INDEX

NAME:_____ STD.:_____ SEC.:_____ ROLL NO.:_____ SUB.: Retina

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T&T/0517081 B

Central Serous Chorio-retinopathy

Def — disease characterized by localized & serous detachments of neurosensory retina with or without focal pigment epithelial detachments & altered RPE.

Forms

- Acute: 3-4 mth; Colour defects
- Chronic: >5yrs; widespread RPE atrophi ↓ FAF with or without SRD; irregular RPE detatchments; Longstanding Cystoid Cavities

Patho

(a) Widespread Choroidal Circulatory Atn
↓
RPE disturbances

(a) Hyperpermeability of choroid

Central Serous Chorio-retinopathy

Definition

Idiopathic disorder characterised by a localised serous detatchment of sensory retina at the macula secondary to leakage from choriocapillaries through one or more hyperpeerbeable RPE sites

Hyperpermeability at choroid → Leak through RPE → NSD

Risk factors

♂ : ♀ 6:1 → 3:1

Young & middle age Caucasian man
Steroid administratⁿ (Triamcinolone) Any route
Cushing syndrome, Endogenous Cortisols
Helicobacter pylori Inf, GERD
Pregnancy
Psychological stress, depression, Antipsychotic medication
Sleep apnoea syndrome.
Psychiatric disturbance — Type A personality
Abn in Coagulatⁿ & Platelet aggregatⁿ factor
Oral MEK Inhibitors (Binimetinib) ✓
PDE-5 Inh.
Unₓ — HTN

Epidemiology

ARMD ↘ DR ↘ RVO ↘ CSCR — Vision threatening disease
Older pt >50yr — Bil. Chronic, > ♂ , >CNV.

Symptoms

Blurred vision. • Rel Central Scotoma. • Metamorphopsia
• Dyschromatopsia • Hypermetropizatⁿ • Micropsy
• ↓ CS. • Sup field defect (If Exu RD) • Prolonged after Images

GURHARIYASHOPINGCENTER
Invoice No:

6/9 - 6/18 hypermetropia improving ⊆ + (convex lens) SRF.

CLASSMATE
Date 2
Page

Signs

Well demarcated round/oval NSD over post pole.
↳ Congenital Optic Pit D.D of NSD
∴ look OD also.

1> serous PEDs

Loss of foveal reflex

Yellow dots at ~~fovea~~ inner surface of RPE — Phagocytosed photoreceptor outer segments

Yellow discolouration of fovea → ↑ visibility of Xanthophyll pigments.

SRF ⊆ Transparent

Subretinal deposits

Tuesid — fibrin deposition

Resolve

Coalesce to form Subretinal fibrosis

↓ in vision permanent.

Inferior Bullous Exudative RD.

RPE change — Atrophy

2° CNV.

PED — Well circumscribed orange coloured elevation with slightly darker rim that can illuminate ⊆ slit beam from side.

When RPE cell & ass BM separate from underlying Bruch's Memb

<0.25DD Occult CNV, PCV, CSCR.

Cause

Classic acute CR.
NSD > 2DD
3-6mth
Colour discriminal defect

Chronic diffuse Retinal Pigment Epitheliopathy (DRPE)

Bullous RD.

Chronic
>12mth
Initially DRE
Widespread tracts of RPE atrophy
Chronic SRF
Irreg RPE detachment
Longstanding Intraretinal Cystoid Cavities

Spont resolution — 3-6mth 80%.
Recurrence — 50%

Type I — NSD Type II — PED

Stress, Isch, Inflammation

Pathogenesis

1. Hyperpermeability of choroid (staining of inner choroid in ICG)
2. Thickened choroid.
   - ↑ Tissue hydrostatic pre
     - Overcome barrier fxn of RPE → PED
     - Breach in RPE → fluid in SR space
3. Chorio-Capillary non perfusion
   - Choroidal venous dilatn & Congestion
   - Actively pumping fluid in SRF.
4. Focal loss of polarity of RPE (pump fluid from choroid-Retina)
5. Aldosterone/Mineralocorticoid pathway
   - Vasodilatn, thickening & leakage of Choroidal vessels
   - elongation of RPE cells. & accumulation of SRF.

Investigation

Amsler grid
OCT
FA
FAF
ICGA

Classic

Ocular findings
As in Signs
PED - Some feuste.
Quiscent CSC + FA - No Leak
Chronic ICG - Hyperperm of CV.

FA . Several hyperf leaks at level of RPE.
Early - focal dot like HF (dye Leak)
Late accumulates beneath NSD.

Smokestack - Sup > Laterally
Minority, dramatic
Convectional currents & protein gradient ≤ protein conc ↑ fluid abs under NSD

Ink Blot - 90%. Uniform filling pattern

Leaks - N>T S>I

ICG Mid phase ≤ in Inner Choroid
Larger but ↓ prominent
↑ Patches in Choroid ≤ Subtle filling of Larger CV in Later

DRPE/Chronic
Thinning of retina.
cystic changes
- Atrophy
- Focal hyperpig
- RPE hyperplasia - Bony spicules

granular ↑F (Atrophy) + Subtle Pindistrict leaks

Bullous RD
Inferior periphery
Subretinal fibrinous exudates
Shifting SRF
Fochoplant pt

↳ → No. of area of hyperp Increase >>

FAF

Acute – ↑ AF (↑ Metab) hypo
Chronic ↓ " ↓ " hyper

◉ Subretinal deposits

fibrin — Lipid → Old pt → CNV — Macrophages — Outer photoreceptor segments

Outer retinal small dots → diffuse yellow flecks

CSC – discrete, hard edged at border of NSD

OCT

Empty vaulted area under NS retina ± NSD
Subclinical cystoid Macular deg / foveal atrophy
PEDs
Poor Visual acuity – IS-OS dysfxn

classmate
Date 4
Page

(1) Pachychoroid conditions

DoD

	ARMD	CNVM	PCV
OCT –	Neovasc complex	Occult – Confused flecks of subretinal lipids / actually CSC	
FA –	No leak		

Harada

Inherited Retinal Macular dystrophy
Pigmentary alteration
↓VA

Subretinal deposits
↓
Retinitis
Choroidal tumour

(2) Anatomical
(3) Vasc disorder
(3) Harada / Post scleritis

~~FFA~~ ICG

CNV

Similar – NSD
PED
mottled depig
↑ plq
PPE dze
Subretinal fibrin deposit

CSCR
Multifocal
Bil
CC ↑ permeab
Temporal &
Topogr character.
» mid phase
Late phase – dispersion of dye ≤ neg staining of 2-3wk later

CNV
Unilateral
Unifocal
↑ Contrast
» late phase — » apparent

Notched PEDs
Subretinal Epithelio Bld
gen of New Bld vessel growth
Punched out Chorioretinal scars

Punched out CR scar
In Ocular Rishoplasmosis Synds

Path Myopia
Lacquer Cracks
Area of Chorioidal atrophy

Ocular CNVI finding

Co rupture
Break in Bruch Mems

ARMD
Drusen
Plg Clumping

FFA
Lacy Vascular pattern of ↑F ≤ ↑ leakage & staining throughout.
Specific feeder vessel

Harada/Post scleritis
→ Intraocular inflammation
Optics, vitreous
patches of yellow disc of post pole
ONH staining on FA.
USG — thickening of choroid — T sign
→ Extraocular — Headache
Neck stiffness
Vomitting

NSD — ONH pits — Bilayer detachment by retinoschisis
x leak on FFA

Appt
Rheg RD — x Leak
✓ Hole/Tear

Vasc disorder — Malignant HTN — Syst HTN, Elschnig spot
Shifting fluid.

DD of SRF — CSCR
Malignant HTN
Toxemia of Preg — HTN, Proteinuria, Edema
Inflammatory condition — Harada VKH
CNVM
Post Scleritis

Cause of Poor Vision in CSCR

Leak from Macula — classmate — Date 5 — Page

Neal

Treatment

Observation

CS Rs → Stopped

Others

Aspirin
β Blockers
Mifepristone
Eplerenone.

Laser — shorter dura" → Micropulse diode laser → RPE site of leakage → resol ↓ of SRF → ↓ Retinal damage

× affect VA.

DRPE — Thermal grid (Argon) laser

Amt of laser uptake ∝
- SRF
- ° of pig on RPE.
- ° of RPE detachment
- λ of laser used

Σ E of laser — CNV Scotoma
RPE scar Expansion

Ix for laser
- Symptoms > 4mth
- Leak sites > 375μ from fixat"
- H/O CSC in fellow eye ≤ unfav Outcome
- Need/desire for Rs

Spot size — 100-200μ
Power — 100-150mW
Time 0.1-0.2s
Peripheral Leak first
Aim — dull gray PC

Laser → Hmrg, ↑ Turbidity of SRF, RPE Thickening adj to Laser — 2° CNV
2° CNV — Nodular, Crescent shape asec of ↑F

PDT
- CNV — Mech Known
- CSR — Not. — Narrowing of Choro capillaries → C. hypoperf → CV remodeling

30-50% of dose for CNV + 50% Light Intensity → ↑ Light Intensity → ≫ choroidal ischaemia

PDT ≤ Verteporfin
↳ csck Chronic

Anti VEGF

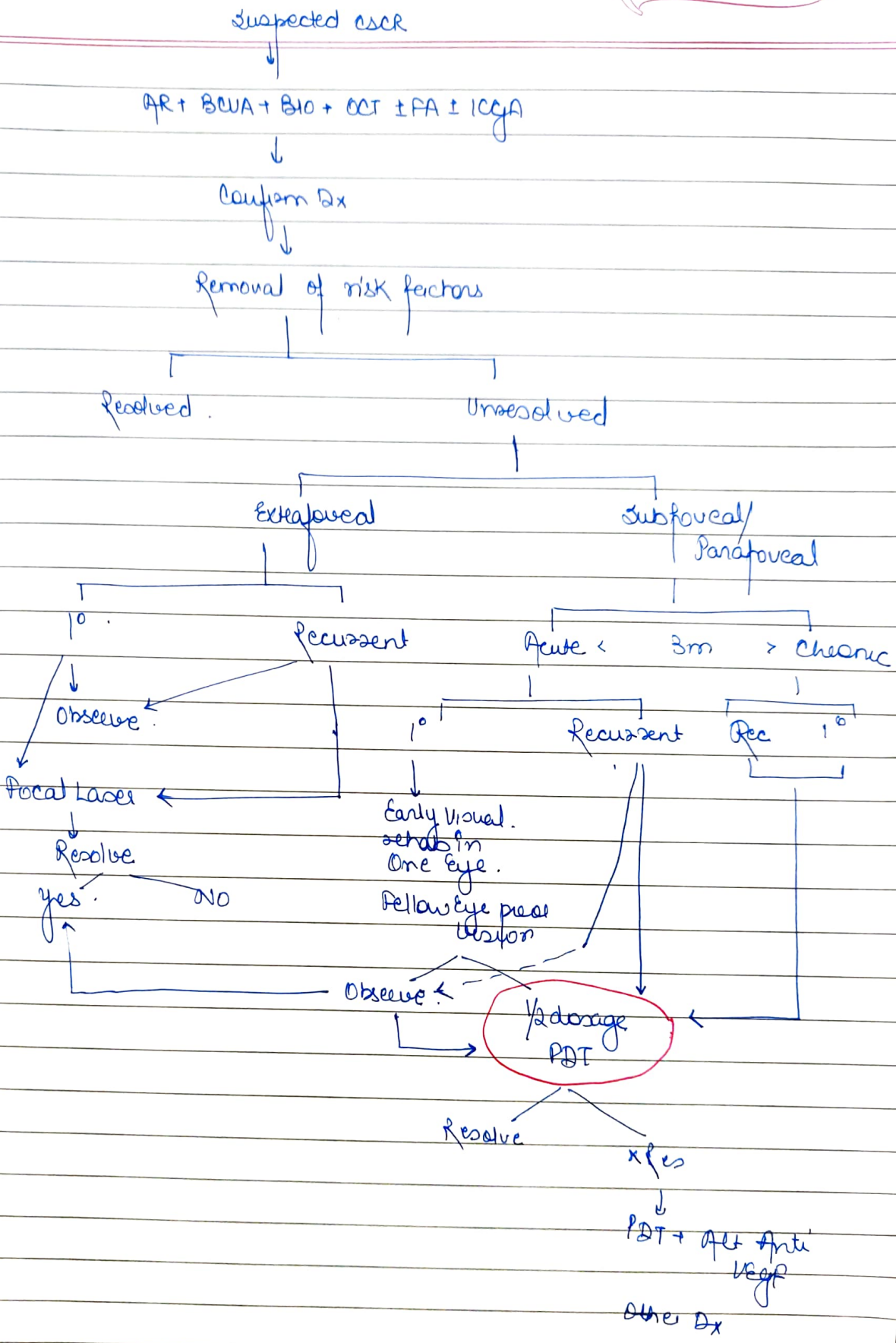
PDT

Conventional	Safety Enhanced	↓ Laser fluence
Normal dose Normal fluence	↓ dosage	
Nedlase AMD		
Verteporfin (Visudyne)		
6 mg/m²	3 mg/m²	
30ml over 10 min infusion		
Laser — 689 nm | 15min later	689nm | 3min later	
Light Energy — 50 J/cm²	50 J/cm²	
I - 600mW/cm²		
T - 83s	83s	
Spot size dia - 1000μ + Lesion		

Post Rx Visual loss
Potential Choroidal Isch
RPE atrophy
develop^n CNV

TTP (Transpupillary thermotherapy)

Long pulse
Low Energy
810nm infrared Laser — Ch. V. Thrombosis
Juxtafoveal leakage


Suspected CSCR
AR + BCVA + BIO + OCT ± FA ± ICGA
Confirm Dx
Removal of risk factors
Resolved
Unresolved
Extrafoveal
Subfoveal/ Parafoveal
1°
Recurrent
Acute < 3m > Chronic
Observe
Focal Laser
Resolve
yes
NO
1°
Recurrent
Rec
1°
Early visual. in One Eye.
Fellow Eye preservision
Observe
½ dosage PDT
Resolve
x Yes
PDT + Alt Anti Vegf
Other Dx

Laser

Discrete, solitary, Extrafoved leakage point & persistent RSD

Diode - 810nm

PDT

Multiple leak, Subfoveal & juxtafoveal leak, Bullous RD

ICGA guided PDT

Coat's Disease

Primary Retinal Telangiectasia
» Capillary bed

- dilatation & tortuosity of retinal blood vessels
- Multiple aneurysm.
- Vascular Leakage
- Deposition of Hard exudates

Types

- Idiopathic Macular T.
  - confused ≠ DR, RVO previous
    - Type 1
      - Aneurysmal.
      - Lesser miliary A.
      - 1 eye
      - Peripheral retina + Macula.
      - ♂ Middle age
      - Vision loss — Mild mod.
      - T, Manens
      - Large A.
      - ME
      - Chr Leak & Lipid dep
      - OCT → Retinal Thick CMO.
      - Localized Exu RD.
    - Perifoveal
      - » Common
      - Deg of Muller Cells
      - Both eyes
      - Perifoveal
      - ♀ = ♂ Middle age
      - Worse
      - Distortion
      - Fine, Superficial, crystalline Retinal deposits
      - ⊕ ⊥ venules
      - ~~Subretinal~~ NV ⇒ Angiomatous
      - Foveal atrophy
      - Small RPE plaque
      - x Lipid dep
      - x A.
    - Occlusive
      - Rare
      - Late
      - Poor Prog
- Coat's Disease

FA

T. C

Late leak.

CMO

Cystoid spaces

Early leak

x CMO

Rx

Lasers.

Anti VEGF

Anti VEGF

OCT

- hyporeflective inner retinal spaces of variable size

Disruptn of IS - OS

Pigment clumps

Foveal thinning

T. → hyperreflective inner retinal dots

FAF

Central foveal ↑AF

↑ extent

Pigment → ↓AF

Macular Pigment Optical density Imaging

6° outward

Oval

Coats

Early Childhood
Idiopathic Retinal telangiectasia

75% ♂. 95% 1 eye
1st decade
NDP gene — Norrie dis

DD → Lebers Miliary An — (mild form)
Leucocoria ~~dis~~ — Retinoblastoma

Symptoms — Unilateral Visual loss
~~Loss~~ Strabismus
Leukocoria

Fundus — T.
fusiform focal A. arteriolar dilatation
Inf / Temp. / Quad B/w Equator & Ora
Intra & Subretinal Exudate
Ext Exudative ~~+RD~~

Complication — Rubeosis Iridis, glaucoma, uveitis, cataract, Phthisis bulbi.

FA — ↑F of T & An.
late staining & leakage

Rx

Observe
Laser Ablation
Anti VEGF.
Intravit Triamcinolone
Cryo → Laser Photocoag
VRS x
Enucleation

Stages of Coat's Disease

1
- Retinal telengiectasia only
- 2 Telengectasia & Exudation.
- 3 Exudative RD
  - a Subtotal.
  - b Total
- 4 Total RD & glaucoma
- 5 Advanced end stage disease

Children

Massive & diffuse → common

Lipid deposit & Exudation

Hmrg

Adult Onset

Limited area of involvement
Slower apparent progression of disease
Hmrg near large vascular dilatation
Equatorial & peripheral region
Localized & limited

» ⊆ bleeding localized to MA

Coat's Disease

Def Idiopathic retinal telangiectasia ass ⊆ intraretinal exudation & freq exudative RD without signs of appreciable retinal & vitreal reaction.

Unil ♂ > 8 - 16 yrs age

Light bulb telengiectasia → due to large amt of exudates accompanying the disease

In Adults r/o Coat's Rxn or Coat's Syndrome.

Pathological processes

Breakdown of BBB at endothelial level

↓

Leakage of plasma into vessel wall

↓

Thickening of parts of vessel wall

↓

Sausage like shape of vessel

Abn pericytes & endothelial cells in RB vessels

↓

Deg → Abn R. Vasc

↓

Aneurysm formation

↓

Closure of Vessel

↓

Ischaemia.

↓

Leakage of cholesterol rich exudates
Hmrg, Cyst, Edema, lymphocytic infiltration
fibrin deposit into Retina.

↓

Thickening of Retina
Exudative RD.

Associations → Consequence of NDP gene
↓
Def of Norrin.
↓
Retinal Vasculogenesis.

Ass c → •) familial renal retinal dysteophy
Senior Loken Syndrome
•) AD fascioscapulohumeral Mal dysteophy
Hallermann steiff Syndrome
•) Turner's syndrome

C/f → Strabismus, Leucocoria, impaired vision
heterochromia of Iris, Nystagmus.

fundus findings → R. telengiectasia
Aneurysmal dilation (light bulbs)
sheathing of vessels
Inf & temp Quad
Aneurysm → fusiform > Saccular

Macular fibrosis
formation of subfoveal nodules
Macular hole
ME +/-

RB

Mass lesion
c Calcification

vs C

Submacular Nodule
Phthisical eyes

~~Incontinentia~~

Incontinentia Pigmenti

Male X linked.
Skin, hair, teeth, nails & eyes

ROP

BL
Premature baby

Ocular toxocariasis

Acq.
Unil.
Uveitis

PFVR

Peripheral retinal
- fibrovasc Mass
- Retrolental Memb
- Exudative RD

PHPV

- Retrolental fibrovasc memb
- Microphthalmic
- Nonrhegmatogenous RD
- Uni/Bil
- Sporadic/Familial

Hemangioblastoma Von Hippel

Phakomatosis ~~cross~~

feeding artery — Drain" Venule

large dilated feeder vessel

Retinal exudates

TRD, VH ✓

D.D

Ix

FFA

Tel → Early hyper F
Exudation — Blocked Hypof
Aneurysm — Light bulb dilatation

USG

Subretinal opacities due to cholesterolosis + ve from exudates + RD.

CT

Rb
Solid Tx ⊆ Calcification
↓
bt. 46% ⊆ out Calci

Coat's
x lesion
↓
20% ⊆ Submacular nodule + Calci

MRI

Rb
Subretinal ~~mass~~
exudation
T1 – Hyper
T2 – Hypo
↑ Solid Tx.

Gadolinium Contrast-

Coat
Solid mass

Hyper on T1, T2

Treatment

UnRx

64% RD

32% 2° gl.

Based on stage of disease

Mild.

Progression

+ → Observation

− → Laser Photocoag Cryo.

Adjuvant

Anti VEGF

IVTA.

Advanced

End stage ≤ painful eye → Enucleation

≤ Comfortable eye → Observe

Adv stage → VRS x → PVD.

Laser & Cryo

Argon laser.

May be 5 sitting

↓

If thick lesion

↓

Cryo Rx — Upto 2 quad

≤ gap of 1mm

EALES DISEASE

Idiopathic occlusive peripheral periphlebitis

Indian young males >
↓ Caucasians

3 overlapping stages : Inflammatory
Occlusive
Retinal neovascular

Etio – Tubercular protein HST
Tubercular vasculitis

Dx » Clinical

Symptoms – Floaters
Sudden vision loss – VH.
Mild ant uveitis
Systemic neurological features

Signs Fundus – Bil
Asymmetrical
Sheathing (Inflam) → Peripheral periphlebitis
Rec VH. (Neovas) – CNP, CME
Sheathing
Sup retinal Hmrg
Cotton wool spots
Pigmented Chorioretinal scars
→ BRVO
→ Peripheral Capillary Non perfusion (temporal >>)
aneurysm, Vascular shunts, NV., Ghost vessels
Recurrent VH (at jxn of perfused/non perfused retina)
NV – fibrous components – FV mems – Traction

NVD

Macular edema may develop

Complicatn
- TRD
- ERM
- NVG
- Cataract

Ix FA — Vasculitis
- CNP areas

CBC
ESR
Retic Count
Bld s/ $\frac{E}{PP}$
Mantoux
Sickle cell Prep
Hb electrophoresis
VDRL
Antinuclear Ab (SLE)
Serum ACE / Lysozyme (Sarcoid)
X ray chest (S, TB)

Vasculitis Cause — Sarcoidosis
- TB

NV — Hypothics

Rx
- Steroids
- AIT
- Scatter photocoag / Cryo to CNP
- complicatn — Vitrectomy

Rec VH

History — Henry Gales

Constipation △ Epistaxis

↓

1° Rec Retinal thrug

↓

Peripheblebitis Retinae (V>>) but A & V both

classmate
Date 14
Page

VMA

Perifoveal vitreous separation with sensing VM attachment

Unperturbed

Reflective lines from perifoveal area into vitreous cavity distorting macular contour with or without accumulation of intraretinal/subretinal fluid

VMT

Abnormal PVD accompanied by anatomical distortion of foveal features

✓

of

foveal morphological features

findings on OCT

Pseudocysts
cystoid macular edema
Macular schisis
SRF

⇓

Symptomatic

↓

Vitrectomy
Ocriplasmin used

VMA | VMT

focal		Broad
<	ISCIGA	>
✓	Vitreolysis Tx	✗

Retinopathy of Prematurity

- Blinding disease of premature babies

Risk factors → POG, BW. ↑ POG → ↓ 27% risk.

10% Premature
↓
30% ROP
↓
10% — Need Rx

Pathogenesis

Vasc. of retina starts posteriorly around ON — 16 wk.
↓
Spindle cells from Adventitia of Hyaloid Artery
↓
Solid cords metamorphose
↓
Come towards Ora

Nasal. 7-8 mth — Temporal. 9 mth

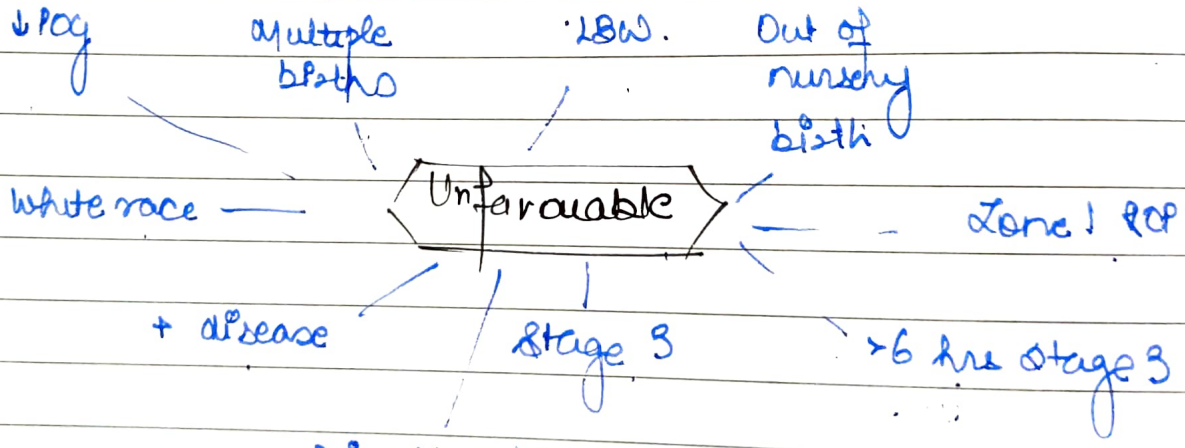
Two phases of ROP develop

Phase I
Hyperoxia.
↓
Cessation of VEGF driven Retinal Vascular growth
↓
Vaso-obliteration
Capillary regression — ↓ ischaemia

Phase II
hypoxia → ↑ vessel growth (after 34 wk POG)
VEGF
IGF - 1
Neovessels around accompany glial cell — contractile properties

Risk factors of ROP

↓ BW
POG
O_2 supplementation.
Light
Vit E def.
Apnoea.
RDS.
Septicaemia, Acidosis, Asphyxia., Anoxia, Shock.
PDA.
Blood transfusion.
IVH.
Bronchopulmonary dysplasia



Classification

Zones

- I
  - d = disc to Macula
  - Zone = 2d
  - ≤ OD at Centre
  - ⇓
  - 2½d
- II
  - ≤ disc at centre.
  - Outer to Zone I
  - touching nasal ora
- III
  - Temporal crescent of retina left behind.

Stages

fn of Vasc ✱ Avascular area.

- I
  - Demarcation line.
- II
  - line gets volume
  - ⇓
  - Ridge.
  - ↓
  - Popcorn Neovascularization
- III
  - Extraretinal Neovascular Proliferation
  - a
    - Sparing Macula
    - Extent
  - b
    - Involving Macula
- IV
  - Subtotal RD
  - a
    - Macula spared
    - No. of clock hrs of highest stage
    - Rush (ARROP)
    - Zone I or Zone II (Post location)
    - → ✓
    - No Ridge
    - NV fronds lie flat
    - AV shunting ≤ in ridge.
    - Ill defined retinopathy
  - b
    - Macula involving
    - + (Plus)
    - Dilatation & tortuosity of ≥ arterioles & veins
    - involving at least 2 Quad
    - non dilating pupil
    - Vitreous Haze
- V
  - Total RD or Retrolental Fibroplasia.

Threshold disease

CRYO-ROP
= chance to progression
regression

Unfav — Macular fold
RD Zone I
PP obsc by Cataract
RL fibroplasia

Def — 5 Contig. & Total | Clock hrs → Stage 3 → Zone I/II (+)

ET ROP
Prethreshold

Type I
15% risk of unfav.
↑ risk, Be threshold Eyes
Early Rx

Stage	Zone	Extent
• any	I	+
• 3	I	+/-
• 2/3	II	+

Type II
↓ severe to req Rx

Stage	Zone	Extent
1-2	I	-
3	II	-

Min risk of poor Outcome →
PMA → 27wk ——→ 45wk —keep screening
till RV comes in Zone III

Screening & Treatment

(US)

All infants < 1500g
≤ 28 wk of POG
> 1500g with unstable course

1st Exam
- Hospital discharge
- 4-6 wk after birth
- 31-33 wk post Menstrual age.

India guidelines → Examine Early in India
< 2kg (1.2kg) (28 wk)
34-35 wk (highest cutoff < 37 wk)
Other risk factors

Timings
- 32 wk PMA
- 4 wk after birth
- Early

Critical period → 30.9 wk — 46.3 wk (PMA)
4.7 — 18.7 wk (Chronological age)

If
- Threshold → wkly.
- ↓ risk of Be-Threshold → fortnightly.

Digital ROP screening < SUNDROP (Stanford) KIDROP. (Karnataka)

Role of Telemedicine.

(a) Laser vs Cryo — % of ROP

LASER is best > Post ROP.

Ix for Cryo → Poor fundus visibility (VH)
Lack of laser
× Surgeon training.

Complicat^n of LASER → As Ischaemia
Cataract.
Burns of corneal iris

Laser tech — Ix
Timings
Steps — ROP deops, Neonatologist nearby

✓ Diode - 810 nm

Parameter — Post retino > energy
long 0.3s 300mW grade 2

✓ Ant to ridge upto Ora
× on Ridge & Post.

Follow up
skyplesion
Sign of regression

Complicat^n — Hyphaema, Iris burn, RD
Accidental burn, Corneal haze
Systemic Complications.

APROP Rx → Avascular retina beneath flat post stage 3 ROP

LASER - 1 → flat stage 3 ROP regresses

↓ 1wk

LASER 2 → Avascular retina — VEGF release

↓

Now exposed ..

Best ROP → Role of Anti VEGF — (32-42wk of PMA)

Risk = (5 clock hr stage 3 ROP

↓

1 Clock hr ↑ α 26% ↑ unfav Macular Outcome

Rx

↓

Stage 4

Lens sparing Vitrectomy

↓

Stage 4A

x Scleral buckle → Aniso. Myopia

→ 2 sitting removal

VBx aim → Undistorted Macula

Total R. detach.

Preservat^n of lens

Central fixation

4A ⊗ 4B/5

Release tractions mainly

Stage 5

In leucocoria lens — sacrificed

↓

Plasmin assisted vitrectomy

↓

for complete V removal

Autologous plasmin

(0.3-0.2IU)

Sequelae of ROP

Changes

Spont Regression.

- foveal pigmentation
- PP pre retinal Memb
- Exudative detachment
- VH.

After tx

- Narrow temp Vasc Arcade
- Tortuosity of PP
- Macular pigments
- Pre-retinal Memb.
- Macular heterotopia

- Strabismus
- Myopia
- Retinal folds & RD.

- Long term → RD. Dx difficult as :-
  - Irregular tears
  - Atrophied peripheral retina
  - Abn of VR interface

- D.D
- Studies

Sequelae

- Peripheral Changes
  - Vascular
  - Retinal
- Post Changes
  - Vascular
  - Retinal

Macular Hole

Idiopathic — 6-7 decade
2:1 ♀

Symptoms — ↓VA, Metamorphopsia, Central Scotoma
Bil — 15-20%.

Pseudohole — Dense ERM sheet with central defect that overlies foveal centre giving ophthalmoscopic appearance of true macular hole

(I) ~~Gass classification~~ Stages of Macular Hole formation

Stage 1 foveal detachment

- 1a → yellow spot in fovea.
- 1b. → Ring in fovea.

Spans closure 50%.

Stage 2, 3, 4 — full thickness retinal defect with complete absence of neural retinal tissue overlying foveal centre.

- < 400µ
- > 400µ
- Complete PVD
- ± occasional operculum overlying

(II)

- 1° VMT
- 2° other condition unrelated to VMT.

(III)

- Small < 250µ
- Medium 250-400µ
- Large > 400µ

OCT classification of macular hole

Stage 0 — UMA ± h/o FTH in other eye.
Stage 1 — VMT
2, 3 — small, medium, large ± VMA present
4 — ± release of VMA

Cause of sub-optimal results after Sx –
- Persistent retinal abn like non restoration of ELM.
- IS-OS dysfxn

Macular Hole

Def — full thickness defect of NS retina involving the anatomical fovea

Prevalence — 2* female prevalence ↑ ± decade

Pathogenesis — Idiopathic in etiology

Laser → Blunt → Trauma & High Myopia, VRSx (Pn. Retinopexy, Lightening)

Tangential traction posterior Vitreous Cortex on NSR at fovea

AP traction

If hole 1 eye other eye — ↑ risk of hole
↑ risk of enlargement

Invasion & Proliferation
↓ of Muller Cell

Contraction of prefoveal vitreous cortex
↓
Tangential traction

Perifoveal Vit detachment
↓
dynamic tractional forces on abn persistent vitreo foveal attachment
↓
AP traction

Muller Cell Cone → Cone shaped, cone of Muller cell forms the central & inner part of fovea centralis
↓
Confers structural support
↓
plug to bind together foveolar PR cells

Traction

Disinsertion of Muller Cell Am underlying PR cells
↓
foveal cyst / foveal schisis

Dehiscence in roof of cyst spreads in pericentric fashion → Crescentric hole
↓
Horse shoe tear
↓
Complete avulsion of cyst roof
↓
fully detached operculum → suspended in PV cortex in prefoveal plank

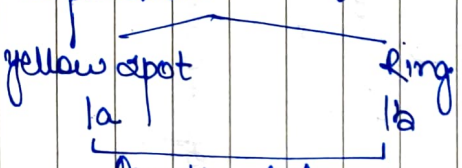
Operculum → Vitreous cortex
glial element ⊆ Variable amt of foveal tissue
↓
R layers → passive centrifugal retraction
↓
full thickness retinal dehiscence ⊆ centrifugal displacement of xanthophyll
↓
edge of hole becomes progressively elevated
↓
cuff of SRF develops
↓
To relieve of traction → Regression of cyst bt × complete closure.

Clinical stage →

Biomicroscopic Exam → Circular defect of NS retina
↓
edges elevated by cuff of SRF
↓
Small yellow spots of xanthophyll pigment may be seen in RPE at base of FT Macular Hole.

DD
- Pseudohole in an ERM
- Lamellar hole ~~in or ERM~~ associated ⊆ arrested macular hole development & CME.

Yass Classification

Stage 1	Impending MH & loss of normal foveal depression  	Intraretinal schisis ↓ " Cyst Masked by semi Opaque contracted Perifoveal vitreous cortex bridging yellow ring	Asymptomatic. Mild Metamorph 20/20 - 20/60	50% regress 40% progress 10% stabilize
Stage 2.	Small full thickness hole <200μ	Pericentric config ass ⊆ persistent vitreo-foveal att	Metamorphopsia Loss of central vision 20/40 - 20/100	15-21% regress 75% progress
Stage 3	Larger full thickness hole (250 - 400μ) with rim of retina & seperation of post hyaloid 4m. Macula.	Fully detached Operculum on Post Hyaloid may be evident	Metamorphopsia. Loss of central vision. 20/60 - 20/200	5% regress. 50% progress
Stage 4	FTH > 450μ Complete. PV seperation 4m OD.	ence of Weiss Ring Operculum evident	Metamorphopsia Loss of central vision 20/60 - 20/400	20% enlarge

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Ix

Watzke Allen slit beam test – Narrow vertical slit projected across the fovea

↓

Dx supported if beam is perceived by pt to be discontinous.

Laser aiming beam test – ✗ perceive Dx laser beam if in lesion bt ✓ if outside on adj retina

OCT

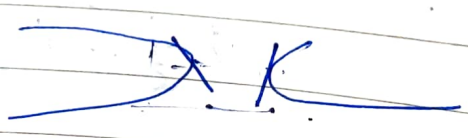
FTH vs. ½ hole
Macular pseudohole/cyst

Stage, Prognosticating factors

HFF	$\frac{a+b}{c}$	> 0.8	good prognosis
HFI	$\frac{Ht}{Base.}$	> 0.5	
DHI	$\frac{Min\ Dia}{BD.}$	< 0.5	Tangential teact°
THI	$\frac{Max\ Ht}{Min\ dia}$	>1.41 better outcome	AP. teaction Retinal hydeation

↓ DHI ↑ THI ⇒ best post op recovery

Min hole dia < 300μ.



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Macular Hole Surgery

To relieve traction & flatten & reappose Macular hole edges

↓

3 ports PPV

↓

Removal of Post Cortical Vitreous / ERM at Macula / ILM peel

↓ ↘ Using Pic or ILM forceps

Traction relieved

↓

Peripheral vitrectomy

↓

Inspect retinal periphery for any breaks

↓

AFE done

↓

C3F8 or SF6 injected.

↓

Prone position x 3-5 days post op.

for if stage 2 hole & < 6 mths

Why ILM peel → ↓ Tangential traction

Removal of potential scaffold for reproliferation of myofibroblasts ⇒ ↓ possibility of late reopening of surgically closed holes

↓

stimulate Muller healing at macula by inducing local expression of undefined growth factors → glial repair

Vital stains in Macular Hole Sx

a) ICG dye 0.5% → cleavage plane
Assist ILM removal
↑ rates of closure

x → Photoreceptor toxicity

-) Infracyanine green
-) Brilliant blue G (2.5%)
-) Trypan blue. — Pet blue (0.1%) 0.06%.
  Membblue (DORC) 0.2%.
-) Triamcinolone.
- Rhodamine 6G
- Bromophenol blue 0.2%
- Patent blue 0.48%

Long term tamponade

- facilitate re-apposition of rim of detached NSR
- Provide interface ⊆ vitreous fluid ⇒ template for glial migration across Macular hole

PFCL ≤

Complications & Prognosis

- Iatrogenic retinal breaks → ↓ by retinopexy
- RD ⇒ chances
- Transient elevation of IOP.
- Post op peripheral VFD
- Cataract development
- Atrophy in RPE.
- CME & post op endophthalmitis

Prognosis factors

- → OCT based visual outcomes ∝ IS-OS line continuity & ELM line
- → Visual recovery — > if vision in fellow eye is subnormal
- → Succesful closure ↑ improve steeocuity.
- → Outcome ∝ stage of hole & duration of symptoms
- → Idiopathic holes have better outcome.
- → Poor prognosis → ↑ Myopia & Submacular RD.
- →

Macular Hole Trials

VMHS — Vitrectomy — 58% / 79%

MMHS — Mooofield — 81% at 24 mth

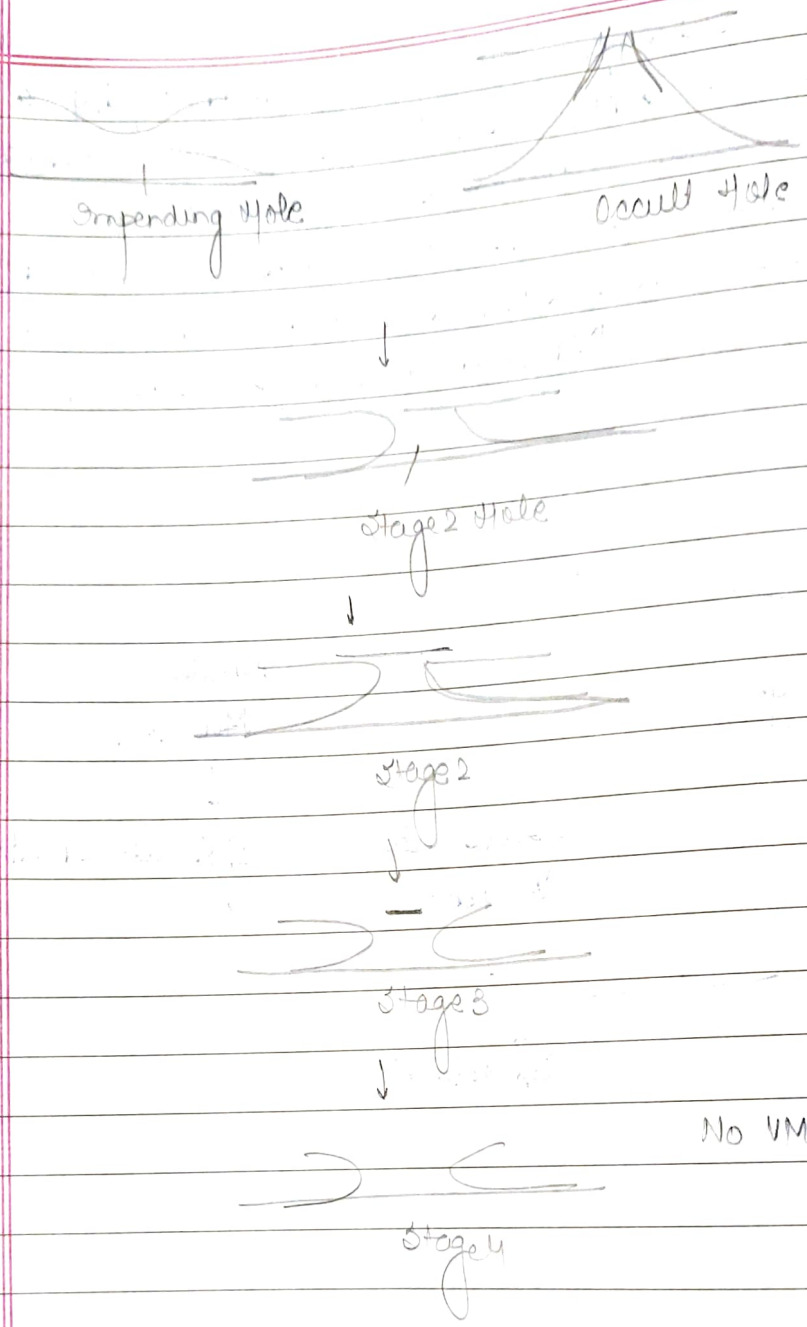
closure rates

Vimpeove

↓

Sx role ✓

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Why ILM Peel

a) Remove foveal traction
b) ERM removal
c) ↑ Cytokine release → glial proliferation.

Rationale of Prone Position :-

Scaffold.

Gas over Oil → gas produce directional force. to macula producing mechanical effect

Hole Closure

- U → Normal foveal Contour
- V → Steep
- W → foveal defect of N&R.

Classifications in ARMD

(A) Clinical.

No apparent Aging changes - x drusen - Abn.

Normal Aging " - druplets ✓ → No prg Abn

Early AMD. - Medium >63 bt < 125μ. No.

Intermediate AMD. - Large > 125μ Any AMD

Late AMD. - NV ee Any GA.

(B) AREDS classification

No	1	few small
Early 2	- multiple small. few I	
Int 2	- Many I + >1 large + GA.	
Late 3		

- Subfoveal GA
- CNV
- PED.
- HE.
- Fibroplasty
- Disc Scar

Angio

- Classic
- Occult
- Min classic / Bed CNV → >/50% classic.

Topographic

- Any
  - Drusen
  - Focal hyperpig
  - Non GA.
  - GA.
- Mel-
  - Choroidal CNV
  - Disciform scarring

Topographic

- Extrafoveal → >200μ
- Juxtafoveal → 1-199μ.
- Subfoveal

ARMD

- Large drusen >125μ
- Pigment abn in Macula

] If for Intermediate AMD

→ RAP

↓

Small discreate hyperreflective lesion in NSR in Outer Nuclear layer

Types of drusen

Subretinal drusenoid deposit
↓
On Top of RPE
Granular hyperreflective material bet RPE & loss Jxn

Cuticular drusen
↓
Uniform, Round Multiple Yellow white punctate accumulation under RPE

Soft drusen

Geographic Atrophy — late Non Neovascular AMD

Blamds

Basal laminar deposits

RPE & its BM
granular material & collagen (IV)
+ Laminin + gag, Cholesterol

Indicator of degree of atrophy & photorecep. deg.

Blinds

Basal linear deposits.

BM of RPE & Bruch's Membrane. (inner collagenous Z)
lipid rich material & coated vesicles

Progression of disease to late stage

Types of ARMD

Dry
- Soft drusen <63μ → Early
- RPE depigmentation

Wet
- late
  - pure GA
  - Exud. ARMD

Dry AMD	Wet AMD
Most common	90% cases of visual loss
Hypo, Hyperpigmentation, atrophy	CNV → disciform scar
Basal deposits Drusen geographic atrophy	CNV disciform scar
Mild Loss RPE atrophy / GA	VA Loss. SRF., RPE detachment OR Hmrg HE, OR fibrosis CME >>

AREDS — Drusen

Cat 1 — Early AMD — <63μ few small

2 — Mild — + few Intermediate 63μ - 125μ

3 — Intermediate — + extensive Intermediate + Large Drusen (>125μ) — Multivit ✓

4 — Advanced — GA in centre or Neovascular AMD — Multivit ✓

Treatment

Dry — Wet AMD

- AREDS1
- AREDS 2
- fenretinide
- CNTF
- Copaxone

AREDS 1 → Lens opacity & AMD Rx

- PDT — TAP, VIP
- Anti VEGF — Combinat° c Avastin
  - Macugen — VERITAS study
  - Lucentis — MARINA, ANCHOR, PIER, PRONTO
  - Eylea
  - CATT result

Vit C – 500mg
E – 400 IU
β Carotene – 15mg
Zn 80mg – genitourinary.
Cu 2mg

Mortality in smokers < lung Ca

AREDS 2 – 2 dietary xanthophyll

Lutein 10mg, Zeaxanthin 2mg

2 Omega 3 LC PUFAs
DHA – 350mg
EPA – 650mg

Conc
Lutein. If diet low/ ↑ → theu. prescribe
✓ Cataract rates. ✗ → ✗ use
↓ Zn – No Benefit
✗ β Carotene – ✓

- Combination c PDT.
- FOCUS.
- si RNAs.
- Receptor tyrosin kinase inhibitors — Bevasiranib / Cand5

Advanced
- CATT Trial.
- Combinat° Rx.

Denali, Mont Blanc, Mount Everest

- VEGF trap.

OCT changes — Loss of 5 letters in VA
fluid on OCT
↑ in CMT >100μ.
New Onset classic CNV.
New Macular thning
Persistent fluid even after 1 mth of inj

Width of Retinal Vein off the ONH → 128μ.

Drusen

Yellowish, white lesions located deep to retina in post pole.

Nodular (Hard)

Smooth surface
dome shaped
B/t RPE & Bruch's
Hyaline material
Stain +ve ⊆ PAS.
Overlying RPE is Hypo
<50μ
↓ Hard
↓ RPE atrophy
✗ ↓ MD.
Window defects.

Soft

Cleavage B/t Bl. & Bl. ⊆ formation of localized detachment
- may become confluent
>63μ dia ⇒ Large
→ atrophic ↓ GA
potential space for CNV
≤ time

Diffuse

Blan. Cuticular deposit
Internal Nodularity
Thickening of inner aspect of Bruch's
B/t PM/& BM/of Bruch's
Collagen + laminin

✗ (young)

Crystalline

discrete, calcific
delineated, B/t D ↓ GA.

Small	Intermediate	Large
<63μ	63μ - 128μ	>128μ
Hypo	Early	Hypo.
Hypo	Late	Hypo x Leak
		Hypo
		Stary sky app

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geographic Atrophy.

well demarcated atrophy of RPE.
End stage of dry AMD

Drusen → Initiation of RPE cell loss → degeneration of outer layer of NSR

↓

Marked atrophy & sclerosis of choriocapillaris

Spares FAZ

Drusen

granular. 250μ.

Soft

Serous → 500μ PED

Memb. → 175μ. → >CNV risk.

Disciform Scar

End stage exudative AMD.

Vascularized ↓ fibrotic Scar tissue

F 0.6% | C 96% | Both.

Loss of neuronal tissue.

Photoreceptor loss ∝ Dia, thickness of Scar

VEGF is critical stimulus.

Patho of dry AMD

a) Changes in Bruch's Membr → ↑ thickness & Co^{2+} deposit
Focal thinning + disruption of EM
↓
↑ cellular activity ⇒ Inflamm patho

b) Changes of RPE — Lipofuscin granules in RPE
Hypo pig & atrophy, attenuat°
Soft drusen, RPE detachment

c) Changes of choriocapillaries - Thinned & sclerosed

d) " in NSR → Aging in Muller cells & axons of eye
Rods disappear Blue >> FG
greatest loss in Parafoveal region.

Rf Syn
Advanced AMD in one eye ⇒ AMD in other eye.

AREDS
- Drusen
- Pigment stippling
- Advanced AMD
- Intermediate drusen
- Area > Size.

CNV — Blood — PED

CNV

New BV formation from choroid
Such changes in BM → Calcification + focal breaks in BM
↓ Thickness & disruption of elastic lamina of Br at Macula

Retinal Bl. — Choroidal Vasc — Combined

Sub RPE CNV — Subretinal

Type 1 — Retro RPE Bench — Type II

RPE & Br — Retro & RPE

Intact retina — Overlying retina Atrophic

few symptoms

Occult type — FFA — classic.

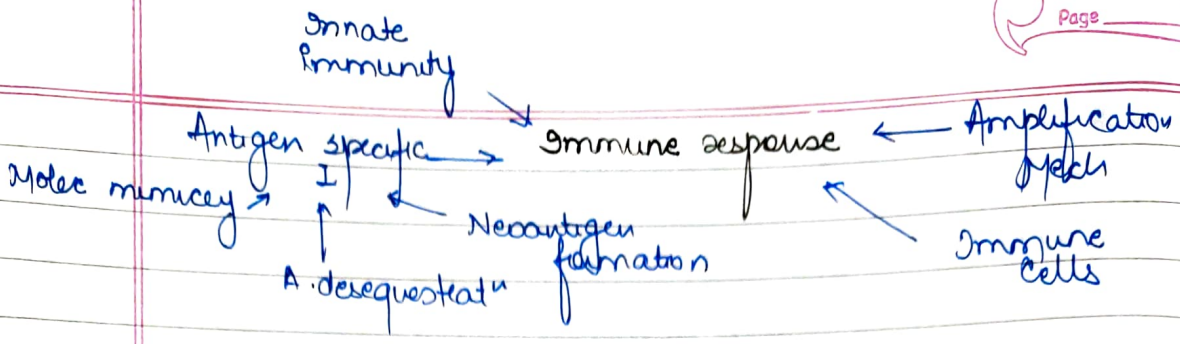
Changes — PEDs < H S (Notched) — Leak on Angio.

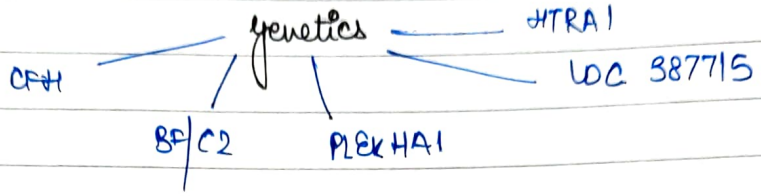
RPE tear, Lipid Exudate

1-12 multiple. — Progressive — few., focal

Hyper (Not as bright as classic) — Early Late — Lacy pattern Hyper > 50% classic

FV PED. (FVPED) — Late Leakage of Undetermined source (LLUS) — but late hyper & Indistinct border → Early Hyper





Risk factors

Age

Gender — ♀ — Late AMD & Soft drusen

♂ → — RPE abn

Race — whites >>

Hereditary — >>

Macular pig ← Lutein Zeaxanthin

Sunlight exposure

Cataract Sx → Ocular factors ← Systemic

- CVS
- Smoking
- HTN
- ↑ S. lipid
- DM
- Chlamydiae

Iris Colour (» Blue)

Refractive Error (» Hyperopic eye)

Sunlight exp.

Systemic factors

RF for CNV

5> drusen

focal hyper

HTN

100> drusen.

Caucasian

Smoking.

Visual acuity
6/6s of dec

Vernier Acuity
3-6s of arc

Symptoms

Sudden Onset ↓ VA.
Metamorphopsia
Scotomas
Self monitoring

Signs

Wet Dry.

Fundus finding ≤ 90°D

↓
PED/CNV = ↑ chances of Visual loss

PDT drugs < Verteporfin
SnET2.

Ix

Self monitoring
- Amsler grid. 10°
- Potential hyperacuity perimeters. 14° in 5min ∝ Vernier acuity.

OCT
- CME also.
- PED — Hemorr Serous
- CNV
- RPE tear/rip
- Natural H/O PED
- CNV Rx

FFA
- if can't localized
- x/diff SRF or solid fibrous tissue

OCT

ICG
Hot spots
Plaques
RCA
PCV

- TAP study → » classic CNV.
- VIP — ~~Occult CNV~~ classic, Patho Hypo, Hemo
  » Size — > 1 DD - classic
  1-4 - minimally classic
  < 5 DD - Occult only lesions

- Macugen — VISION study 0.3, 1, 3 mg
- VERITAS

PDT
Triamcinolone + Pegaptanib

- Lucentis — Xpina — Subfoveal occult. minimally classic CNV
  Anchor — classic L vs PDT.

PIER 1 mth * 3 mth
1/3 mth * 2 yrs
Dosing
• PRONTO — OCT usage
+ PDT — FOCUS

- Avastin.

Mx

Risk factor modification
Home based Amsler grid
Prevention of VA
~~Anti VEGF + Ix~~

Maculopathies

dry
- Obs AREDS 2.
- Antioxidant supplementation

Wet AMD

- Macular → Anti VEGF
- PDT
  - Macular / Subfoveal
    - L 50% lesion
    - < 5400μ
  - Occult CNV
    - Vision < 20/50
    - CNV < 4 MPS
    - → Juxtafoveal
- Laser
  - Extra ~~Juxta~~foveal
  - Juxtapapillary

Other Rx (New in AMD)

TTT
Teletherapy (EBRT)
Beachy therapy (Plaque Radiotherapy)
Anecortave acetate
si RNA
Vatalanib
Squalamine lactate
Sphingomab
Volociximab
DARPins
Sirolimus
Infliximab
Eculizumab
PEDF
Bevacizumab
Alprostadil
Maculoplasty
Macular translocation
Rhufab V2

VHL Von Hippel Lindau Disease

Eye → Retinal Hemangioma Capillary

Kidney → Renal Carcinoma, Pheochromocytoma

CNS → Hemangioblastoma

Neuroendocrine Tx

Pancreatic cysts

Inheritance AD. VHL gene — 3p 25-26 → tumour suppresser

a.a → Hypoxia

× pVHL → ∴ ↑↑ VEGF production

Median age 20-25

♀ = ♂

Mutation α → Juxtapapillary

β — Peripheral

Ophthalmic

Retinal Angioma

R. Hemangioblastoma

Capillary retinal Angioma.

↳ if multiple → R. angiomatosis

Sporadic — part of VHL

VHL — Multifocal, Bilateral

Early stages — Hardly visible angioma & × feeder vessel

Late stages — complicat° → RD, ERM, VH

Juxtapapillary — Peripheral
Poorly defined. — Borders
Less demarcated
Variable — growth pattern
Subretinal space, — may grow.
Intraretinal, vitreous
fluid exudation — Vision loss

DD - C. Hemangioma.
Amelanotic C. Melanoma.
CNV M

Cf) → Painless loss of vision / VF
Painful 2° glaucoma
» Development of Blindness

Early detection bcos — prevent blindness
slow growth.

Histo) Large capillaries displaying normal endothelium, BM & pericytes are ± astrocytes.

Classification

Nail's Stage

- I — Early stage ⊆ dilation of feeding artery, vein & Angioma formation
- II — Hmrg + Exudation
- III — Massive Exudation + RD
- IV — Uveitis, Absolute glaucoma, Loss of eye

Retinal distribution

- Peripheral
- Juxtapapillary

Morphology

- Endophytic
- Sessile
- Exophytic

Effects on retina

- Exudative
- Vitreoretinal

Systemic features

- without VHL disease
- ⊆ VHL disease

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VHL classification

	Type 1	Type 2 A	Type 2 B	Type 2 C
Retinal Hem.	+	+	+	+
CNS Hemangi	+	+	+	
RCC	+	−	+	
Pancreatic Neop & Cysts	+	−	+	
Phaechromocytoma	−	+	+	+

Dx Criteria

+	−
Retinal CH	2 or > RCH.
CNS.	2 or > CNS
Visc Lesion	

Any 1

- Renal Cysts
- Renal Ca.
- Pheochromocytoma
- Epididymal Cystadenoma.
- Pancreatic Cysts
- Endolymphatic Sac Tx.
- Islet Cell Tx
- Adenexal papillary cyst adenoma.

Coat's
Racemose H.
R. Cavernous.
VPRT
RPE Adenoma.

Uveal Melanoma
Uni. Papilloedema
Juxtapapillary Choroiditis

Every yr.
Urinary Catecholamine
Ophthalmoscopy
Abd USG

1-2 yrs
Abd CT

2 yrs (11-60)
MRI Brain & Spine

61+ (3 yrs)

<500μ → Observe
× Exu.
× SRF

<1.5mm. → Laser Photocoag.

>3mm → Cryo.

>4-5 → Plaque. Radio Rx

- Observe
- Laser
- TTT
- Photopoorphyrin DT
- PDT
- Cryo
- Plaque
- Ext beam Radio
- Proton Beam.
- PPV / Scleral buckling
- Enucleation.

Juxta.

difficult
as destruction of Nf ⇒ Scotomas

Asymptomatic → Observe

Symp → Laser
Ext Beam
Vitrectomy
PDT

Modified PDT — Pulmonary fusion
83-125s

Peripheral

Lasers.

Larger / Cryo

Ruthe 106 — Radio &
Proton Beam.

PDT → RD

VRSx for 2° Complicat"

Serous - Drusen = PCV
PED

	Serous PED	Abnormal Vascular PED
Symptoms	Blurred Central Vision. Induced Hypermetropia. Metamorphopsia	By MPSC its form of Occult CNV
Signs	Orange, dome shaped elevation. ⊆ sharply delineated edge ⊆ pale margin of SRF. If pigment band – Chronicity Bld, Lipid Exu, CR fold, Irregular. SRF — CNV. x drusen ⇒ PCV	» Irregular in outline " in elevation
FFA	well demarcated hyper ↑I x Area of notch – CNV	Irregular, granular hyperf. ⊆ uneven filling of PED. Leakage & Late staining
ICG	Hypo ⊆ surrounding hyper ring. focal hot spot	» CNV.
OCT	Seperation of RPE from Bruch's CN → Notch. ↑ Main elevat" & small Mound.	Less uniform « Serous both fluid & fibrous proliferat" (Irregular shattered effect
Natural Course	» ↑ atrophy & ↓ VA >60 → Worse Prog Sudden loss – RPE tear, Hmrg from CNV. ⅓ → clinical CNV ⊆ Sys.	∴ Serous ⊆ CNV.
Mx.	<60, x CNV → Observe. ✓ → Anti VEGF PDT + IVTA / Anti VEGF	RPE tear risk

Drusenoid PED.

develop from confluent large soft drusen, BrM

shallow elevated pale area c scalloped edges

Early diffuse Hypof c patchy relatively faint early hyperf → irregular late staining

Hypo predominates

Homogenous hyperreflectivity c/n PED x SRF

Better
gradual vision loss
Once 10yrs — 75% GA
25% CNV.
3yrs → 1/3 → GA/CNV
Long term stability

Observation

Hmrg PEDs

CNV
PCV → Pf x drusen.
Sudden Impairment of central vision
Elevated dark dome shaped lesion c well defined outlines
Bld +nt Pn SR space

Dense masking of background fl. c overlying vessels.

→ PCV Mx
CNV c small PED ✓
Large - Hmrg AMD

Macular dystrophies

- Stargardt's
- Fundus flavimaculatus
- Vitelliform dystrophy
- Foveomacular VD
- Familial Dowsen
- Central Areolar Pigment Epithelial (CAPE) dystrophy
- North Carolina Macular D.
- Dominant slowly Progressive MD of Singerman Berkau Patz
- Dominant Cystoid Macular D
- Fenestrated sheen MD
- Butterfly shaped Pigment D of M
- Stogen's Reticular D of RPE.
- Pigment Epithelial D of Noble Carr Siegal
- Benign Concentric Annular MD.
- Central Areolar Choroidal D.
- Inner Retinal & Vitreo-retinal D.

Hereditary Central Retinochoroidal dystrophy

NFL
↓
Sex linked
Juvenile
Retinoschisis

Pigment epithelium
- Best
- Retinal D
- Butterfly shaped dystrophy of fovea
- Fundus flavimaculatus
- Grouped pig of foveal area

Choroid
↓
Central Areolar
Choroidal
atrophy

Neuroepithelium
- Stargardt
- Dominant foveal
- Inverse RP
- Progressive Cone dystrophy
- Familial lipid degeneration

Emixustat (Acucela) — Inhibitor of RPE specific protein RPE-65.
Genes → CNTF, Gene silencing

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Stargardt's — Fundus flavimaculatus

Onset — Bil — Ophthalmoscopic diff ✓
Symm — >> children ✓
AR. short arm Chr 1 — AR.
Juvenile Macular degeneration

HPE — PAS +ve lipofuscin like material in engorged RPE cells

>> Macular Atrophy
Beaten Bronze atrophy 1
Mutat^n of ABCA4 gene
3 Types

>> flecks 2)

CF — Progressive loss of Central vision
Fundus ↓VA. (time)
1st Involved (6-20yrs)

Multiple angulated, fish tail shaped yellow white lesion. confined to RPE of posterior pole.
of variable size shape & confluence
New clusters appears periodically as old one fades.

Fishman classification
Parafoveal, mid peripheral yellow white flecks ~~× beyond~~
Periphery — Round, granular
Stock spots of ↑ Pigmentation
& surrounding depig areas
Atrophy ↑ → GA.
∩ Central Areolar CD
= Bony Sprcule ∩ RCD.
Central Scotoma

× Atrophic Macular lesions 1)

× ~~beyond~~ Equator.

FFA — dark or Silent Choroid → ↓ effect of Blockage of Choroidal F. from accumulation of Lipofuscin (A2E)
Red green Dyschromatopsia

FAF — Hypof — RPE deg
Hyper → & surrounding flecks loaded & Lipofuschin.

Electrophysiological test
ERG, EOG - Normal
VEP - Subnormal (Early changes)

ERG — Subnormal (
Non Recordable — Tapeto retinal dystro
EOG - Subn.

OCT — deepening of foveal
HPE — defects in photoreceptor ↓° to RPE defects /Atn.

Dod — ∴ Rod Cone dystrophy CACD.
× give Vit A — as ~~toxic~~

Vitelliform dystrophy

Best's, Polymorphic Mac deg of Bealey, Central exudative detatchment of Retina, Hereditary Macular Pseudocysts.

BEST-1 gene

Adult type

Pseudo vitelliform MD

QD.

Hallmark – focal loss of PR & RPE atrophy

VA – better

c/f

good VA → if ↓ / Atrophic / Macular changes / Scarring

VFD +nt

Hyperopia → Emmetropia → ↓. Amblyopia

Bil. Symmetry ↑

Yellow Subretinal lesions

3 D central pigmented spot in fovea of each eye

Stage 0

(N) Macula.

foveal hypoplasia

Non EOG

Stage 1

Egg yolk lesion

Round, Homog opaque yellow lesion c discrete margin

Stage 2

deg lesion

Scrambled egg appearance

SRNV.

Diff ←

1) Smaller

Recent later

× Evolve in similar fashion

III

yellow material + fluid

Pseudohypopyon

IV

Advanced

Atrophy

SRNV

Fibrous Scarring

FFA

hypo +nt

ERG – (N)

EOG → ↓ defect at level of RPE & Bruch's

Central hypo due by hypo.

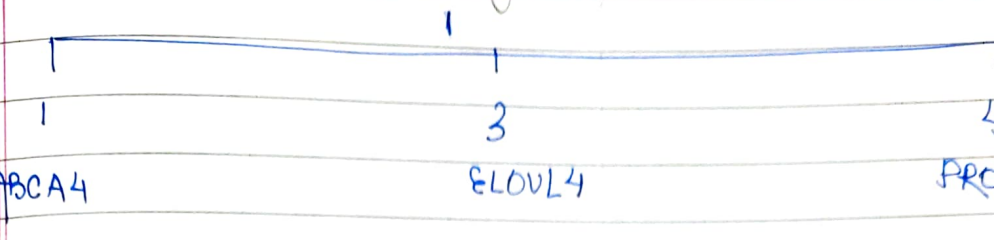
Arden Ratio < 1.8.

Dark Adaptometry – (N)

Colour defect (+)

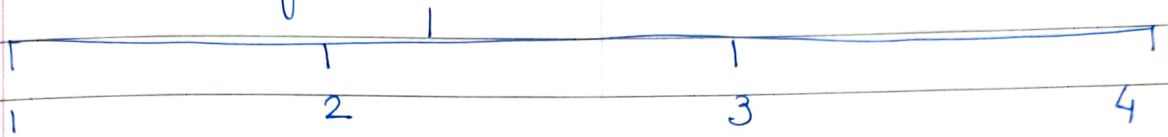
OCT – Vitelliform material ant to RPE

Stargardt Disease



- Rel sparing of peripapillary area

Fishman classification



Fundus	Beaten bronze	flecks	flecks + Atrophy at macula	throughout
ERG	(N)	(N)	Sub (N)	
DA		Prolonged		↓
Field defect			Central field defect	Moderate to extensive

Pathology → Lipofuscin from Inner to Outer segments of Rod Cells.

Choroidal Hemangioma

- Circumscribed
- Diffuse
  - ⇓
  - Sturge Weber Syndrome

Confused with Uveal Melanoma

Congenital & noticed if — SRF, Macular edema or direct involvement of Macula

During Embryogenesis — Choroidal Vasculature — persistent AV shunts.

Age related Vitreous Changes

By age of 45-50 → Liquefaction (Synchisis)

↓

Small cavities coalasce → Big

Collapse of Collagen Network, (Syneresis) → (+)

Through defect in Post hyaloid

fluid escapes

↓

Seperating Post hyaloid surf frm ILM

↓

PVD

Synchisis + Syneresis ← ↑ Myopic eye
IO inflammation
Surgical aphakia
Trauma
Wagner
Stickler's

Width of Vitreous base post to Ora ↑ c age from 2-3 mm in radial dimension in temporal periphery.

↓

PVD → increased trac^{n} → Peripheral break RD

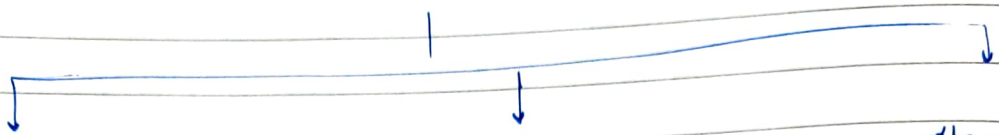
(*) Temporal fade

→ Disci ocas

RAP

Aromalous Retinal Vasc Complex. + R + SR NV

misdiag as Occult CNV.
Bil



Stage I	Stage II	Stage III
Intraretinal NV	R + Subretinal	II + CNV.
Paramacular	NV is tangentially	
IR thickg +	thickg	SRFD, Disciform
IR Edema.	Edema, NSD.	Scar.

focal staining — FFA — diffuse leakage
Hot Spot

Choroidal inflammations

White Dot Syndromes

Idiopathic immunological disorders having visual disturbances & multiple whitish subretinal lesions located at level of outer retina, RPE & choroid.

∴

Choroidal Inflammatory disorders ∈ help of ICG site localized to choroid.

- Inflammatory choriocapillaropathics
  - Non perfusion
    - 1°
      - MEWDS
      - AIBSE.
      - MFC
      - PIC
      - APMPPE.
    - 2° to inflam of RPE & Ch. stroma.
      - Toxoplasma retinochoroiditis
      - Birdshot (BC) chorioretinopathy
  - On ICG
    - Early
    - Late
- Stromal Choroiditis
  - hyper i.e leakage
  - hypo f
    - 1°
      - VKH
      - SO
      - B.C.
    - 2°
      - T.B
      - Syphilis
      - Sarcoidosis

Serpiginous choroiditis
Peripheral. Tubercular.
Serpiginous like choroiditis
Azoor.

SFU

	MEWDS & AIBSE.	APMPPE	MFC | PIC.
Age	young. middle aged	young 20-50 yrs	Recurrent
Sex	females	27 yrs.	
Laterality	Uni	Bil/Uni	Bi – MFC Uni – PIC
Symptoms	Visual loss, photopsiae, field loss	→ Rarely – OD edema & exudates. Enlargement 0.5 to several DD multiple placoid yellowish white lesions in post pole & mild. to moderate AC & Vitreous in Acute phase Convalescent stage – CRA. scars.	→ Myopic ♀ → Macular Mild Vitreous
Signs	Normal fundus discrete. discolouration of midperipheral fundus.		AC & Vitreous – inflam. yellowish white lesions of post pole & midperiphery →
Preceding f	Viral flu like illness	→ Viral / Cerebral Vasculitis – Stroke	2° CNVM (30-40%)
ICG E	hypo	geographic hypof.	hypo → Late phase
I		↓	» MEWS & APMPPE
L	hypes	Late phase.	
	Absent findings		
FFA	patchy & peripapillary hypo → late phase hypo.	Early hypo. Late hypes.	Acute / Convales → E – hypo, L – hypes, L Mixed hypo & hypes pattern.
		HLA B7 – DR2.	
Others/ Course	AIBSE → subclinical counterpart	Adenovirus 5, HLADR2, CNS imaging LP.	
Rx	Spont.	X. Scarring at post pole Steroids	Bil – systemic steroids Uni – PST. & Immunosuppressive CNVM – Anti VEGF PDT

Geographic Choroiditis
Helicoid peripapillary c.
Macular g. Helicoid c.

(*) RPC = APMPPE + SC
Relentless
PPM (Persistent Placoid Maculopathy)

Serpginous choroiditis	Serpginous like choroiditis	Adult.
Recurrent 30-60yrs Progressive, AI Bil >> whites ♂ > ♀	Recurrent- Progressive 2° to Tubercular HST. Uni Bi	Late., Middle Aged females.
→ PPM Peripapillary Macular Amp T	→ MF Mixed. Diffuse.	Sudden onset field Loss ⊆ prominent photopsiae which. are worse in Bright light & show movements
a) yellow white lesions around disc, but elevated b) Late → ↓VA c) start as MF APMPPE like lesion & coalesce to classic Serpginous Lesion	MF — Most common. disc yellow white SR lesion in. post pole & periphery which coalesce to form SC like. lesions. Diffuse — ≅ to SC since start	Acute — No abn. Late — Sectoral RP like. picture.
Heal — Pigmentation, diffuse CRA & CNVM formation Early Hypo → Late	AC & VHe inflam →→→ ↓↓ RPE pigmentation ⇒	N.
Acute: E - Hypo, L - Hypo Chronic — Window defect	⇒ ⇒	N. Late stages — Window defects in area of RPE loss
Etio HSV, EBV, CMV, VZV		VF — Temporal affected
Histo - extensive loss of RPE & death of overlying retina. CC - lymphocytic infiltrate		ERG — ↓a, b amplitude delayed 30Hz flicker affection Cones >> rods
Pulse steroid c IS.	Oral steroids + IT I, R (9-12 mth) I, E stopped after 2 mth.	EOG — light ↓ X → Spont
⇒		

Stromal Choroiditis

Inflammation of stroma. ↳ by granulomatous disease
Leakage of normally fenestrated larger choroidal vessels

CC, RPE & outer retina involved —↓ VA

Ocular Sign — Disc Edema
Exudative RD
yellow white subretinal lesions
Hypopigmented scars

ICG

Early — Leakage from choroidal vessels
Hypo (HDD) — granuloma. which prevent leakage of ICG from vessels in that area

Late
Hypo — if full thickness
Iso — ½ thickness

↓

Choroidal vessels — Loose their distinct outline & have fuzzy borders ⊆ leads to late diffuse hyperF.

VKH	SO	BC
Bil granulomatous panuveitis	↑ ⊆ Trauma Sx – Post PPV	Bil granulomatous Posterior Uveitis both C & R involvement >> around disc
Prodrome of Tinnitus, headache, Meningismus, hearing loss, fever ↓		HLA A29.
Acute Uveitic stage of KP, uveitis, Disc edema, Multiple Exu RD		Uveitis >> Small & Large vessel vasculitis, CME, Disc edema & Choroidal involvement – granuloma in. stroma in periphery of fundus
Conv – Poliosis, Vitiligo, Sunset glow fundus & Nummular Hypopigm		Healed – Yellow white depig lesions in periphery & Pseudo RD in the numerous HDD ↷ also in. Late & fuzzy Choroidal vessels
ICG – Early CV leak HDD, fuzzy choroidal vessels & disc hyperf.		Conv – Disapp of HDD & Hypof (CRA)
Long term CS & IS.		~~FFA – Pseudo.~~ Long term steroids & IS agents
FFA Pin point hyperF in Early Pooling in Late Windows defects		Pseudodelay of AV transit due to massive dye leakage CME, disc hyperf

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Disorders of Viteous

a) Posterior Viteous Detatchment
b) Asteroid Hyalosis
c) Viteous Amyloidosis
d) Cholesterolosis Bulbi
e) Intra Ocular lymphoma.
f) familial VR disorders. — Stickler, Wagner.

Posterior Viteous Detatchment

♀ > ♂. Age: <50 — 10%; 60-69 — 27%; 70 — 69%.

> in Aphakic eyes & in modern day surgery after YAG cap.

cf: Flashes — Mech irritation of retina in process of seperation

Floaters — Casting of shadows in collapsed collagen fibrilar network of Viteous on Retina. (entoptic phenomenon)

Detatched Viteous

Reacts forward. Sits behind lens → flashes & floaters goes away.

Retinal breaks ± VH.

VH: Traction → Tear; Avulsion of BV → VH

Abn firmer att of Vitr & Retina → Lattice deg
- Enclosed Oral Bays
- Retinal tuft
- Post advancing Vitreous Base.

Dx

Clinically
- Weiss Ring (Surest sign)
  - few mm in front of retina
  - Collapsed vitreous gel behind lens
  - Empty fluid space sup behind gel
- condensation of collagen fibrils of V. body (PVD)

Ix
- OCT.
  - Post perifoveal vitreous detachment spreading radially to involve larger areas
- USG
  - Thin white line bounding the V. gel.

Mx

A. Symptomatic → RD risk.
- Indentation to look for Break
- Cryo/Laser.

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Complications — VH.

Retinal Breaks

Tractional dislocation & Traction / Elevat^{n} of Macula

↓

VMT syndrome

foveal cavitation & M. hole formation.

ERM formation

CME & Macular cyst formation.

Asteroid Hyalosis

Benign condition characterised by small yellow white spherical opacities throughout V. Cavity

>50yr — 0.8-2%
Unilateral

POD → Small round-Oval opacities suspended in Vitreous 3-100μ.
× effect of gravity
× effect on Vision. as VH.

Calcium containing Phospholipid
If × Unil — No systemic association.

DR assessment hindered. but FFA ✓
(×) — OCT, USG

Ix for PPV — Poor visualization of fundus
Inability to localize break in RD
Rarely if visual symptoms +

Complications — ↓ PVD rates
Difficult PVD induct^{n} ⇒ Stronger VR att.
Iatrogenic breaks during PPV

Vitreous Amyloidosis

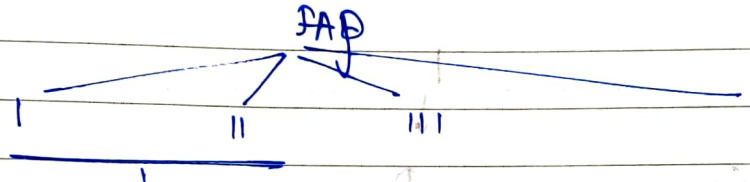
Part of FAP → familial Amyloidotic Polyneuropathy (FAP)

↓

Mutation in transthyretin gene (TTR) → aggregation of β pleated sheet of protein in various tissues.

Characterised by →
- V. Opacities
- Cardiomyopathy
- Neuropathies — Peripheral, Autonomic, Central.

AD transmission



↓

Vitreous Amyloidosis

Peripheral Neuropathy.

» Uvil

Signs

Dry Eyes. Pupillary light near dissociation.
Peripheral Microaneurysm in Cor
↓ Corneal Sensitivity
Scalloped Configuration of pupil
Flecks / deposit ⇒ Pseudoexfoliation
Pseudopodidentis → Multiple small dots / footplates on back of lens
Prominent Corneal Nerve.

- ↑ IOP, GON
- Diffuse deposits of fibrillar opacity throughout
  - ∟ cobweb / glasswool / cotton wool
- Perivascular infiltrates & sheathing
- Perifoveal intraretinal Amyloid deposit — Grey ring
- Dot & blot Hmrg — Equatorial region.

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FFA
Perivascular filling defect
└ M. edema
Multiple MA
staining of cotton wool like
infiltrates

EOG
↓ dark & light trough

ERG
↓ Scotopic response
↑ P100 latency.

Prognosis
poor

Dx
Vitreous Bx
confirmatory

Rx
PPV → visual rehab
↓
Complete Vitrectomy

D.D	Asteroid H.	Vitreous A.
	Uni°	Bil.
Vision	* effect	↓
Progression	slow	Rapid
Appearance of deposits	small, yellow spherical	Cob Web, glass wool.
Systemic Association	None	Neuropathy Cardiomyopathy.

Cholesterolosis Bulbi (Synchisis Scintillans)

yellow white, gold glistening, angular crystalline bodies formed of cholesterol

(*) Settle inf like golden rain

Repeated. Severe & x trauma. ⊆
↑ IO Hmeg
Origin 4m leaking Retinal Vessels

	Asteroid Hyalosis	Synchisis Scintillans
Vitreous	formed.	Liquefied. PVD +
	grey - white.	golden yellow.
	Small round Oval	Angular, crystalline, glistening
	suspended in V.	shower of golden rain.
	x ⊆ gravity	Settle inf
	» diabetic	Severely traumatized eye ⊆ IO Hmeg
	Ca containing Phospholipid	Cholesterol crystals.

Intraocular Lymphoma

Masquerade Syndrome
- NHL
- Systemic NHL — Metastatic to Eye
- HL.

features → Sick pt — fever, LN pathy, Wt loss
NHL of CNS → Dx dilemma

50-60yrs
Uni ↷ Bil
Panuveitis ✓ → Viteous cells in sheets

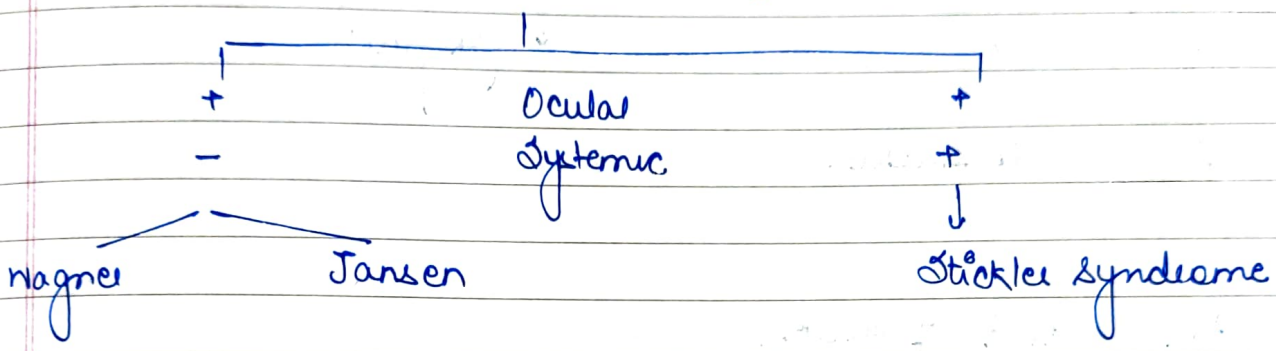
CNS Involven —
- Seizures
- Headache
- focal weakness
- Sensory deficit
- Confusion
- Difficulty in gait

MRI, lumbar puncture.
Viteous Bx → Cytological Analysis

Familial VR disorders

Hallmark → • Optically empty Vit cavity ± sparse Intedsetmall vitreal memb & strands

- Deg Pig Epi alterations
- Lattice like changes in the zebra
- Subnormal ERG.



Stickler	Wagner	Jansen
Astheo - Ophthalmopathy	AD ± 100% penetrance × sex predilection	
facial def, Ocular features, SNHL hearing loss. Bone, JT involvement. Pierre Robin Mal complex	(Versican gene) Cs 19q2 → on long arm q of 5th Chr	
AD	Cataract ✓	
Type I → COL2A1	Mild-moderate Myopia	
Type II → COL11A1	RD risk < Stickler	
Stickler like → COL11A2	Nyctalopia	

Optically empty VC ± sparse Intedvitr Memb & steands

↑Myopia, Develop C. OAG. Prone to RD.

RD prone. if occurs difficult to Rx
→ Multiple post, large breaks ⊆ PVR
- CRT in young
- Recivascular Pigmented

Simple Rx of RD

RD ass ⊆ Pedigree & poor prognosis

ERG ↓ ↓ (cf fundⁿ)

HVF Constricted.

Mx Rx Rx of break.
Early Cataract Extr
Genetic Counselling] ✓
x systemic

Face → cleft palate, glassoptosis, Marfanoid

Bone → Jt hyperextensible
Precocious Arthritis
Mild spondylo epiphyseal dysplasia

H Swiss family

J Dutch family

Vitreous Hmrg

Blood Vitreous Barrier →

a) Blood Vitreous Barrier → Vascular Endothelium
Pigmented/Non Pig Epi of CB

b) Active transport pump Mech
c) Basal lamina at Vitreoretinal Interface.
d) V. hyaluronic acid — block the movement.

Etiopath a) Blunt Trauma Torn retina, B.V bridging may bleed. Rupture of CB.

b) Proliferative Retinopathies → Vessels grow on Post hyaloid face. Bleed → VH. Even ⊆ mobⁿ of eyeball

c) ↑ of Subarachnoid pressure → Sub hyaloid Hmrg (Terson Syndrome)

d) Chronic Uveoretinal Inflamm
e) IO Tx → Invading retina.
f) Blood dyscrasias
g) Retinal NV
h) Pars planitis

C/F Sudden painless loss of vision
Direct pupillary Rxn — dense VH+
Iris — examine for rubeosis iridis
Fundus — Indent
No View — USG B Scan.

Types

- Intravitreal
  - ↓
  - Hmrg in Vitreous body
    - Complete
    - Incomplete
      - Ant
      - Central
      - Post
- Subvitreal
  - ↓
  - In Post hyaloid face & ILM of retina
  - ↓
  - Subvitreal / Subhyaloid / Pre-retinal Hmrg.

Appearance of VH

- whether Vitreous is gel / liq
- The amt of bleeding
- Change that occur in vit due to + of bld in it

- Small bleeding & occurs in formed vitreous
  - ↓
  - red → yellow
  - fluffy ↓ opacity
- Bleeding in Synchitic Vitreous
  - ↓
  - Unclotted
  - >> depend part
  - ↓
  - Better Vision in morning than other days
- Massive bleeding
  - ↓
  - ✕ glow appreciated
  - Layering seen
    - yellow plasma
    - Red at bottom
- Subvitreal Hmrg
  - Boat shaped

>> Terson's Diabetes Pt

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Fate of VH

- Amt of blood
  - Small VH resolve earlier
    - Small → resolve
    - Large → Lose transparency become opaque
- State of Vitreous
  - Hmrg in formed V. take longer to resolve
- Proximity to Retina.
  - Post resolve ».

Blood in vitreous → fibrinolysis → RBC hemolysis → Absorption of breakdown products of bld by mΦ

Diagnosis

- Scleral Indentation → Cause
  - H/O DM, HTN] → clues in other eye.
  - H/O Trauma. → choroidal rupture.
  - ↓ NVE/NVD → PVR causes — Vasculitis, BRVO, CRVO.
- Dense VH → Rule out RD, IOTx by USG & Scan
  - Patch, Head End raised 7-10d → IDO, USG
- Diff b/t PVD & RD

Rx → Self resolving

↓

2mth x resolve

↓

Non clearing VH

↓

PPV

↓

Traumatic

↓

≤ in 4d

Early

Reg, TRD

≤ EL.

goal → clear media.

remove V. scaffold

" Post hyaloid

Identify & Rx retinal breaks

↓

Retinal laser Photocoagulation.

↓

Recurrent Hmrg

Laser.

Hyaloidotomy

↓

Subhyaloid Hmrg

» Macula region

Enzymatic Vitreolysis

↓

Enz hyaluronidase in pure form.

Complications

- V. Syneresis
- Bands & Memb → ERM, TRD
- 2° glaucoma → RBC lose its elasticity
  khakhi coloured (ghost cells)
  ↓
  Block TM. » if Ant hyaloid face involved.
- Erythroclastic ly → RBC, Hb, mφ obstruct TM
- Complicated Cataract →
- Sideotic Change
- OA

VH in Vitrectomized eye

Within 1 week
- Release of RBC entrapped in uncut V. base

Inadequate intraop hemo

Delayed
- NVI
  - Delayed type hrng in fibrovasc ingrowth
    ↓
    ∴ RDI ».

Congenital & developmental disorders of Retina.

- familial Exudative Viteoretinopathy
- Persistent fetal Vasculature Syndrome
- Coat's disease
- Norrie's "
- Congenital retinoschisis.

Familial Exudative Viteo-Retinopathy Osswick Schepens Syndrome

full term infants
c/o out history of O_2 supplementation.
85% Bil.

Etio

Malformation of Normal Retinal Vasculature
↓
Vacable area of Avascular Peripheral retina
↓
drive VEGF expression
↓
Leakage, Exudation.
↓
TRD.

Genetics → frizzled 4 mutations
LRP - 5, 6
Norrie disease
T. Span - 12.
] WNT Signalling pathway

Staging

1 → Avascular retinal periphery ± out Extrafoveal

2 → " with ✓

3 → RD subtotal ± out Exudate
fovea → × involve

4 → ✓

5 → Total, Open funnel, closed funnel

Dx

Dragged disc, RD-Exu, TRD, Radial folds — adhere to post lens

Can reactivate throughout lifetime.

Post Capillary deep-Out ← PhotoCoag
↓
Exud

Rx
- Peripheral laser ablation of Avascular periphery
- Vitrectomy ± Urokinase Enz to clear VR
  junctures, SOI
- Anti VEGF ± Caution.

Persistent Fetal Vasculature Syndrome (PFVS) (PHPV)

Disease of hyaloid system as well as tunica Vasculosa lentis

Vasc

Cf

Unil - 90%

Ant form	Post form	Combined
Smaller eye	Clear post lens capsule	Plaque & stalk upto OD
elongated ciliary processes & a plaque of vessels that extend to ciliary processes	vessels remaining in post pole at ON	≤ TRD.
↓	↓	
Tie into hyaloid vessel & stalk	Vessels dia 1-1.5 mm	
↓		
Opaque material in the area of lens to ON		
↓		
Opaque area replaced lens capsule ∴ cannot be peeled off		

Presentation

1st
- Around birth
- Leucocoria in eye
- ↓
- Rule out Rb.

2nd
- 6-10 yrs
- ↓
- Strabismus
- Dragging disc + TRD.

Uni
Bil
> Retinal Dysplasia

D.D Norrie's dis
but > Systemic issues

℞ Lensectomy
- Division of stalk
  - ↓
  - as TRD +
  - → avoid damage to Post lens Capsule
  - → floccullent Cataract formation
- Diathermy to stalk vessel

Association

- Post Coloboma
  - foveal involvⁿ
  - Dfferential VA results
- MicroCornea
  - Post lenticonus
  - PFV &
  - Colobama

Retinal dysplasia

- Gross (+)
  - Easily visible ⊆ Ophthalmoscopy
- Microscopic (−)

Norrie's disease

Malformation of Vasculature & Neural tissue
↓ ↓
FEVR Norrie dis phenotype

CNS + hearing + Ocular Malformations

Presentation

- avascular peripheral retina.
- Oval appearing str of retina in vitreous cavity c stalk extending frm centre of this to back of lens

↑ x VEGF release. as thin retina can manage c choroidal circulation

End stage
↓
Dysplastic PFVS
∴ PFVS + ~~Coat's~~ FEVR + Norrie
↓
Subretinal hmrg + Exudates

Vascular changes, Retinal dysplasia] House in PFVS + Norrie

Treatment → Defect in WNT signalling
Childhood screening
Dx Rx

Congenital retinoschisis

Involve 6 exon gene

100% foveal schisis 50% peripheral

Bil Males affected ♀ — silent Carrier

Classification

		Foveal cystic	Lamellar. Macular schisis	Peripheral Schisis
Type I	foveal	+	−	−
Type II	foveo-lamellar	+	+	−
III	complex	+	+	+
IV	foveo-peripheral	+	−	+

(Large retinal break

H/O Amblyopia & Strabismus.

c/f

Atrophic Macula

↓

Macular Deg

Large Schisis cavities that impede vision ⊆ RD

Recurrent VH.

Splitting occur at mid retina level not NFL. ✓

OPL

ERG ↓ B wave

℞

Combined schisis ≤ full thickness RD overhanging Macula

↓

Occiplasmin assisted vitrectomy

↓

Drainage of schisis cavity thru small sclerotomy.

- 39G Cannula to drain intraschisis fluid.

Tenascin C → ECM

↓

Schisis Cavity

70-80% silicon oil bubble

↓

face down fluid both lens surf

full RD.
FTRD

↓

Scleral buckling Vitrectomy tech

Retinal Vasculitis

Inflammation of retinal vessel wall resulting in evident clinical manifestations such as
- Vascular sheathing
- Leakage
- Occlusion

Etio

- Out systemic disease
  - Eale's
  - Frosted Branch Angiitis
  - Pars Planitis
  - Bird shot Retino Chorioidopathy
  - IRVAN (Idiopathic Retinal Vasculitis Aneurysm & Neuroretinitis)
- Systemic disease
  - Neurological — MS
  - Neoplastic — Acute Leukemia
    - Ocular lymphoma
  - Systemic inflammatory
    - BD
    - SLE
    - Sarcoid
    - WG
    - PAN, RA, RP
    - HLA B27
- Infectious
  - Bact
    - TB
    - Syphilis
    - Brucellosis
    - Lyme
    - Cat-Scratch
  - Viral
    - HSV
    - HZ
    - CMV
    - Hep B, C
    - HIV
  - Parasitic
    - Toxoplasma.
    - Lara.

On Basis of vessel involved.

Vein	Arteries	Both
- TB	- SLE	- Toxoplasmosis
- Sarcoid	- PAN	- Frosted Branch Angiitis
- Behcet	- Syphillis	- Wegner's
- MS	- HSV	- Crohn's.
- Eale's	- HZV	
- HIV	- IRVAN	

cf. Painless rapid ↓ of vision preceded by floaters

Large area of scotoma ⇔ area of ischaemia

Staging

Stage of Active Retinal Vasculitis →

- Sheathing, cuffing of BV & Vitreous Cells
- Dilated veins, Tortous & perivasc Exudates
- Superficial flame shaped Hmrg
- Sheathing
- Cotton Wool Spots
- Retinal edema.
- Intraretinal Hmrg

(Early signs of Occlusion)

Stage of Occlusion →

- Occlusion occurs CNP areas
- Jxn b/t CNP & P area → microAn
  - Veno-Venous shunts
  - Hard exudates
  - Cotton Wool Spots

Stage of NeoVascularization → CNP → VEGF → NVE

Retina Hypoxic

<90° → NVE

>90° → NVD

↓

fxn of CNP, P areas

NVE → Fragile, Leak & Retinal trauma like Haemorrhage of eye → VH

fibrovascular glosses → TRD

Advanced Stage of Disease → ↑ Hypoxia → VEGF permeate Ant Rubeosis Iridis → NVG → TRD.

Cause of Vision impairement → CME.
VH.
TRD.

RV.

NVE > NVD

Quick progression to NVE, NVD

Site of Occlusion

No of "

BRVO

at AV crossing

> 1

NVE >>

Specific presentation in diff Disease

a) T.B (Tuberculosis)

Choroiditis, Retinal periphlebitis
↓ obliterative ⊆ thick perivenous sheathing
↓
PVR.

Recurrent VH, TRD
Uveitis ± AC sxn.
Bil. young adults 3-4 decade ♂ > ♀

Ix — TST, +ve PCR, Quantiferon TB gold

Rx — Oral steroids ⊆ ATT
PVR → Laser PhotoCoag
Non resolving VH + TRD — PPV + EL
(*) good prognosis unlike PDR. ✓

(b) Sarcoidosis

Ant granulomatous Uveitis
Conj Nodule
Sd
Uveitis.
Peripheral phlebitis
Non Occlusive type ⊆ Segmental Cuffing
Extensive sheathing + Perivenous exudates ⇒
Candle Wax drippings.
Multiple small round Chorioretinal lesions

Xray chest, -ve Mantoux, ↑ Serum Ca, ACE levels

Laser. — PVR
Steroids. — Active stage
PPV — VH + TRD.

Behcet's Disease

HLA B51

Oral + genital + Skin + Eye.

Non granulomatous Panuveitis ± hypopyon (Steule)
Retinal Vasculitis

Leaky peri phlebitis
Recurrent | Vaso-Occlusive episodes

Recurrent attacks & remission

Aphthous Ulcer of Oral mucosa ± (2 or >) → genital ulceration
Eye lesions
Skin "
+ve pathergy test.

Multiple Sclerosis

Optic neuritis
PP
Retinitis
Periphlebitis — Occlusion

HIV

+ CMV

CMV Retinitis → Scattered yellow white area of Necrotizing retinitis ± Hmg ⇒ Pizza pie Retinopathy

Perivascular sheathing

Eale's Disease

VH

Constipation — Epistaxis

Idiopathic Inflammatory Veno-Occlusive disease of young ♀ adults.

↓

PVR.

± Vitritis, Uveitis

Retinal phlebitis

CNP. — NV.

& same. 3 parts.

State/UT

Syphilis STD T. pallidum

Leutic Vasculitis

a) focal & MFC. (Chorioretinitis)
- Vitritis
- Retinal Vasculitis
- Neuroretinitis

- thrive in all 3 layers
- ON
- IU, Panuveitis
- Pseudo RP.

ARN HSV 1,2, VZV

Peripheral Necrotizing Retinitis, R. arteritis ± Severe Vitritis
AC oxn.

Vision loss → RD, Macula Involve^n, Optic Neuropathy
Vasculitis

SLE Small Vessel Occlusion. Retinal Vascular lesions
Arteriolar Occlusion. Cotton Wool Spots ± RH.
Diffuse arteriolar occlusion ± extensive CNP

SLE + ↑APLA → » Vaso-Occlusive dis
Anti ds DNA, ↑ ANA, +ve SLE Cell phenomenon
↑ γ gls, ↑ circulating immune complexes
↓ serum Complement

PAN Necrotizing Vasc of small & medium sized arteries CW spots, Hmrg, edema, CRAO.
PUK, Necrotizing scleritis, Non granulomatous Intis, Vitritis, Papillitis, ION.

Toxoplasmosis — Focal necrotizing retinitis + Macula
Atrophic scar ⊆ pigmented borders
Reactivation seen
2° to TOXO → FBA, Occlusive Vasculitis
Granulomatous Ant Uveitis

Wg — Necrotizing granulomatous V.
involving Kidney & Respi tracts
Ocular — RAO, CAO, RVO, ON Vasculitis
Episcleritis, Scleritis, CU, Paranasal granuloma

Dx → ANCA
HPE — Inflammation of Small Vessels, Necrosis, granuloma formation.

Rested Branch Angitis — Diffuse PeriVasculitis
R. Edema, Severe Sheathing of R. Vessels
RH, HE, RD

FFA — Leakage of dye ✓ Rt × Occlusion.

Ass ⊆ every disease

Mx of RV → FFA, TLD, DLC, ESR, VDRL
FTA-ABS, Mantoux, HIV, X ray Chest.

FFA
Leakage of dye
Staining of vessels
Cx → CME, NVE, NVD, Vascular Occlusion + CNP.

Specific Ix

TB
+ve Mantoux
↑ESR
PCR 4m Aq.
Quantiferon TB gold blood
Other test
VA BSL-BD
DRS-SLE.
Uveous biopsy - Ocular lymphoma.
MRI - MS

Sarcoid
-ve Mantoux.
↑Serum Ca^{2+}
Serum ACE levels
CECT chest
Transbronchial lung Bx

Infectious dis
TOXO - Serology.
PCR - VZV, HSV, 1,2 CMV.

Systemic dis
RF
ANA
PANCA
ds DNA
CRP
LE Cell phenomenon

Complications
CME
VH.
TRD
Rubeosis irides
NVG

Treatment

RV

- Active Infective.
  - Steroids - 1mg/kg/day.
    - Tapering under Anti Bact / Viral / Parasitic Cover
- T.B. Presumed TB.
  - ATT x 9 mth + Oral steroids
- Non infective T.B
  - Steroids
    - Immuno Suppression

Rx of Complications

- CME
  - BT
- NVE.
  - NVE
    - Focal laser
  - NVD
    - PRP.
- Rec VH + TRD
  - PPV.
  - Anti VEGF. (To ↓ Intra-Op bleed)
- Rubeosis Iridis = NVG
  - PPP.
  - Glaucoma filtering Sx.

Prognosis

» than PDR

- younger ↑
- ↳ healthy Macula

Vogt Koyanagi Harada Syndrome

Chronic bilateral diffuse granulomatous panuveitis with accompanying integumentary, neurologic & auditory involvement.

Etio' & Patho

Autoimmune

T_{H1} ⟶ against melanocytes of all Organ System.

↑ IL 6, 8, IFN γ

Ag

- Tyrosinase related protein
- S100 ↓ against Melanocytes.

♀ > ♂ ? HLA DR B1 haplotypes

Stages of VKH

1. Prodromal
   - Diffuse Non Necrotizing granulomatous inflamm × CC involve
   - Dx: CSF ✓ .
   - Later fuchs Nodules
2. Acute Uveitic
   - Protein deposit under NSD.
3. Convalescent
   - Inflamm Rxn. lacks epithelioid cells & Loss of Melanocytes ↓ Sunset glow fundus.
4. Chronic Recurrent
   - granulom. chorioditis ± damage to CC.

c/f

Prodromal stage
Headache
Nausea.
Meningismus
fever
Tinnitus
Orbital pain.
Photophobia
Hair of skin & hair

focal neurologic signs & CSF pleocytosis

Auditory problems
- Central dysacusia involving ↑ freq.
- Tinnitus

Ac. Uveitic Stage
Bil granulomatous uveitis

↓

Blurring of vision

↓

Ant uveitis ≤ mutton fat KPs
- Iris Nodules
- Vitritis
- Thickening of post choroid
- Hyperaemia
- Edema of ON.
- NSRDs.
- Large bullous exudative RDs

↑IOP.

Convalescent Stage
Resolution of Exu RD

↓

deping of choroid

Sunset glow fundus

Perilimbal vitiligo

↓

Sigiura sign.

Vitiligo.
Alopecia
Poliosis.

Chronic recurrent stage
Bouts of gran. Ant uveitis ≤
- KPs
- Post synech.
- Iris Nodules
- " depigmen.
- steomal atrophy

↓

RSC.
Glaucoma
CNV
Subretinal fibrosis

Ix

FFA.

↓

Numerous punctuate hyperf foci at level of RPE in early phase

↓

Late phase - Pooling of dye

DISC leakage +
CME +

OCT
NSD
CME
CNV

ICG
detection & follow up of Subclinical cases

USG

∴ Thickening of post choroid (» Peripapill
Exudative RD
Vitreous Opacificat"
Post thickenn of Sclera

Diagnostic Criteria.

Manifestation

Early

1) Diffuse Choroiditis
   - focal area of SRF
   - NSD
2) focal delayed C. perfusion
   Pinpoint leakage
   Pooling of dye

USG – Diffuse Choroidal thickening
Cout evidence of Post scleritis

Late ← Similar finding (2)

- Depigmentation
  - Sunset glow fundus
  - Sigiwa sign.

Nummular scars or RPE clumps

Neurologic / auditory findings
Integumentary findings

(A) Meningismus
Tinnitus
CSF pleocytosis

(B) Alopecia
Poliosis.
Vitiligo.

VKH

Complete.

Incomplete.

Probable

I – No H/O penetrating Ocular Tx or Sx
II – No. clinical/lab evidence of other ocular dis
III – Early ocular disease (Early + Late)

I-III

I-III + A + B

I-III

4/5

℞

Acute – i.v CH_3 Pred

↓

Oral steroids

Intravitreal steroid inj

Immunosupression.

Triple ℞ ← Oral Pred.
Azathioprine
Cyclosporine

Sympathetic Ophthalmia

Sympathetic Uveitis.

Rare, bilateral diffuse granulomatous uveitis

- Exciting eye
- Sympathizing eye.

5d — 66yrs avg > 2wk.

- Can occur after any ocular surgery / Trauma

↓ incidence due to aggressive Mx of severely traumatized eye.

♂ > ♀

Etiopath T cell response — ↑ of soluble protein ags
- ⊆ retinal photoreceptor Memb → ↑ Ag.
- HLA association (+)

Thickened choroid
Infiltrating lymphocytes
Spacing of CC
↑. Infiltrates
R. perivasculitis & RD
Scleral involvement +
Phaco-Anaphylaxis

Dalen Fuch's Nodules

focal aggregates of epitheloid histiocytes admixed with RPE of Bruch's Memb & RPE
Small, yellow white deposit underneath RPE.
+nt in VKH, SO.

In SO → 1/3 pt +
Mixture of Ia +, OKM1+ cells
depigmented RPE cells — Ia −, OKM1 −

CF

↓ Vision, photophobia, ↑ lacrimation, blurring, paresis of accomodatn

- Ciliary inj.
- dilated poorly responsive pupil
- Thickened iris
- Clouding of Vitreous
- KPs (+)

Papilitis
Optic R. Edema,

classmate
Date 69
Page

Jx

clinical Dx

FFA

Multiple subretinal enlarging hyperf. spots
& pooling of dye
Late staining of R. Vessels

Later — block f in early phases
Hyper f in Late "

cicatricial — Atrophic
Window defects

ICG

Intermediate hypo ↷ fading

Atrophic scar — Hyper throughout

Complication — Cataract, 2° glaucoma, Exudative RD, Chorio-Retinal scaring, Choroidal NV, OA, Subretinal fibrosis

℞ Prevention — Proper Ocular toilet

x Enucleation of Exciting eye ⇒ may provide good vision

Steroids
Subtenon inj of steroid, mydriatic, Cycloplegics

- Azathioprine 2-2.5 mg/kg/day
- Chlorambucil 6-8 mg PO OD
- Ciclosporin 5mg/kg.

i.v cyclophosphamide is given.

Retinal Vein Occlusions

CRVO

DR → RVO

Incidence

RVO in 1 eye — Hayreh report

↓

After/in 4yrs RVO

- Same eye → 2.5%
- Other eye → 11.9%

♂ > 65yrs > Unil.

BRVO = 3 * CRVO

CRVO.

♂ >

BRVO.

3* »

♀ = ♂

Hemi CRVO.

NVE, NVD > common

CRVO.

NVI, NVA ✓✓

Pathogenesis

CRA & CRV share common sheath as they exit ONH & pass through narrow opening in lamina cribrosa

↓

Narrow opening/entry — Tight Compartment

↓

Anat position — predispose to Thrombus formation

↓

Slowing of blood steam (stasis of Bld)

↓

Virchow Triad

- Changes in Blood (Hypercoagulability)
- Changes in vessel Wall (Endothelial Injury)

↓

Occlusion of CRV

↓

Back pressure build up in Venous System.

↓

↑ resistance to Bld flow

↓

Stagnation of blood

Ischemic damage to Retina

↓

↑ VEGF in vitreous cavity

↓

Stimulate NeoVasc of Post/Ant segment

↓

Capillary leakage

↓

CME.

Site of Occlusion

Non I.
↓
further back
↓
than Retrolaminar region

9
↓
Lamina Cribrosa.
Thrombus near it
» Complete.

Severity of Retinopathy ∝ Site of Occlusion

↓ severe if further as
» collateral channels.

No. of Risk factors → Advancing Age. — young — 25%
HTN — > 50yr — 64%
DM, CAD
Hyperlipidaemia

(*) Evaluate pt if < 50 yrs
Bilateral CRVO

- Hyper Homocysteinemia
- Factor V leidan Mutation
- Protein C & S.
- Abn fibrinogen level
- APLA
- Lipid Anticoag factor.

↑ Hematocrit
Hyperviscosity — Polycythemia, Leukemia, Sickle Cell Disease, Multiple myeloma, Waldenstrom Macroglb dehydration

Ocular condition predispose to CRVO
ION, Tilted Disc, ONH drusen, Thyroid related Ophthal

OCPs, Diuretic, Hep B vaccine.

Classification

Non Ischaemic CRVO	Ischaemic CRVO
80%	20%
Venous stasis	Haemorrhagic
↓	
Thrombosis is incomplete R. is ½ perfused mild form	Complete Now perfused severe
> 6/60	< 6/60
×	✓
RAPD	
Hmg +nt	» dense
ME +/-	Tomato ketchup R.
Disc edema +	ME + ✓
- inflam	DE ✓, VH ✓
- Occlusive	Cotton Wool Spots → ✓
Papillophlebitis	
Prolongation of Arm Circulation Time & decr of Non Perfusion	State of perfusion
Block of flow in Cilio Retinal ♀	CVP > IOP on post pole.

RFA

Non Ischaemic		Ischaemic
x	Perimetry	Dense Central Scotoma
(N) <5%	ERG	↓↓ Wave applitude 45-80%.
	NVI	

Course

Non ⟶ Isch

3yrs

as progression of thrombus from proximal to distal part of retrolamina.

Reason for Vision loss

- Macular Edema.
- ERM
- Dense Intraretinal Hmrg
- Macular Ischaemia
- VH
- TRD.
- NVG

Complications

- NVI + NVA
- NVD » ⊆ in 1m/h
- CME — Intractable in Isch. CRVO
- VH
- ERM
- Optociliary shunts vs NVD
  - Disc Collateral » calibre
  - x leak. FFA
  - x allow. ⟶ As. NV.
  - flat.

raised

Exam → H/O
Family H/O
Risk factors
Undilated Ophthalmoscopy — NVA
90D, 20D
ERG
Perimetry
FFA → wait 4 wks for bleeding to subside
↓
CNP to come
OCT — ERM, ME.

Prog → dR — Bil >
OIS → dilated veins x tortuous
RH → Midperiphery
H/O Amaurosis fugax, TIA / Orbital pain

Rx → PTO

Follow Up

VA
≥ 6/12 → 1-2 mthly x 6m
< 6/60 → mthly / 6m
2mthly / 6m

Prognosis → Neu > Isch.
∝ development of collaterals.

Conclusion

Various Studies

Surgical Rx

Rx

Medical Rx

- Rx of associated Medical conditions
- x Smoking
- Rx glaucoma
- x established role of pentoxyphyline
- x role of systemic Anticoag!

LASER Rx

grid pattern laser

↓

- ME
- Early Rx to prevent NVI & NVA.

⇓

NVG.

↓

CRVO study

↓

LASER only in young ≤ 50ch CRVO waiting for NVI development at 2 clock hr

> Indian scenario

→ Chorio-Retinal Venous anastomoses — by Laser at BRV to septicle Post Wall & Bench's stents.

TPA.

↑

↓

Intravitreal Anti VEGF drugs

Avastin (0.05ml/1.25mg)

Anti VEGF.

↳ NVG.

↓

Intravitreal steroids

NTA — 1mg/0.1ml

> Better in Non Isch

» RD Cataract

SCORE Study

Ozurdex — 700µg

micronised dexa + co-polymer of lactic & glycolic Acid.

↑ IOP; Cataract format

✓ → Aphakic

→ Pseudophakic

→ Steroid Non responders

genova study

Delivery Instructions

CLASSMATE Date Page

Surgical Rx

PPV → Non clearing VH

Radial Optic Neurotomy

Rx compartment Syndrome

PPV + Transvitreal incision of Nasal Scleral ring to release pressure & to Dx. decompress CRV at level of scleral outlet

Intravitreal tPA

CRVO

Scatter PRP ✓

× Macular grid

Hemi CRVO

least common type.

Cf

Sup & Inf field defect
Vision loss
Perimetry → Scotoma + Segmental defect ≤ large Isopter

Dd

Hemi CRVO.		CRVO
I/NI		Isch
Optic Nerve	Site of Occlusion	AV crossing
Edematous	Disc	(N)
Near OD or over it Collaterals		Away frm disc.

NVE, NVD
∴ rarely NVG ✓

Rx

= CRVO.

Prognosis

Better V. prognosis

BRVO

> Temporal
> Sup Quad

R/f → HTN, DM, dyslipidaemia, OAG, Age related atherosclerosis

Major. Minor.

Pathogenesis — Ischaemic type.

Atherosclerosis → Thickening of Central A.

↓

Compression of Adj vein

↓

Turbulent blood flow.
Endothelial cell damage
Thrombotic Occlusion

↓

↑IOP. → Poor health
Bleeding.

ME + Ischaemia +

c/f
- Sudden painless ↓ DOV
- Blind spot in field of vision
VA > CRVO

Early → Late.
ME + CNP + (by FFA)

Dd vs Hemi CRVO

Retinal vasculitis — If site of occlusion is not AV Jxn

Mx

- Medical Rx
  - ↓ Rx risk factors.
- Laser Therapy
  - 0.1s (green laser) 532 Nd YAG
  - 100µ spot size
  - Power – Med white. burn
  - → CME → NVE → VH
- Anti VEGF.

grid pattern → Thinning of retina so that choroidal vasc supplies inner retinal needs

↓

Autoregulatory Constriction of R. Vasc in leaking area

↓

↓ Edema

- Intra Ocular Cs.
  - ↓ IVTA
  - ↑ Cataract formatn
  - ↑ IOP
  - Endoph
  - VH.
  - Lens injury
  - RD
- Sx Rx
  - PPV
    - Non clearing VH
    - TRD Involv" Macula
    - ERM
    - Complicat" of ghost cell glaucoma
  - PPV + PVD
  - AV adventitial sheathotomy
    - decompress Vein

Diff

Macular BRVO.		Exudative ARMD
Uni		Bil
+	Ass systemic dis	-
patch of sup hmrg CWS spots + ME	Clinical picture	patch of subret hmrg + exudates, NSD RPE detatchment + soft drusen
CWP area. Petalloid pattern HF µAn	FFA	Lacy pattern of HF
↑ thickness at site of lesion.	OCT.	classic | occult CNV.

BRVO LASER → 3mth for spon recovery & Collateral ✓ of ME & NVE beyond 3mth

CI → Macular isch
↓ VA due to RPE changes

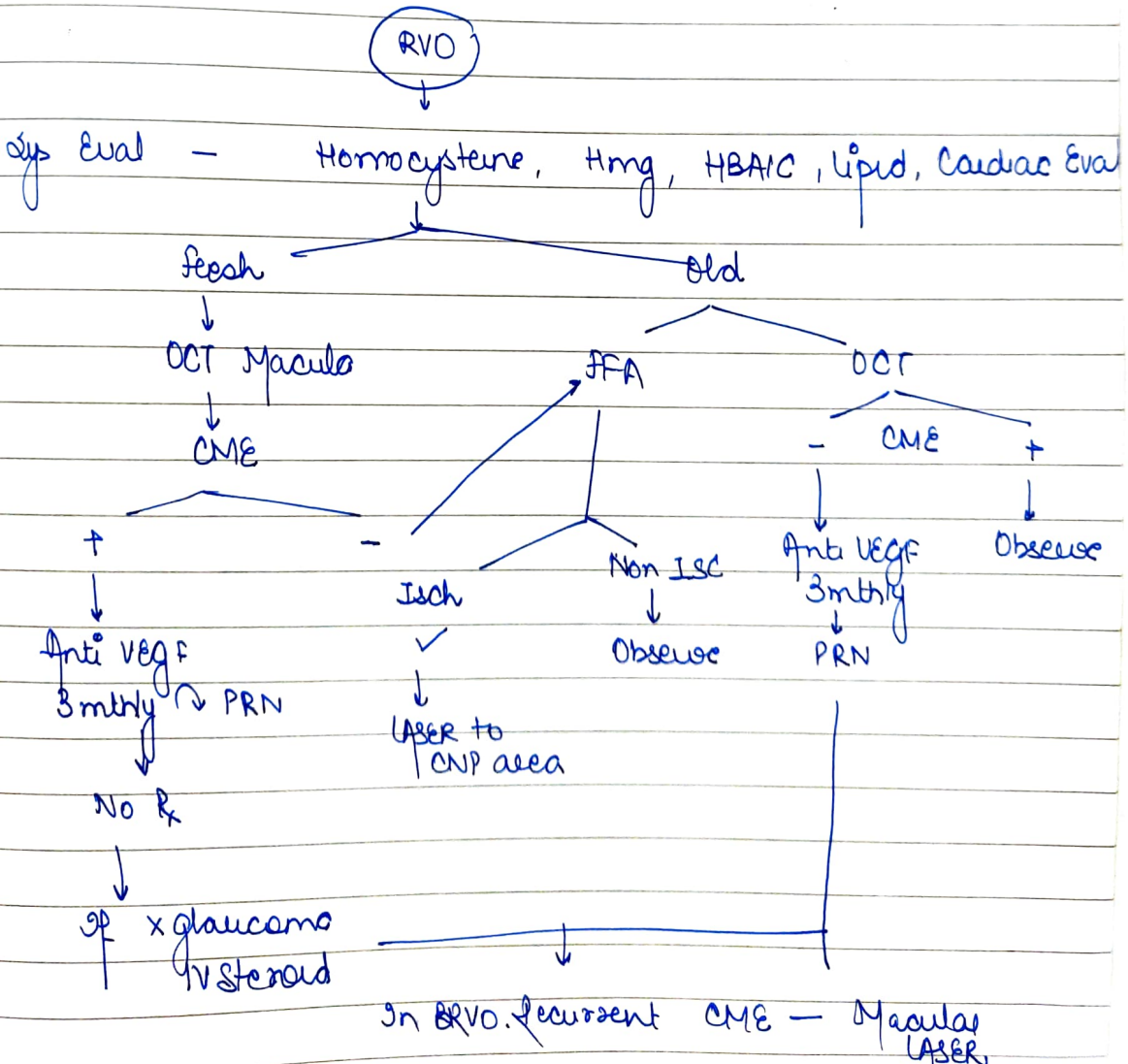
Scatter laser ✓
DD beyond fovea

Macular and outside FAZ

Tanzanite study — Supachoroidal inj of TA + Aflibercept

grading of NVI

grade	
0	No Iris NeoVascularization
1	< 2 Quad of NV at Iris pupillary zone
2	> 2 Quad " " "
3	grade 2 + < 3 Quad of NV at Iris ciliary zone & Ectropion uvea
4	> 3 Quad of NV at Iris ciliary zone or ectropion uvea.



Idiopathic Juxtafoveal Telangiectasia

Other d.d of telengectasia — Coat's
RVO
DM
Irradiation
Carotid artery obstruction

Teminology

yass

I — A, B
Congenital
Unilateral
ME+

II — A, B
Acq.
Bil
Atrophy of fovea
↓
5 Categories

III — A, B

II A > I > III

Aneurysmal telangiectasia

Perifoveal (II A) ← Yannuzi

Aneurysmal Telangiectasia
IJT / Macular T.

Perifoveal. or Type II
Coat's found in Macula

Cf

♂, unil., Cong
T. Capillaries with Aneurysmal dilatation
ME+, lipid deposition +

Acquired, Bil
Metamorphopsia ✓
5 Stages

Stage 1
↓
Asymp
loss of retinal transp
grayish app

Stage 2
Slight graying
- Thinned out foveal centre
Occult telangies

Stage 3
↓ vision
Vertically oriented slightly dilated right ∠ venules

Stage 4
RPE clumps

Stage 5
SRNV
(Sub retinal NV
- Remodelling
- Proliferation
- Invasion of outer retina

Telengiectasia, foveal atrophy
NO visual loss

SRNV + fibrosis
✓ Visual loss

~~Occult~~	Visible.	Telengiectasia	Occult
		Superficial Cystals	+
✓		Ballooning or Cystic spaces in OPL	×

SRNVM

CNVM

✗

<

does not deteriorate.

PE detachment ✓
final size
VA

IJT - 1

Vessels filling both deep & Sup
↓ CNP areas

IJT - 2

teleng capillaries
deeper vasculature involved.

ME +
↑ retinal thickness
fluid filled spaces
shallow M. detachment

foveal ateophy
Inner lamellar cyst ⊆ ILM deape
Blunting of foveal pit
Intraretinal NV near foveola
Central Intra-retinal hyperreflective L

✓✓ Role FAf
Microperimetry.

RVO, DR, Radiation R.
Sickle Cell M, OIS
Coat's

Other. Ass.

Macular Hole.
RVO
Radiation R
Best's
SRNV — ARMD

Rx

(SRNV)

Non Prolif
↓
Avastin

Proliferative
↓
LASER
PDT ⊆ IVTA
TTT
Ox removal

LASER
IVTA —
Anti VEGF — ME +