in the tissue for a long time can produce impairment of their function called METALLOSIS. The common metallic foreign bodies which penetrate the eye are iron or steel, copper, aluminium, lead pellets Most frequently encountered metallosis is ocular siderosis [2].

Pathophysiology
Bivalent iron (ferrous) is more toxic to ocular tissue than the trivalent ion (ferric). The toxicity is caused by interference with the essential enzyme system. Studies of the pathologic features of eyes with siderosis have revealed iron deposits accumulated in the regions of ocular pumps i.e., corneal endothelium and Descemet's membrane, trabecular meshwork, pupillary constrictor muscles, dilator muscle, non-pigmented ciliary epithelium, lens epithelium, retinal pigment epithelium, and internal limiting membrane. Histologically, Prussian blue stains the iron blue and shows it to be present in all ocular epithelial structures and in areas of trabecular meshwork scarring and retinal gliosis. By electron microscopy, intracellular sideromes are present in the lens epithelium and in corneal keratocytes[3].

In siderosis,ferrous pigmentation causes a rusty coloration of the cornea, iris ,or lens in addition ,a series of chronic

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degenerative changes of the retina (with poor night vision and restricted fields), retinal detachment, or open angle glaucoma. These complications usually occur between two months and two years after the injury, but may occur within several days. They are severe enough to warrant major surgical attempts to extract the foreign body as early as possible[4].

According to some author's deposition of iron in the sphincter of the iris leads to mydriasis. On contrary to this, our patient presented with the fixed normal size pupil. The cause may be posterior synechiae due to previous iritis[5].

Iron granules may deposit in any layer of cornea but are usually more prominent in the deeper cornea.siderosomes are noted within the keratocytes[6].

Lens

In siderosis or hemosiderosis the lens epithelium takes on a yellow-brown or rusty appearance, from minute dots of intracellular iron, identifiable by Perls' or other iron stains. Focal rusty-brown nodules of subcapsular cataract may develop. When the foreign body is in the lens there may be progression to a mature cataract, with diffusion of ionizable iron throughout the lens fibers. The iron appears to bind to enzymes within these cells, becoming insoluble and incorporated in phagolysosomes, with eventual cellular degeneration[3].

This visual outcome and clinically normal retina may be due to the favourable location of foreign body as documented in literature that there is poorer visual outcome in posterior segment intraocular foreign body as compared to anterior segment foreign body[5].

Intraocular iron may become oxidized and produce localized siderosis if the foreign body lodges in the sclera or siderosis bulbi when the foreign body is intravitreally diffuse[7].

Intraocular migration of iron from iron foreign bodies in an extraocular or intrascleral location was studied in rabbits. Focal changes occurred in the choroid and retina beneath the foreign bodies. Significant amounts of iron diffused through the sclera into the globes. This iron was typically deposited in the epithelium of the ciliary processes and in the tissues at the choroid-scleral junction in the posterior pole[8].

Even in the presence of good vision, a patient with an intraolenticular ferrous foreign body should be followed closely, and the foreign body should be removed before irreversible siderosis bulbi occurs [9].

Dan De Angelis et al found iron foreign body intraoperative. They could not find on standard radiography and CT. size of foreign body was 0.7mm by 0.7mm[10].

Conclusions:

Presence of intraocular iron particle is not a necessary precondition fordevelopment of siderosis bulbi. Removal of iron foreign body results in siderosis bulbi. Hence a close follow-up for prolong period (6-12 months) should be maintained in such patients. Post-operative visual prognosis is good.

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Fig 1:Preoperative RE siderosis bulbi

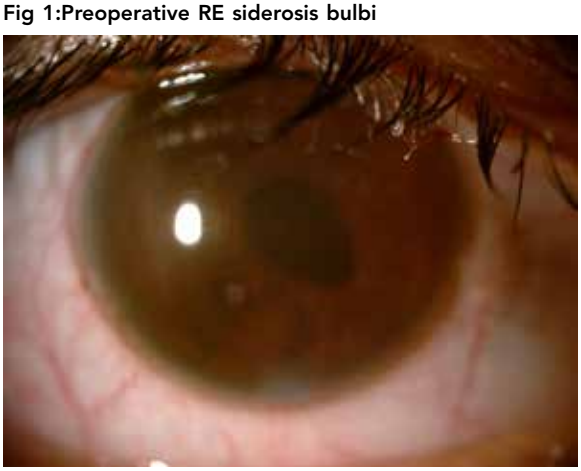

Fig 2:Postoperative RE Pseudophakia- Endothelial dusting

Fig 3:RE corneal opacity

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