Ocular manifestations of systemic diseases - how you can save lives in your everyday practice

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Auckland
Aotearoa / New Zealand

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Greenlane Clinical Centre, Auckland, NZ

	2003	2025
Ophthalmologists	19 P/T	50 P/T
Optometrists	1 P/T	9 P/T
Orthoptists	2 F/T	4 F/T
OPC / year	52,000	118,000
Acute consults / year	9,000	17,5000
Optom clinics	Retinal screening, paeds, CL	DRS, paeds, CLs, LV, Glaucoma, Corneal, Medical Retina, EEC, virtual Med Ret, Virtual Glaucoma

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Learning objectives:

- Present some cases of ophthalmic conditions with a systemic disease association or aetiology
- Review the differential diagnoses of these conditions
- Discuss the clinical findings that are suggestive of a systemic disease association or aetiology
- Discuss the optometric and collaborative management in these cases
- (to interact with the audience to ensure deep and considered thinking about the cases, differentials and workups)…

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The ones that we're used to:

- Undiagnosed diabetes (*)
- Chronic hypertensive retinopathy (*)
- Corneal arcus (*)
- Xanthelasma (*)
- Papilloedema (* refer to neuro?)
- Thyroid dysfunction
- Multiple sclerosis
- GCA

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Case 1 - presentation

- 47 year-old Polynesian male was seen in a visiting outreach clinic in the Pacific Islands.
- c/o bilateral recent vision reduction over the previous week.
- He had no prior ocular history and his general health was reported to be good, although he did mention experiencing unintentional weight loss of 10 kg over the previous six months (*).
- He had experienced no night sweats, fatigue or anorexia and he gave no significant neurological symptoms or history of neurological problems (*)
- Family ocular history was unremarkable.

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Case 1 – 2018 examination

- Vision (unaided) right 6/48 (no improvement with pinhole) and left 6/36 (no improvement with pinhole).
- IOPs were right and left 14 mmHg.
- OE, the anterior segments were unremarkable.
- A dilated fundus exam was performed:

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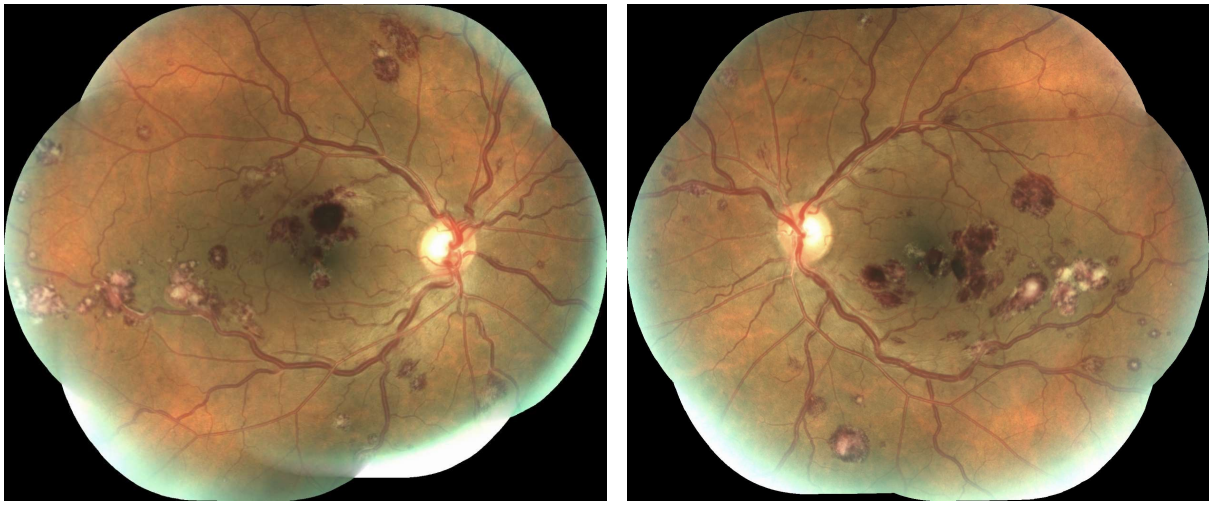
Case 1 – composite fundi images



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Case 1 – diagnosis and differentials

- A provisional diagnosis of CML was made with differential diagnoses of:
- diabetic retinopathy
- infective endocarditis
- Anaemia
- bilateral simultaneous central retinal vein occlusions.
- Blood testing was ordered (*) locally
- Showed some anaemia (Hb 100, normal 130 – 175) but marked leukocytosis (white blood cell count > 500, normal 4 - 11)
- Results were consistent with the diagnosis of CML. He was referred to New Zealand for treatment after discussion with Haematology in NZ

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Case 1 – bloods (in NZ)

Haemoglobin	**102**	**g/L**	**L**	**130-175**
RBC	**3.08**	**x E12/L**	**L**	**4.3-6.0**
Haematocrit	**0.29**		**L**	**0.4-0.52**
Mean Cell Volume	95	fL		80-99
Mean Cell Haemoglobin	**33.1**	**pg**	**H**	**27-33**
RDW	**19.4**		**H**	**11.5-15.0**
RBC (nucleated)	**15.57**	**x E9/L**	**H**	
Platelets	175	x E9/L		150-400
MPV	10.3	fL		9.0-12.2
WBC	**521.2**	**x E9/L**	**H**	**4.0-11.0**
Blasts	**5.2**	**x E9/L**	**H**	
Neutrophils	**218.9**	**x E9/L**	**H**	**1.9-7.5**
Basophils	**20.9**	**x E9/L**	**H**	**0-0.2**
Eosinophils	**10.4**	**x E9/L**	**H**	**0-0.5**
Monocytes	**0.0**	**x E9/L**	**L**	**0.2-1.0**
Lymphocytes	**10.4**	**x E9/L**	**H**	**1.0-4.0**
Immature Granulocytes	**255.4**	**x E9/L**	**H**	**0.0-0.06**
Blood Film				
Haematologist's Comment				
Authorised by	Lynn Palmer, RMLS			
Referred	REFERRED SPECIMEN			

Blood Film
RED CELLS - normal morphology WHITE CELLS - marked neutrophil left shift, blast cells present PLATELETS - giant platelets present
Reported by: Lynn Palmer, RMLS

Haematologist's Comment
Haematology comment: Marked leukocytosis with leukoerythroblastosis, basophilia and eosinophilia. Neutrophils are left-shifted to the blast stage with the majority being myelocytes. Blasts comprise 1% of the leukocytes and basophils 2%. The features are consistent with chronic myeloid leukaemia. A bone marrow biopsy has been organised for further evaluation.

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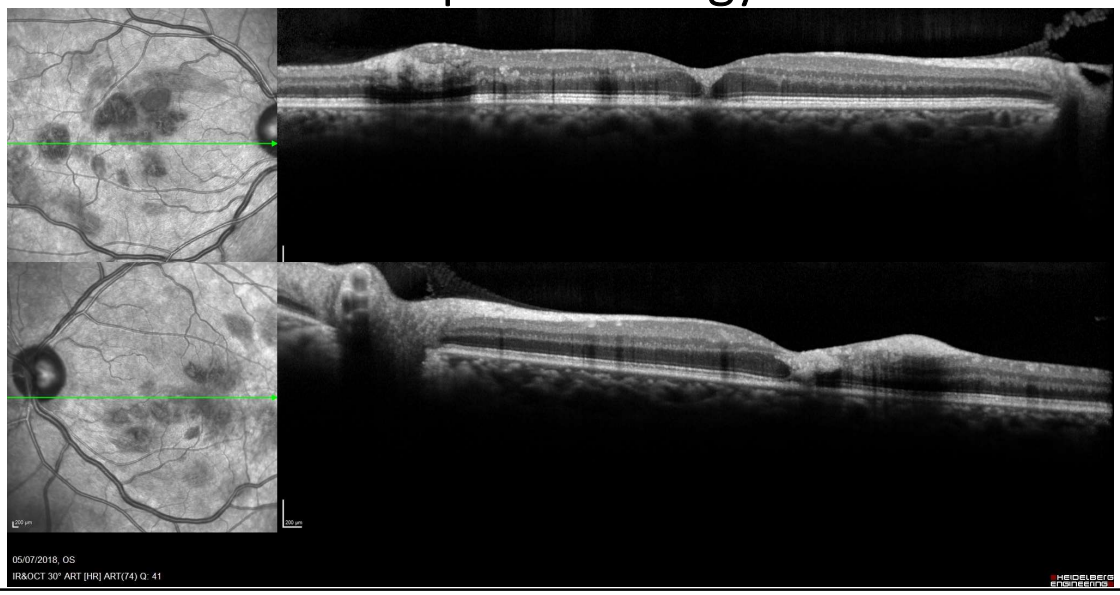
Case 1 – seen in Ophthalmology



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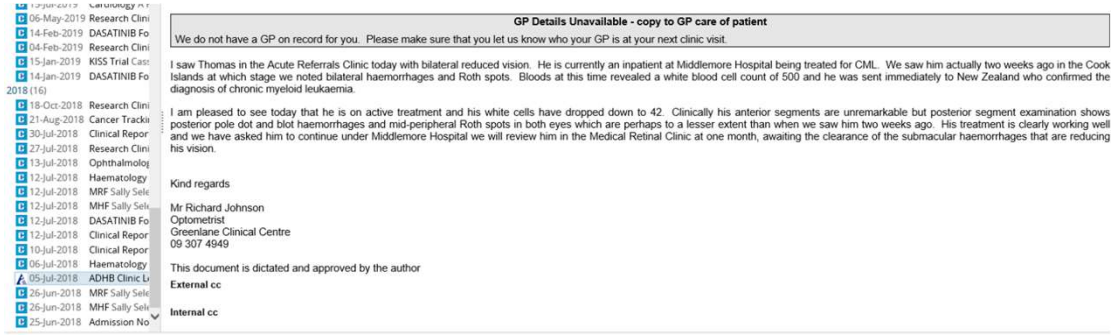
Case 1 - correspondence



06-May-2019 Research Clini
14-Feb-2019 DASATINIB Fo
04-Feb-2019 Research Clini
15-Jan-2019 KISS Trial Cas
14-Jan-2019 DASATINIB Fo
2018 (16)
18-Oct-2018 Research Clini
21-Aug-2018 Cancer Tracki
30-Jul-2018 Clinical Repor
27-Jul-2018 Research Clini
13-Jul-2018 Ophthalmolog
12-Jul-2018 Haematology
12-Jul-2018 MRF Sally Sele
12-Jul-2018 MHF Sally Sele
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12-Jul-2018 Clinical Repor
10-Jul-2018 Clinical Repor
06-Jul-2018 Haematology
05-Jul-2018 ADHB Clinic L
26-Jun-2018 MRF Sally Sele
26-Jun-2018 MHF Sally Sele
25-Jun-2018 Admission No

GP Details Unavailable - copy to GP care of patient

We do not have a GP on record for you. Please make sure that you let us know who your GP is at your next clinic visit.

I saw Thomas in the Acute Referrals Clinic today with bilateral reduced vision. He is currently an inpatient at Middlemore Hospital being treated for CML. We saw him actually two weeks ago in the Cook Islands at which stage we noted bilateral haemorrhages and Roth spots. Bloods at this time revealed a white blood cell count of 500 and he was sent immediately to New Zealand who confirmed the diagnosis of chronic myeloid leukaemia.

I am pleased to see today that he is on active treatment and his white cells have dropped down to 42. Clinically his anterior segments are unremarkable but posterior segment examination shows posterior pole dot and blot haemorrhages and mid-peripheral Roth spots in both eyes which are perhaps to a lesser extent than when we saw him two weeks ago. His treatment is clearly working well and we have asked him to continue under Middlemore Hospital we will review him in the Medical Retinal Clinic at one month, awaiting the clearance of the submacular haemorrhages that are reducing his vision.

Kind regards

Mr Richard Johnson
Optometrist
Greenlane Clinical Centre
09 307 4949

This document is dictated and approved by the author

External cc

Internal cc

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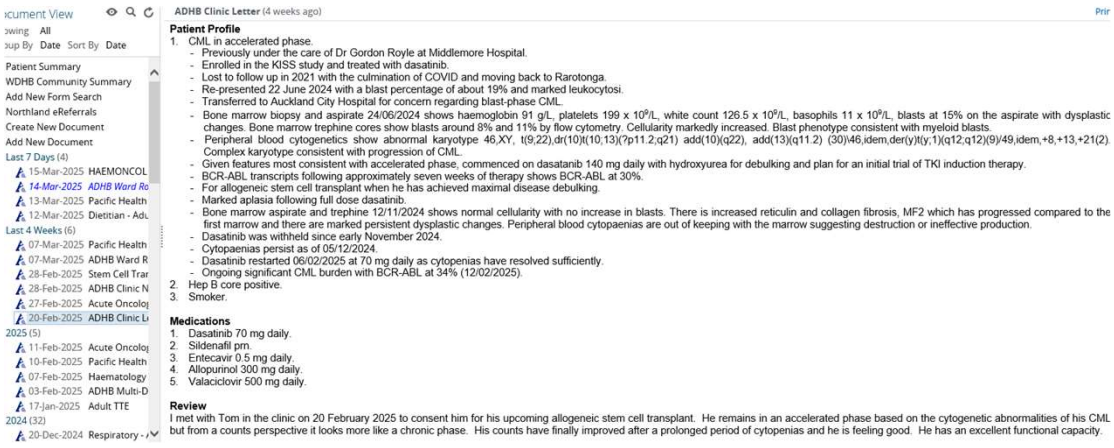
Case 1 – 2025 update



ıcument View
owing All
oup By Date Sort By Date
Patient Summary
WDHB Community Summary
Add New Form Search
Northland eReferrals
Create New Document
Add New Document
Last 7 Days (4)
15-Mar-2025 HAEMONCOL
14-Mar-2025 ADHB Ward Ro
13-Mar-2025 Pacific Health
12-Mar-2025 Dietitian - Adu
Last 4 Weeks (6)
07-Mar-2025 Pacific Health
07-Mar-2025 ADHB Ward R
28-Feb-2025 Stem Cell Trar
28-Feb-2025 ADHB Clinic N
27-Feb-2025 Acute Oncolog
20-Feb-2025 ADHB Clinic L
2025 (5)
11-Feb-2025 Acute Oncolog
10-Feb-2025 Pacific Health
07-Feb-2025 Haematology
03-Feb-2025 ADHB Multi-D
17-Jan-2025 Adult TTE
2024 (32)
20-Dec-2024 Respiratory -

ADHB Clinic Letter (4 weeks ago) Prir

Patient Profile

1. CML in accelerated phase.
   - Previously under the care of Dr Gordon Royle at Middlemore Hospital.
   - Enrolled in the KISS study and treated with dasatinib.
   - Lost to follow up in 2021 with the culmination of COVID and moving back to Rarotonga.
   - Re-presented 22 June 2024 with a blast percentage of about 19% and marked leukocytosi.
   - Transferred to Auckland City Hospital for concern regarding blast-phase CML.
   - Bone marrow biopsy and aspirate 24/06/2024 shows haemoglobin 91 g/L, platelets 199 x 10^9/L, white count 126.5 x 10^9/L, basophils 11 x 10^9/L, blasts at 15% on the aspirate with dysplastic changes. Bone marrow trephine cores show blasts around 8% and 11% by flow cytometry. Cellularity markedly increased. Blast phenotype consistent with myeloid blasts.
   - Peripheral blood cytogenetics show abnormal karyotype 46,XY, t(9;22),dr(10)t(10;13)(?p11.2;q21) add(10)(q22), add(13)(q11.2) (30)\46,idem,der(y)t(y;1)(q12;q12)(9)/49,idem,+8,+13,+21(2). Complex karyotype consistent with progression of CML.
   - Given features most consistent with accelerated phase, commenced on dasatanib 140 mg daily with hydroxyurea for debulking and plan for an initial trial of TKI induction therapy.
   - BCR-ABL transcripts following approximately seven weeks of therapy shows BCR-ABL at 30%.
   - For allogeneic stem cell transplant when he has achieved maximal disease debulking.
   - Marked aplasia following full dose dasatinib.
   - Bone marrow aspirate and trephine 12/11/2024 shows normal cellularity with no increase in blasts. There is increased reticulin and collagen fibrosis, MF2 which has progressed compared to the first marrow and there are marked persistent dysplastic changes. Peripheral blood cytopaenias are out of keeping with the marrow suggesting destruction or ineffective production.
   - Dasatinib was withheld since early November 2024.
   - Cytopaenias persist as of 05/12/2024.
   - Dasatinib restarted 06/02/2025 at 70 mg daily as cytopenias have resolved sufficiently.
   - Ongoing significant CML burden with BCR-ABL at 34% (12/02/2025).
2. Hep B core positive.
3. Smoker.

Medications

1. Dasatinib 70 mg daily.
2. Sildenafil prn.
3. Entecavir 0.5 mg daily.
4. Allopurinol 300 mg daily.
5. Valaciclovir 500 mg daily.

Review

I met with Tom in the clinic on 20 February 2025 to consent him for his upcoming allogeneic stem cell transplant. He remains in an accelerated phase based on the cytogenetic abnormalities of his CML but from a counts perspective it looks more like a chronic phase. His counts have finally improved after a prolonged period of cytopenias and he is feeling good. He has an excellent functional capacity.

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Case 1 – take home messages

- Consider the type, appearance and location of retinal haemorrhages
- Consider the symmetry of retinal haemorrhages
- Bilateral is usually systemic (eg CML, diabetes, hypertensive retinopathy)
- Unilateral is usually local (eg, conversion of AMD to wet)
- Speed of onset of symptoms also helps differentiate
- If suspect a systemic blood dyscrasia, as an optometrist, discuss with GP (or ophthal)
- Telemedicine is perfect for eyes…

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Case 2 – a short story…

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Case 2 - G6PD

- G6PD is an enzyme that has an essential role in the defence against cellular oxidative injury.
- Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzyme deficiency in the world
- Approx 400m people world wide, mainly of African, Mediterranean and Asian descent.
- X linked inheritance, therefore males only, female carriers
- Symptoms & signs include anaemia, tachycardia and fatigue when RBCs are haemolysed
- Premature cataract
- Protection vs malaria
- RBCs get haemolysed by Fava beans, blueberries, moth balls and red wine as well as some sulphur drugs, especially anti malarials and can go to haemolytic crisis (anaemia)
- Test for G6PD costs approx $USD 20.

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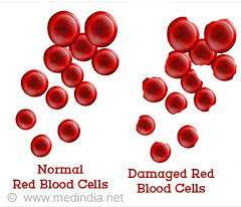
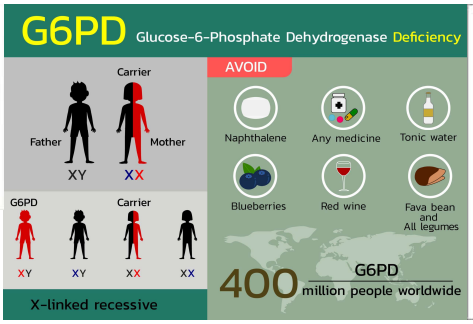
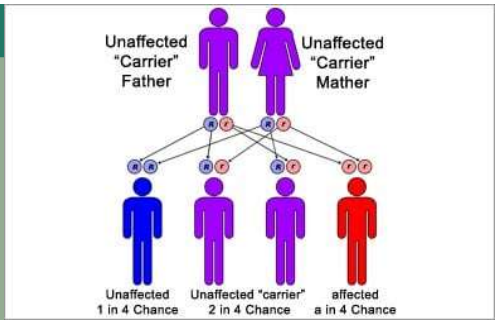
Case 2 - G6PD vs SCA







Images from MedIndia.net

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Case 2 – Issues of G6PD and SCA in diabetics

- HbA1c not accurate in conditions that cause as RBC haemolysis (eg G6PD and sickle cell anaemia)
- Testing is based on life span of a RBC of 3/12.
- Therefore diagnosing diabetes is often late and undertreating occurs in this group due to false HbA1c readings.

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Case 2 – take home messages

- Test for G6PD in all cases of premature cataracts
- (Or at least ask about symptoms)
- Reiterate the need to avoid the triggers of haemolysis
- Be aware that HbA1c may be inaccurate during acute episodes

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Case 3 – ~~presentation~~ background

- 53 y o Samoan female
- Imaged in a Virtual Medical Retinal clinic Nov 2024
- Last seen June 2023
- Bilateral moderate diabetic retinopathy
- Macula clear, no foveal IRF or SRF
- VA with glasses RE 6/6 LE 6/6
- IOPs RE 14 mmHg LE 14 mmHg
- 15/4/23: HbA1c 91 mmol/mol

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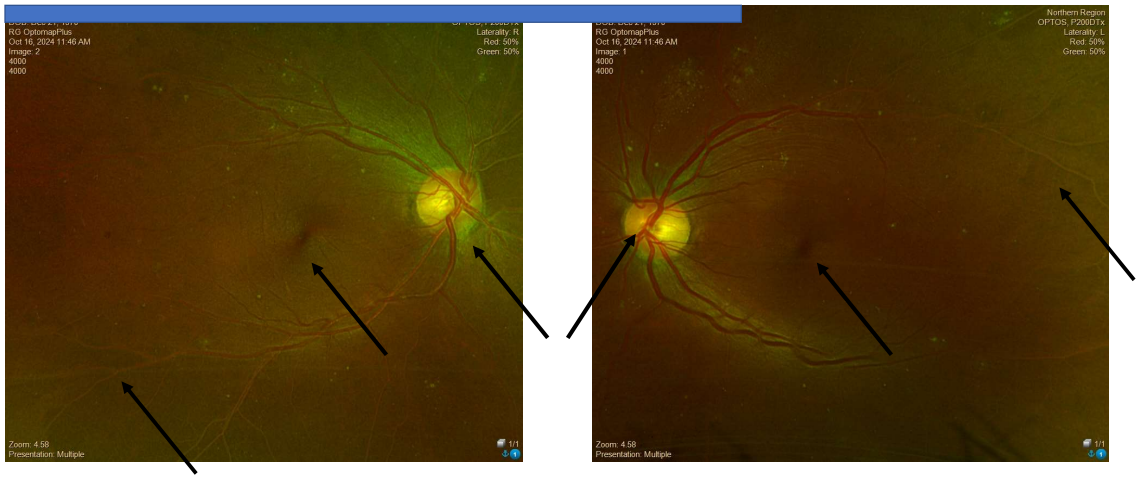
Case 3 – Optos images (central)



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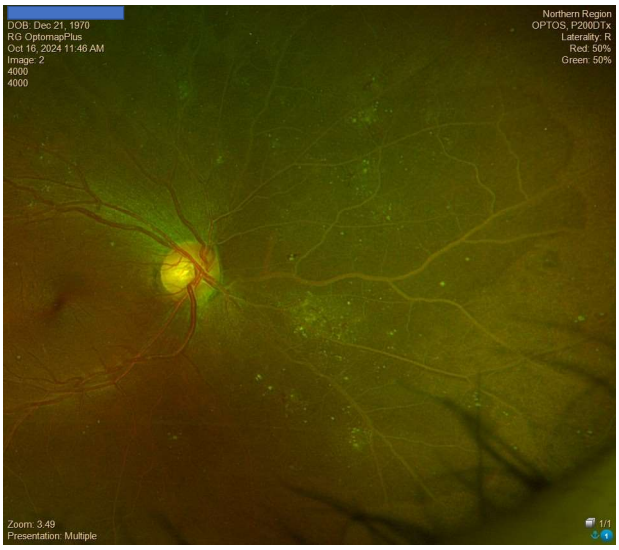
Case 3 – Optos images (peripheral)



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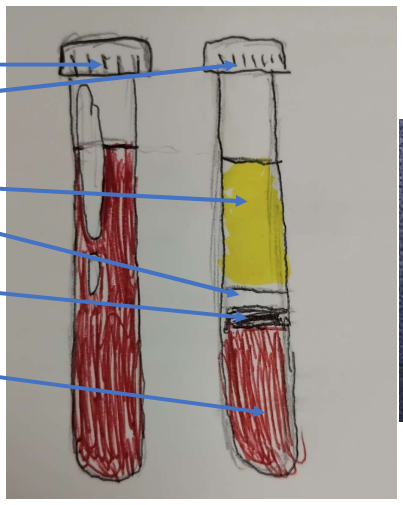
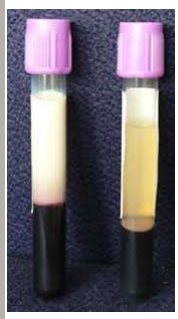
Blood – a quick refresher

- Whole blood
- Fractionated blood
- Plasma (52-62%)
- WBCs (< 1%) (-phils & lymphocytes)
- Platelets (<1%) (thrombocytes)
- RBCs (38-48%)





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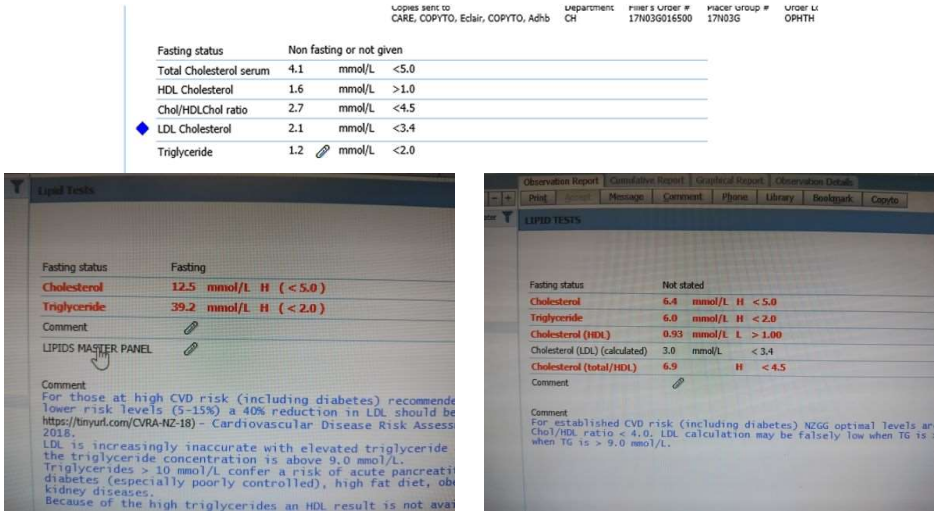
Case 3 – blood results



Copies sent to: CARE, COPYTO, Eclair, COPYTO, Adhb | Department: CH | Filler's Order #: 17N03G016500 | Placer Group #: 17N03G | Order Li: OPHTH

Fasting status	Non fasting or not given		
Total Cholesterol serum	4.1	mmol/L	<5.0
HDL Cholesterol	1.6	mmol/L	>1.0
Chol/HDLChol ratio	2.7	mmol/L	<4.5
LDL Cholesterol	2.1	mmol/L	<3.4
Triglyceride	1.2	mmol/L	<2.0

Lipid Tests

Fasting status	Fasting		
Cholesterol	12.5	mmol/L H	(< 5.0)
Triglyceride	39.2	mmol/L H	(< 2.0)
Comment			
LIPIDS MASTER PANEL			

Comment
For those at high CVD risk (including diabetes) recommende
lower risk levels (5–15%) a 40% reduction in LDL should be
https://tinyurl.com/CVRA-NZ-18) - Cardiovascular Disease Risk Assess
2018.
LDL is increasingly inaccurate with elevated triglyceride
the triglyceride concentration is above 9.0 mmol/L.
Triglycerides > 10 mmol/L confer a risk of acute pancreati
diabetes (especially poorly controlled), high fat diet, ob
kidney diseases.
Because of the high triglycerides an HDL result is not ava

Observation Report | Cumulative Report | Graphical Report | Observation Details
Print | Accept | Message | Comment | Phone | Library | Bookmark | Copyto

LIPID TESTS

Fasting status	Not stated		
Cholesterol	6.4	mmol/L H	< 5.0
Triglyceride	6.0	mmol/L H	< 2.0
Cholesterol (HDL)	0.93	mmol/L L	> 1.00
Cholesterol (LDL) (calculated)	3.0	mmol/L	< 3.4
Cholesterol (total/HDL)	6.9	H	< 4.5
Comment			

Comment
For established CVD risk (including diabetes) NZGG optimal levels ar
Chol/HDL ratio < 4.0. LDL calculation may be falsely low when TG is
when TG is > 9.0 mmol/L.

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Case 3 - Lipaemia retinalis

- LR occurs in the presence of extremely elevated triglyceride levels
- usually exceeding 1,000 mg/dL (55.5 mmol/L) to 2,500 mg/dL (138 mmol/L) (seems very high to me)
- Begins initially with changes in the peripheral vessels
- No (clinical or histological) disturbance of retinal function has been associated with these changes
- Most frequently seen in association with DM in patients with acidosis and ketosis.
- Triglycerides > 10 mmol/L confer a risk of acute pancreatitis. Consider familial causes, diabetes (especially poorly controlled), high fat diet, obesity, excess alcohol, liver and kidney diseases.

https://www.healio.com/news/optometry/20120225/milky-white-retinal-vessels-seen-during-routine-exam

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Case 3 – Differentials and treatment

- Vessel sheathing (eg RVO or RAO) – wouldn't affect all the vessels
- Vasculitis – would be symptomatic (do visual fields), inflammatory markers would be elevated. Could order an FFA but risks.
- When statins just don't cut it:
- Olbetam (acipimox) is a niacin derivative used as a lipid-lowering agent. It reduces triglyceride levels and increases HDL cholesterol.

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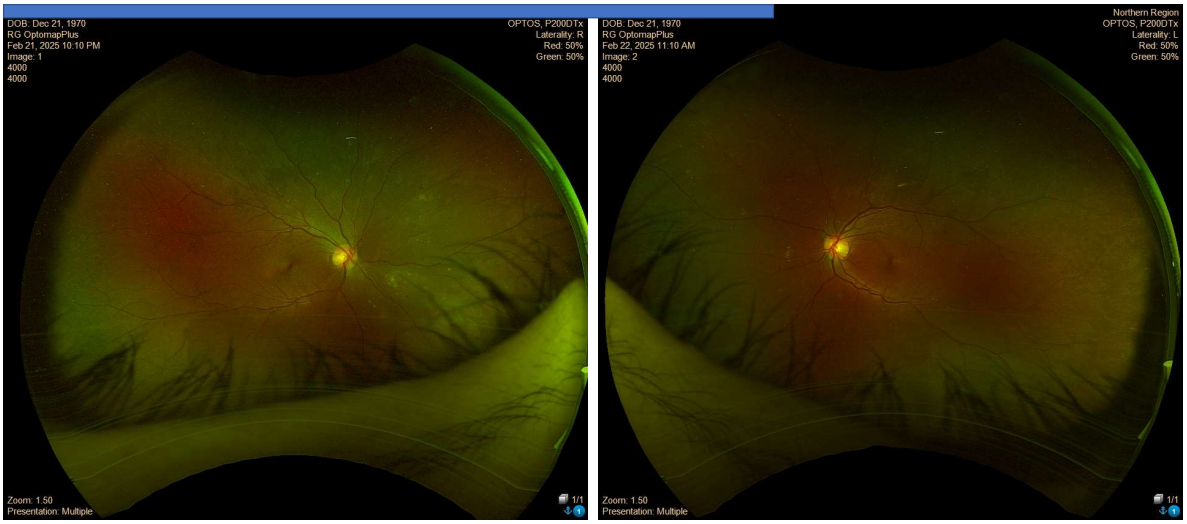
22.2.25 Back on Olbetam x 4/12



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Case 3 - outcome

It was nice to see Mary in Clinic today. We were keeping an eye on her diabetic retinopathy in the Virtual Medical Retinal Imaging Clinic. When I was reading her photographs in October of last year, I noted that she had lipaemia retinalis in both eyes. Reviewing her blood tests from that time showed that her triglyceride levels were 39 (normal < 2). She was previously on Olbetan, but she had stopped taking this, hence the increased elevated triglyceride profile.

She has been back on it for some time now, and I am pleased to say that the blood vessels have returned to a normal colour. She shows moderate diabetic retinopathy in both eyes, but there is no macular oedema in either eye. Her vision with he glasses is right eye 6/5- and left 6/6+. The intraocular pressures are excellent at right eye 14 mmHg and left eye 15 mmHg.

As the lipemia retinalis has settled, we will return her back to the Imaging Clinic, to be seen in six months' time.

Just as aside, I note that Mary has run out of metformin. She has an appointment with you, and the earliest she can get there is in two weeks' time. I have given her a covering prescription for this in the interim.

Kind regards

Mr Richard Johnson
Optometrist
Greenlane Clinical Centre
09 307 4949

This document is dictated and approved by the author

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Case 4 - Background

- Bilateral simultaneous CRVOs diagnosed in EEC / Med Ret input
- I saw **** in clinic today. This was on six-week review of bilateral three (of 3) intravitreal Avastin injections.
- There has been no real response to the injections and his vision has actually dropped further to 6/120 in the right, 6/30 in the left.
- Pressures are right and left 9 mmHg.
- Test?
- BP = 220 / 140
- Transfer to Gen Med stat

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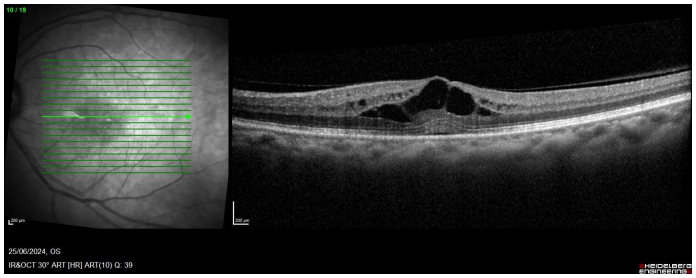
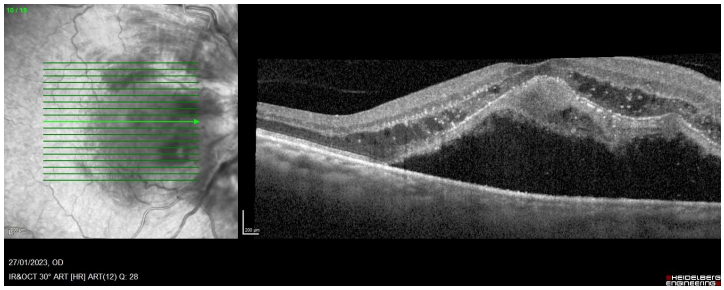
Case 4 – OCTs (note IRF / trace SRF vs SRF / trace IRF)





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Case 4 – Diagnosis (1/12 later)

- He has bilateral malignant hypertensive retinopathy with optic nerve fluid. Given the reduction in vision in the right eye, we will put him through for a right Avastin today with a guarded prognosis for improvement. We will review him here in two months' time.
- The main issue is obviously the hypertension. His blood pressure today on his current medication is 180/86 mmHg but this still needs to be much lower to try and reduce the oedema around the nerves. He is coming to see you to discuss his medications and the possibility of 24-hour BP monitoring.
- Kind regards
- Mr Richard Johnson
- Optometrist
- Greenlane Clinical Centre

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Malignant hypertensive retinopathy

- ***AV Crossing Changes***
- Salus's sign: Deflection of retinal vein as it crosses the arteriole.
- Gunn's sign: Tapering of the retinal vein on either side of the AV crossing.
- Bonnet's sign: Banking of the retinal vein distal to the AV crossing.
- ***Arterial Changes***
- Decrease in the arteriovenous ratio to 1:3 (the normal ratio is 2:3).
- Change in the arteriolar light reflex (light reflex appears as copper and/or silver wiring)
- ***Retinal Changes***
- *Retinal hemorrhages:* Dot-blot hemorrhages: Bleeding in the inner retinal layer
- Flame shaped hemorrhage: Bleeding is in the superficial retinal layer
- *Retinal exudates:* Hard exudates: Lipid deposits in the retina
- Soft exudates: These are also known as cotton wool spots which appear due to ischemia of the nerve fibers
- ***Macular Changes***
- Macular star formation due to deposition of hard exudates around the macula.
- ***Optic Nerve Changes***
- Optic disk swelling (also known as hypertensive optic neuropathy)

Modi P, Arsiwalla T. Hypertensive Retinopathy. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK525980/

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Case 4 - Malignant hypertension (MHT) definition

- Originally defined as diastolic BP > 130 mmHg at the time of the diagnosis and hypertensive retinopathy grades III or IV in the Keith et al.'s classification
- Now MHT has been reconceptualised to emphasis multi-organ damage

Domek, M., Gumprecht, J., Lip, G.Y.H. et al. Malignant hypertension: does this still exist?. J Hum Hypertens 34, 1–4 (2020). https://doi.org/10.1038/s41371-019-0267-y

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Chronic hypertensive retinopathy changes

- ***Keith-Wagner-Barker classification***
- Group 1: Slight constriction of retinal arterioles
- Group 2: Group 1 + focal narrowing of retinal arterioles + AV nicking
- Group 3: Group 2 + flame-shaped haemorrhages + cotton-wool spots + hard exudates
- Group 4: Group 3 + optic disc swelling
- ***Scheie Classification***
- *For Hypertensive Retinopathy*
- Stage 0: No visible abnormalities
- Stage 1: Diffuse arteriolar narrowing
- Stage 2: Stage 1 + focal arteriolar constriction
- Stage 3: Stage 2 + retinal hemorrhage
- Stage 4: Stage 3 + hard exudates + retinal edema+ optic disc swelling
- *For Arteriosclerosis*
- Stage 0: Normal
- Stage 1: Broadening of arteriolar light reflex
- Stage 2: Stage 1 + AV crossing changes
- Stage 3: Copper wiring of arterioles
- Stage 4: Silver wiring of arterioles

Modi P, Arsiwalla T. Hypertensive Retinopathy. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK525980/

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Malignant hypertensive retinopathy

- Chronic hypertensive retinopathy rarely causes significant visual loss.
- The retinal changes can be halted when hypertension is treated.
- However, arteriolar narrowing and AV changes persist.
- For untreated malignant hypertension, the mortality is high as 50% within 2 months of diagnosis and almost 90% by the end of 1 year.
- Vision loss in hypertensive retinopathy is because of either secondary optic atrophy after prolonged papilloedema or retinal pigmentary changes after exudative retinal detachment…
- …or that the patient is no longer alive

Modi P, Arsiwalla T. Hypertensive Retinopathy. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK525980/

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MHT (retinopathy) take home messages

- If your patient has:
- Bilateral SRF around each optic nerve or,
- “a bilateral simultaneous CRVO” then:
- Measure the BP (yourself, because then you know it’s done)
- For untreated malignant hypertension, the mortality is high as 50% within 2/12 of diagnosis and almost 90% by 12/12.
- i.e time is crucial…

Keith NM, Wagener HP, Barker NW. Some different types of essential hypertension: their course and prognosis. Am J Med Sci. 1974 Dec;268(6):336-45

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Case 5 a. (High street) optometry referral

URGENT referral for possible RE vitreous haemorrhage/proliferative diabetic retinopathy. I have already spoken with the on-call ophthalmologist.

Nick came in today due to very blurred RE vision for the past 3 days, it started with seeing a ‘twig’ like line in the R corner. Nick is diabetic and he does mention his blood sugars have been quite high the past few days. There is no pain or redness. The LE is unaffected. He is also on medication for heart and hypertension. Nick has had bilateral cataract removal but has no other ocular history.

Aided vision was:
R 6/15 (No improvement with pinhole)
L 6/6

On dilated fundus examination, the vitreous appears very hazy in general in the RE but especially worse in the temporal, inferior and inferio nasal sides, could not get much of a view there. The ONH and macula appears unremarkable though. There were some dot and blot haemorrhages in the nasal and superior peripheries. The LE was unremarkable.

IOP was R 13mmHg L 14mmHG (NCT@1100)

Anterior segment was unremarkable. IOL was clear BE.

I am concerned the hazy vitreous and blurred vision is due to proliferative diabetic retinopathy and thus would appreciate if Nick is seen under your expert care.

Thank you.

Kind regards,

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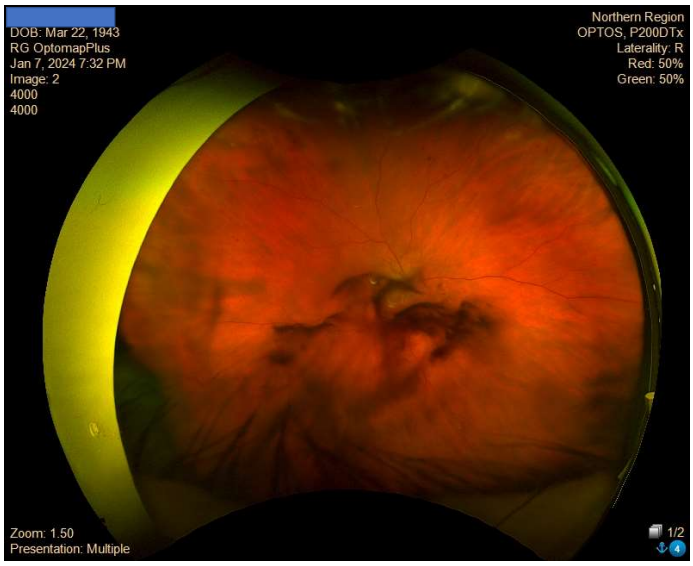
Case 5a. RE fundus 7/1/24



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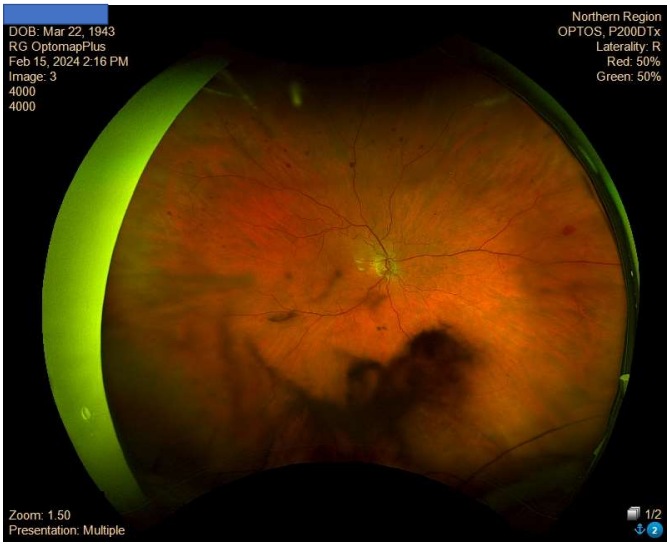
Case 5a. 15/2/24



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Case 5a. Investigations

Date of exam: 27/02/2024
Examination(s) included in this report:
US DD Carotid Bilateral

INDICATION: Ocular ischemic syndrome in the right eye.

COMPARISON WITH PREVIOUS IMAGING: Carotid US on 28/6/23 reported a densely calcified right ICA occlusion.

FINDINGS:
RIGHT:
Plaque: Dense calcification in the ICA origin which causes complete acoustic shadowing preventing visualisation of flow. No flow detected distally - occlusion suspected as previously reported on 28/6/23 US scan.
CCA systolic: 25 cm/sec
CCA diastolic: 4 cm/sec
ICA systolic: Likely occluded
ICA diastolic: Likely occluded
ICA/CCA Ratio: -
Grade: Occlusion suspected
ECA: 56 cm/s
Vertebral: antegrade

LEFT:
Plaque: Diffuse calcified atheroma throughout the CCA and ICA, not causing a significant stenosis.
CCA systolic: 47 cm/sec
CCA diastolic: 14 cm/sec
ICA systolic: 69 cm/sec
ICA diastolic: 24 cm/sec
ICA/CCA Ratio: 1.6
Grade: <50%
ECA: 122 cm/s
Vertebral: antegrade

SUMMARY:
As previously reported on the 28/6/23 carotid US, densely calcified right ICA with no flow seen throughout, occlusion suspected.
Previous vascular surgeon opinion sought and noted to be for best medical therapy only (29/06/2023).
No significant stenosis in the left ICA.

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Case 5a. 4/3/24 Post PRP

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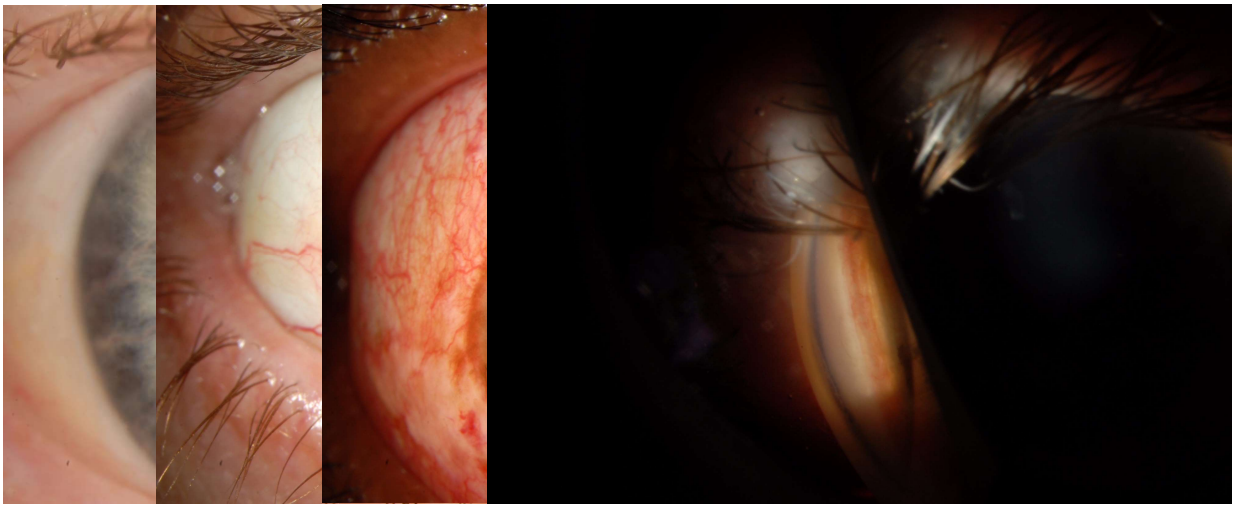
Case 5b. Glaucoma referral – bilateral ↑ IOPs



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Case 5b. Investigations

To: Dr Richard JOHNSON
OPHTHALMOLOGY DEPT
GREENLANE
-
-

[redacted]
DOB: 01/08/1940

Patient Referred from: GCCOPHTHAL
Exam performed at: North Shore Hospital Radiology
Reference: R240214269
Date of exam: 14/02/2024
Examination(s) included in this report:
US DD Carotid Bilateral

**** ABNORMAL REPORT - DOCTOR TO SIGHT - ACTION REQUIRED ****

INDICATION:
Bilateral iris neovascularisation, please rule out carotid stenosis as aetiology. Bilateral iris neovascularisation, please rule out carotid stenosis as aetiology.

FINDINGS:
There is a mild amount of soft plaque in the mid CCAs bilaterally. There is soft plaque in extracranial internal carotid arteries bilaterally which are totally occluded. There is elevated peak systolic velocity of 195 cm/s in the left ECA. There is antegrade flow in both vertebral arteries.

INTERPRETATION:
Total occlusion of the Internal Carotid Arteries bilaterally.

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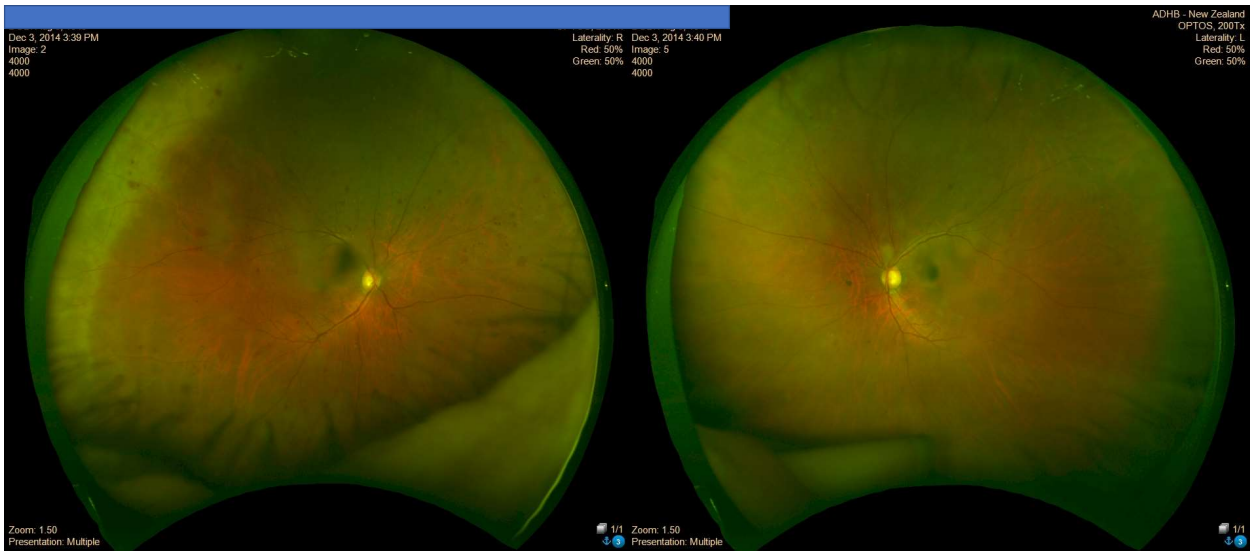
Case 5c. Asymmetrical diabetic retinopathy



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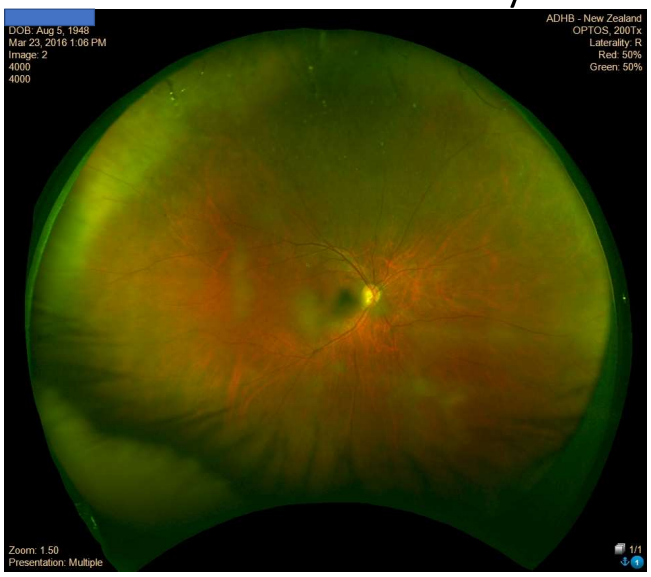
Case 5c. Post R endarterectomy



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Case 5 – Suspect Ocular Ischaemia Syndrome in:

- All new cases of glaucoma esp IOP asymmetry – look for NVI & NVA
- Diabetic retinopathy – suspect OIS if > 2 levels of severity between the gradings of the 2 eyes
- Cases of (non-diabetic) vitreous haemorrhage in the elderly
- The haemorrhage may take time to clear to be able to view possible causes (OIS vs tear)
- Must have PRP if not having an endarterectomy else may develop new vessels (NVD, NVE, NVI, NVA) due to global retinal ischaemia

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Case 6 - background

- 21 year old male referred with bilateral gradual reduced vision
- ? Media opacity / irregularity → perhaps mild cataract but or keratoconus
- General health good but reduced hearing
- VA unaided RE 6/12 (PH 6/7.5-) LE 6/12 (PH 6/7.5-)
- IOPs RE 12 mmHg LE 12 mmHg
- Tests?
- Scissoring with retinoscopy
- Corneal topography normal BE

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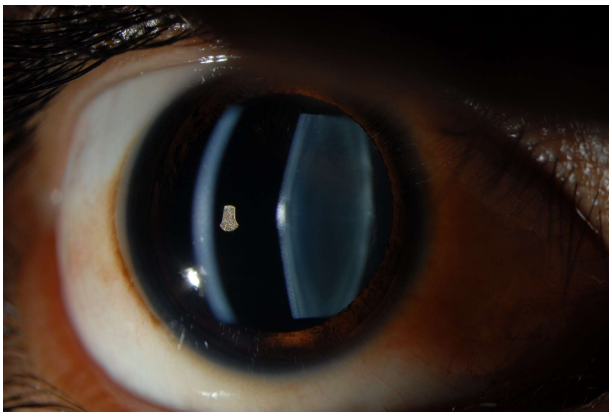
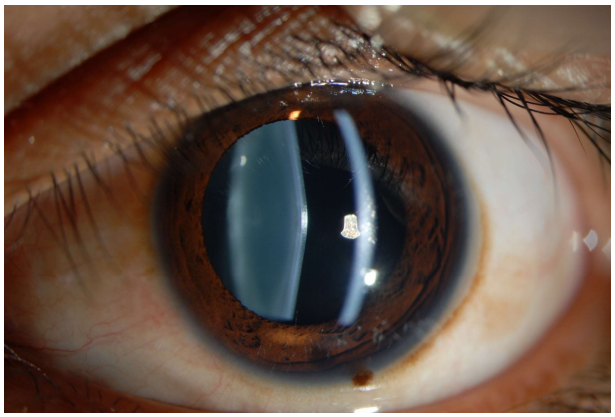
Case 6 – anterior segments





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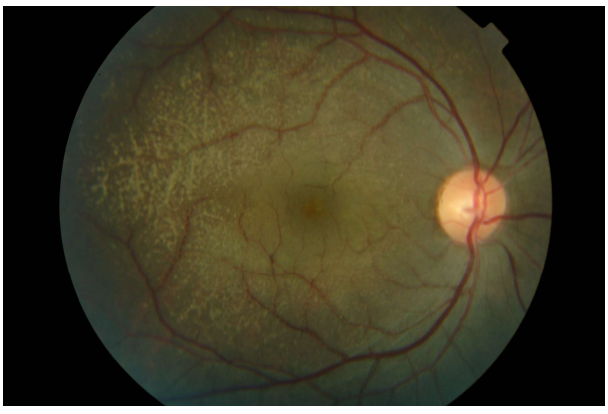
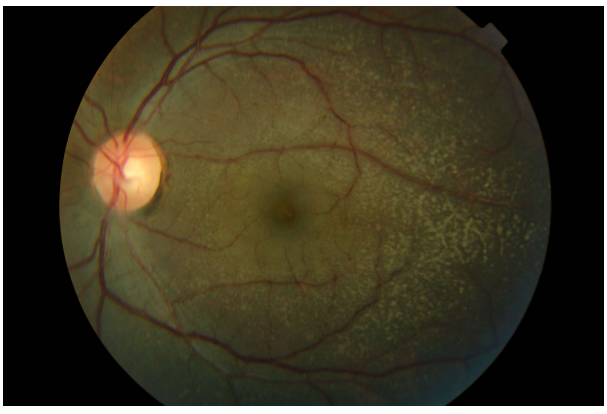
Case 6 - Fundi





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Alport syndrome (AS)

- Alport syndrome is a rare, inherited disorder characterized by kidney disease, hearing loss, and eye abnormalities (usually anterior lenticonus)
- Caused by mutations in genes responsible for type IV collagen (a key protein in the kidney's filtration system)
- Inheritance: X-Linked AS, Autosomal Recessive AS, ADAS
- Males who have X-Linked AS and anyone with ARAS often develop kidney failure and hearing loss before 30.
- Females who have XLAS usually have an average lifespan

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Alport syndrome take home messages

- Check for lenticonus (not just keratoconus) in media opacities / distortion, especially in young males
- If you see lenticonus, check for flecked retina
- Ask about hearing
- Ask about kidney function (hypertension, haematuria, proteinuria, leg oedema, fatigue)
- Do eGFR and creatinine
- Refer to nephrologist

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Others we could do…

- Ocular colobomata
- Marfan's syndrome
- Wilson's disease
- NF-1

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Thank you

- See you tomorrow

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