Red eye 2

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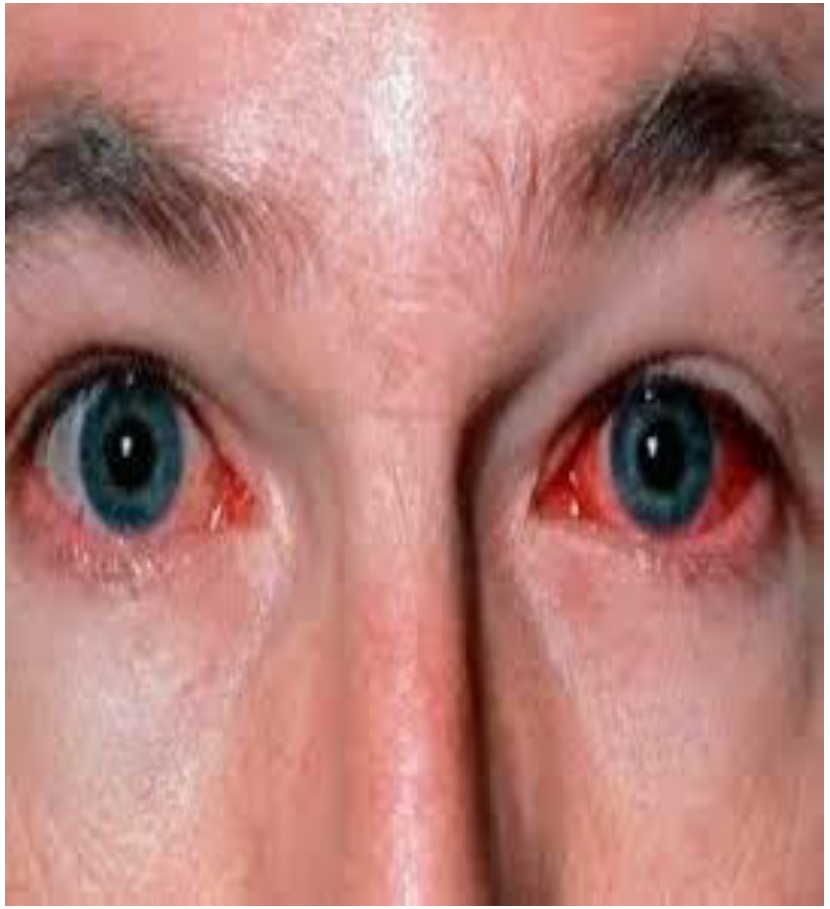
Scenario

- A 35 yrs male patient comes to opd with redness irritation for the last few days . His vision is 6/6 Bes. He is having watery discharge. Many people are affected In the area. What is most probable cause.?
- A acute bacterial conjunctivitis
- B acuta allergic conjunctivitis
- C acte viral conjunctivitisd
- D vernal conjunctivitis

- Dx C

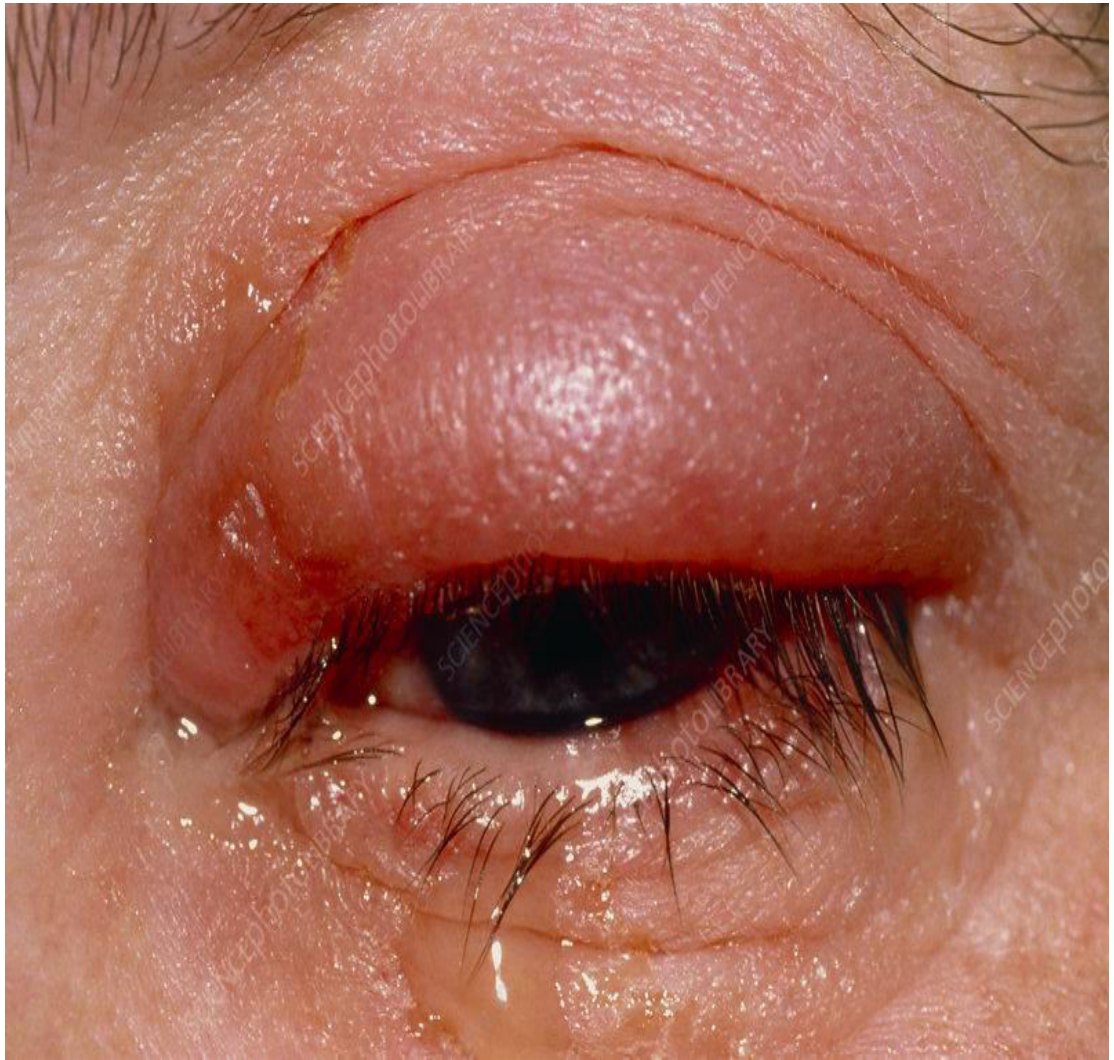
Viral Epidemic keratoconjunctivitis(EKC)

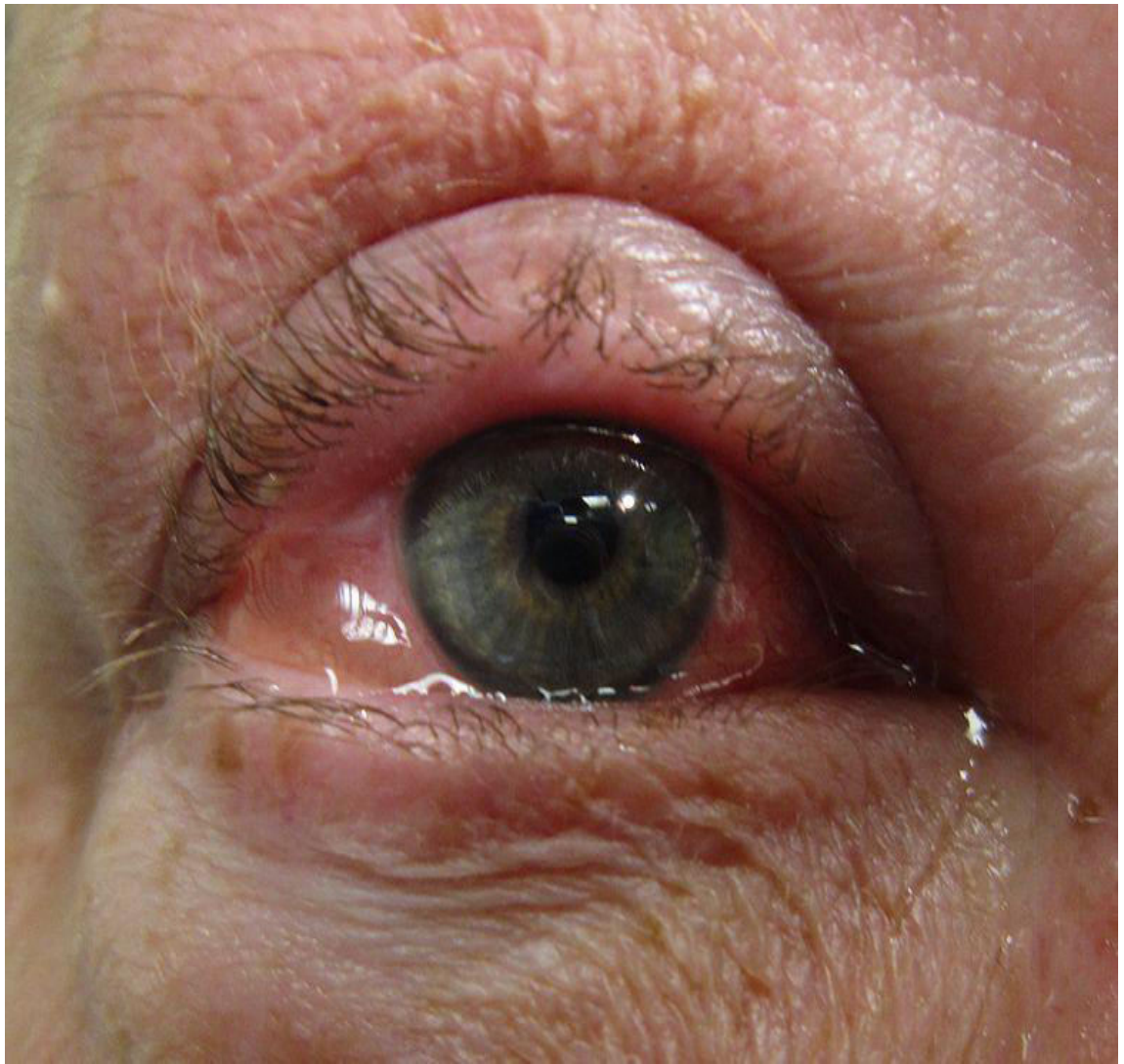
Viral conjunctivitis is a highly contagious acute conjunctival infection usually caused by an adenovirus type 8,19 &37. highly infectious .
Incubation period 7-8days
Symptoms include irritation, photophobia, and watery discharge.
Diagnosis is clinical; sometimes viral cultures or immunodiagnostic testing is indicated.
Infection is self-limited, but severe cases sometimes require topical corticosteroids.



Clinical features

- After an incubation period of about 5 to 12 days, conjunctival hyperemia, watery discharge, and ocular irritation usually begin in one eye and spread rapidly to the other.
- Transmission through conjunctival secretion
- Follicles may be present on the palpebral conjunctiva.
- A preauricular lymph node is often enlarged and painful. Many patients have had contact with someone with conjunctivitis, a recent upper respiratory infection, or both.





- In severe adenoviral conjunctivitis, patients may have photophobia and foreign body sensation due to corneal involvement (SPK).
- Lid edema & Chemosis may be present.
- Pseudo-membranes of fibrin and inflammatory cells on the tarsal conjunctiva, focal corneal inflammation, or both may blur vision. Even after conjunctivitis has resolved, residual corneal subepithelial opacities (multiple, coin-shaped, 0.5 to 1.0 mm in diameter) may be visible with a slit lamp for up to 2 years. Corneal opacities occasionally result in decreased vision and significant halos and starbursts.

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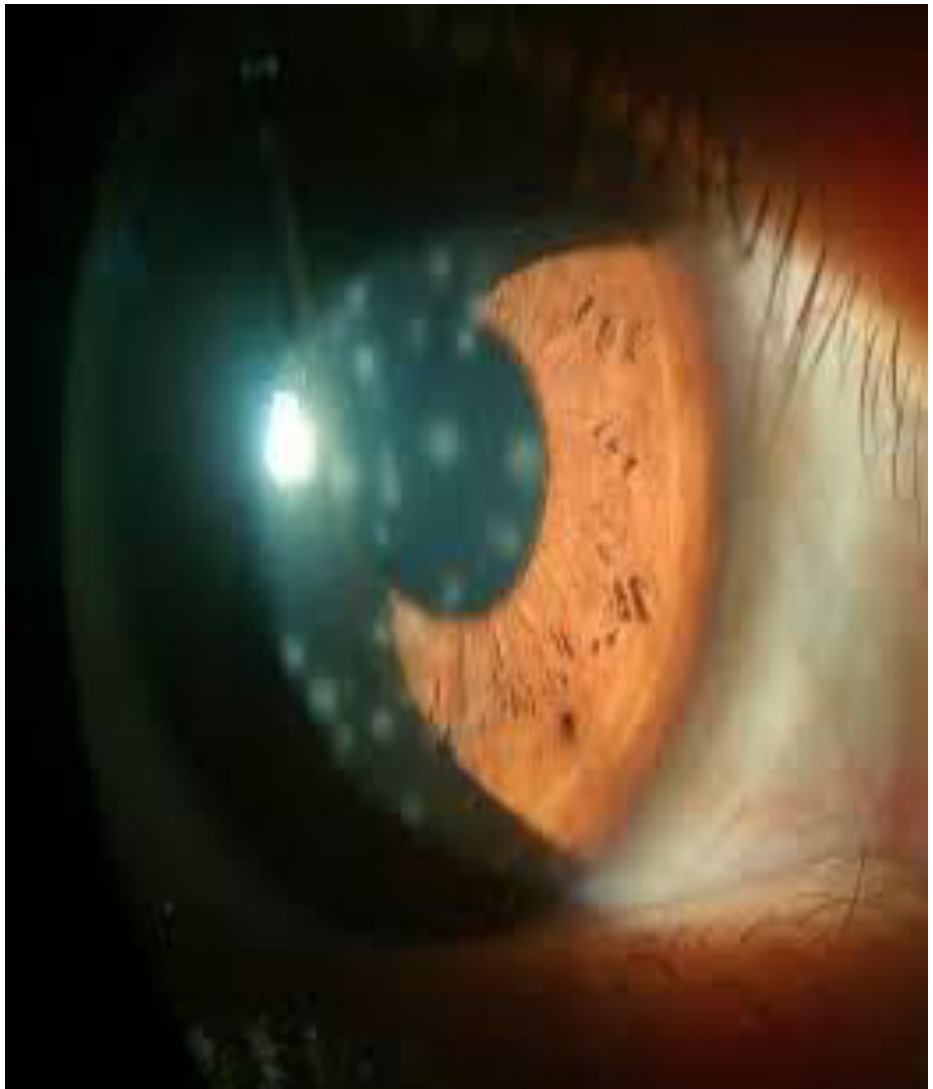


Diagnosis

- Features that may help differentiate between viral and bacterial conjunctivitis can include purulence of (a) ocular discharge, (b) presence of preauricular lymphadenopathy, and, in epidemic keratoconjunctivitis (c) flu like systemic symptoms, (d) chemosis.
- Patients with photophobia are stained with fluorescein and examined with a slit lamp.
- Epidemic keratoconjunctivitis may cause punctate corneal staining.
- Secondary bacterial infection of viral conjunctivitis is very rare. However, if any signs suggest bacterial conjunctivitis (eg, purulent discharge), cultures or other studies may be useful.

Treatment of viral Conjunctivitis

- **Preventive &Supportive measures**
- Viral conjunctivitis is highly contagious, and transmission precautions must be followed.
- To avoid transmitting infection, **physicians** must
- Use hand sanitizer or wash their hands properly (fully lather hands, scrub hands for at least 20 seconds, rinse well, and turn off the water using a paper towel)
- Disinfect equipment after examining patients

Contnd

- **Patients** should do the following:
- Use hand sanitizer and/or wash their hands thoroughly after touching their eyes or nasal secretions
- Avoid touching the noninfected eye after touching the infected eye
- Avoid sharing towels or pillows
- Avoid swimming in pools
- Eyes should be kept free of discharge and should not be patched. Small children with conjunctivitis should be kept home from school to avoid spreading the infection.
- Should sleep on the affected side

Topical antibiotic steroid combination

- Viral conjunctivitis is self-limiting, lasting 1 week in mild cases to up to 3 weeks in severe cases. It requires only cool compresses for symptomatic relief.
- ***Topical*** However, patients who have severe photophobia or whose vision is affected may benefit from topical corticosteroids (eg, 1% prednisolone acetate 4 times a day). Corticosteroids, if prescribed, are usually prescribed by an ophthalmologist.
- keratitis must be ruled out first (by fluorescein staining and slit-lamp examination) because corticosteroids can exacerbate it. Topical cyclosporin A eye drops are effective but are helpful if corticosteroid drop use is limited by adverse effects.

- Topical antibiotic for any superadded bacterial infection
- ***Sytemic***
- Antihistamine, antibiotics & sun glasses for photophobia if there.

Type	Reaction	Itching	Discharge	Lymphadenopathy	Fever sorethroat
Viral	Follicular	Minimal	Watery	Common	Present
Bacterial **Non-Gonococci** **Gonococcal**	Papillary	Minimal	Purulent Mucopurulent Hyper-Purulent	Uncommon	May or may-not
Chlamydial	Follicular	Minimal	Mucopurulent	Common	No
Allergic	Papillary	Severe	Watery mucoid	None	No

Allergic Conjunctivitis(Non-infective)

- Seasonal allergic conjunctivitis
- Perennial allergic conjunctivitis
- Vernal keratoconjunctivitis
- Atopic keratoconjunctivitis
- Giant papillary conjunctivitis
- Keratoconjunctivitis sicca (dry eyes)
- Superior limbic keratoconjunctivitis (SLK)

. Chemical or irritative conjunctivitis

Vernal keratoconjunctivitis

- **Introduction**
- Vernal keratoconjunctivitis(VKC) is an important sight-threatening, chronic, inflammatory disease of the cornea and conjunctiva characterized by recurrent flare-ups of ocular surface inflammation, causing intense ocular symptoms of itching, redness, and photophobia associated with corneal damage and impairment of visual function and quality of life.
- VKC is a clinical form of allergic conjunctivitis diseases, together with seasonal and perennial allergic conjunctivitis (AC) and atopic keratoconjunctivitis (AKC) .

- In fact, VKC shares some clinical features and pathogenic mechanisms with other forms of AC, including ocular itching, swelling, redness, and conjunctival papillary reaction associated with immunoglobulin (Ig) E-mediated release of histamine and other allergic reaction mediators from mast cells.
- However, this is likely not the only mechanism involved in VKC immunopathogenesis, as only 50% of cases of VKC show allergic sensitization. Several studies have demonstrated that the inflammatory reaction occurring in VKC also involves a lymphocyte T-helper (Th) type 2-driven reaction, a late-phase allergic reaction with eosinophil infiltration, and extracellular matrix remodeling
- In addition, the demographical, geographical, and clinical characteristics of VKC suggest that other endocrine, environmental, and/or genetic factors may play a role in the pathogenesis of this challenging condition.

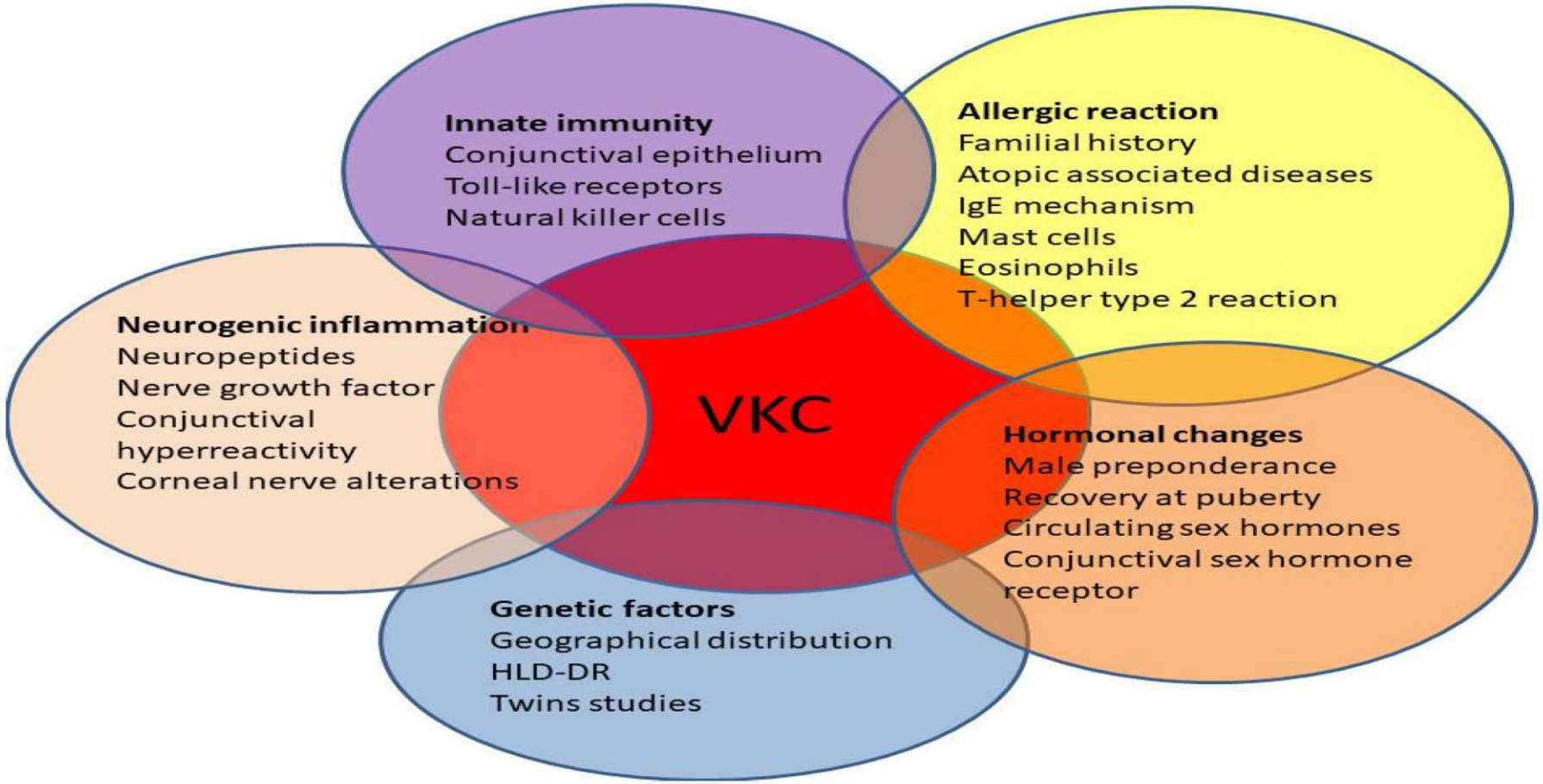
- Is an allergic conjunctivitis to an exogenous allergan which act as an antigen
- It’s a type I & type IV hypersensitivity reaction.
- Age. 5-15 yrs
- Sex. male are common as female
- Season. common in summer & spring
- Family Hx. 2/3 have family Hx

- Specifically, VKC mostly affects children, with higher propensity in boys than girls, and, in most cases, spontaneously resolves after puberty, suggesting that an imbalance of sex hormones may play a role in its pathogenesis.
- A higher prevalence of the condition is observed in warm regions, such as the Mediterranean area, Central and South America, Japan, Central and West Africa, and the Middle East, which also suggests a potential pathogenic role of genetic and/or environmental factors.
- Finally, recent studies point to a possible role of innate immunity, including toll-like receptors (TLRs) and natural killer (NK) cells, in the development and severity of

Pathology

- Exogeneous allergan act as an antigan & induce the immunoglobulin production (IgE).
- Antigen antibody reaction occur over the surface of the mast cell- causes degranulation – release of chemical mediators- serotonin, histamine & slow release substance P- causes inflammation in conjunctiva & cornea

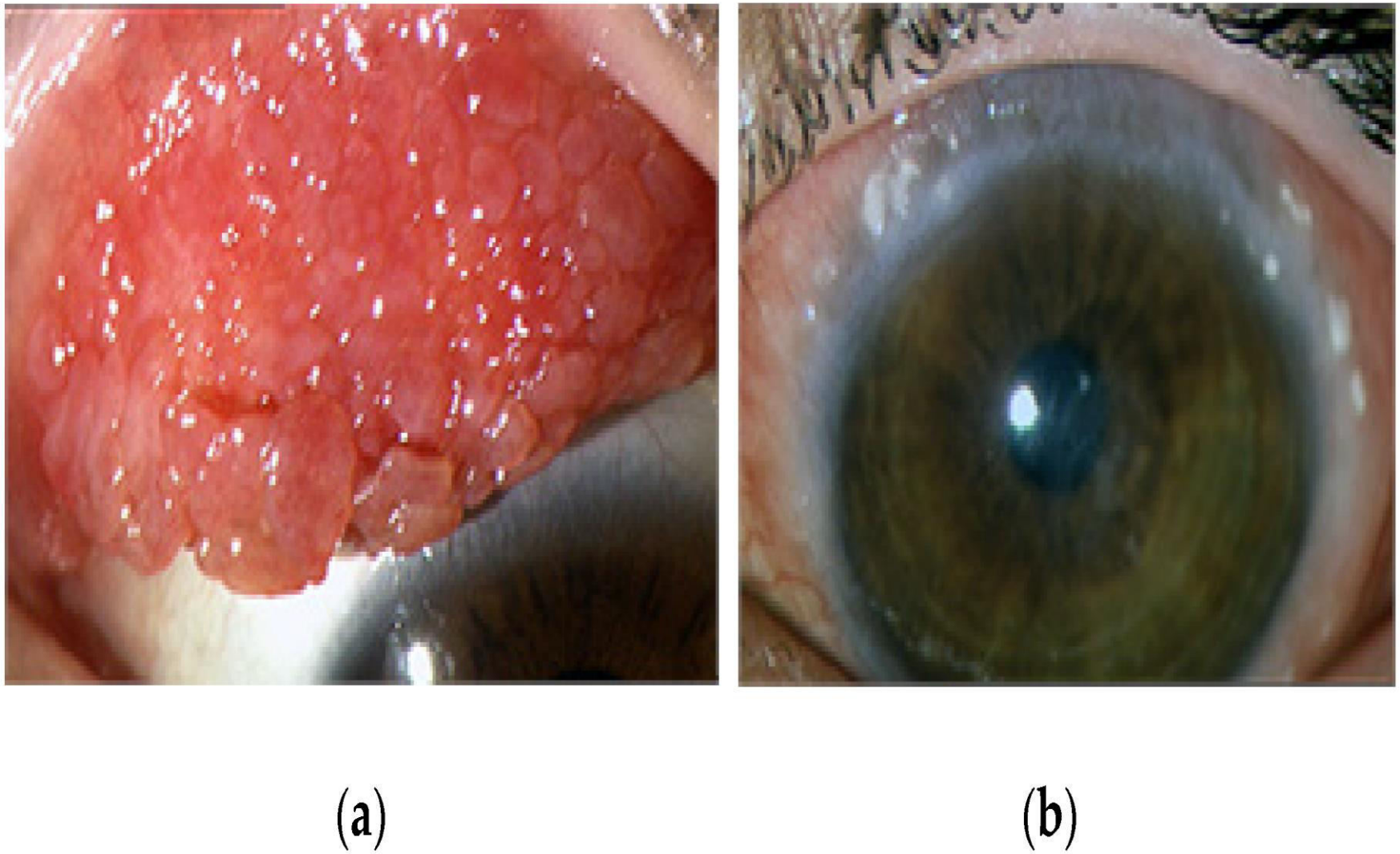
Figure. Allergic reaction represents the main pathogenic factor of VKC; however, several sources of evidence show that innate immunity and neuroinflammatory response, as well as genetic, hormonal, and environmental factors, also participate in the development and severity of VKC



Types

- Three types
- A clinical characteristic sign of VKC is the presence of a giant conjunctival papillary reaction of the upper tarsal conjunctiva and/or limbal region resulting from chronic inflammation and extracellular matrix remodeling.
- Palpebral/tarsal VKC is characterized by giant hypertrophic papillae at the upper tarsal conjunctiva with a cobblestone appearance,
- Bulbar/ limbal form is characterized by gelatinous infiltration around the cornea. in which the bulbar conjunctiva is mainly involved
- Mixed type in which both the tarsal & bulbar conjunctiva are involved & shows papillary reactions.

- Vkc
- A palpebral type
- B Bulbar type

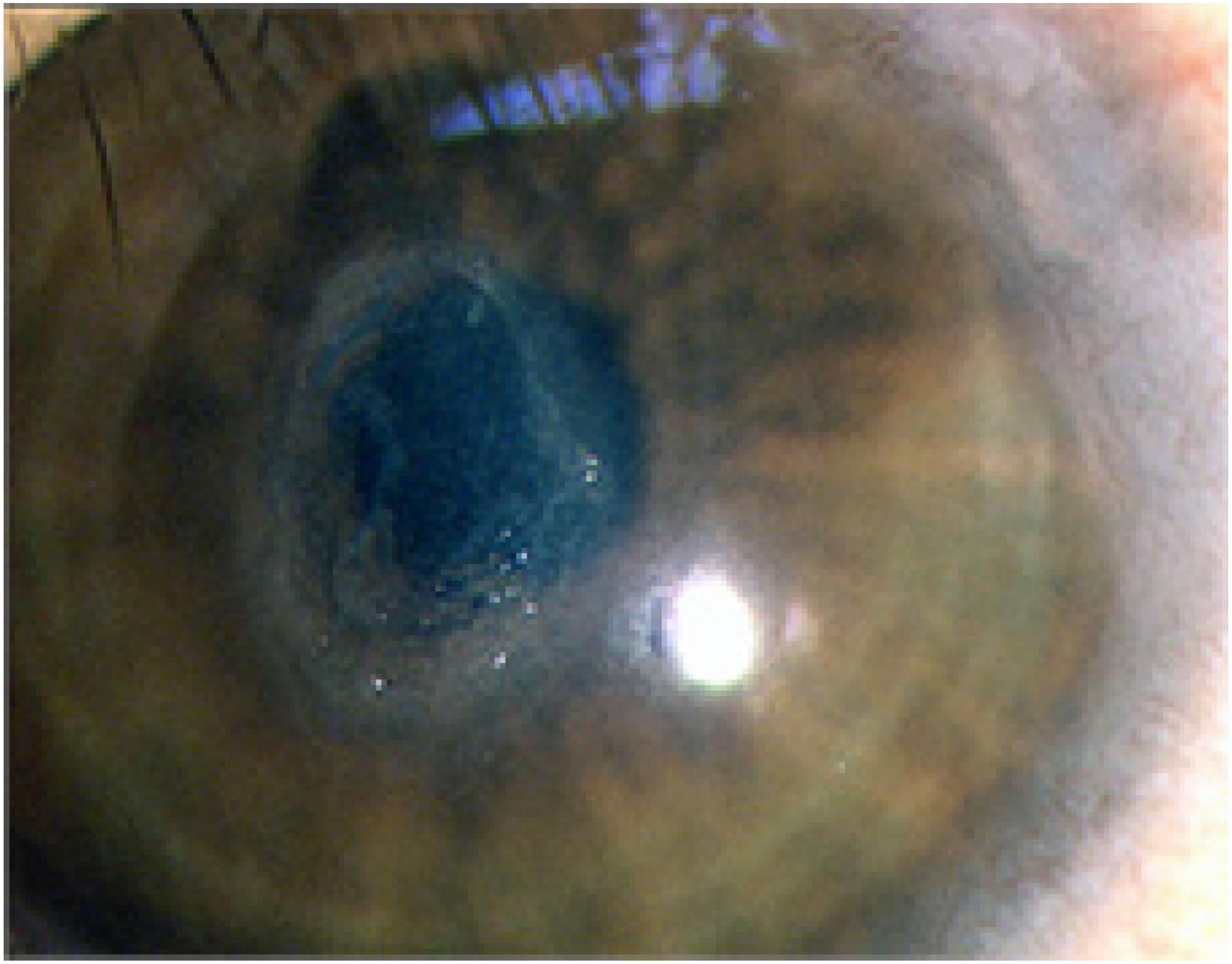


(a) (b)

- Horner-Trantas dots surrounding the cornea represent a typical sign of active inflammation and are mostly due to eosinophil infiltration.
- Corneal epithelial defect
- More severe cases, with long-standing disease, may develop corneal neovascularization and scarring, associated with permanent impairment of visual function

- Corneal ulcers are
- reported in 3–11%
- of cases and may
- cause pain and
- impairment

of visual function



Management

- A challenge for ophthalmologists.
- Cold Compressing
- **Antiallergic** eye drops such as antihistamines and/or mast cell stabilizers are effective only in very mild cases.
- Mast cell stabilizer such as sodium cromoglicate, lodxamide
- Antihistamine such as emedastine, epinastine
- **Topical steroids** The majority of patients in the active phase of ocular inflammation require the use of topical steroids and/or other immunosuppressive drugs. Topical steroid are very effective in controlling the signs and symptoms of active disease. However, their chronic use is associated with the development of severe ocular complications, such as glaucoma and cataract.

- **Immunosuppressive,** Therefore, steroid sparing agents, such as topical cyclosporine A (immunosuppressive) (CsA) or topical tacrolimus another immunosuppressive +antibiotic, are currently used for the chronic treatment of VKC
- Drops & ointment form are used
- **Supratarsal steroid injection,** in resistant & non-complianace condition is given . Dexa & triamcinolone
- Systemic antiallergic & steroid can be added

Complications

- Keratoconus
- Glaucoma may be due to steroid use
- Cataract formation may be due to steroid use
- Corneal defect like shields ulcer, scaring, vacularization

Chronic conjunctivitis

- Trachoma

Poor hygiene

overcrowding

Scenario

- A female patient of 17 yrs comes to opd. She is complaining of redness, discharge for the last 2-3months. Her vision is 6/6 Bes. There is mild conjunctival congestion with follicles on lid eversion. She took many treatment but no relief. On Hx she has poor background with many family members lives in small house. What is the most probable cause.?
- A acute allergic conjunctivitis
- B acute bacterial conjunctivitis
- C acute viral conjunctivitis
- D trachomatous conjunctivitis

- Dx D

Chlamydial conjunctivitis; Trachoma

- It is a chronic conjunctivitis caused chlamydia trachomatis
- It is usually bilateral, may be unilateral
- Caused by Chlamydia Trachomatis type A,B, & C
- Incubation period is 5-12 days
- Common in poor communities with overcrowding & poor hygiene
- Contagious with personal contacts
- Spread through conjunctival secretion
- Leading cause of preventable blindness

Transmission

- *C. trachomatis* spread through direct contact. Infected young children serve as a reservoir of infection.
- The bacteria are then transmitted by close physical contact with family members and other caregivers.
- The bacteria are also spread through shared blankets, pillows, and towels. The
- Bazaar fly *Musca sorbens* lays its eggs in human feces that can be contaminated with trachoma bacteria.
- These flies pick up bacteria on their bodies and can transmit them to humans.

- ***Certain conditions promote the spread of trachoma bacteria. These include:***
- a) poor personal hygiene
- b) poor body waste and trash disposal
- c) insufficient water supply for washing
- d) shared sleeping space with dirty hands and common face towels & clothes/shawls
- e) close association with domestic animals

The Life Cycle of Trachoma

INFECTING THE EYES

Flies carrying the micro-organism land on children's eyes, to feed on discharge.

FAMILY CONTACT

Women who take care of children also get the infection.

SPREADING OUT

Flies that breed in human feces spread the disease to others.

Dirty hands or face cloths also spread the disease.

HOW TRACHOMA BLINDS

Infections inflame and thicken the upper eyelid.

Scarred eyelids turn inward.

The lashes scratch the cornea, leading to blindness.

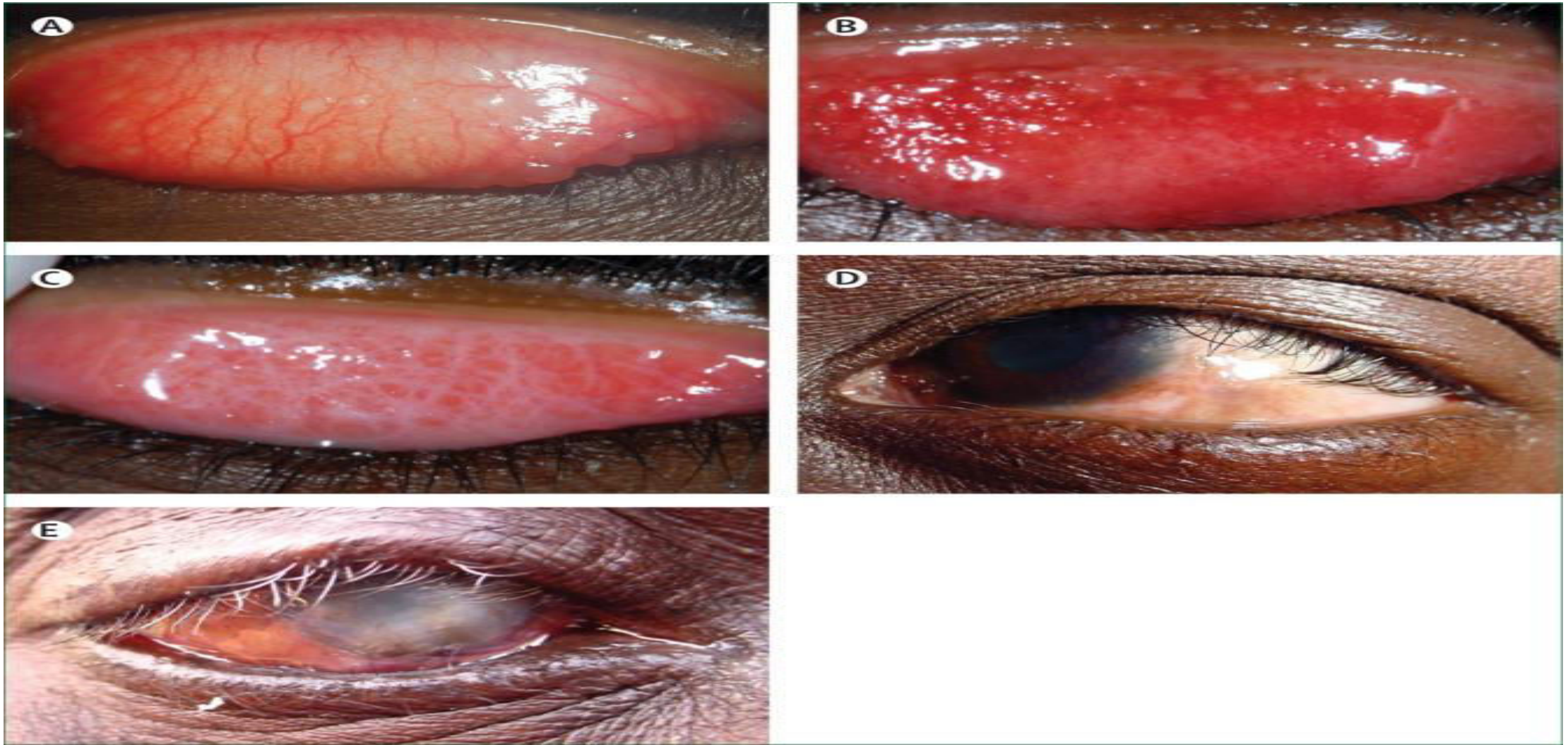
Sources: The Carter Center, International Trachoma Initiative

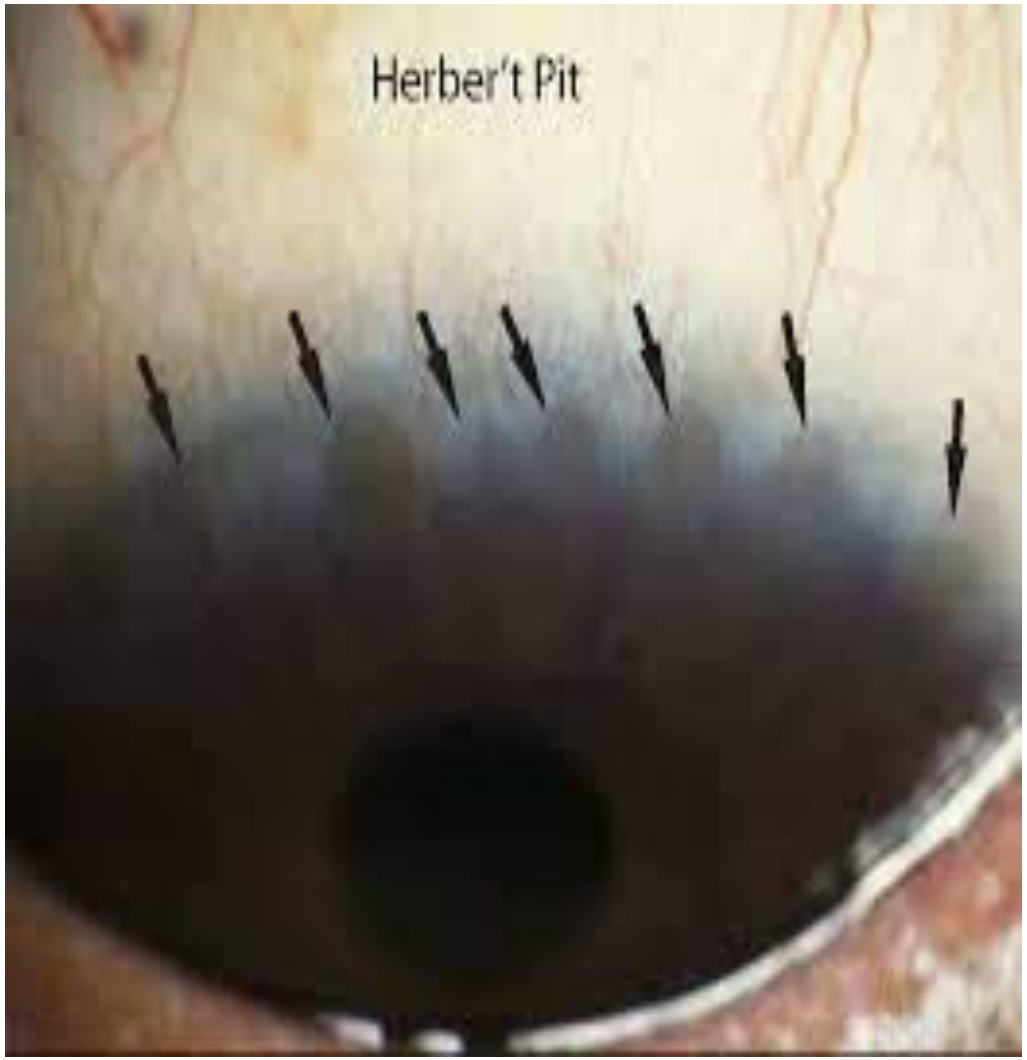
Al Granberg/The New York Times

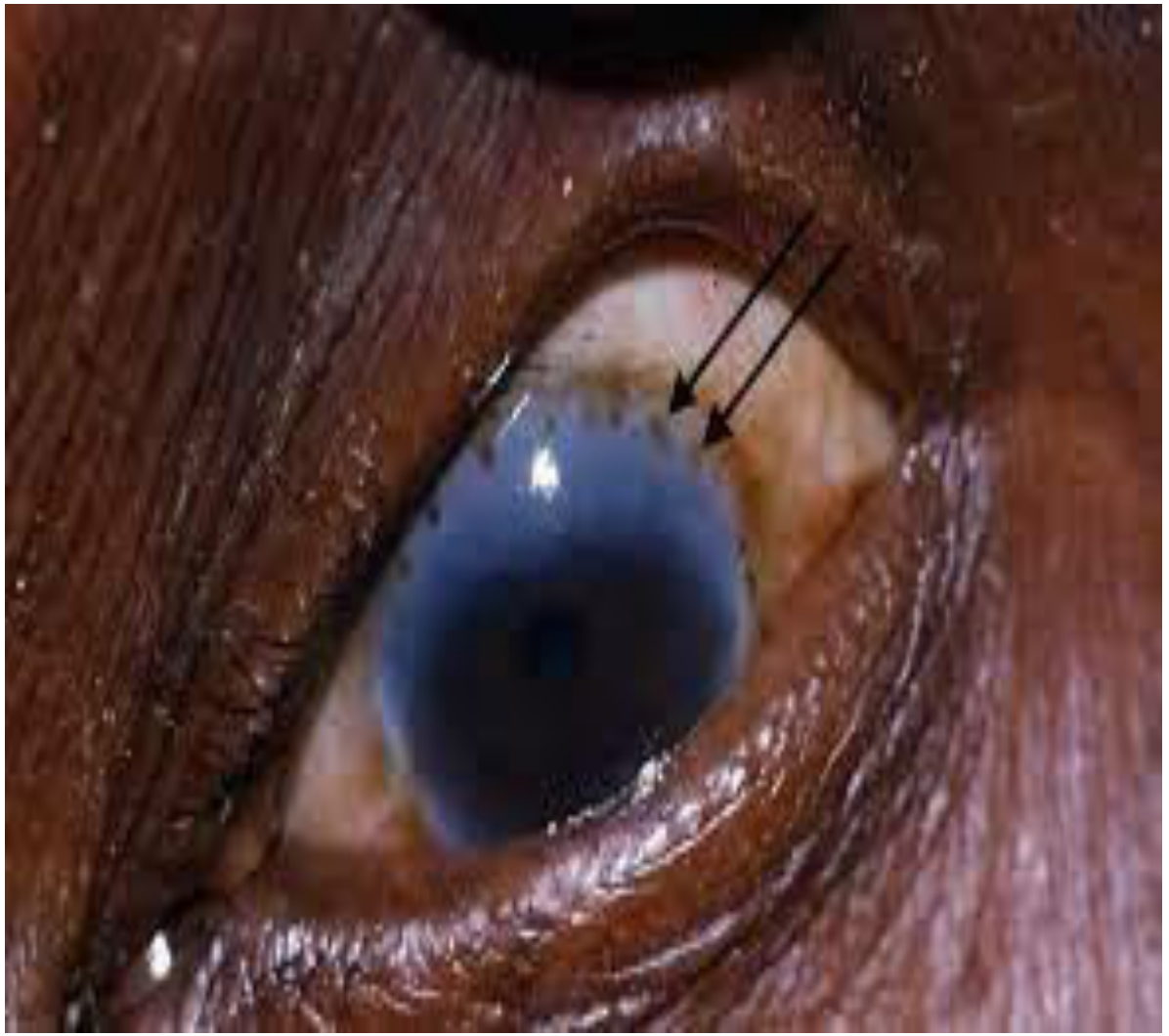
Clinical feature (WHO 1987) (FISTO)

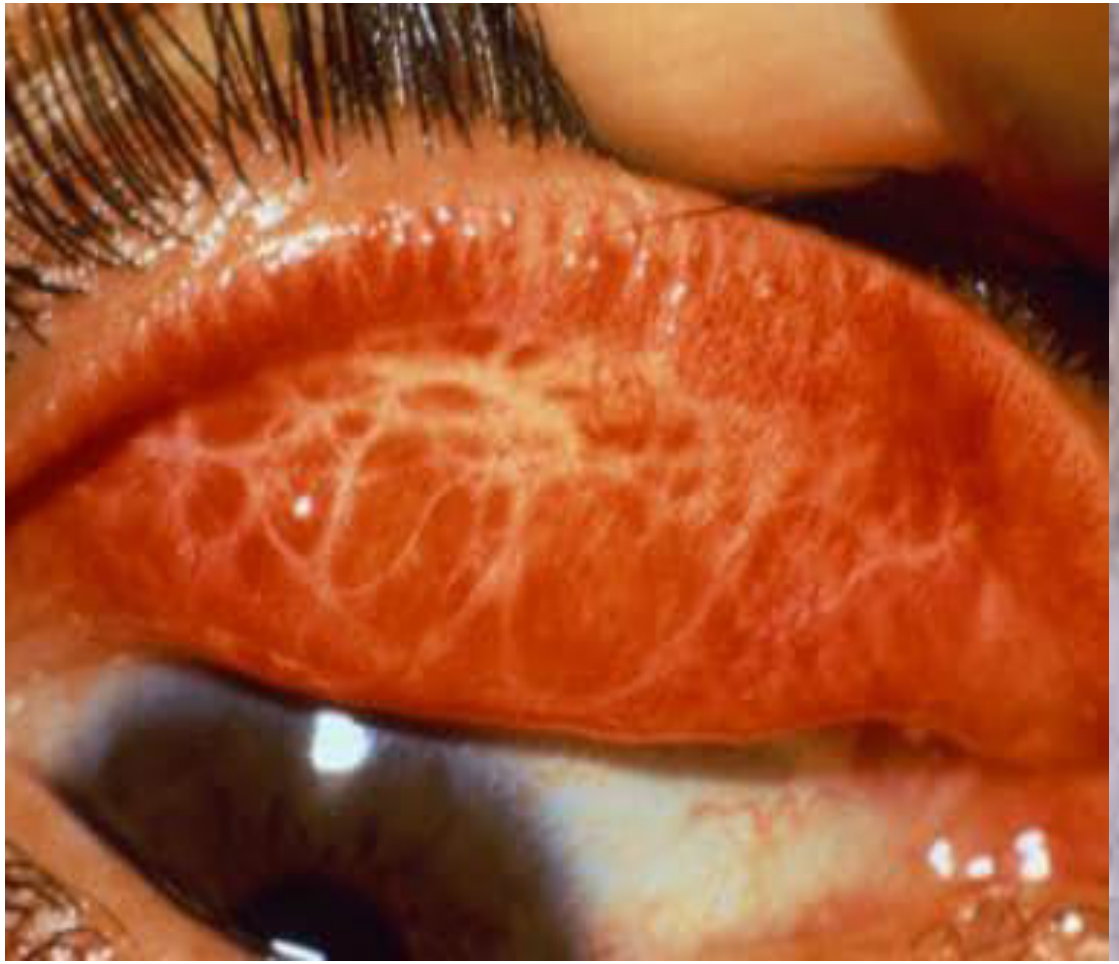
- According to grading important
- 1. *Follicular* stage 0.5mm or more
- 2. *Inflammatory* stage with intense inflammation of the tarsal conjunctiva with more than 50% vessel obscuration
- 3. Conjunctival *Scarring*
- 4. *Trichiasis* is the stage in which eyelashes becomes misdirected touching the cornea
- 5. Scarring stage with *corneal opacity* formation leading to blurring and loss of vision

Clinical features of trachoma. (A) Active trachoma in a child with follicle formation(TF) (B) intense inflammation (TI). (B) Tarsal conjunctival scarring (TS). (C) conjunctival scarring (TS) (D) Entropion and trichiasis (TT). (E) corneal opacity CO with entropion and trichiasis (TT).




Herber't Pit





Treatment (SAFE) policy

- Surgery of the complications
- Antibiotic use topical & systemic
- Face wash means improve hygienic condition
- Environmental improvement , to increase cleanliness, water supply, to improve poverty and living condition and sanitation of the public

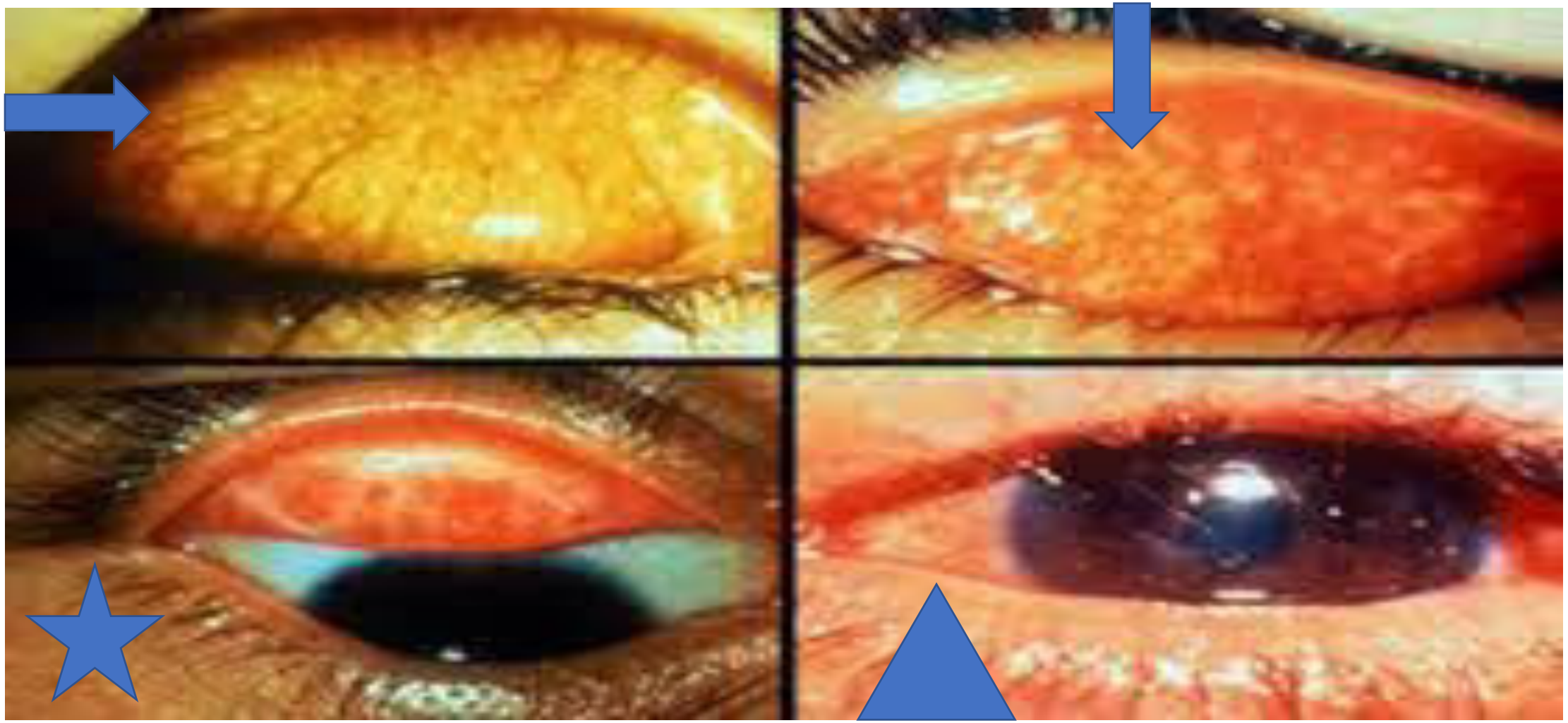
Safe strategy; surgery(S), antibiotics(A), face wash(F), environment improvement(E)

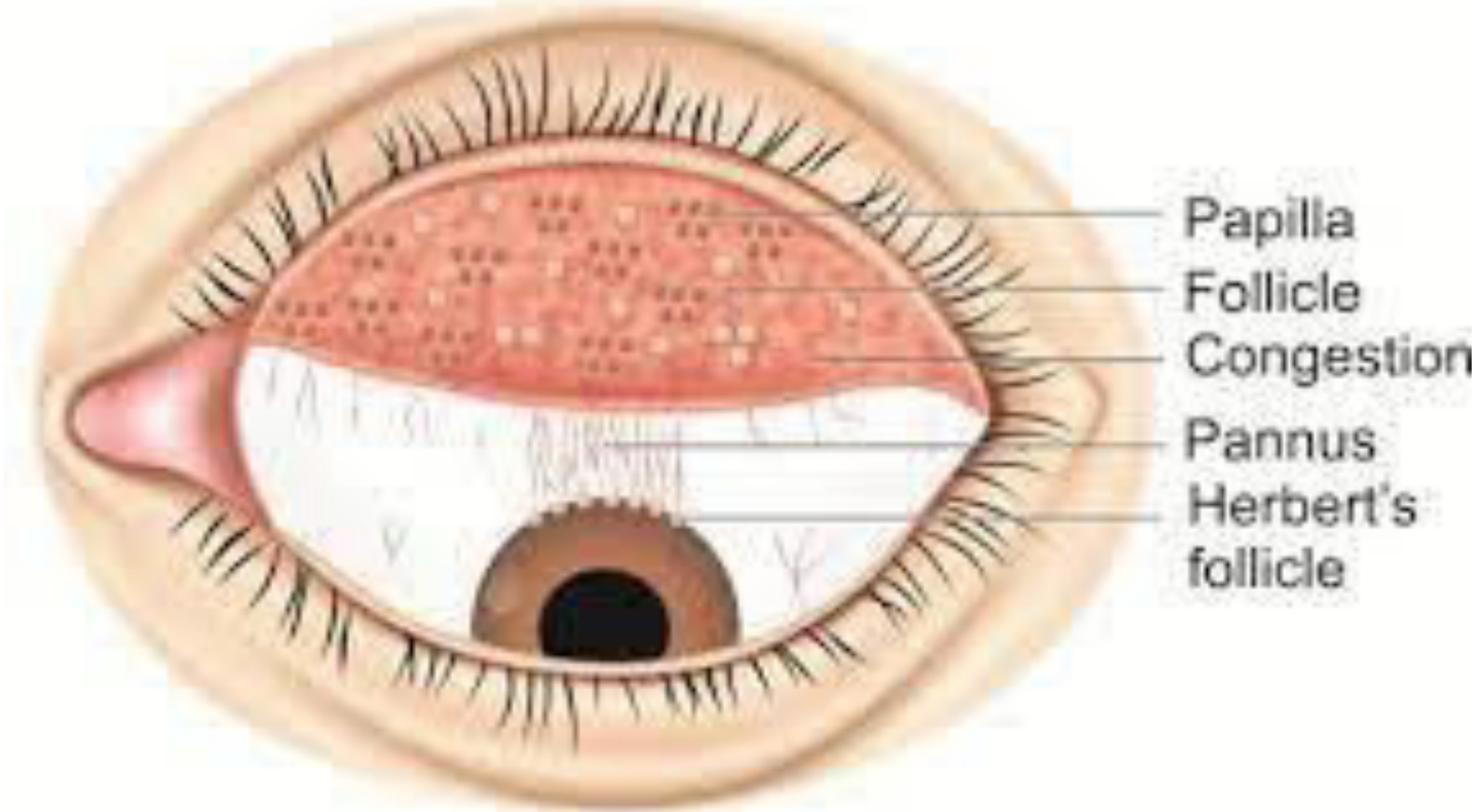
Clinical features (Mac Callan 1908)

- Four stages
- Follicular hyperplasia
- Papillary hypertrophy
- Conjunctival scarring
- Corneal involvement

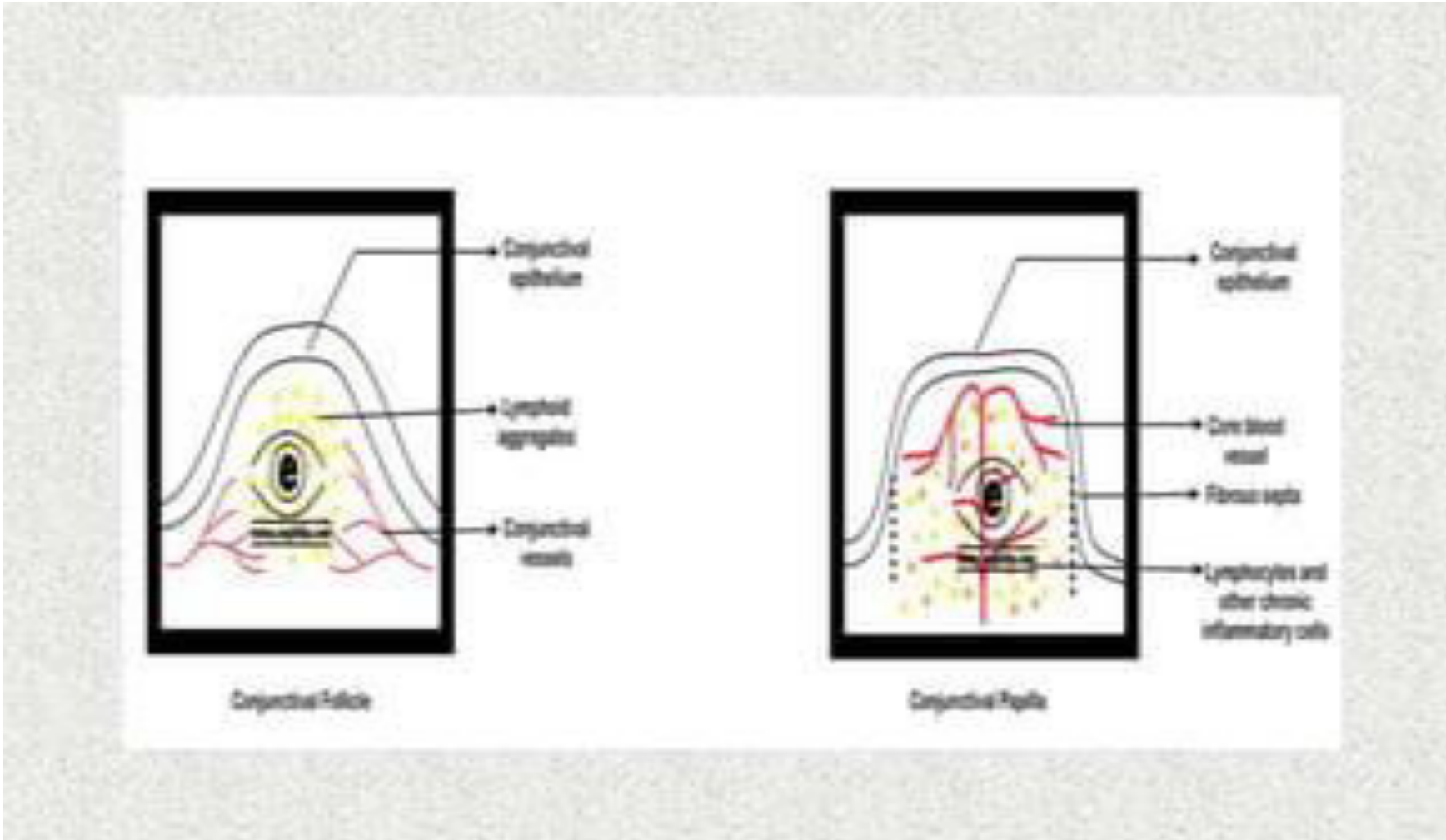
- Stage 1 with minimal symptyoms & low conjunctival reaction
- Minimal discharge
- Follicle are there in up tarsal conjunctiva & immature
- Corneal involvement +_
- No other complications
- Stage 2 with watering photophobia foreign body sensation
- Follicule formation 0.5 - 5mm
- Intense inflammation with papillary hyperplasia which may obscure the follicules
- Corneal pannus formation at the up part of the cornea
- Corneal ulceration may or may not be present

- Stage 3 with corneal scarring still an active stage but conjunctival scarring . The inflammation subsides with necrosis and the follicues ends with linear scarring called Arlets lines
- Herbert's pit are are pits at the corneal limbus due to necrosis of limbus follicules
- Regression of the corneal vascularized pannus with scar formation
- Stage 4 is the complicatins formation. The inflammation is subsided but the complications are due to cicatrization
- Corneal scaing
- Triachiasis
- Entopian formation etc etc




Papilla
Follicle
Congestion
Pannus
Herbert's follicle

- Thanks


Conjunctival epithelium
Lymphoid aggregates
Conjunctival vessels
Conjunctival epithelium
Core blood vessel
Fibrous septa
Lymphocytes and other chronic inflammatory cells

SPREAD OF INFECTIONS

- DIRECT- contact with airborne or waterborne infections
- VECTOR- flies (Musca domestica)
- MATERIAL- most important