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The use of oral doxycycline and vitamin C in the management of acute corneal hydrops: a case comparison

Abstract- We compare two patients presenting to clinic with an uncommon complication of keratoconus, acute corneal hydrops. Management of the patients differs. One heals quickly, while the other has a delayed course to resolution.

I. Case A
   a. Demographics: 40 yo AAM
   b. Case History
      i. CC: red eye, tearing, decreased VA x 1 day OS
      ii. POHx: (+) keratoconus OU
      iii. PMHx: depression, anxiety, asthma
      iv. Meds: Albuterol, Ziprasidone
      v. Scleral CL wearer for approximately 6 months OU
      vi. Denies any pain OS, denies previous occurrence OU, no complaints OD
   c. Pertinent Findings
      i. VA cc (CL’s)- 20/25 OD, 20/200 PH 20/60+2 OS
      ii. Slit Lamp
         1. Inferior corneal thinning and Fleisher ring OD, central scarring OD, 2+ diffuse microcystic edema OS, Descemet’s break OS (photos and anterior segment OCT)
         2. 2+ diffuse injection OS
         3. D&Q A/C OU
      iii. Intraocular Pressures: deferred OD due to CL, 9mmHg OS (tonopen)
      iv. Fundus Exam- unremarkable OU

II. Case B
   a. Demographics: 39 yo AAM
   b. Case History
      i. CC: painful, red eye, tearing, decreased VA x 1 day OS
      ii. POHx: unremarkable
      iii. PMHx: hypertension
      iv. Meds: unknown HTN medication
      v. Wears Soflens toric CL’s OU; reports previous doctor had difficulty achieving proper fit OU; denies diagnosis of keratoconus OU
      vi. Denies any injury OS, denies previous occurrence OU, no complaints OD
c. Pertinent Findings
   i. VA cc (spectacles) 20/25 OD, 20/200 PH 20/150 OS
   ii. Slit Lamp
      1. 1+ inferior papillae OU
      2. Mild inferior corneal thinning OD, diffuse corneal haze OS, 2+ microcystic edema OS, 1+ bullae OS, Descemet’s break OS (photos and anterior segment OCT)
      3. 2+ diffuse injection OS
      4. D&Q A/C OU
   iii. Intraocular Pressures: 12 mmHg OD, 13mmHg OS (tonopen)
   iv. Fundus Exam- unremarkable OU

III. Differential Diagnosis
   a. Leading
      i. Acute corneal hydrops OS
         1. A break in Descemet’s membrane allows fluid to enter the cornea causing severe corneal edema.
   b. Other
      i. Fuch’s corneal endothelial dystrophy
         1. A progressive, bilateral corneal disorder caused by a reduction in number of endothelial cells. The lack of endothelial cells causes corneal edema.
      ii. Posterior polymorphous corneal dystrophy
         1. A rare, autosomal dominant corneal dystrophy characterized by abnormal vesicular lesions at the level of Descemet’s membrane and the endothelium. Rarely, this can cause corneal edema.
      iii. Iridocorneal endothelial syndrome
         1. A rare eye condition composed of a triad of corneal edema, heterochromia and glaucoma.
      iv. Infectious keratitis
         1. An infection of the cornea usually from contact lens wear or trauma. Presents with pain, reduced vision, light sensitivity and tearing or discharge from the eye.

IV. Diagnosis/Discussion
   a. Corneal Hydrops OS
      i. Relatively uncommon, occurs in 2.6-2.8% of patients with keratoconus (incidence of keratoconus estimated to be 1:2000)
      ii. Mean age of onset is 25 years of age, more commonly seen in males
      iii. Increased prevalence in patients with corresponding allergic eye disease, vernal keratoconjunctivitis, eye rubbing, Down syndrome
      iv. Symptoms – pain, photophobia, reduction in VA, and epiphora
v. Signs – reduced VA, conjunctival injection, severe corneal edema, evidence of underlying corneal ectasia

vi. Etiology
1. Weak corneal collagen leads to a break in Descemet’s membrane allowing fluid from the aqueous to invade the cornea causing severe corneal edema.

vii. Pathophysiology
1. For years keratoconus has been labeled a “non-inflammatory” disease based on the lack of neovascularization and infiltrative response. Several studies have shown elevated levels of pro-inflammatory cytokines such as IL-6 and TNFα in tears of keratoconic patients. Tissue degrading enzymes such as MMP-9’s have also been higher in tears from keratoconic eyes.
   a. The pathophysiology of keratoconus may be related to an imbalance between pro-inflammatory and anti-inflammatory cytokines.
2. New research shows anterior hyper-reflective cells in Bowman’s layer prior to the development of corneal hydrops. The hyper-reflective cells correspond to fibrillar degeneration and fibroblastic cells, which suggests an inflammatory component to corneal hydrops.

viii. Corneal hydrops is self-limiting over a period of 2-4 months. Medical therapy intends to improve comfort and reduce risk of complications such as corneal perforation or infection.
1. Treatment is often a combination of topical antibiotics to prevent potential infection from ruptured bullae, topical hypertonic solutions to reduce swelling, topical cycloplegics for pain management and copious artificial tears to help with surface irritation.

ix. Long-term therapy aims to improve visual function with either rigid CL’s or keratoplasty depending on the degree of visual impairment.

V. Treatment/Management
a. Case A
i. Treatment
1. Initial – Polytrim qid OS, Muro 128 ung tid OS, PF AT’s q1h OS, 50 mg of doxycycline bid po, 1 tablet of 1000mg vitamin C/day po
2. Day 7 – Same as initial with addition of Pred Forte qid OS
3. Day 21 – Same as day 7 with discontinuation of Polytrim qid OS
4. Day 28 – Same as day 21
ii. Response
   1. Improvement of symptoms day 7
   2. Resolved Descemet’s break and significant improvement to microcystic edema day 7 (photos and anterior segment OCT)
   3. Evidence of scarring and 1+ corneal haze day 28

b. Case B
   i. Treatment
      1. Initial – Ciprofloxacin qid OS, Muro 128 gits qid OS, Cyclopentolate bid OS, PF AT’s q1h OS
      2. Day 3 – Same as initial
      3. Day 8 – Same as initial with addition of Muro 128 ung qhs OS and reduction of Cyclopentolate to prn OS
      4. Day 14 – Same as day 8
      5. Day 21 – Same as day 8 with addition of 50 mg of doxycycline bid po
   ii. Response
      1. Improvement of symptoms day 6
      2. Mild improvement of bullae and corneal edema, although still significant day 17 (photos and anterior segment OCT)
      3. Significant improvement of bullae and corneal edema day 21 (photos and anterior segment OCT)

c. Case comparatives
   i. Both patients presented with acute corneal hydrops
   ii. Both cases were treated with a combination of topical antibiotics, topical hypertonic solutions and copious preservative free artificial tears
   iii. Case A was also initially treated with oral doxycycline and oral vitamin C
   iv. Case A was started on topical steroids on day 7
   v. Case A resolved quicker than case B
   vi. Case B demonstrated poor compliance with medications and follow up

VI. Conclusion
   a. Medical therapy for corneal hydrops aims to improve comfort, decrease complications and expedite recovery time.
   b. New research suggests a potential inflammatory component to keratoconus and corneal hydrops.
      i. As demonstrated with Case A, it is possible that treatments such as oral doxycycline and vitamin C may quicken corneal healing.
      ii. Oral doxycycline inhibits activity of MMP-9’s and has anti-inflammatory properties. Doxycycline is beneficial in the treatment of recurrent corneal erosions as it promotes corneal
healing. Oral doxycycline may facilitate corneal recovery in conditions like acute hydrops as well.

iii. Vitamin C is an excellent anti-oxidant. Topical and oral vitamin C reduced the degree of opacification in cases of infectious keratitis. Vitamin C may also diminish scarring from corneal hydrops.

c. Patients must understand the importance of long-term therapy and slow healing time with corneal hydrops.

d. Corneal scarring with the resolution of corneal hydrops flattens the cornea, and both patients will require a new CL fit once resolved.

VII. References


