

CORNEA

MOHAMED ANBAR

M.D

Anatomy and physiology

General aspect

The cornea is a complex structure which, as well as having a **protective** role, is responsible for about three-quarters of the **optical** power of the eye.

The normal cornea is **avascular**

Nutrients are supplied and metabolic products removed mainly via the aqueous humour posteriorly and the tears anteriorly.

The cornea is the most **densely innervated** tissue in the body, with a subepithelial and a deeper stromal plexus, both supplied by the 1st division of the trigeminal nerve.

Dimensions

The anterior surface of cornea is elliptical with an average horizontal diameter of 11.7 mm and vertical diameter of 11 mm.

The posterior surface of cornea is circular with an average diameter of 11.5 mm.

Thickness of cornea in the centre is about 0.52 mm while at the periphery it is 0.7 mm.

Radius of curvature. The central 5 mm area of the cornea forms the powerful refracting surface of the eye.

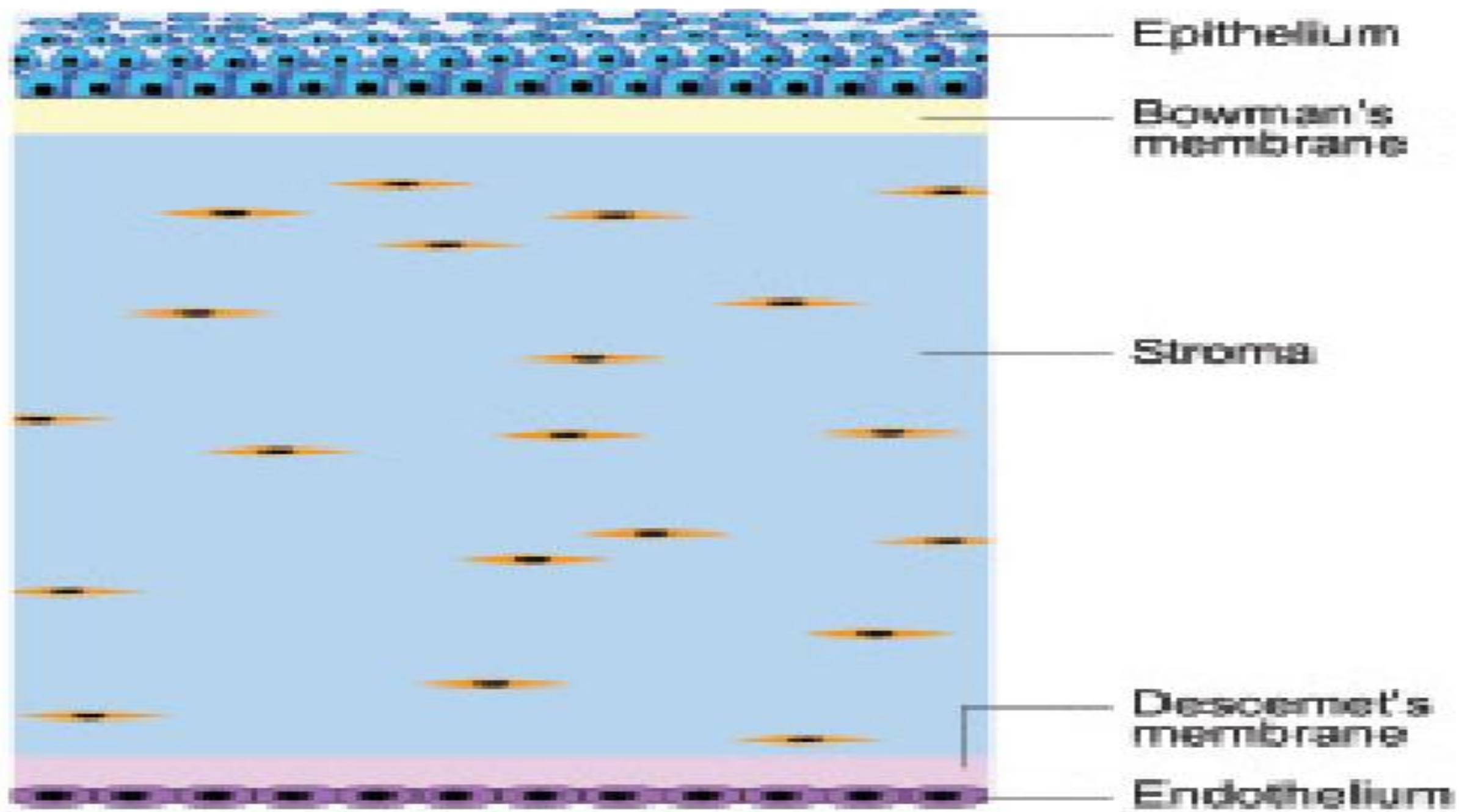
The anterior and posterior radii of curvature of this central part of cornea are 7.8 mm and 6.5 mm, respectively.

Refractive power of the cornea is about 45 dioptres, which is roughly three-fourth of the total refractive power of the eye (60 dioptres)

Layers

1- The epithelium

is stratified squamous and **non-keratinized** and becomes continuous with the epithelium of bulbar conjunctiva at the limbus.



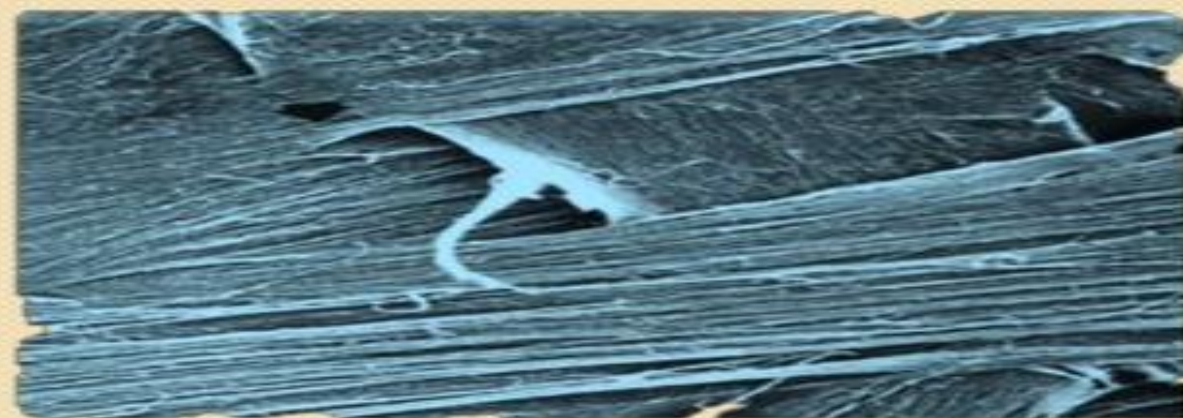
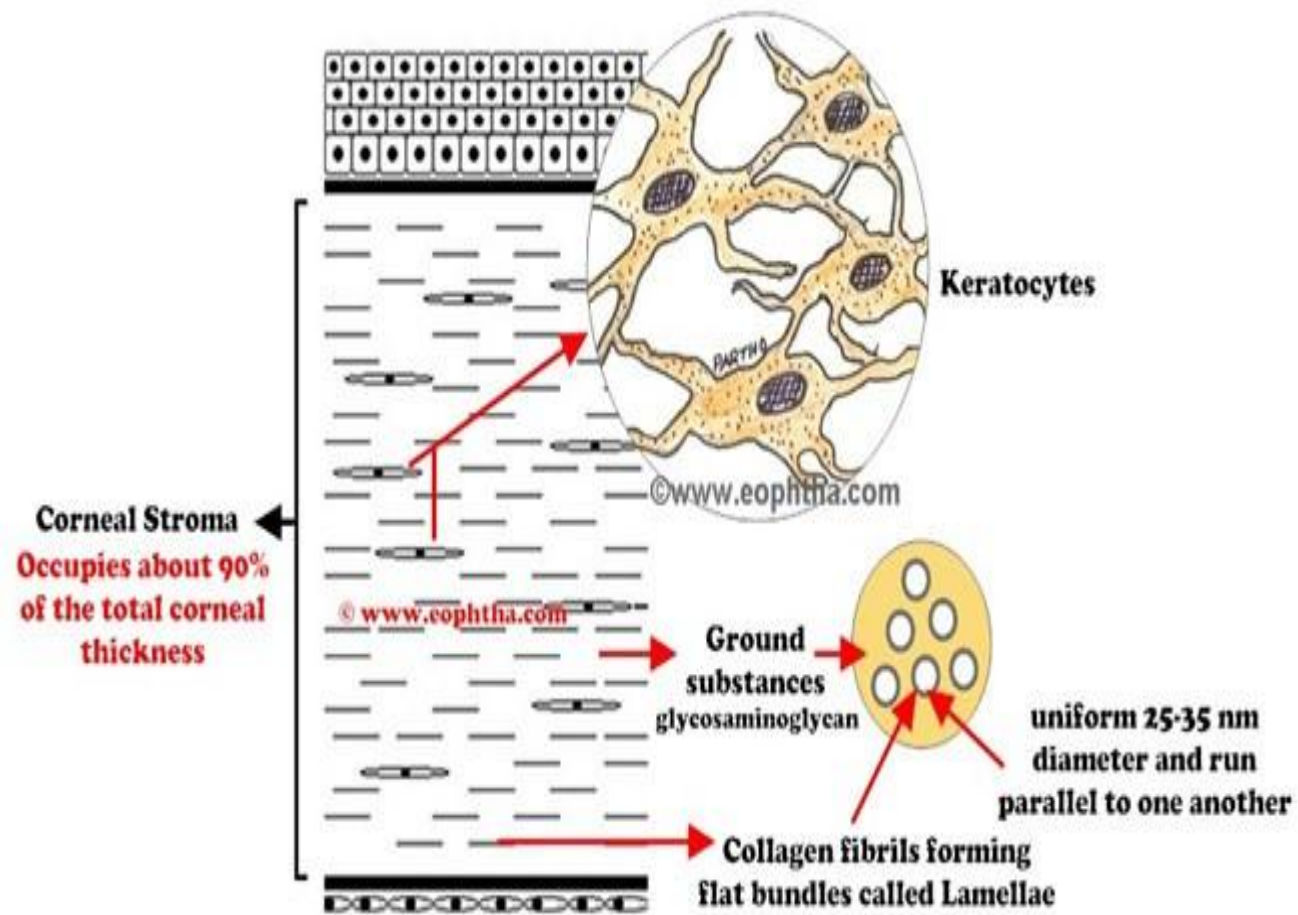
2- Bowman's membrane

is the acellular superficial layer of the stroma formed from collagen fibres

3- Stroma (substantia propria)

makes up 90% of corneal thickness.

It is arranged in regularly orientated layers of **collagen fibrils** whose spacing is maintained by **proteoglycan ground substance** (chondroitin sulphate and keratan sulphate) with interspersed modified fibroblasts (**keratocytes**).



4- Descemet membrane

is a discrete sheet composed of a fine latticework of collagen fibrils that are distinct from the collagen of the stroma.

It is very resistant to chemical agents, trauma and pathological processes. Therefore, 'Descemetocoele' can maintain the integrity of eyeball for long.

Unlike Bowman's membrane **it can regenerate**



5- The endothelium

Consists of a monolayer of **polygonal cells**.

Endothelial cells maintain corneal deturgescence throughout life by pumping excess fluid out of the stroma.

The adult cell density is about 2500 cells/mm². The number of cells decreases at about 0.6% per year and neighbouring cells enlarge to fill the space; the cells **cannot regenerate**.

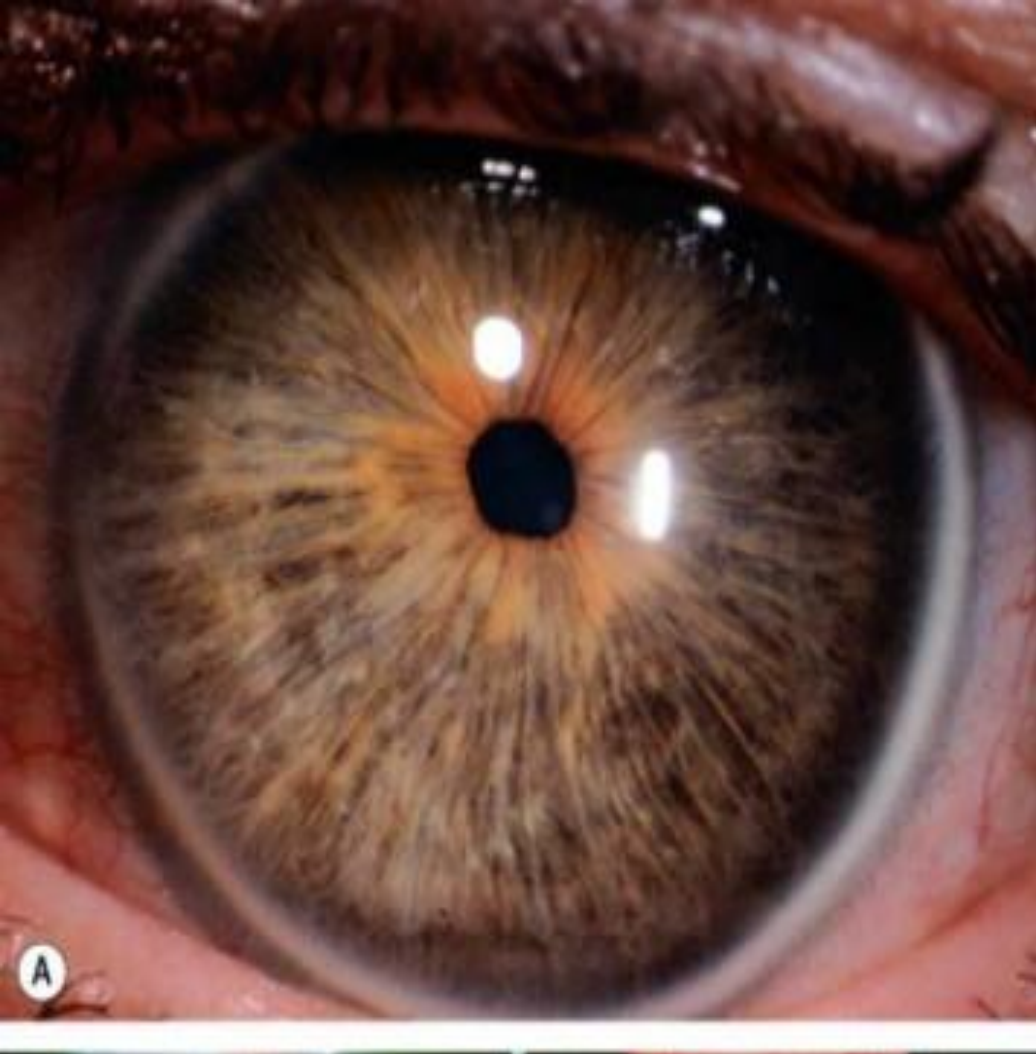
At a cell density of about 500 cells/mm² corneal oedema develops and transparency is reduced.

CONGENITAL ANOMALIES

Megalocornea

Horizontal diameter of cornea at birth is about 10 mm and the adult size of about 11.7 mm is attained by the age of 2 years.

Megalocornea is labelled when the horizontal diameter of cornea is of adult size at birth or 13 mm or greater after the age of 2 years.



The cornea is usually clear with normal thickness and vision.

The condition is not progressive.
Systemic association include
Marfan's, Apert, Ehlers Danlos and
Down syndromes

Microcornea

Microcornea is a rare unilateral or bilateral condition.

Signs

The adult horizontal corneal diameter is 10 mm or less.

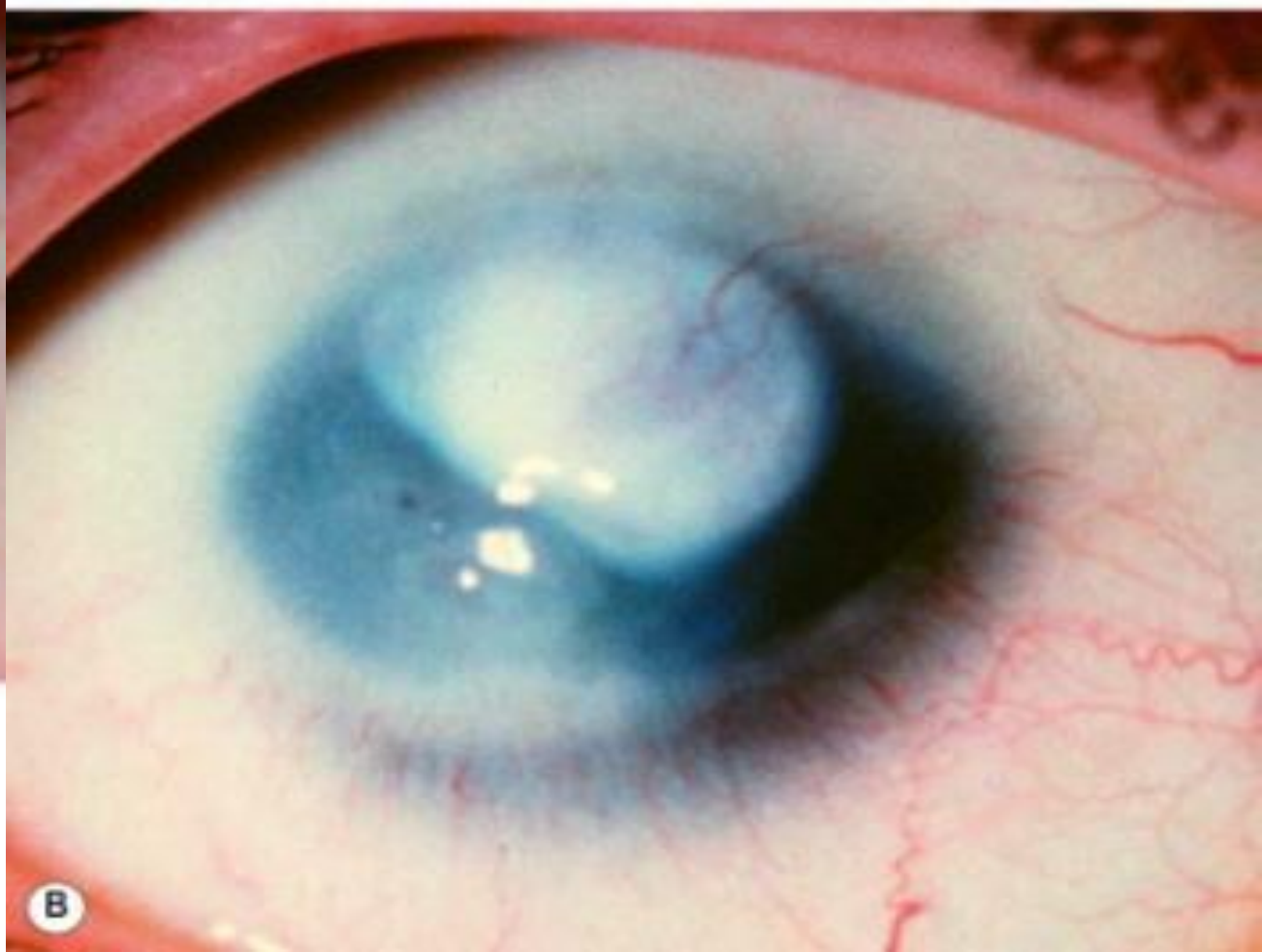
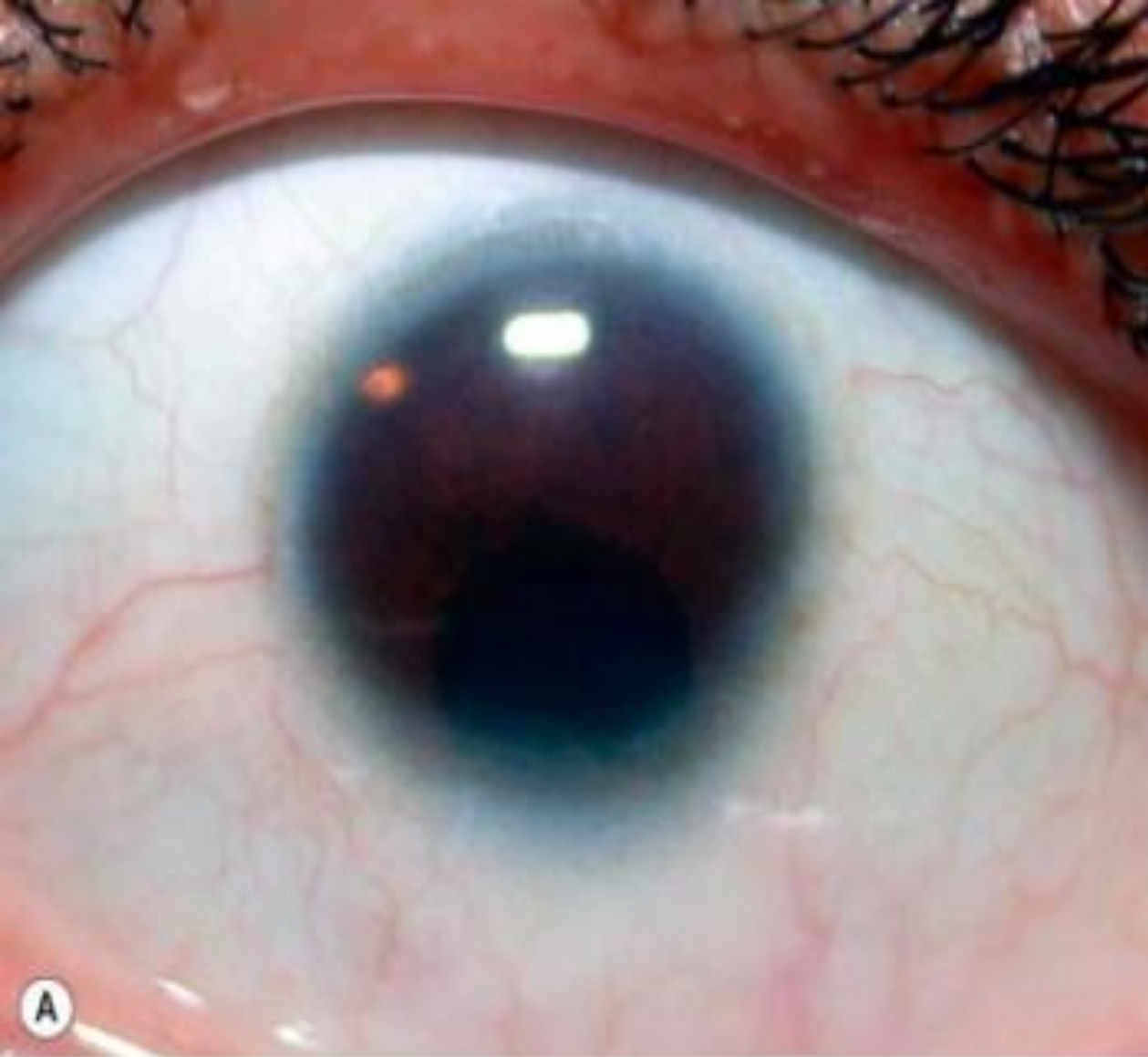
Hypermetropia, shallow anterior chamber but other dimensions are normal.

Ocular associations

Glaucoma (closed and open angle), congenital cataract, leukoma, cornea plana, Rieger anomaly, microphakia and optic nerve hypoplasia.

Syndromic systemic associations

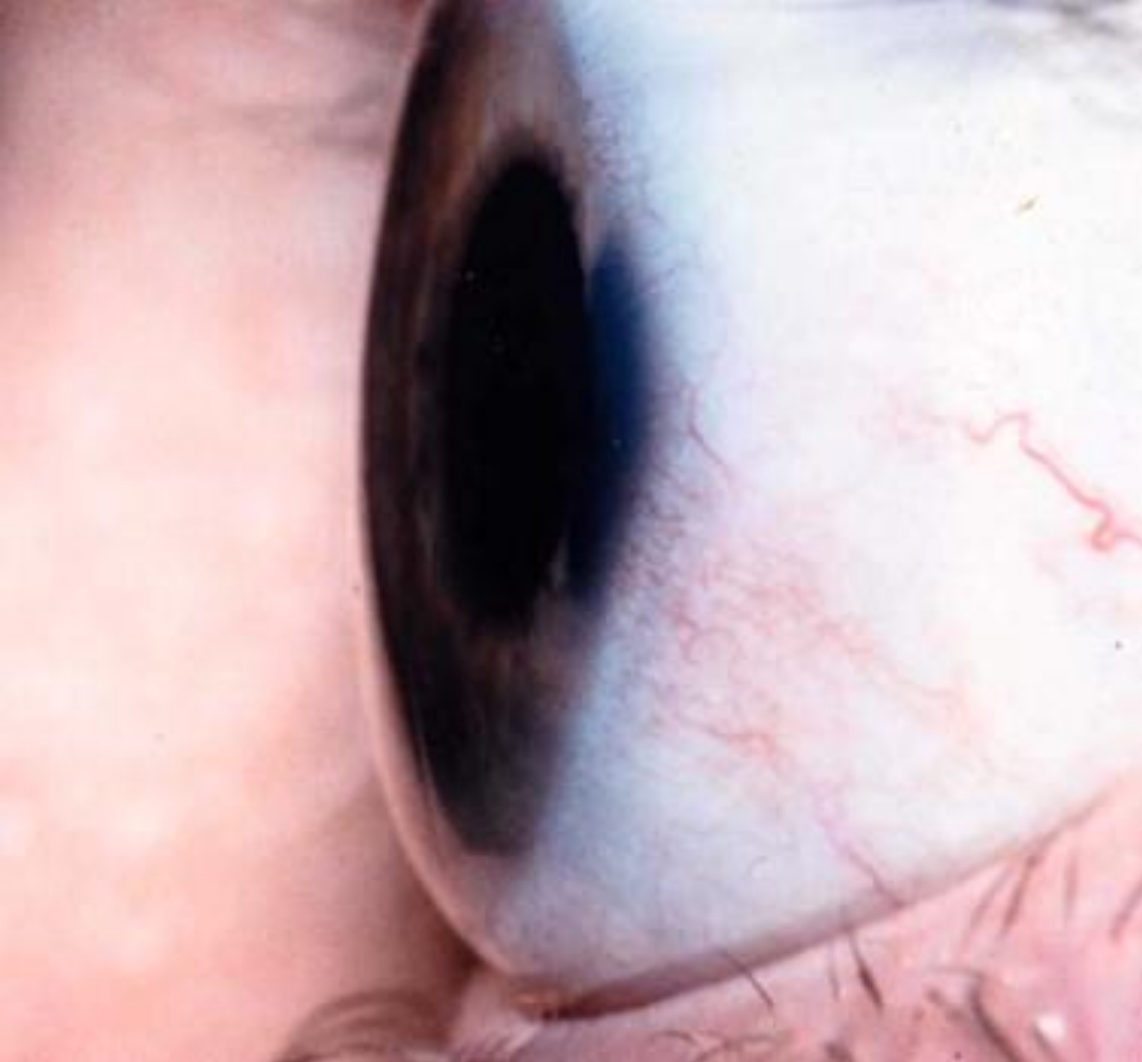
include fetal alcohol, Ehlers–Danlos, Weill–Marchesani, Waardenburg, Nance Horan and Cornelia de Lange syndromes.



Cornea plana

This is a rare bilateral condition.

Flat cornea, and a corresponding reduction in refractive power resulting in high hypermetropia



Associations

include shallow anterior chamber with predisposition to angle-closure glaucoma, scleroderma, microcornea and microphthalmos, Peters anomaly, and iris abnormalities including irido-corneal adhesions

Microphthalmos

Microphthalmos is a developmental arrest of ocular growth.

Total axial length is reduced because of stunted growth of the anterior or posterior segment, or both. The condition is typically sporadic and may be unilateral or bilateral.



Simple microphthalmos

Not associated with other major ocular malformations

Complex (colobomatous) microphthalmos

Associated with coloboma, usually of the iris





Microphthalmos with cyst

Caused by failure of the optic fissure to close, leading to the formation of an orbital cyst that communicates with the eye. The extent of the cystic component is best delineated on MR or CT

Posterior microphthalmos

is a rare subset of microphthalmos in which TAL is reduced in the setting of normal corneal diameter, resulting in high hypermetropia and papillomacular retinal fold.

This differs from nanophthalmos, which is described as eyes with microphthalmos, microcornea and a tendency toward uveal effusions.

Anophthalmos



Simple anophthalmos

Complete failure of budding of the optic vesicle or early arrest in its development



Anophthalmos with cyst (congenital cystic eyeball)

a condition in which the globe is replaced by a cyst

Signs of corneal inflammation

Superficial lesions

1- Punctate epithelial erosions (PEE)

Tiny epithelial defects that **stain** with fluorescein and rose bengal.

Causes

include a wide variety of stimuli; the location of the lesions may give an indication of aetiology as follows

Superior

Vernal disease, chlamydial conjunctivitis, superior limbic keratoconjunctivitis, floppy eyelid syndrome and mechanically-induced keratoconjunctivitis.

Interpalpebral

Dry eye (can also be inferior), reduced corneal sensation and ultraviolet keratopathy

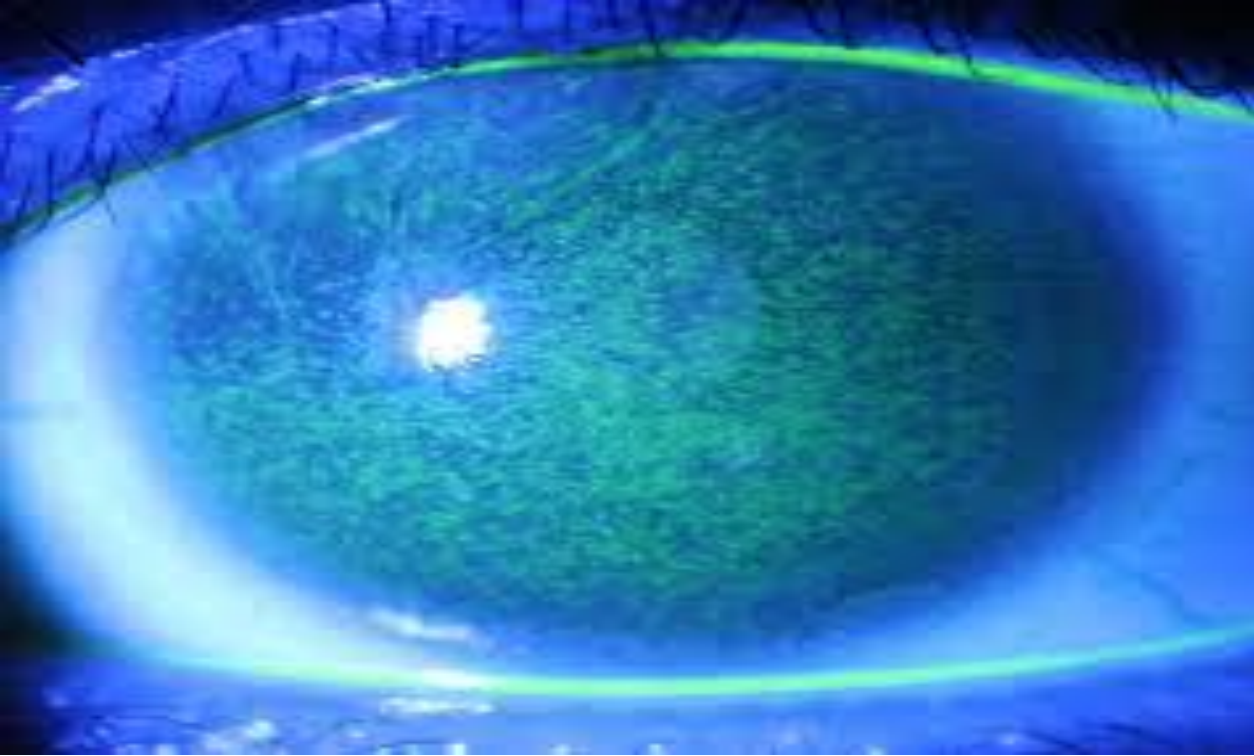
Inferior

Chronic blepharitis, **lagophthalmos**, eye drop toxicity, self-induced, aberrant eyelashes and entropion.

some cases of viral and bacterial conjunctivitis, and toxicity to drops

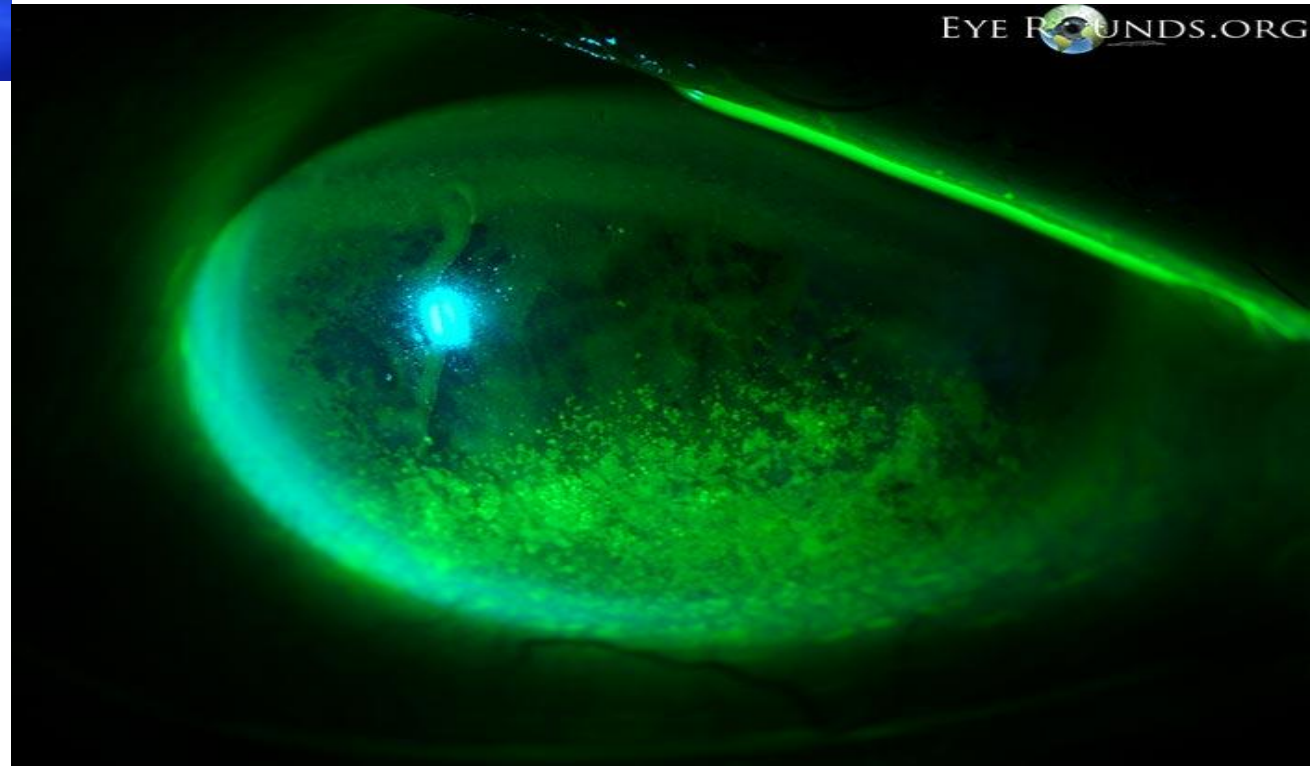
Central

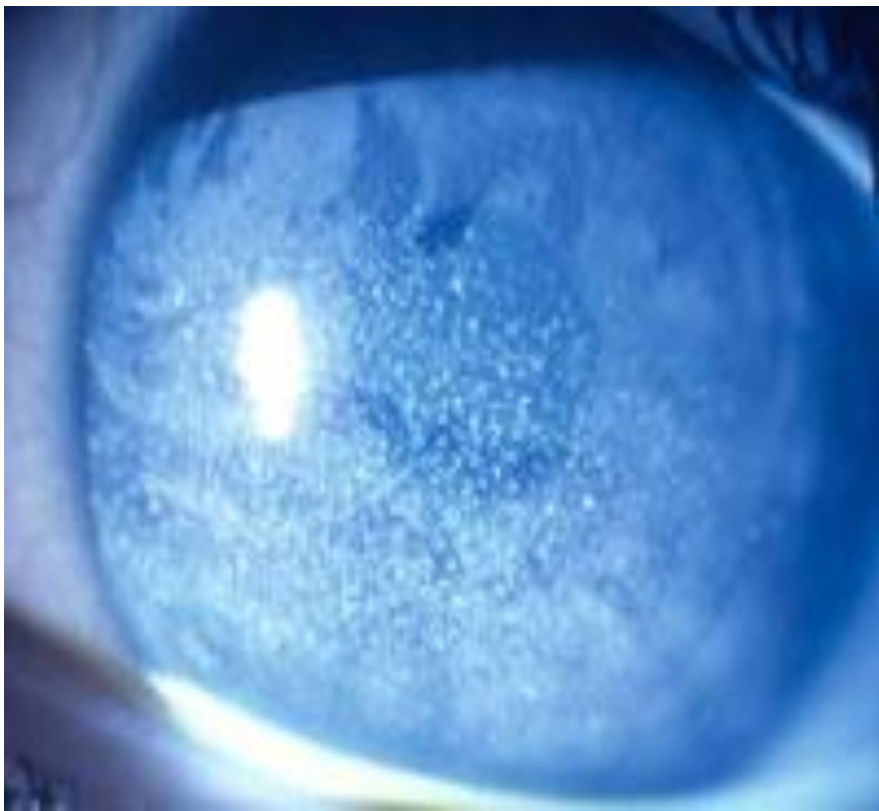
prolonged contact lens wear.



Diffuse

Exposure Keratopathy





Dry eye

Vernal keratoconjunctivitis



2- Punctate epithelial keratitis (PEK)

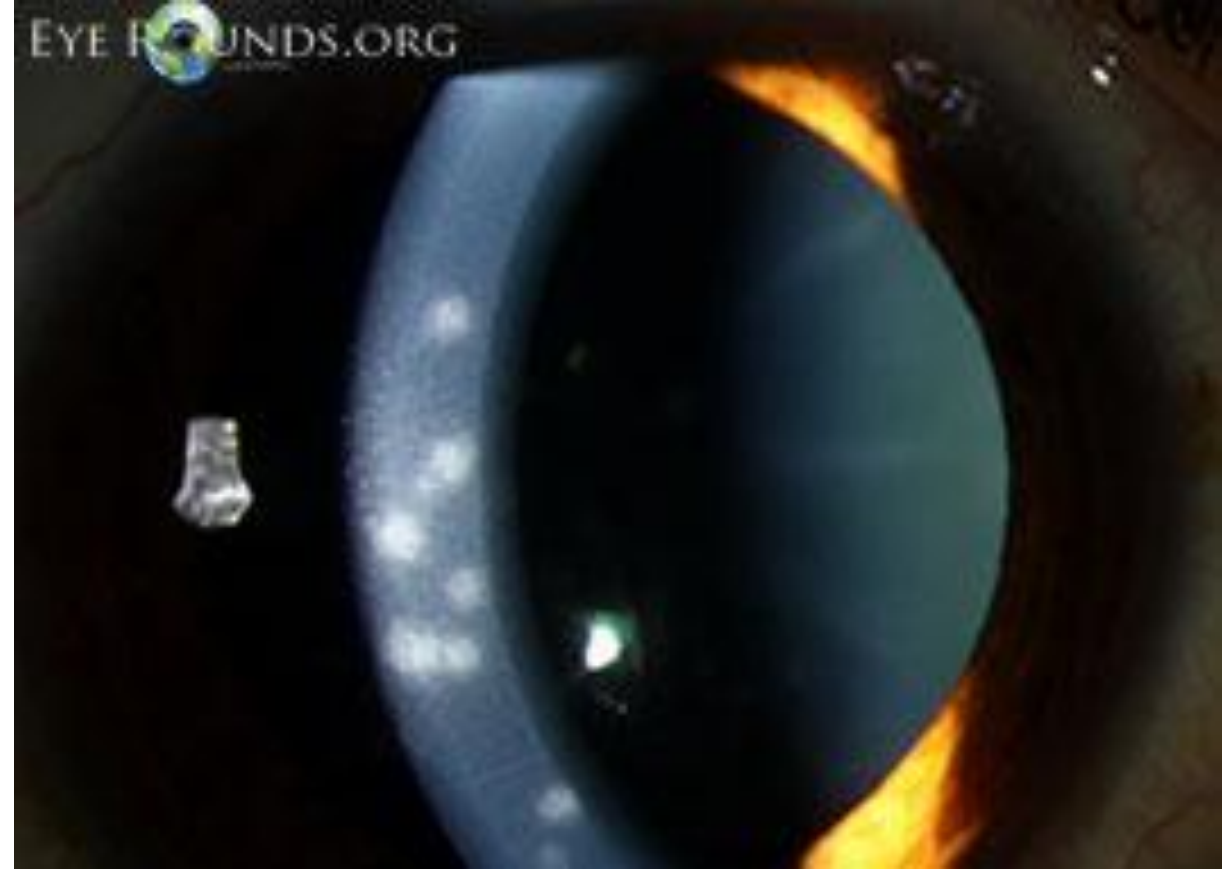
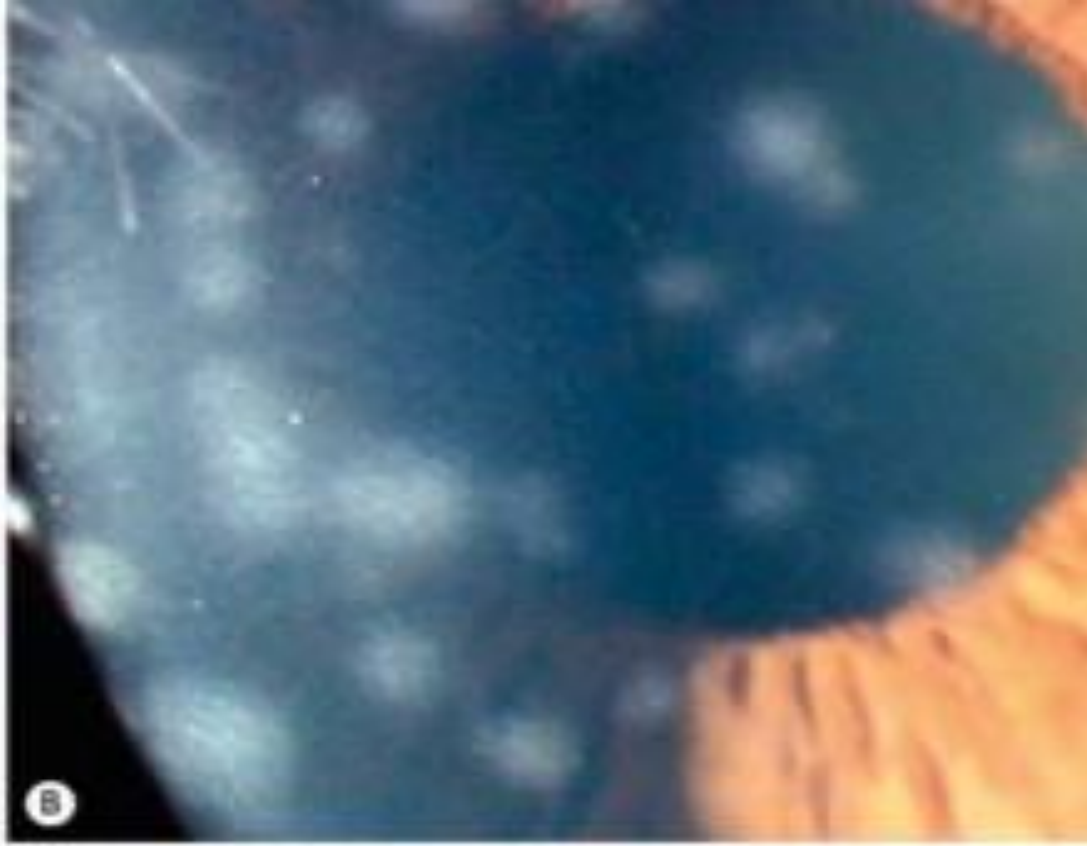
Granular, opalescent, swollen epithelial cells, with focal intraepithelial infiltrates.

They are visible unstained but stain well with rose bengal and variably with fluorescein

Causes.

Infection such as ***adenoviral***, chlamydial, molluscum contagiosum, early herpes simplex and herpes zoster, and systemic viral infections (e.g. measles, varicella, rubella).

Miscellaneous such as Thygeson superficial punctate keratitis and ***toxicity to drops.***



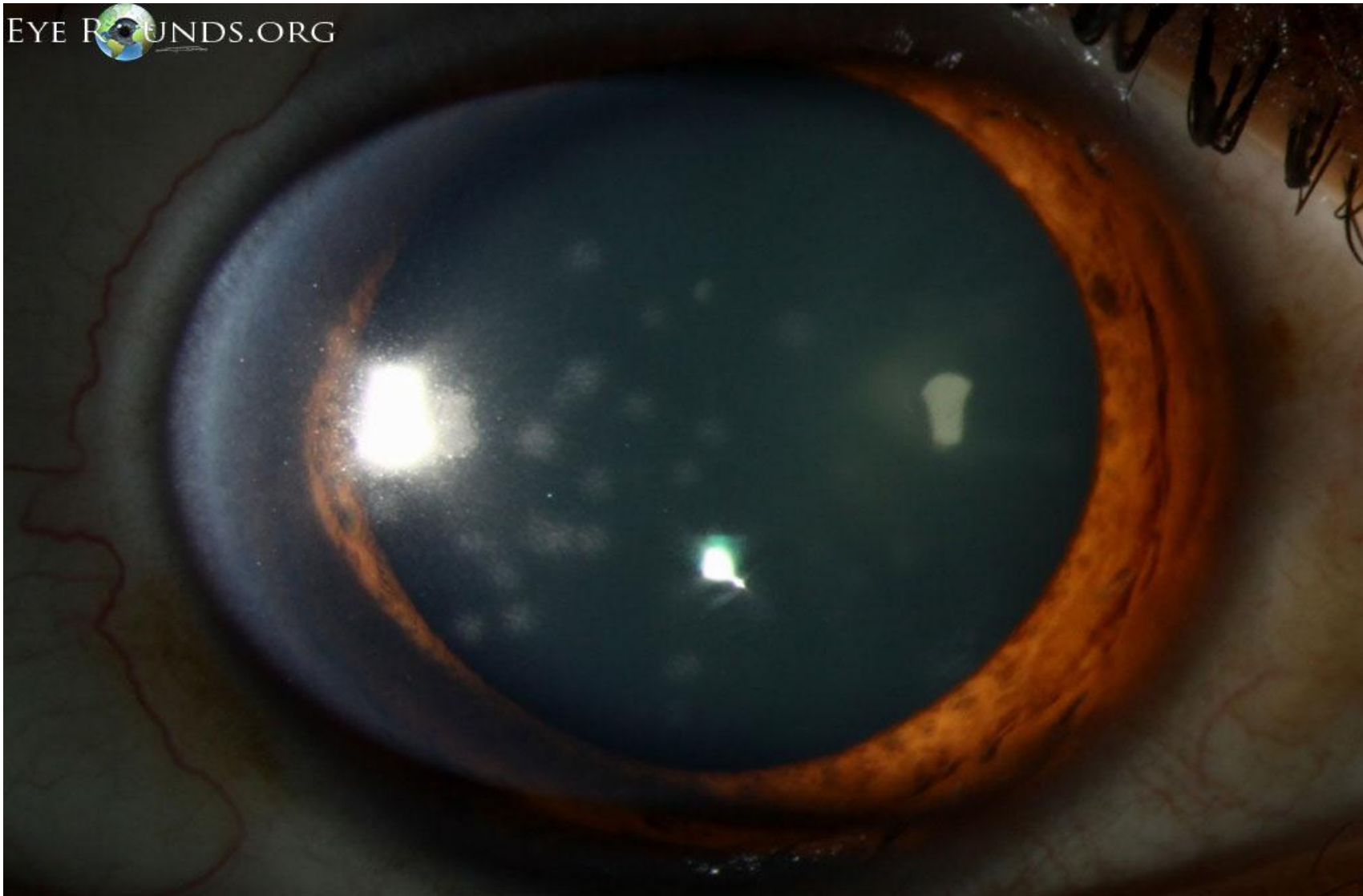
Punctate epithelial keratitis (PEK)

3- Subepithelial infiltrates

Tiny subsurface foci of non-staining inflammatory infiltrates.

Causes

Severe or prolonged **adenoviral** keratoconjunctivitis, herpes zoster keratitis, adult inclusion conjunctivitis, marginal keratitis, rosacea and Thygeson superficial punctate keratitis.



Subepithelial infiltrates

4- Filaments

Strands of mucus admixed with epithelium, attached at one end to the corneal surface, that stain well with rose bengal.

The unattached end moves with each blink. Grey subepithelial opacities may be seen at the site of attachment.

Causes

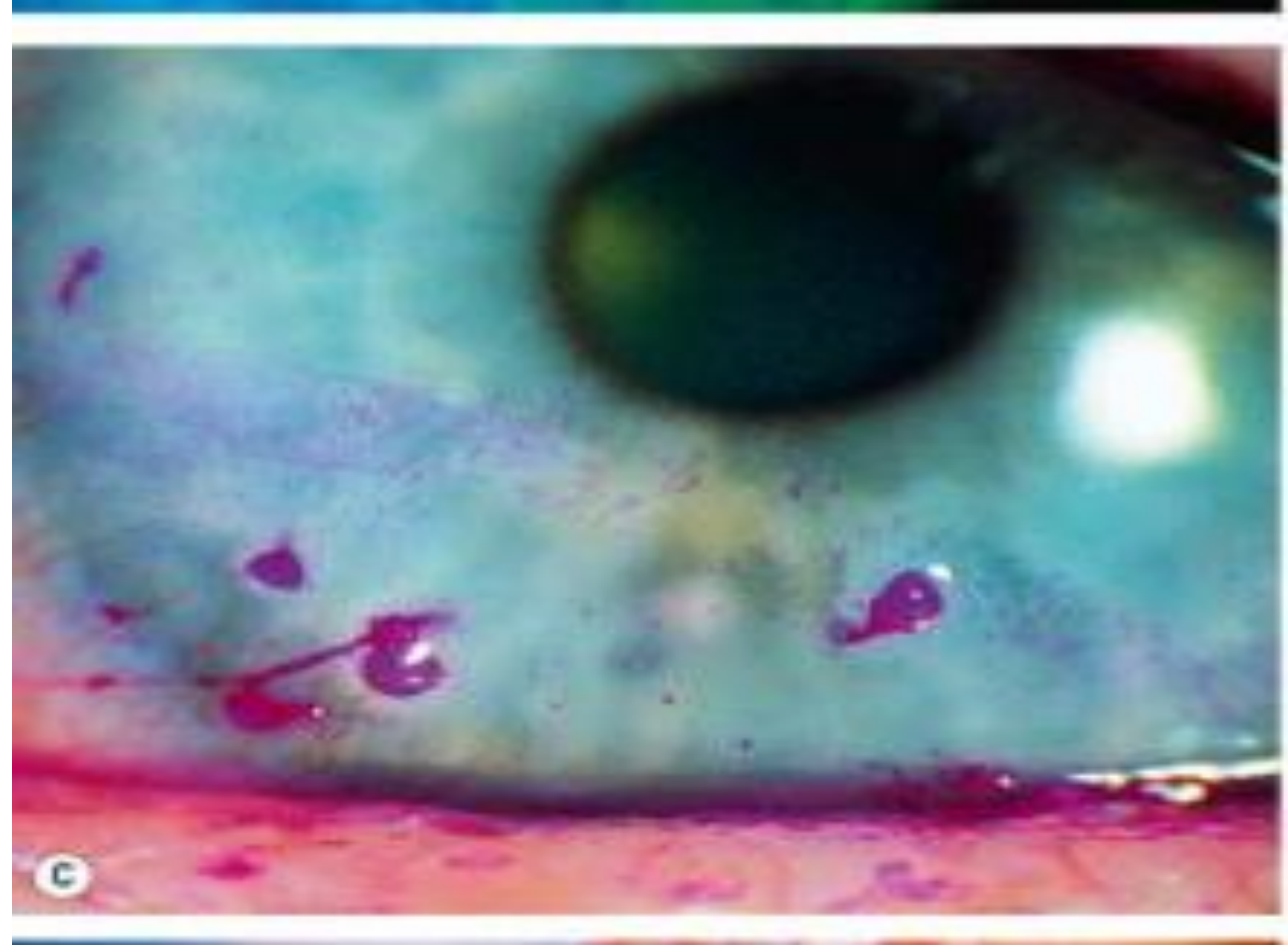
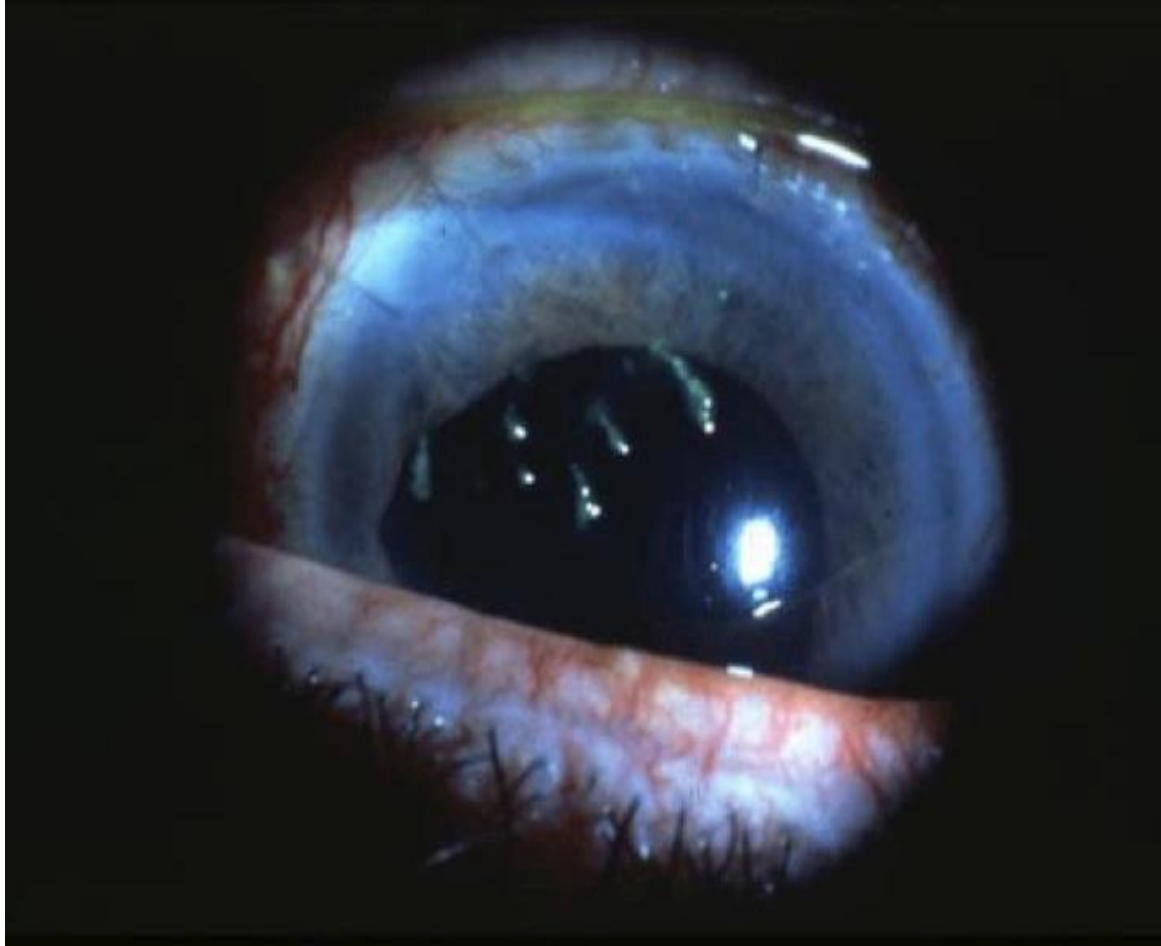
Dry eye

Superior limbic keratoconjunctivitis

Neurotrophic keratitis

Long-term ocular patching

Essential blepharospasm.



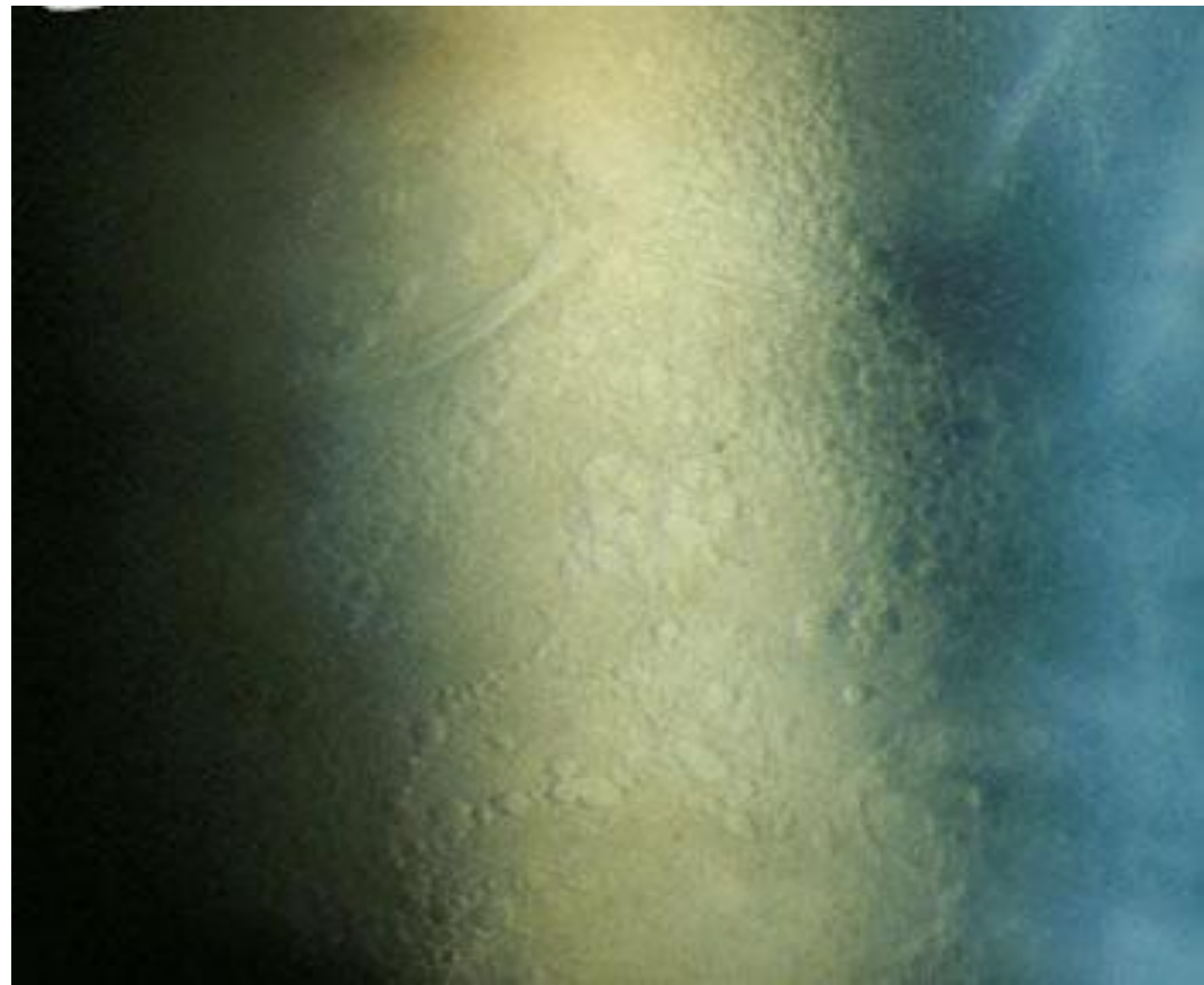
Filamentary Keratitis

5-Epithelial oedema

Subtle cases may manifest with loss of normal corneal lustre, but more commonly by abundant tiny epithelial vesicles; bullae are seen in moderate-severe cases.

Causes

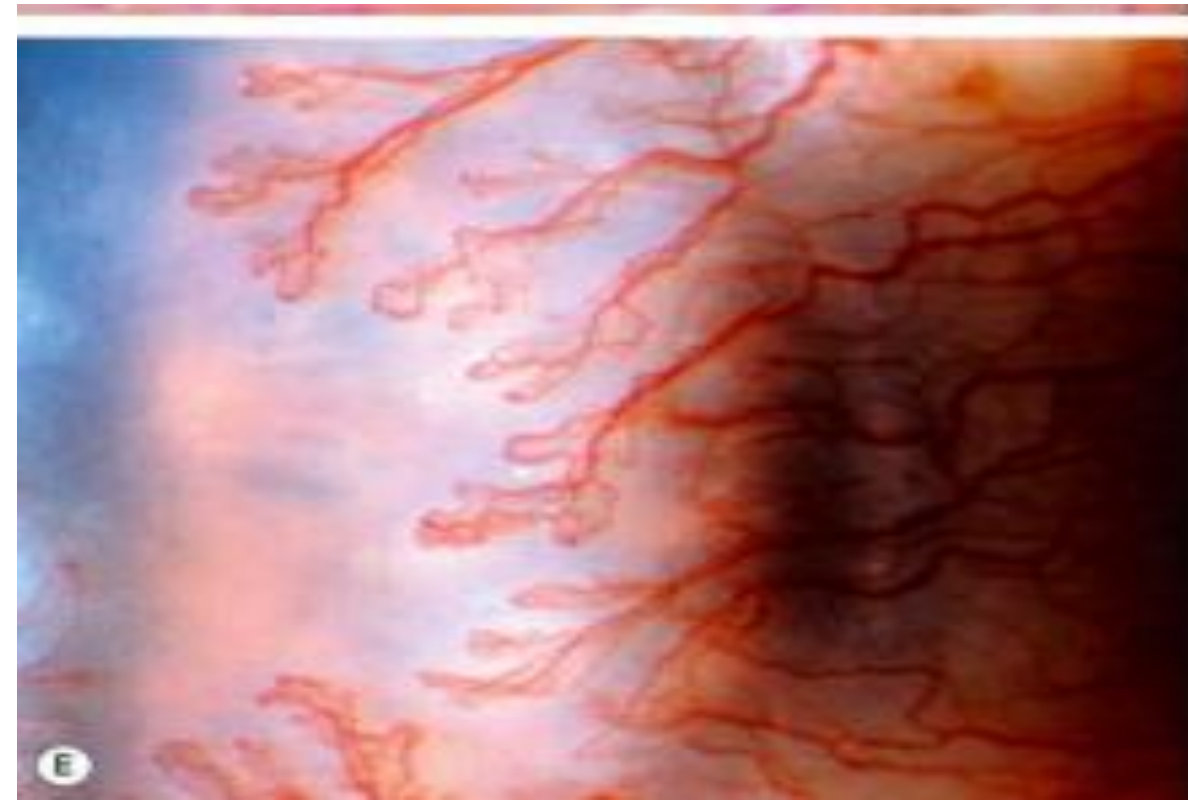
Endothelial decompensation, including that due to severe acute elevation of intraocular pressure



Epithelial oedema

6-Superficial neovascularization

Is a feature of chronic ocular surface irritation or hypoxia, as in **contact lens wear**



7- Pannus

Describes superficial neovascularization accompanied by degenerative subepithelial change extending centrally from the limbus.



Pannus

Deep lesions

1- Infiltrates

are focal areas of acute stromal inflammation composed of inflammatory cells, and cellular and extracellular debris including necrosis.

Signs

Yellow- or grey-white **opacities** located initially within the anterior stroma associated with limbal or conjunctival hyperaemia.

Causes

1- **Suppurative keratitis** is caused by active infection with bacteria, viruses, fungi or protozoa.

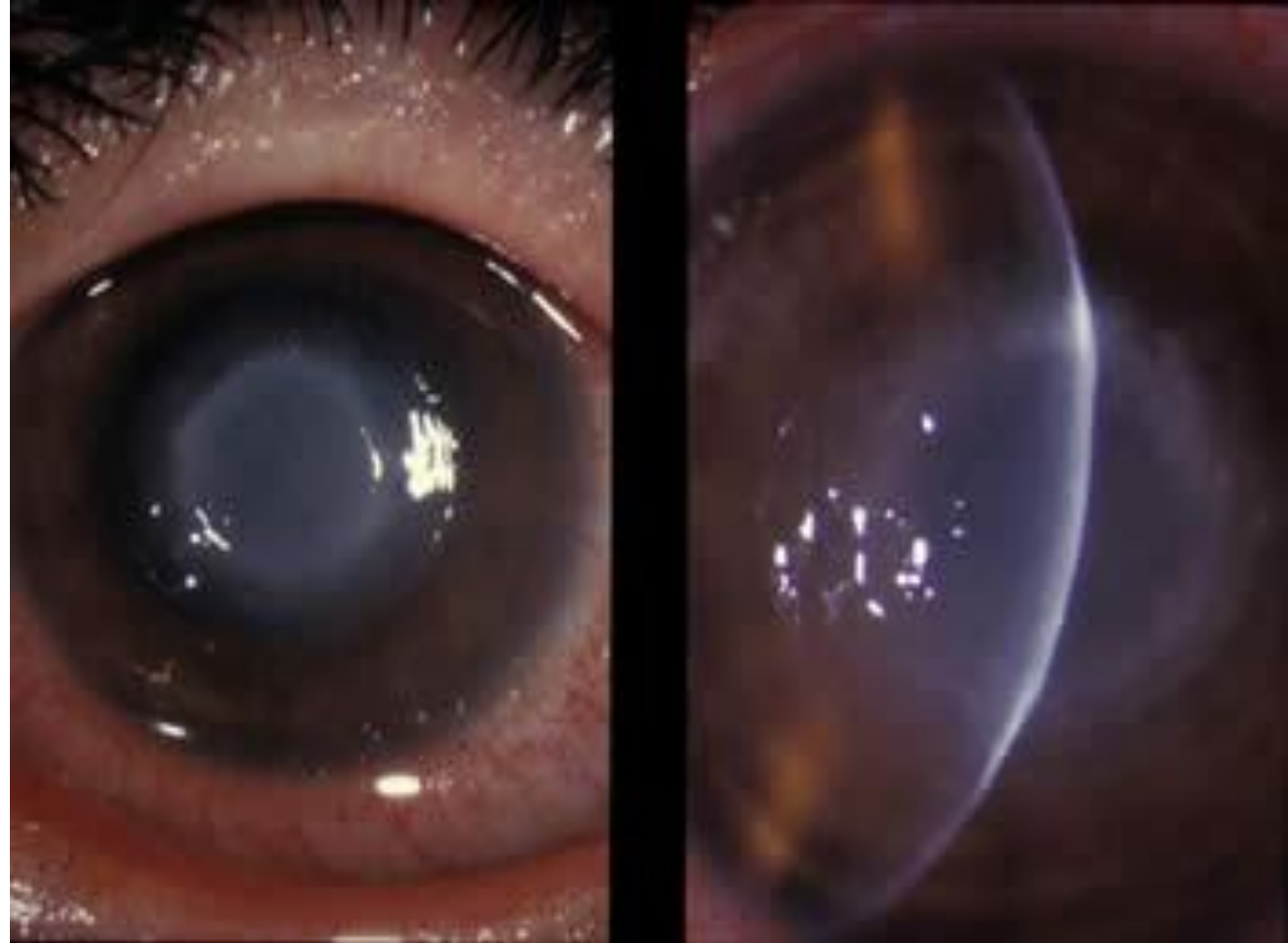
2- Non-infectious 'sterile keratitis' is the result of an immunological hypersensitivity response to antigen as in marginal keratitis and with **contact lens wear**.

The key distinction of clinical importance is between **sterile** and **infective** lesions; '**PEDAL**' mnemonic: **P**ain, **E**pithelial defects, **D**ischarge, **A**nterior chamber reaction, **L**ocation.

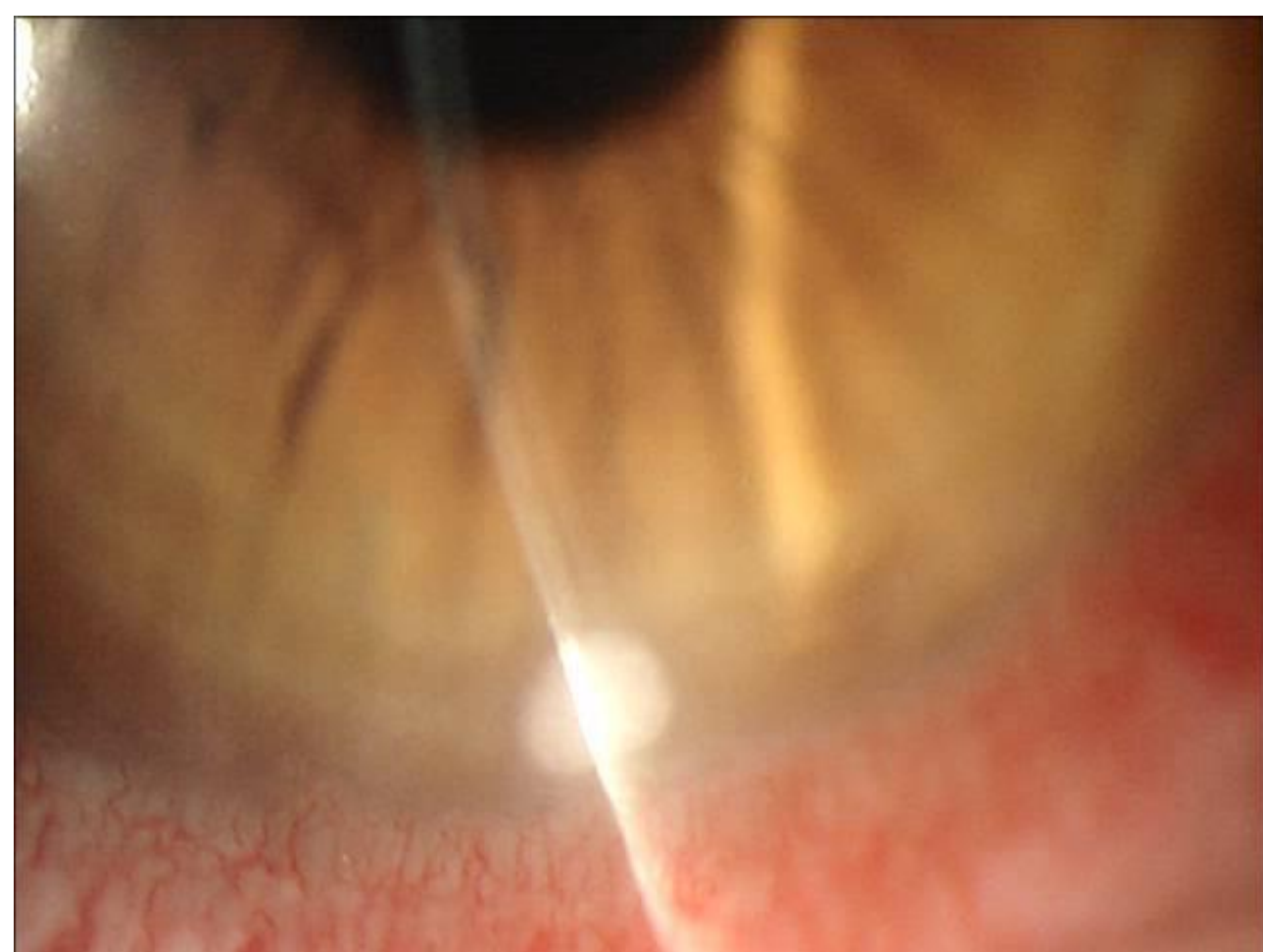
	<i>Infective</i>	<i>Sterile</i>
Size	Tend to be larger	Tend to be smaller
Progression	Rapid	Slow
Epithelial defect	Very common and larger when present	Much less common and if present tends to be small
Pain	Moderate-severe	Mild
Discharge	Purulent	Mucopurulent
Single or multiple	Typically single	Commonly multiple
Unilateral or bilateral	Unilateral	Often bilateral
Anterior chamber reaction	Severe	Mild
Location	Often central	Typically peripheral
Adjacent corneal reaction	Extensive	Limited



Fungal keratitis



Acanthameba keratitis



Sterile Infiltrate

2- Ulceration

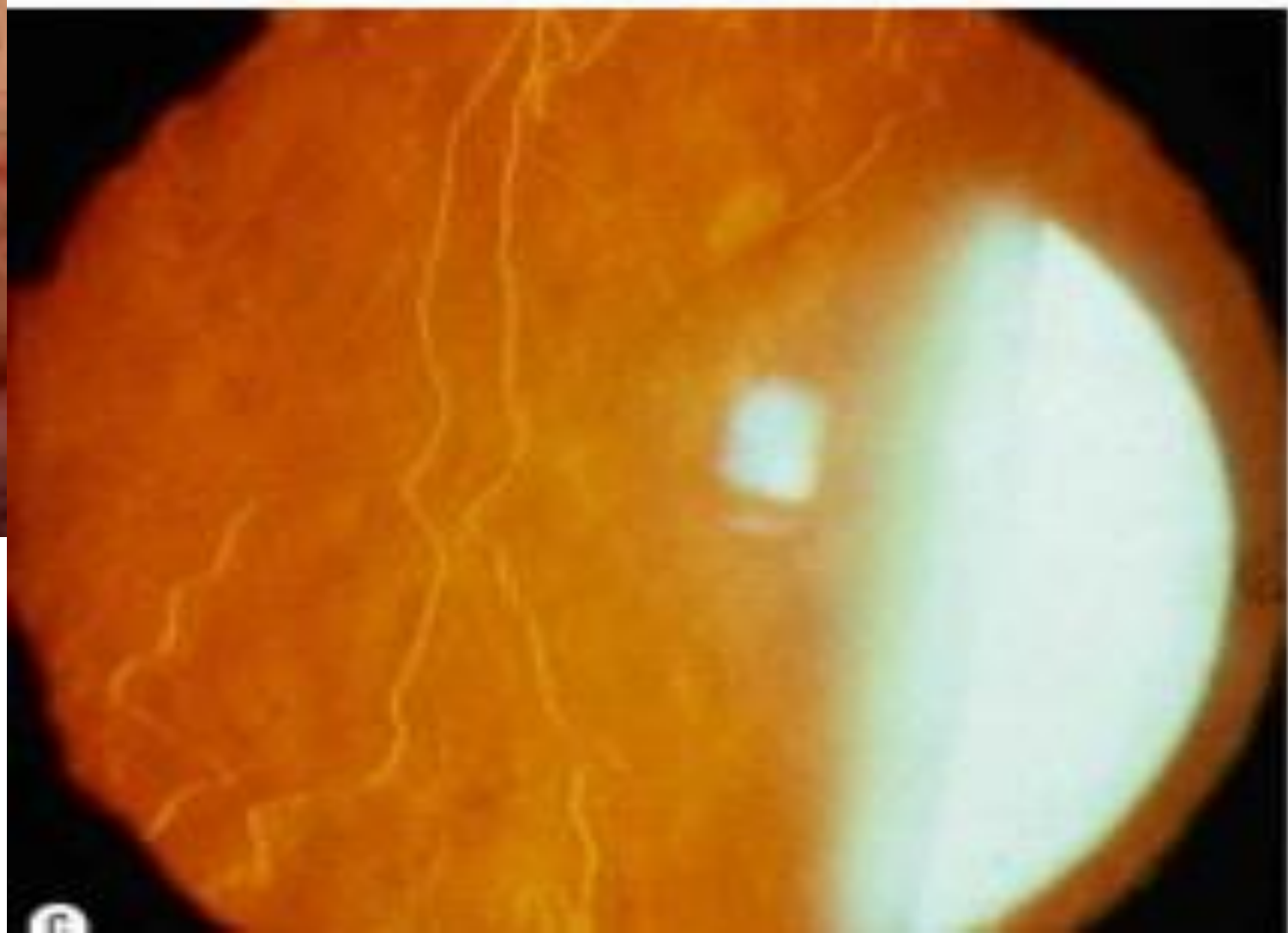
refers to tissue excavation associated with an epithelial defect. Connective tissue 'melting' occurs in response to enzymatic activity, as in peripheral ulcerative keratitis.



3- Vascularization

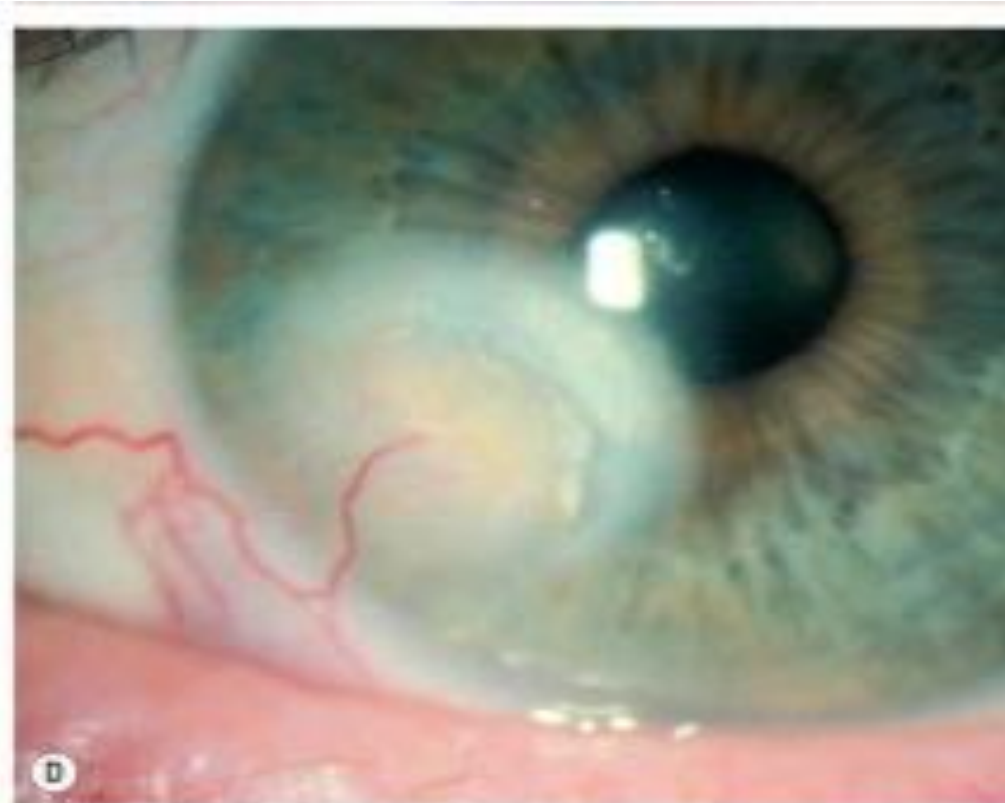
occurs in response to a wide variety of stimuli. Venous channels are easily seen, whereas arterial feeding vessels are smaller and require higher magnification.

Non-perfused deep vessels appear as 'ghost vessels', best detected by retroillumination



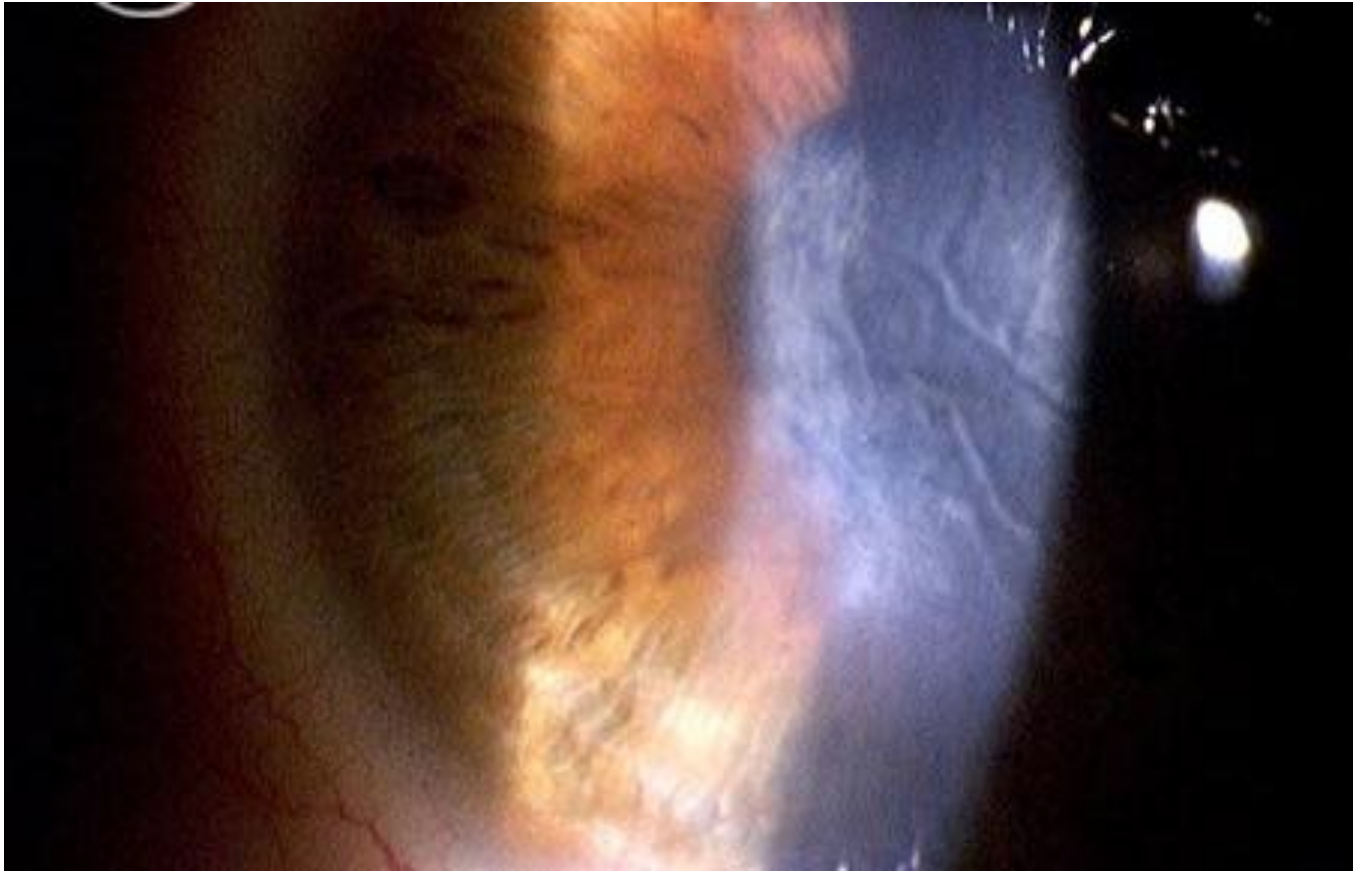
4- Lipid deposition

may follow chronic inflammation with leakage from corneal new vessels



5- Folds in Descemet membrane

may result from corneal oedema exceeding the capacity of the endothelium to maintain normal turgescence. Causes include inflammation, trauma (including surgery) and ocular hypotony.



6- Descemetocoele



7- Breaks in Descemet membrane

Haab striae in infantile glaucoma

Keratoconus

Birth trauma

Birth trauma



Haab striae

