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Case Report on Corneal Keloid

KEYWORDS

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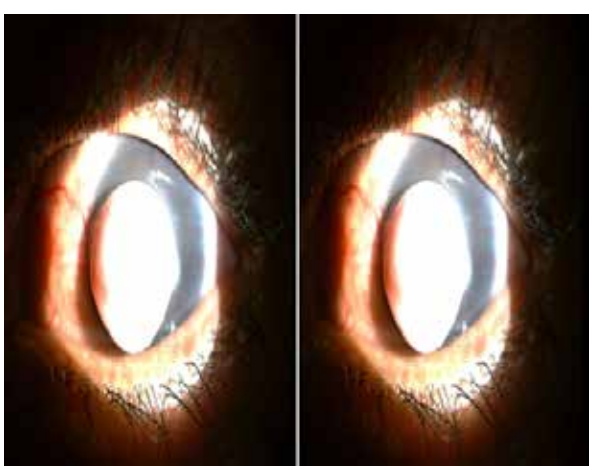
OBJECTIVE:
to learn management of patient with corneal keloid and to study literature review on it.

CASE REPORT:
30 year young female patient residing at rajasthan came to c.h. nagari eye hospital with chief complains of mass over cornea in left eye since 7 months gradually increasing in size with presenting size 5-6 mm with visual acuity of

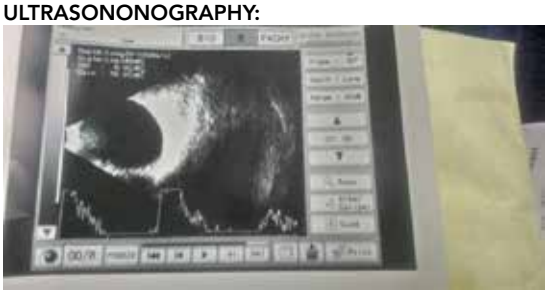
OD 6/6
OS HM+ PL+ PR4+
Patient gave history of injury to left eye with needle few years back no other significant history was present.



INVESTIGATIONS:
BLOOD INVESTIGATIONS:

Hb: 12	rbs: 98
Tc: 8000	urea: 14
Dc:65/30/4/1	creat:0.6
Esr:19mm/hr	SGPT: 17U/l
Bt:1'30"	S.bili: 0.7 mg/dl
Ct:2'45"	HIV/HBsAg: non-reactive

ULTRASONONOGRAPHY:

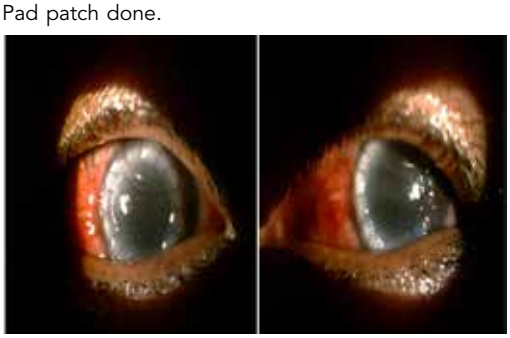


Vitreous cavity anechoic retina on

ULTRASOUND BIOMICROSCOPY:
UBM report indicates that mass arise from the cornea no attachment to iris was found.

HISTOPATHOLOGICAL EXAMINATION:
Keratin and fibrocollagen present in mass s/o keloid.

SURGERY : *optical penetrating keratoplasty sx was planned..*
Peribulbar anaesthesia given
Painting & draping done
Lids retracted
Corneal mass was excised with trephine 7.5 mm
Anterior chamber formed with viscoelastic substance
Donor tissue obtained with trephine 8.0 mm
Donor tissue placed over recipient bed and secure with suture ethylone 10.00
Anterior chamber formed with air
Subconjuctival geramycin + dexamethasone given
Pad patch done.



after penetrating keratoplasty surgery was done:

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patient was prescribed oral and topical steroids and antibiotics along with cycloplegics, lubricating eyedrops and antiglaucoma drops.

REVIEW OF LITERATURE:

INTRODUCTION:

Corneal keloid clinically appears as a solitary, gray-white, firm, elevated, smooth, shiny nodule that is well-demarctaed from adjacent tissue, although it may involve the entire corneal stroma.[10,17,24] There is often a clinical history of penetrating or non-penetrating injury preceding the development of the keloid. Corneal thickening is caused by fibrovascular hyperplasia secondary to scar formation. The lesion often covers a large area of the corneal surface, well beyond the presumed localized sit of injury.[2,11,17,24,26]. Ultrasound evaluation of corneal keloid usually shows a solid lesion with high echogenecity1[13], a thickened cornea, poorly defined angle anatomy and possible iridocorneal touch.[10] Anterior chamber and lens structures are usually normal unless there is an associated heritable syndrome.[11] If the keloid arises secondary to penetrating trauma, iris incarceration into the wound may also be present.

HISTOPATHOLOGICAL EXAMINATION:

The diagnosis of most cases of corneal keloid has been made by pathologic examination rather than clinical examination.[11,17] The thickened epithelium may exhibit hyperplasia, keratinization, parakeratosis and basilar edema. Bowman's layer is usually disrupted or absent.[26] The stroma may contain irregularly arranged fibroblasts, collagen bundles, and vascular channels[7,10,15,16,19,23] along with activated fibroblasts, myofibroblasts and hyalized collage in later stages.[7,24,26] Neovascularization may be located anteriorly or diffusely throughout the stroma with the origin of the vessels from either the peripheral cornea or incarcerated iris.[17,24] The keloid from our patient had many of these findings, including hyperplastic epithelium overlying edematous, heavily collagenized fibrovascular tissue. The underlying Bowman's layer was disrupted and activated fibroblasts along with vascular channels were present within the keloid.

HISTOGENESIS:

The histogenesis of keloid may be divided into an early inflammatory stage followed by fibroblastic, fibrous and hyaline stages. Initially, there is increased vascualrity, hyperplasia of juvenile fibroblasts, and production of collage type III fibrils during the inflammatory and fibroblastic stages. During the fibrous stage, collage type I becomes tightly packed and there is a reduction in both vascularity and juvenile fibroblasts. Compact collagen stroma then fuses and becomes homogenous to form the the final hyaline stage.[7,24,26] It is believed that in keloids, myofibroblasts which are transformed fibroblasts that express smooth muscle phenotypes persist and often dominate the cell population. Eventually they deposit material that slowly collagenizes int eh center but does not retract. This produces a bulky slow growth of active fibroblasts and my ofibroblasts.[7,11,17,19,20] From the histopathologic findings of the diffuse fibrovascular tissue, vascular channels, heavy collagenization, and limited amounts of hyaline tissue in our patient, the keloid was in the late fibroblastic or fibrous stage of development

A/W CONGENITAL ANOMALIES:

Corneal keloids are also common with certain genetic syndrome. Lowe's syndrome, an X-linked recessive disorder characterized by hydrophthalmia, cataracts, intellectual disabilities, aminoaciduria, and vitamin D-resistance rickets, has been associated with congenital corneal keloids.[14,18,22,28] It is believed that in Lowe's syndrome, increased levels of amino acids such as tyrosine leaking from abnormal new corneal vessels or from within the anterior chamber through defective endothelium stimulate fibroblast proliferation.[14,20,22]Another genetic syndrome associated with corneal keloids is Rubinstein-Taybi syndrome(RTS). Rao and co-workers reported bilateral corneal keloids along with the RTS constellation of symptoms of broad thumbs and great toes, short stature, mental retardation, congenital heart defects, and characteristic faces.[10] Besides these two genetic syndromes, authors have reported that congenital corneal keloids are associated with ocular anomalies such as peripheral iridocorneal adhesions, subluxated lenses[21]; aniridia and cataract with anopthalmia.[15] In those idiopathic cases, it is postulated that there was a failure of normal corneal tissue differentiation during embryogenesis resulting in the occurrence of keloids and concomitant failure of the development of normal ocular tissue.[15,20,21] Congenital and genetic syndrome keloids appear to be a result of anterior-segment mesenchymal dysgenesis.

TREATMENT:

Successful treatment of corneal keloid has included superficial keratectomy, lamellar excision, lamellar keratoplasty or penetrating keratoplasty. Lamellar keratectomy is possible unless there is deep stromal involvement of the lesion.[18] Several authors have reported success with superficial lamellar keratectomy with amniotic membrane coverage.[11,13] Sclerokeratoplasty has also been performed successfully as an alternative procedure to preserve the globe when conventional keratoplasty could not be performed secondary to severe destruction of the cornea. Visual results from all the procedures are variable but anterior reconstruction of the eye is usually good.

Treatment goals are directed at removing the existing lesion and preventing recurrence by inhibiting fibroblastic proliferation and collagen synthesis. In the ultrastructural analysis of corneal keloids, it was noted that the persistence of mast cells promotes recurrent inflammation and utilizing topical steroids or mast cell stabilizers may be useful.[14,18] Other options that have been proposed include topical cyclosporine[22] or the use of physical forms of treatment such as ultrasound, cryotherapy, pressure therapy and laser.[18] The utilization of amniotic membrane after Superficial keratectomy has been successful in preventing recurrence and epithelial break down due to its antifibroblastic and anti-inflammotory properties.