# Decreased vision due to retinal disorders

Siamak Moradian MD Professor (Associate) LMC

## **Central Retinal Vein Occlusion**

- characteristic fundus appearance of dilated and tortuous retinal veins, a swollen optic disc, intra retinal hemorrhages.
- CRVO is now classified by 2 ends of the spectrum of disease: *nonischemic*, a milder form sometimes referred to as partial, perfused, or venous
- stasis retinopathy ischemic, a form characterized by at least 10 disc areas, as demonstrated by fluorescein angiography, of retinal capillary nonperfusion on a posterior pole view;
- also known as nonperfused, complete, or hemorrhagic

#### CRVO have a common mechanism:

- thrombosis of the central retinal vein at and posterior to the level of the lamina cribrosa.
- Occlusion of the central retinal vein is often a result of both local and systemic causes.

## Risk Factors for Central Retinal Vein Occlusion

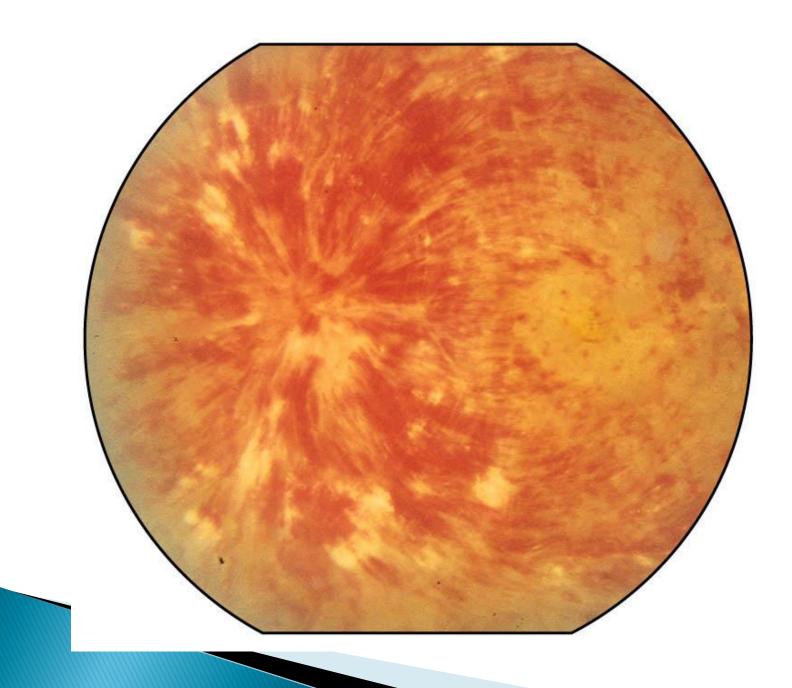
- Increased Risk: Systemic hypertension, Diabetes mellitus Increased erythrocyte sedimentation rate (women only), History of glaucoma
- Decreased Risk: Increasing levels of physical activity, Increasing levels of alcohol consumption, Exogenous estrogens (women only)

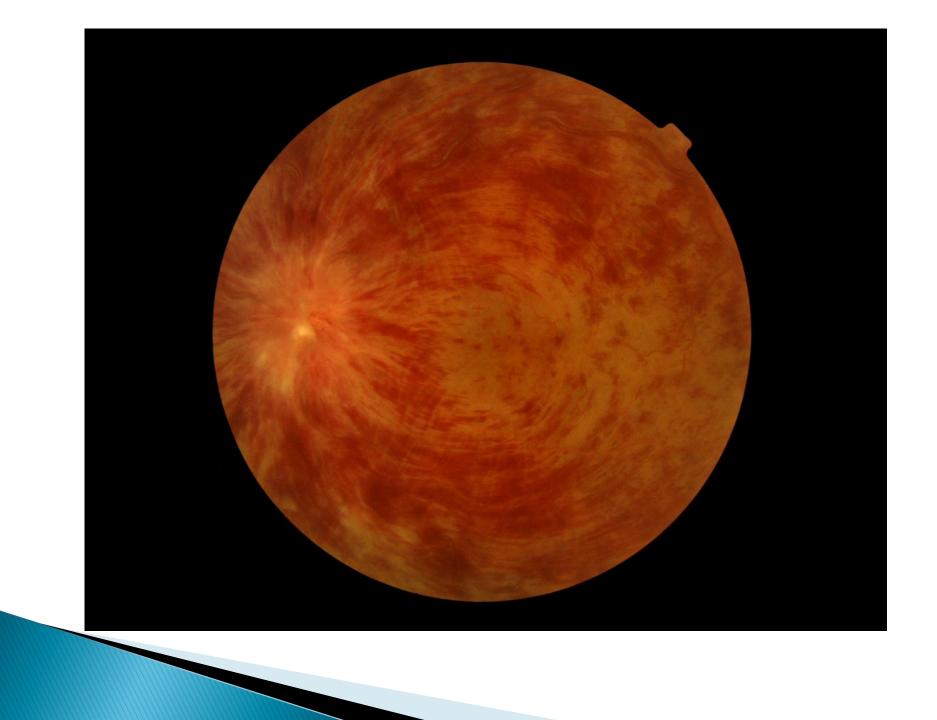
## Risk Factors for Branch Retinal Vein Occlusion

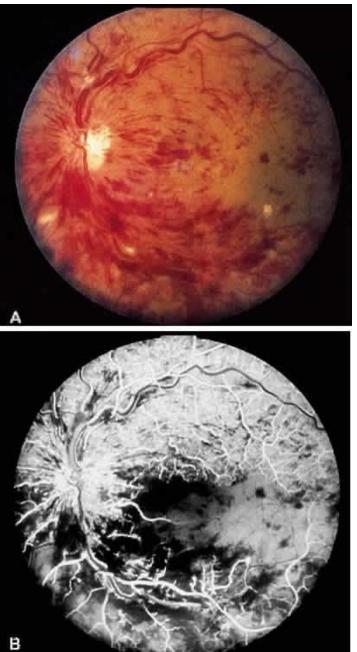
**Increased Risk** :Systemic hypertension, History of cardiovascular disease ,Increased body mass index at 20 years of age, History of glaucoma ,High serum levels of  $\alpha_{2-globulin}$ 

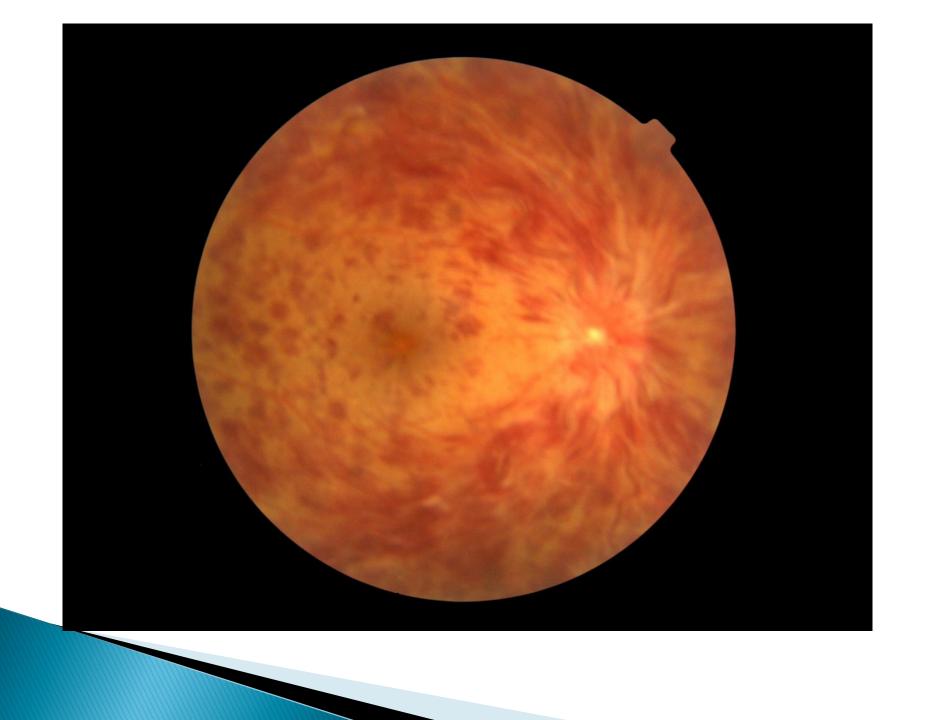
 Decreased Risk Higher levels of alcohol consumption, Higher serum levels of highdensity lipoprotein cholesterol

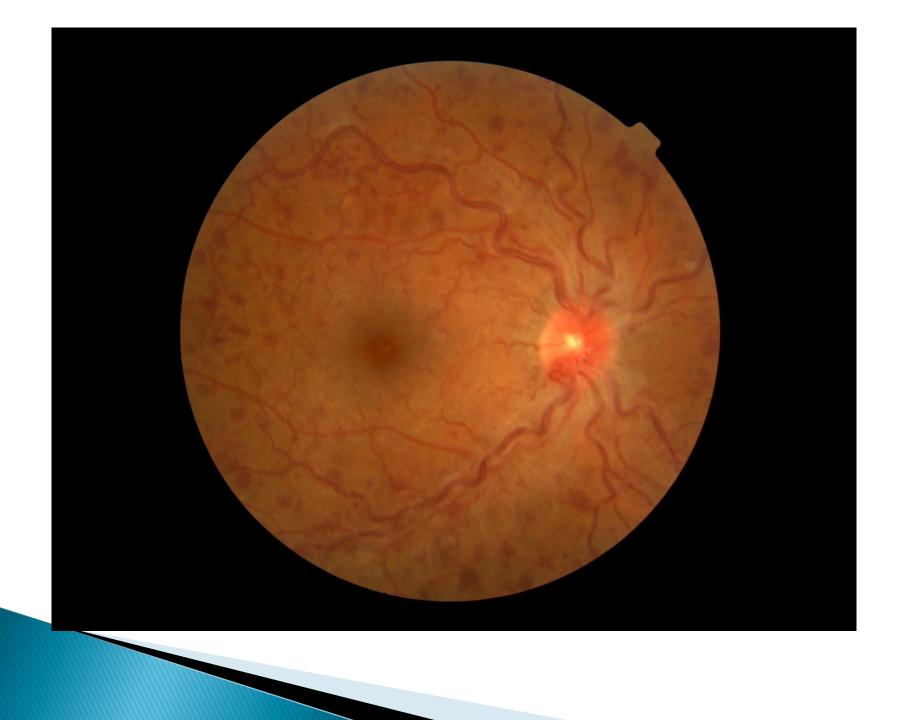
- CLINICAL CHARACTERISTICS
- Ocular neovascularization in venous Occlusion
- Papillophlebitis
- HEMICENTRAL AND HEMISPHERIC RETINAL VEIN OCCLUSION

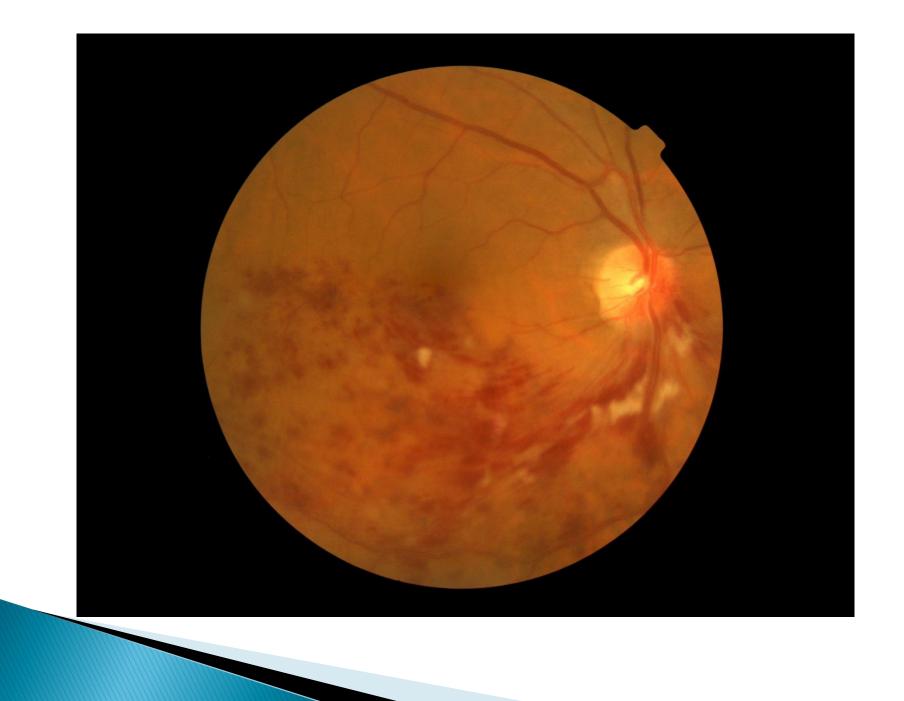


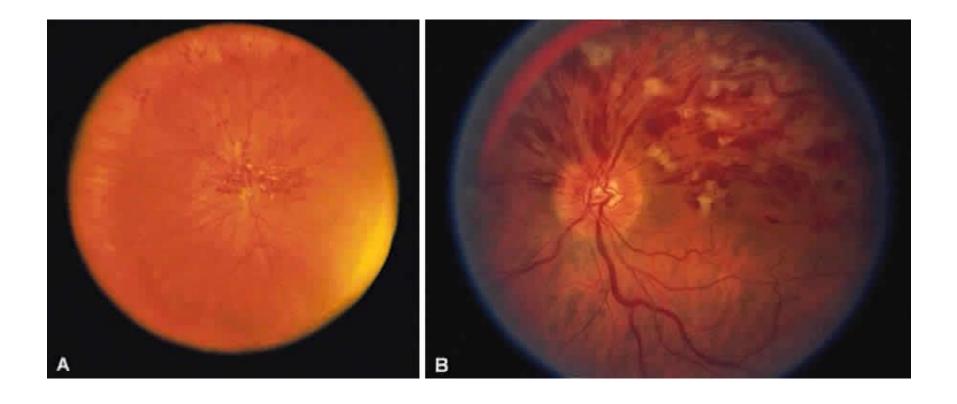


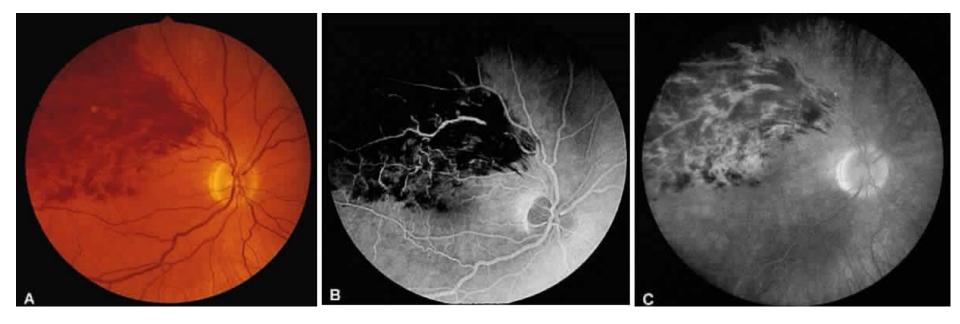


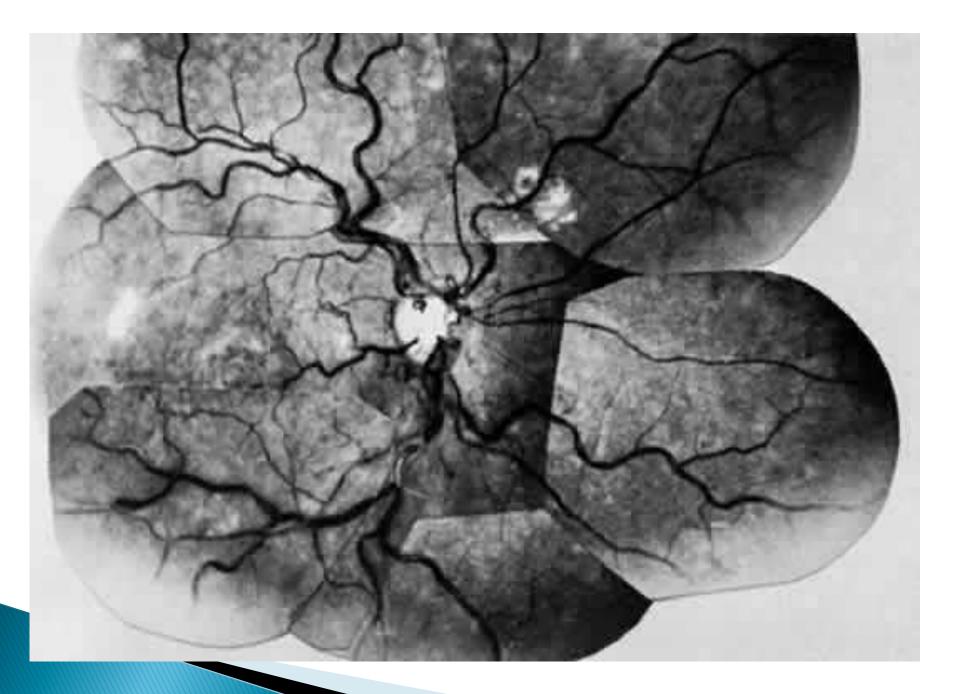












## TREATMENT

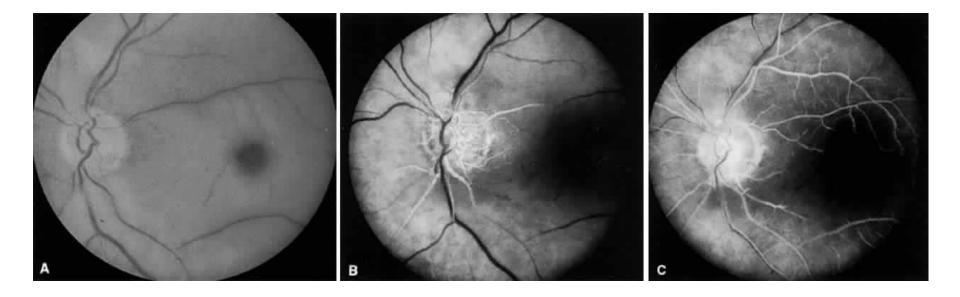
Treatment of an underlying systemic condition, anticoagulants, fibrinolytic agents,, carbogen inhalation, cholesterollowering agents (clofibrate), corticosteroids, prostacyclin, aspirin, ticlopidine (an inhibitor of platelet aggregation), isovolemic hemodilution, and a surgical procedure that involves cutting both the scleral ring and the dura of the optic nerve.

- Once neovascularization in the anterior segment is detected, panretinal photocoagulation should be instituted promptly. neovascular glaucoma responds poorly to any type of treatment.
- In BRVO macular edema were treated with the argon laser in a "grid" pattern in the area of capillary leakage

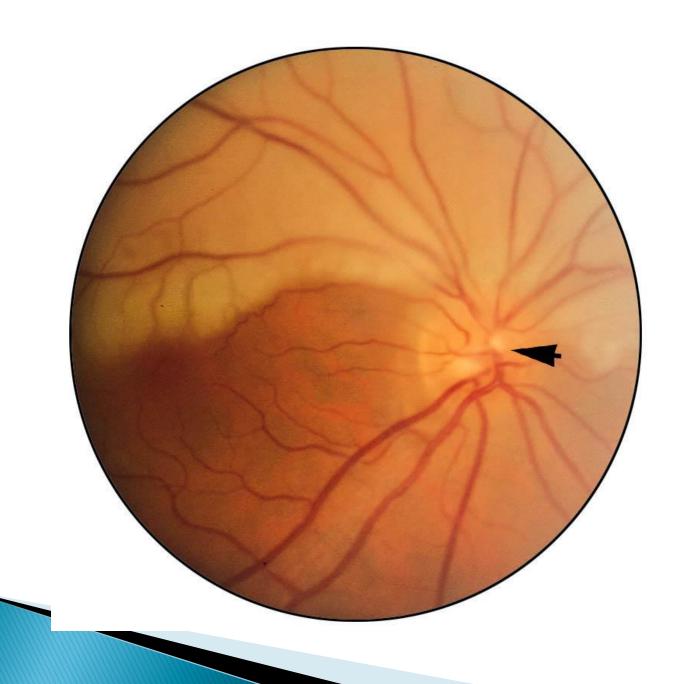
### CENTRAL RETINAL ARTERY OBSTRUCTION

 sudden painless loss of vision. The appearance of a cherry-red spot in the fundus is characteristic. Segmentation (boxcarring) of the blood column

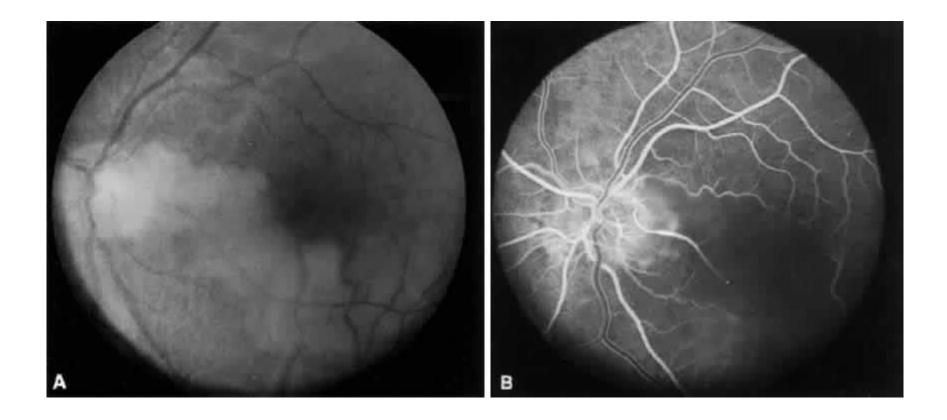














- Conditions Associated With Arterial Obstructions
- Systemic Conditions associated with embolus formation
  - Arterial hypertension<sup>30,31</sup>
  - Carotid artery disease
  - Cardiac or valvular disease<sup>30,240</sup> Myocardial infarction (mural thrombus)<sup>257</sup> Tumors
- Pancreatitis<sup>273,274</sup>
- Purtscher's retinopathy<sup>275,276</sup>
- Intravenous drug abuse<sup>29,277-280</sup>
- Scalp, nasal, facial, or orbital corticosteroid injection<sup>281-288</sup>
- Subacute bacteiral endocarditis<sup>31</sup>
- Heart surgery<sup>289</sup>
- Atrial fibrillation
- Cardiac arrhythmias
- Amniotic fluid
- Systemic cholesterol microembolization syndrome<sup>290</sup>

- CoagulopathiesPlatelet and factor abnormalities<sup>29,31,45,291-298</sup>
- Pregnancy<sup>29,31,299,300</sup>
- Oral contraceptive<sup>29,31,301,302</sup>
- Sickle cell disease<sup>30,302-307</sup>
- Homocysteinuria<sup>308-310</sup>
- ▶ Use of nasal spray<sup>311</sup>
- Hyperglobulinemia in hair cell leukemia<sup>312</sup>
- Anticardiolipin antibody 55,313,314
- Lupus anticoagulant antibody<sup>31,313</sup>
- Thrombocytopanic purpura
- Dysplasminogenemia<sup>315</sup>
- Ulceraturic colitis<sup>316</sup>
- Homocystenemia<sup>46</sup>

#### Ocular and Periocular

- Blunt trauma<sup>304,352</sup>
- Compression <u>56,147,353-355</u>
- Penetrating injury
- Retrobulbar injection<sup>303,356-359</sup>
- Orbital fracture repair<sup>360</sup>
- Belpharotplasty<sup>361</sup>
- Common Carotid dissection<sup>362</sup>
- Increased intraocular pressure<sup>305</sup>
- Prepapillary arterial loops<sup>363-369</sup>
- Optic nerve drusen<sup>323,366,367</sup>
- Optic neuritis<sup>368</sup>
- Orbital mucomycosis<sup>369,370</sup>
- Toxocara canis infection<sup>371</sup>
- Toxoplasmosis retinochoroditis<sup>372</sup>
- Papilledema<sup>373,374</sup>
  - Carotid artery disease

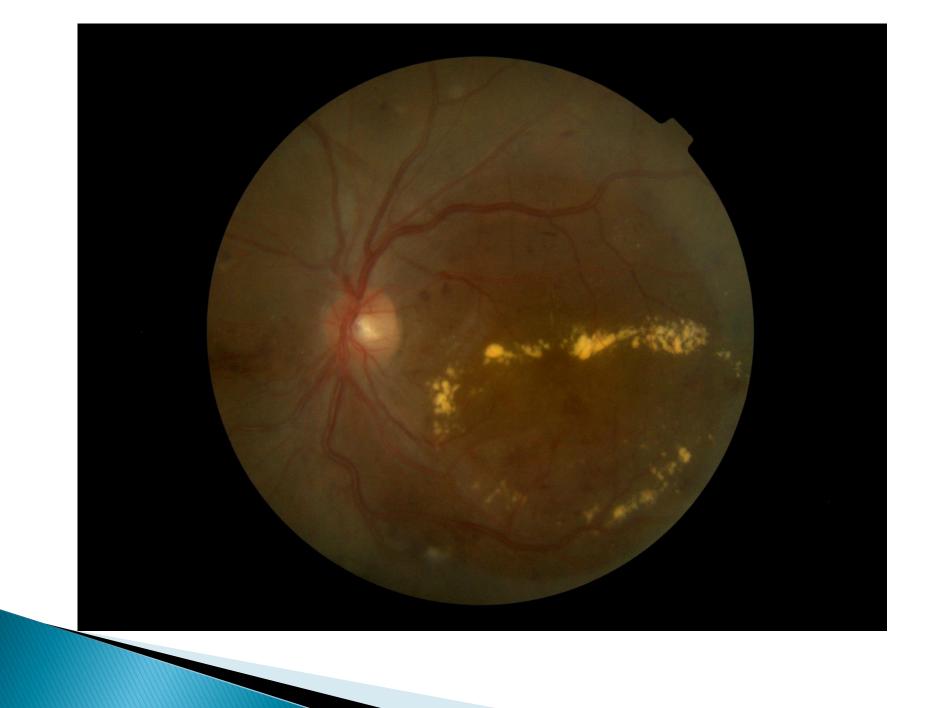
## **Diabetic Retinopathy**

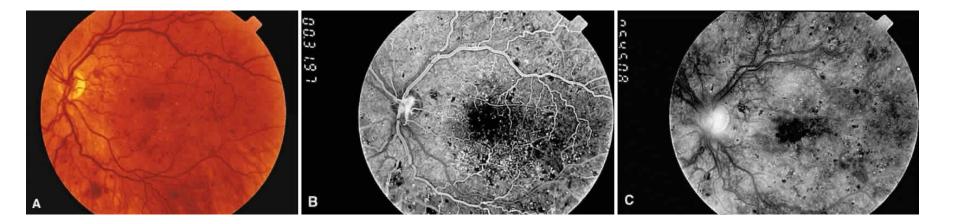
- Exposure to hyperglycemia over an extended period results in a number of biochemical
- and physiologic changes that ultimately cause vascular endothelial damage. Specific
- retinal vascular changes include the loss of pericytes and basement membrane thickening,

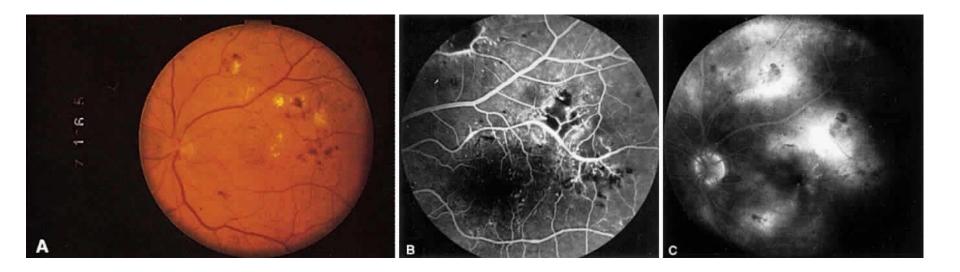
- increased platelet adhesiveness
- increased erythrocyte aggregation
- abnormal serum lipids
- defective fibrinolysis
- abnormal levels of growth hormone
- upregulation of vascular endothelial growth factor (VEGF)
- abnormalities in serum and whole blood viscosity

- The potential for visual loss in patients with diabetic retinopathy can be associated with
- the following conditions:
- sequelae from ischemia-induced neovascularization
- diabetic macular edema
- ischemic macular changes

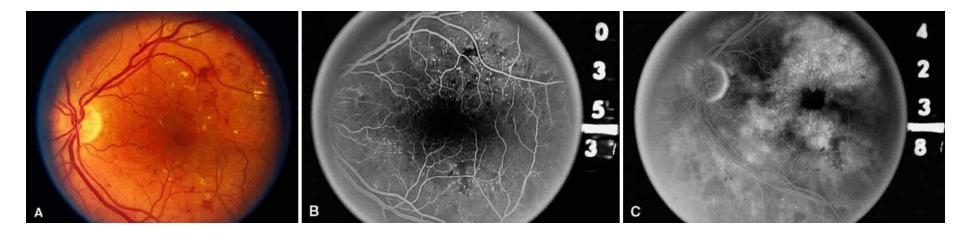
- Diabetic retinopathy is classified into an early stage, 11Onproliferative diabetic retinopathy
- (NPDR), and a more advanced stage, proliferative diabetic retinopathy (PDR).
- Characteristic
- findings in NPDR include microaneurysms, dot-and-blot intraretinal hemorrhages, retinal
- edema, hard exudates, dilation and beading of retinal veins, intra retinal microvascular
- abnormalities (I RMA), nerve fiber layer infarcts, arteriolar abnormalities, and areas of
- capillary non perfusion.

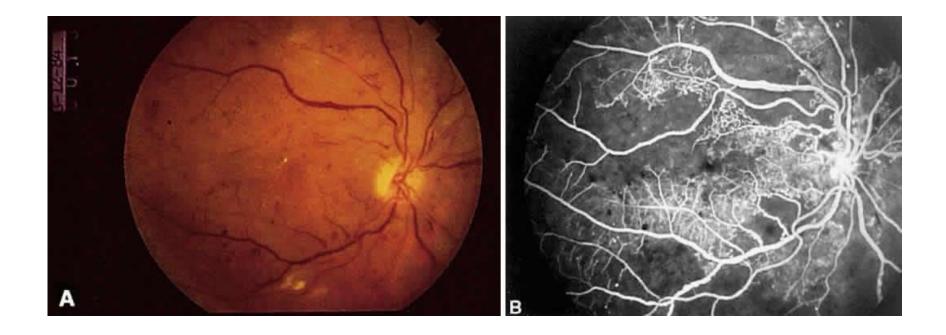


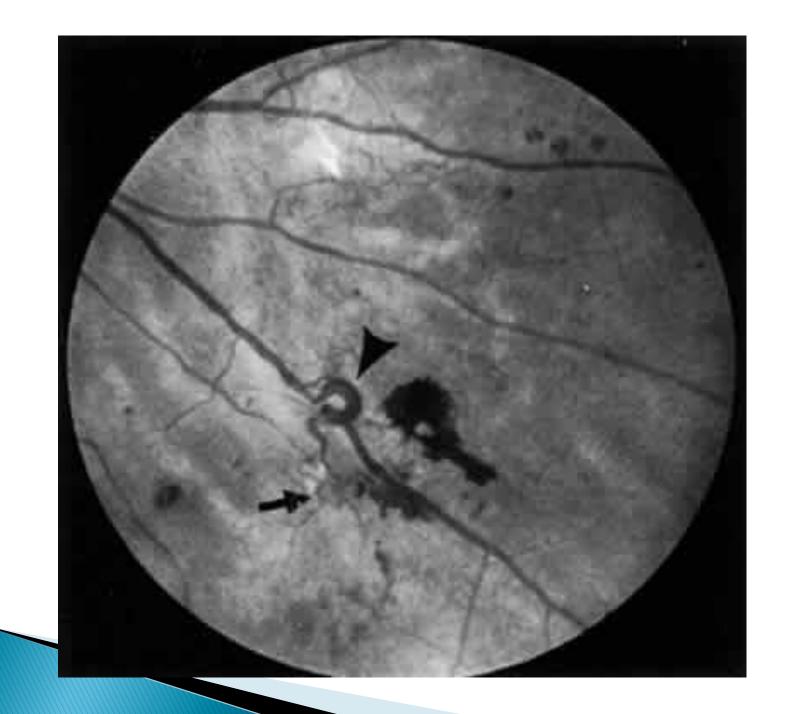










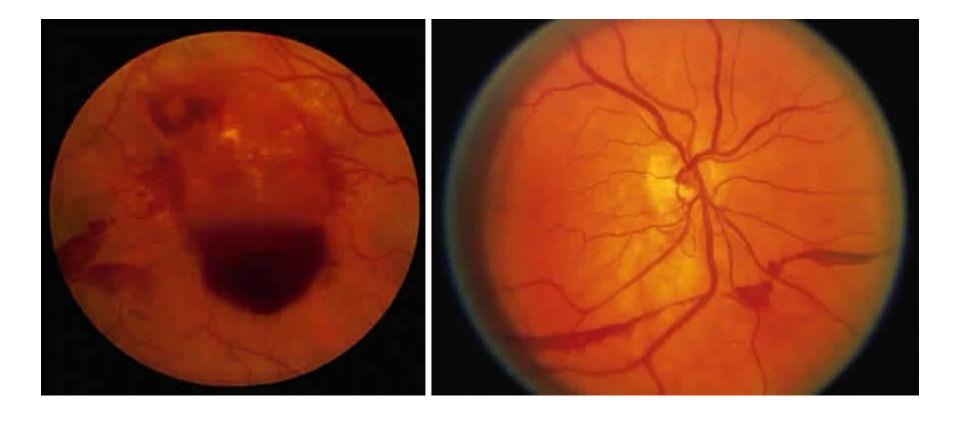








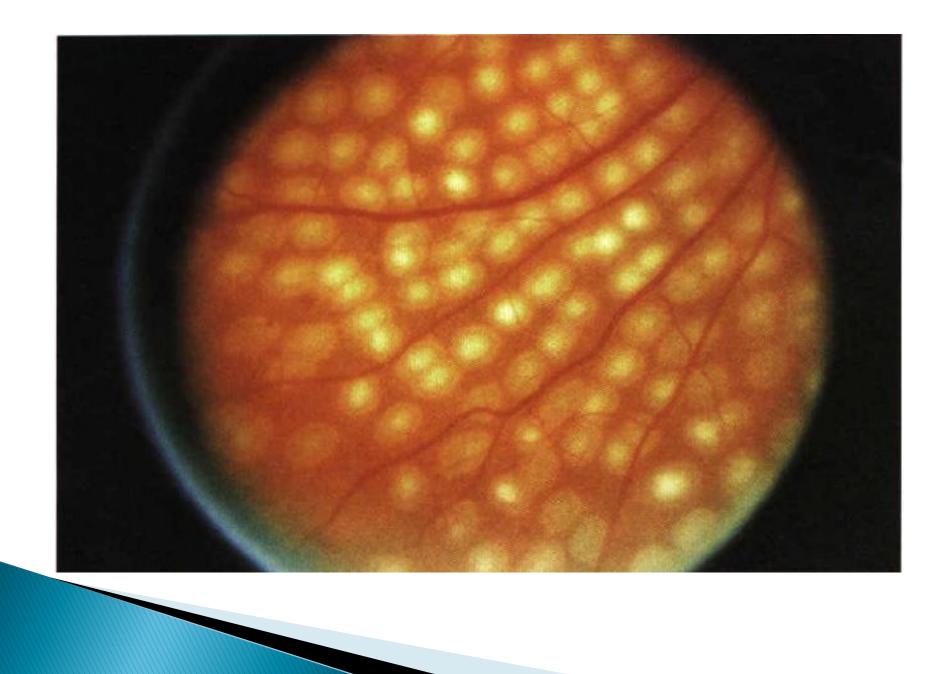


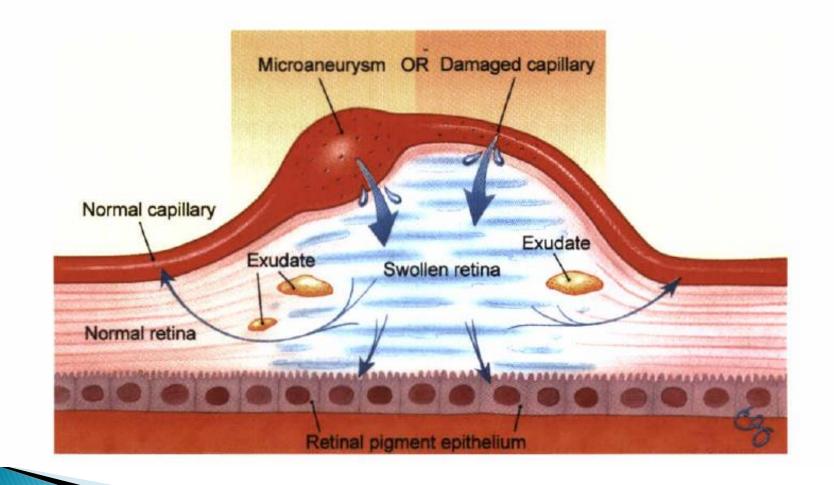




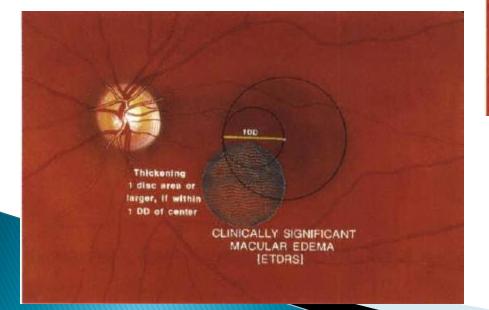








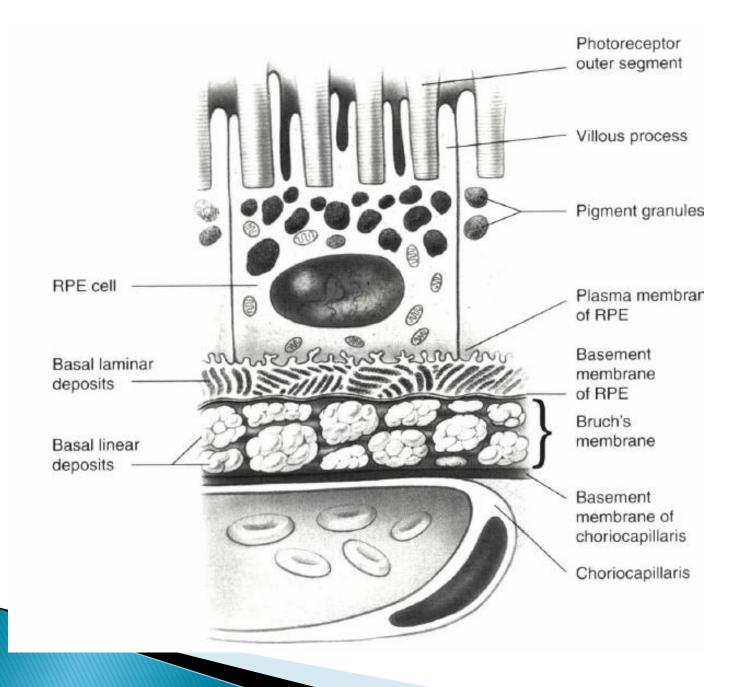


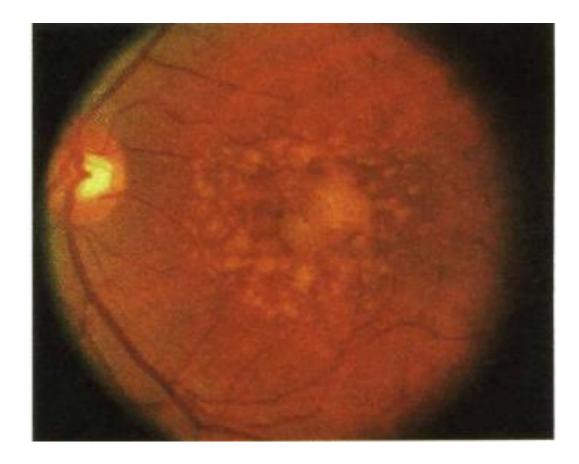




#### Age-Related Macular Degeneration

- Nonneovascular Abnormalities in AMD
- The hallmark of the nonneovascular (nonexudative) form of AMD is drusen; other indicators are abnormalities of the RPE, including geographic atrophy and areas of hyperpigmentation.





#### Neovascular AMD

- The hallmark of the neovascular form of AMD is the presence of CNV
- the presence of subretinal fluid
- subretinal or sub-pigment epithelial blood
- subretinal or intraretinal lipid
- subretinal pigment ring
- irregular elevation of the pigment epithelium
- subretinal gray-white lesion
- cystoid macular edema
- a sea fan pattern of subretinal small vessels

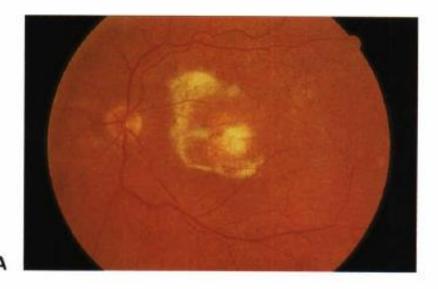
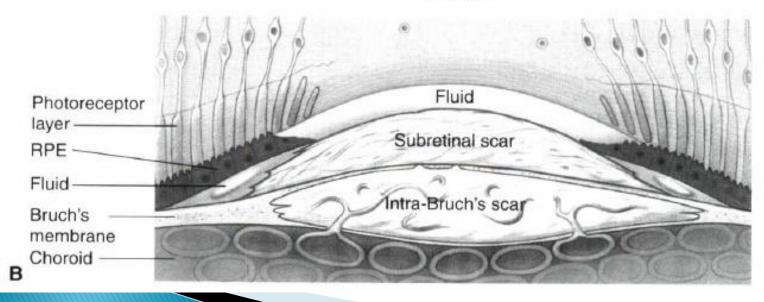
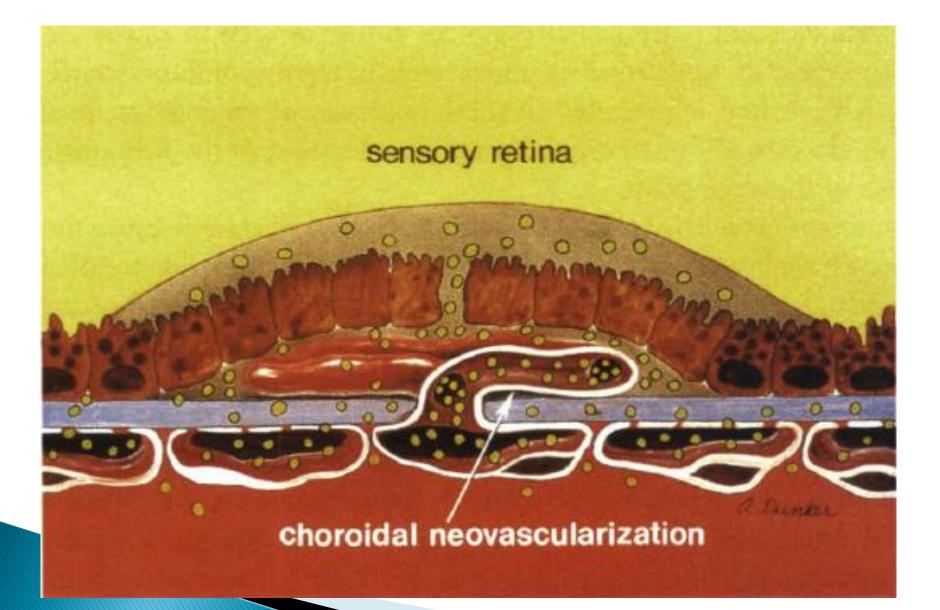


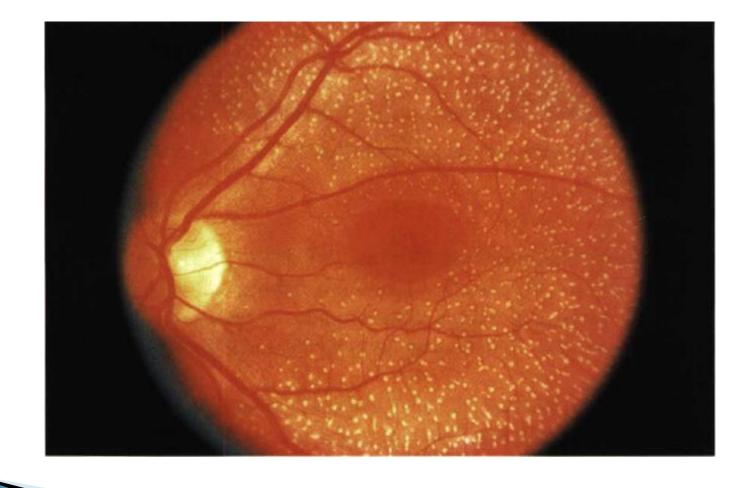
Figure 4-4 A, Endstage CNV that has progressed to fibrovascular scar, often called a disciform scar. B, Schematic cross section of a disciform scar depicting sub-RPE fibrovascular component and subretinal fibrocellular component. Note partial loss of photoreceptor layer overlying the disciform process and disturbance of the RPE and Bruch's membrane. (Part A reproduced with permission from Bressler NM, Bressler SB, Fine SL. Age-related macular degeneration. Surv Ophthalmol. 1988;32:375–413. Part B illustration by Christine Gralapp.)





# Hereditary Retinal and Choroidal Dystrophies

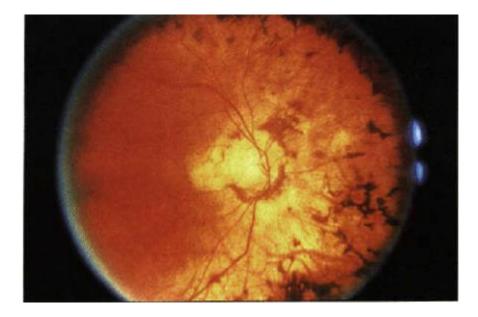
- Leber congenital amaurosis
- Retinitis pigmentosa (RP)
- Cone degenerations
- Achromatopsia
- X-linked blue-cone monochromatism
- X-linked cone dystrophy with tapetal-like sheen
- Congenital stationary night blindness
- Rod-cone degenerations (RPI
- Choroideremia
- Secondary RP, including some storage diseases
- Progressive retinitis punctata albescens
- Cone-rod degenerations/dystrophies
- Autosomal dominant
- Autosomal recessive
  - X linked recessive

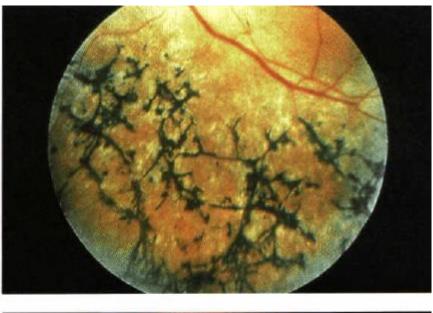


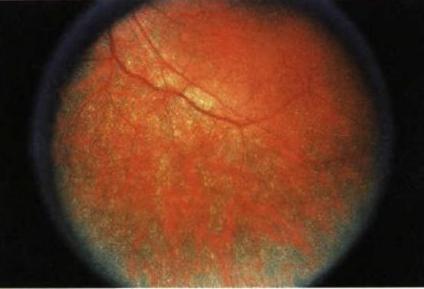
# Retinitis Pigmentosa

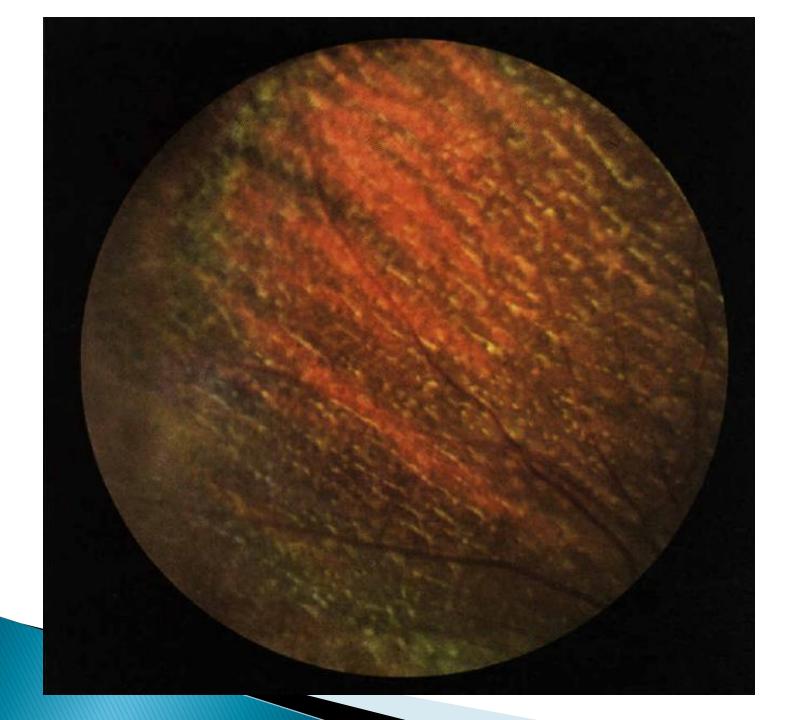
- Retinitis pigmentosa was defined as a group of hereditary disorders that diffusely involve photoreceptor and pigment epithelial function characterized by progressive visual field loss and abnormal ERGs. The pigmentary retinopathies can be divided into two large groups: (I) primary RP, in which the disease process is confined to
- the eyes, with no other systemic manifestations, and (2) secondary pigmentary retinopathy,
- in which the retinal degeneration is associated with single or multiple organ system
- disease.

Typical fundus findings in RP include arteriolar narrowing, variable waxy pallor of the disc, and variable amounts of bone spicule-like pigment changes.









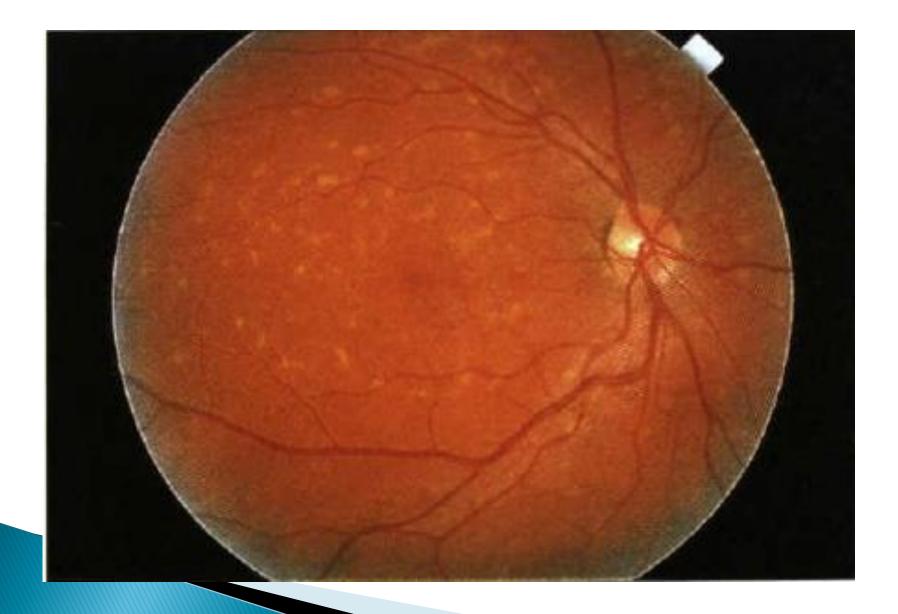
### LCA

severely reduced vision from birth associated with wandering nystagmus and severely impaired ERG responses from both cones and rods. seldom obvious fundus changes. Later, round subretinal black pigment clumps develop in many patients, Visual function can range from 20/200 vision in some cases to no *light* perception in others. the *oculodigital* reflex. Cataracts and keratoconus may be seen in older children.



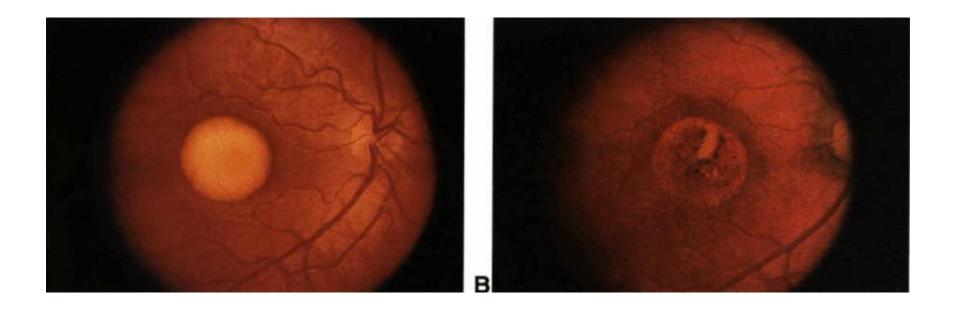
# **Macular Dystrophies**

- Stargardt Disease (Fundus Flavimaculatus)
- is the most common juvenile macular dystrophy and a common cause of central vision loss in adults under the age of 50.

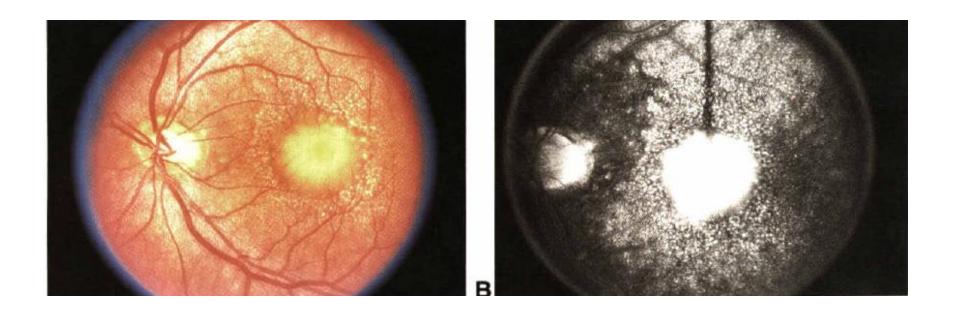


- Differential Diagnosis of Bull's-Eye Maculopathy
- Stargardt disease
- Cone and cone-rod dystrophies
- Chloroquine retinal toxicity
- Age-related macular degeneration
- Chronic macular hole
- Central areolar choroidal dystrophy
- Olivopontocerebellar atrophy
- Ceroid lipofusciniosis

# Best disease, or Best vitelliform dystrophy



## Familial (Dominant) Drusen



## Choroidermia

