Short Communication

Bilateral Moorens Ulcer

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A 36 year old male presented with chief complaints of foreign body sensation watering, redness, diminished vision ocular pain and photophobia in both eyes for last 2 months, On examination right eye revealed a perforated Mooren ulcer with uveal tissue herniation at 4 – 5 o'clock position. In both eyes, an undermined overhanging edge with grey white opacification and extending centrally and circumferentially could be appreciated. No hypopyon or AC cells could be discovered on examination, A diagnosis of bilateral Mooren's ulcer was made and systemic immunosuppressive therapy was started in terms of oral methotrexate 10 mg once a week and oral prednisone 1 mg / kg / day, Bandage Contact Lens was applied in right eye and conjunctival recession was performed.

Key Words: Moorens Ulcer, peripheral ulcerative keratitis

Ulcus rodens Corneae or Mooren's ulcer has been described as an agonizing, unrelenting, unabating ulcerative keratitis that initiates from peripheral cornea and then enlarges circumferentially and centrally. If primarily location of ulcer is at the periphery of the cornea and there is no separation, it becomes an indicator to investigate for collagen vascular diseases. The general ophthalmologist needs to be meticulous if the peripheral corneal disease has the following characteristics: if the epithelium is not intact, if there is some loss of stroma, if it is a real keratitis and inflammation is present, if white blood cells have infiltrated into the peripheral cornea. The fact to keep in mind is that PUK is a diagnosis of exclusion.

A 36 year old male presented with chief complaints of foreign body sensation watering, redness, diminished vision ocular pain and photophobia in both eyes for last 2 months. The symptoms were pronounced OD with excruciating pain since last two weeks. No history of ocular or systemic disease however could be elicited.

Ocular examination revealed a visual acuity of 6/9 OD and 6/6 OS. Ocular examination demonstrated a 360 degree circumferential zone of scarring and ectasia of the right cornea but no associated scleritis. Scarring and neovascularization encroached upon but did not obscure visual axis. 360 degree circumferential area of scarring and ectasia of cornea was also present in left eye.

On examination right eye revealed a perforated Mooren ulcer with uveal tissue herniation at 4-5 o'clock position. In both eyes, an undermined overhanging edge with grey white opacification and extending centrally and circumferentially could be appreciated. The pupil revealed peaking nasally OD due to impending uveal tissue herniation. No hypopyon or AC cells could be discovered on examination. Dilated Indirect ophthalmoscopy revealed no abnormality. There was no history of trauma or joint pain or systemic disease. Circumcorneal congestion was present. Extraocular movements were full OU. Posterior segment examination in both eyes was inconclusive.

A battery of investigations was conducted which included – Complete Heamogram with ESR, X-ray chest and small Joints, Urine routine and microscopy, VDRL, RA factor, ANCA (antinuclear cytoplasmic antibodies), ANA (antinuclear antibodies), HBSAG (hepatitis b surface antigen), SGPT, HCV (hepatitis c virus). Scraping of the ulcer were done, which was inconclusive. Finally a diagnosis of Bilateral Mooren's

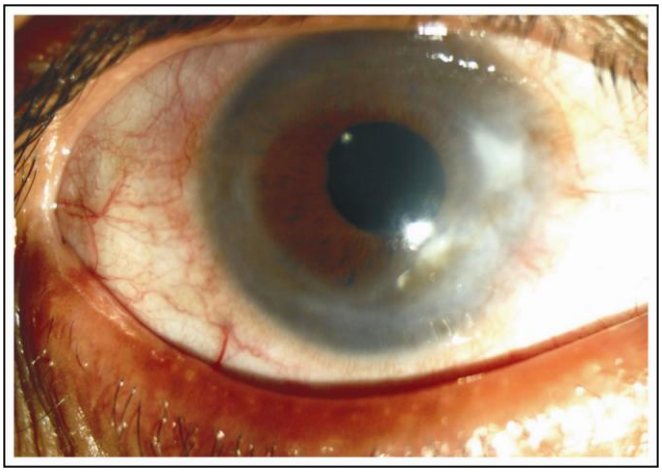

Fig. 1: 360 degree circumferential zone of scarring and ectasia of the left cornea.

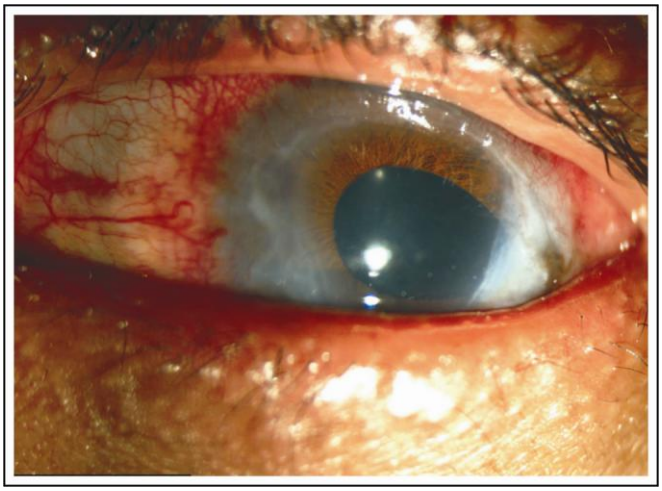

Fig. 2: 360 degree circumferential zone of scarring and thinning of right cornea but no associated scleritis with perforation at 4 -5 oclock, partially epithelised.

ulcer was made and systemic immunosuppressive therapy was started in terms of oral methotrexate 10 mg once a week and oral prednisone 1 mg / kg / day. Bandage Contact Lens was applied in right eye and conjunctival recession was performed.

DISCUSSION

Mooren's ulcer also known as chronic serpiginous ulcer of cornea has been defined as an entity with cascade of unknown events existing in absolute absence of any ocular infection or systemic rhematological diseases accountable for the ongoing devastation of the cornea. It has been recognized as an immensely destructive corneal lesion starting from corneal periphery and spreading centrally centrifugally and Posteriorly. Absence of scleritis is of substantial importance. Precise pathophysiology still remains unknown, although evidences suggest cell mediated and humoral immune mechanisms as a basis of pathogenesis. Though many modern approaches have been devised in step approach management of Moorens ulcer, notable amount of cases are recalcitrant to accessible treatments and end in severe visual morbidity.

Presenting complaints in Mooren's ulcer usually are redness, epiphora, and Photophobia, but excruciating agony out of proportion to inflammation is typically the prominent feature. Related iritis, central corneal involvement and irregular astigmatism due to peripheral corneal ectasia may lead to decreased visual acuity. The disease begins with many variegated, peripheral stromal infiltrates that merge later on, more frequently in the medial and lateral quadrants than in the superior and inferior ones. This is followed by formation of epithelial defect and a shallow furrow in this area[1].

Involvement of anterior one-third to one-half of the stroma occurs typically with a sloping, overhanging edge. This is followed by healing and vascularization with the lesion gradually taking its course in 4 – 18 months. Iritis, glaucoma, cataract and very rarely hypopyon are associated with Moorens ulcer[2].

The ulceration encompasses corneal periphery leaving a central island of oedematous opacified cornea else progresses transversely and relentlessly replaces stroma with thin scar tissue. Corneal perforation is a much common occurrence in MU leading to visual morbidity.

The adjacent scleral and DM tissue is largerly spared[3].

On the basis of clinical presentation and the low-dose anterior segment fluorescein angiographic findings, Mooren's ulcer has been classified into three main types: Unilateral Mooren's ulcer is usually recognized as excessively painful progressive keratitis in elder age group in this variety of MU superficial vascular plexus of the anterior segment remains non-perfused. Bilateral aggressive Mooren's ulceration (BAM), clinical entity more frequent in young individuals, progresses circumferentially and, only later, centrally in the cornea. Angiography shows

vascular leakage and new vessel formation at the base of the ulcer.

Bilateral indolent Mooren's ulceration (BIM) common in middle-age group patients results in progressive peripheral corneal guttering in both eyes, with minimal inflammatory response. vascular architecture is usually normal on angiography with an exception that extension of new vessels into the ulcer may be noted[4,5].

If one uses a fine instrument to explore the ulcer, one will find that it has an overhanging lip; if this fine instrument is exploring the ulcer toward the center of the cornea, one can be absolutely astonished at how far into the cornea the instrument can go before it meets resistance. In other words, there's a lot more destruction than is clinically apparent at the slit lamp.

The systemic evaluation should not discover that the patient has an elevated C-reactive protein, an elevated sedimentation rate, auto-antibody production of any type but particularly antineutrophil cytoplasmic antibody."

Moorens ulcer has been linked with different systemic entities including toxoplasma, hepatitis B and C, herpes simplex and zoster, syphilis, TB and intestinal hookworm[1,2] histopathology of Mooren's ulcer reveals increased number of antigen-presenting cells, mast cells and immunoglobulins[6]. Bilateral Mooren's ulcer as seen in our case is frequently found in Indian subcontinent and in patches of West Africa. The age group most commonly involved is 14 – 40. They may present with unilateral typical lesion in one eye but soon may develop lesion in the other eye. Angiography reveals altered architecture of episcleral vessels along with some areas of closure. There are no changes in conjunctiva but the angiogram reflects a breakup of the limbal arcade, extension of the vessels into the ulcer bed and leaky vessel tips. This variety has a tendency to perforate spontaneously and if not paid attention can lead to significant visual morbidity[7].

CONCLUSION

Bilateral Mooren's ulcer in young population needs to be given an immediate attention as visual morbidity remains significant in this variety**.** Only if the aggressive systemic evaluation turns out inconclusive and the adjacent sclera is not involved, it's appropriate to hang the label Mooren's ulcer. Aggressive systemic immunomodulatory medication is absolute if cause of patient's PUK is discovered to be a consequence of polyarteritis nodosa or microscopic polyangiitis and granulomatosis with polyangiitis.

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Dr. Anuj Sharma
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Dr. Ravinder K Gupta
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