Cas cliniques

UNIVERSITÉ PARIS DESCARTES

ASSISTANCE PUBLIQUE HÔPITAUX DE PARIS

HÔPITAUX UNIVERSITAIRES PARIS CENTRE

Antoine Brézin

♂☐ 16 ans
Pas d’antécédent médical
Né à terme de parents non consanguins
Pas d’antécédent familial ophtalmologique notable

Symptômes visuels depuis 3 semaines
Sensation d’amputation du champ visuel périphérique, du côté gauche, puis bilatérale.
Puis, baisse d’acuité visuelle bilatérale - 5/10^{e} ODG le 29 OCT 2019

Analyse de champ unique

Oeil: Gauche

Nom: [redacted] ID: DDN: 31-10-2003

Test de seuil central 30-2

Contrôle de fixation: ARRET
Cible de fixation: Central
Pertes de fixation: 0/0
Erreurs faux pos.: 0 %
Erreurs faux nég.: Sans objet
Durée du test: 05:14

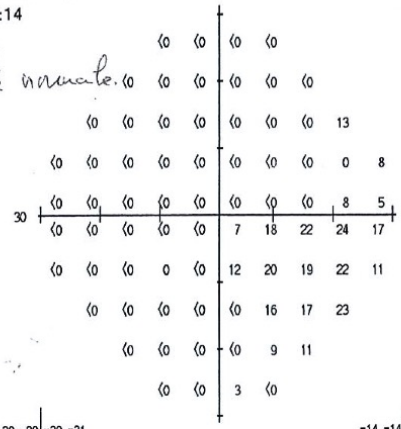
Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: SITA-Fast

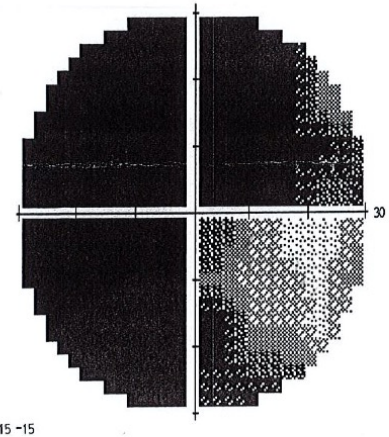
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

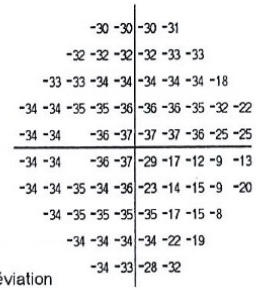
Date: 21-10-2019
Heure: 07:53
Age: 15

Fovea: 29 dB

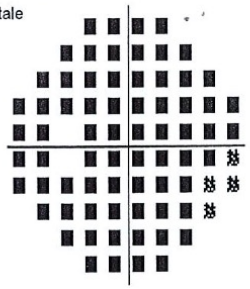
il y a la normale.

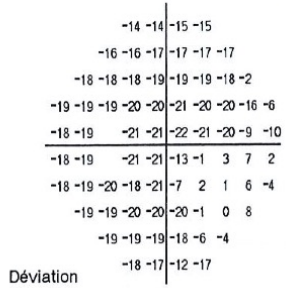




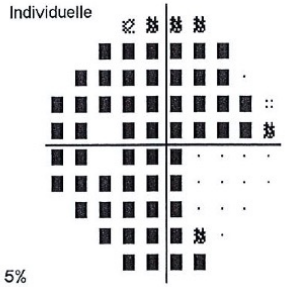


Déviation Totale





Déviation Individuelle



THG
Hors limites normales

MD -29.52 dB P < 0.5%
PSD 9.60 dB P < 0.5%

:: < 5%
⁘ < 2%
⁙ < 1%
■ < 0.5%

CABINET D ORTHOPTIE YAZALI
AV ALLAL EL FASSI IMM ALLOUMAIRI
TEL 044-31-16-49

Analyse de champ unique

Oeil: Droit

Nom: [redacted] ID: DDN: 31-10-2003

Test de seuil central 30-2

Contrôle de fixation: ARRET
Cible de fixation: Central
Pertes de fixation: 0/0
Erreurs faux pos.: 0 %
Erreurs faux nég.: Sans objet
Durée du test: 05:00

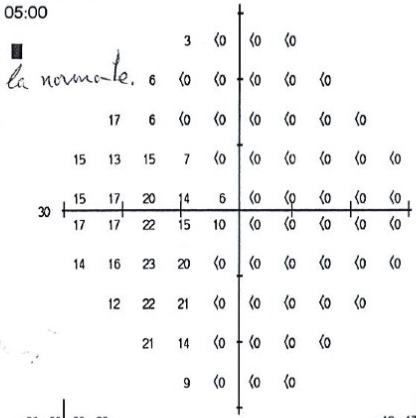
Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: SITA-Fast

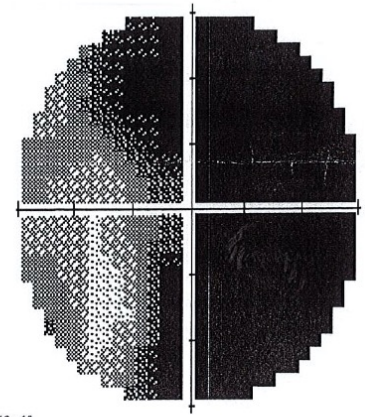
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

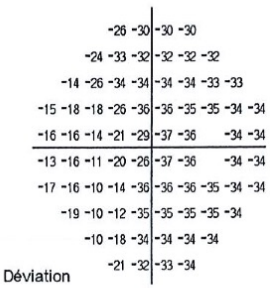
Date: 21-10-2019
Heure: 08:02
Age: 15

Fovea: 28 dB

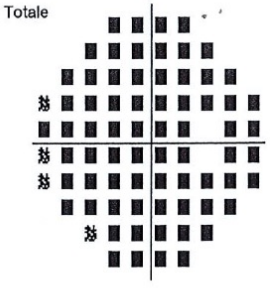
il y a la normale.

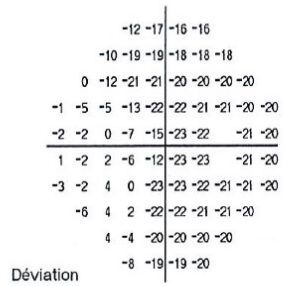




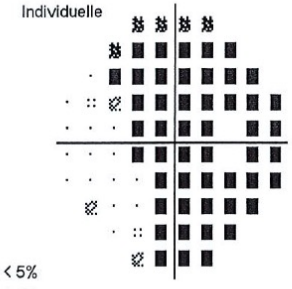


Déviation Totale





Déviation Individuelle

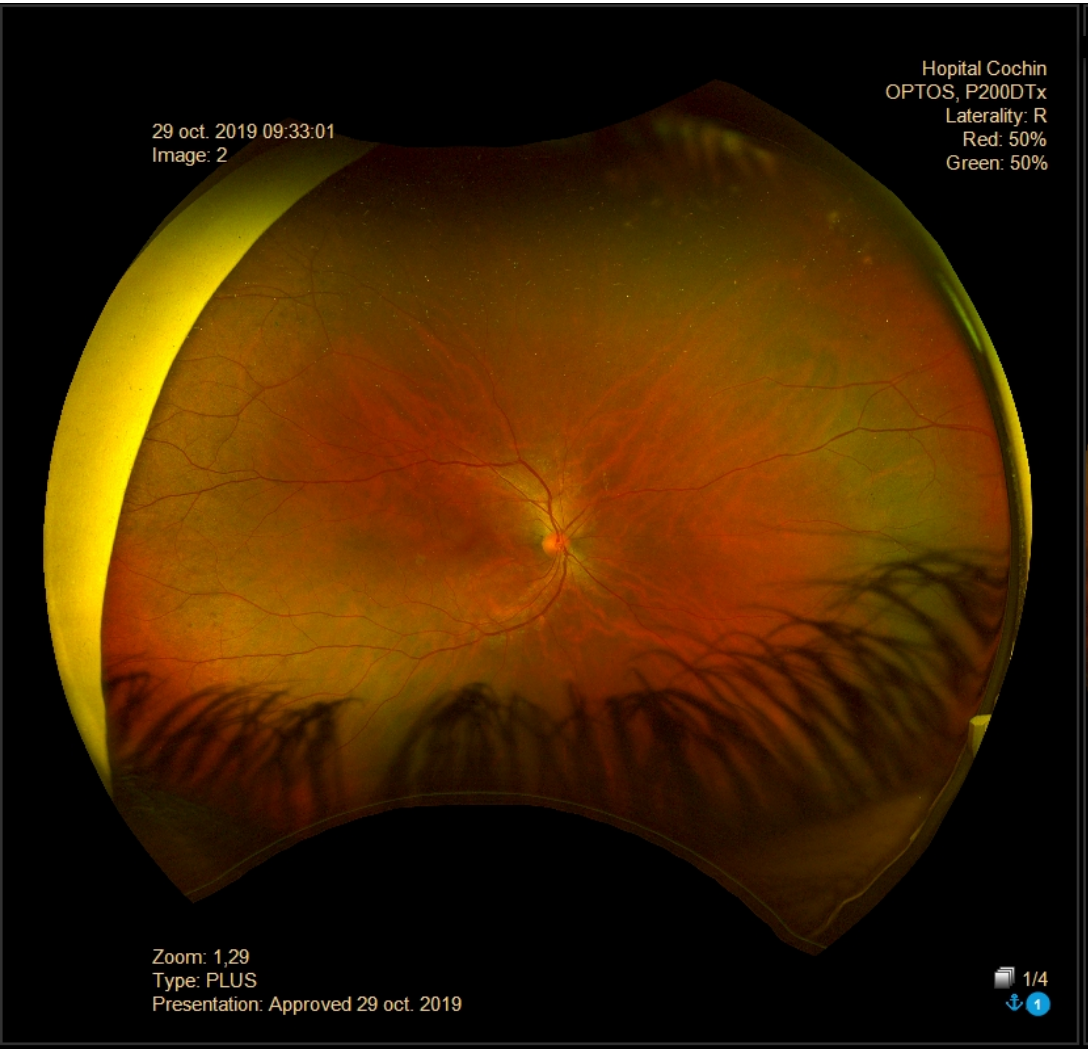


THG
Hors limites normales

MD -27.34 dB P < 0.5%
PSD 10.36 dB P < 0.5%

:: < 5%
⁘ < 2%
⁙ < 1%
■ < 0.5%

CABINET D ORTHOPTIE YAZALI
AV ALLAL EL FASSI IMM ALLOUMAIRI
TEL 044-31-16-49


29 oct. 2019 09:33:01
Image: 2
Hopital Cochin
OPTOS, P200DTx
Laterality: R
Red: 50%
Green: 50%
Zoom: 1,29
Type: PLUS
Presentation: Approved 29 oct. 2019
1/4
1


29 oct. 2019 09:33:43
Image: 6
Hopital Cochin
OPTOS, P200DTx
Laterality: L
Red: 50%
Green: 50%
Zoom: 1,42
Type: PLUS
Presentation: Approved 29 oct. 2019
1/3
1

Examens déjà pratiqués avant l'arrivée à Cochin :

IRM cérébrale : normale.

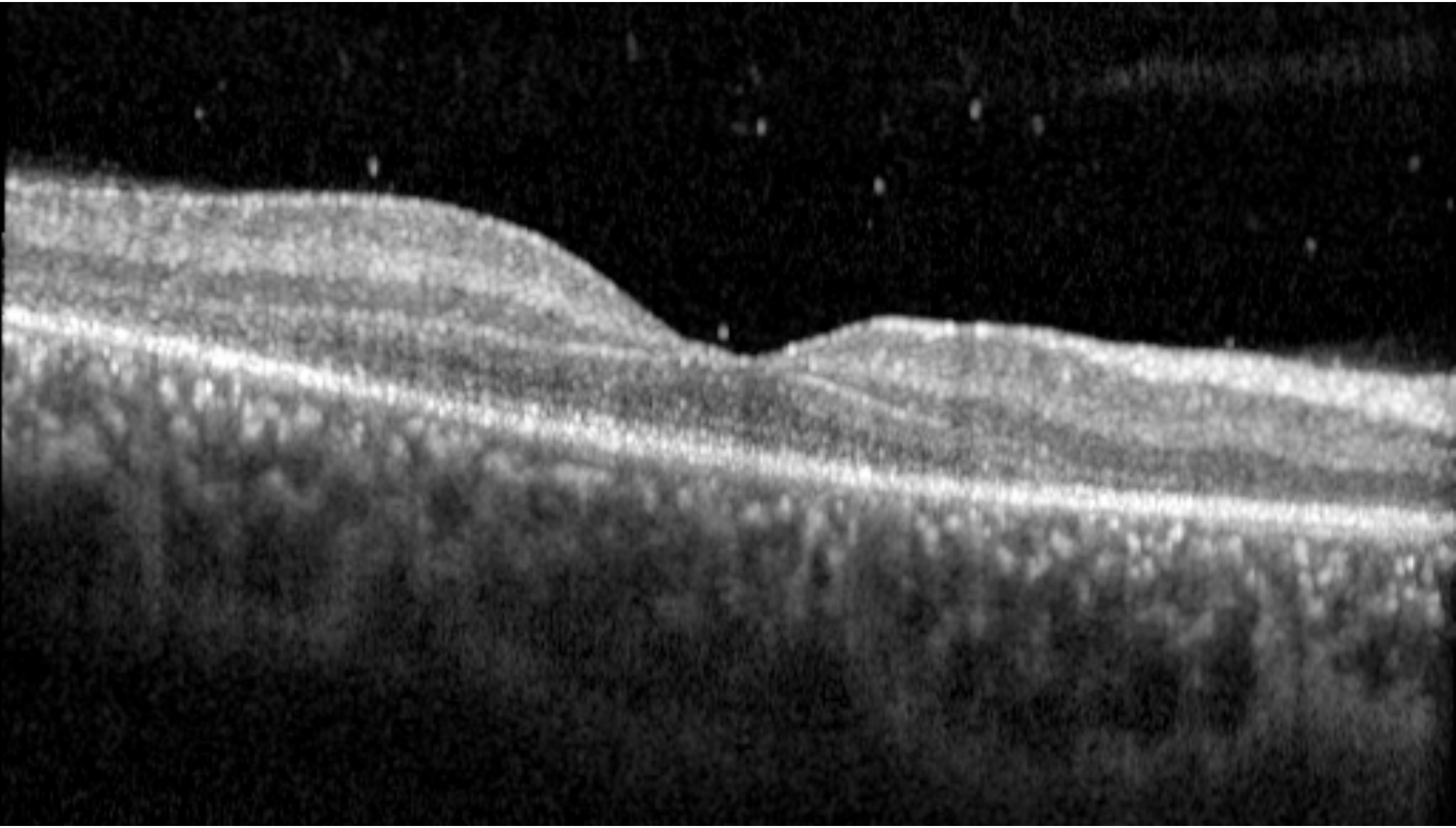
VS 5 mm à H1, CRP 1 mg/l.

NFS et créatininémie normales.

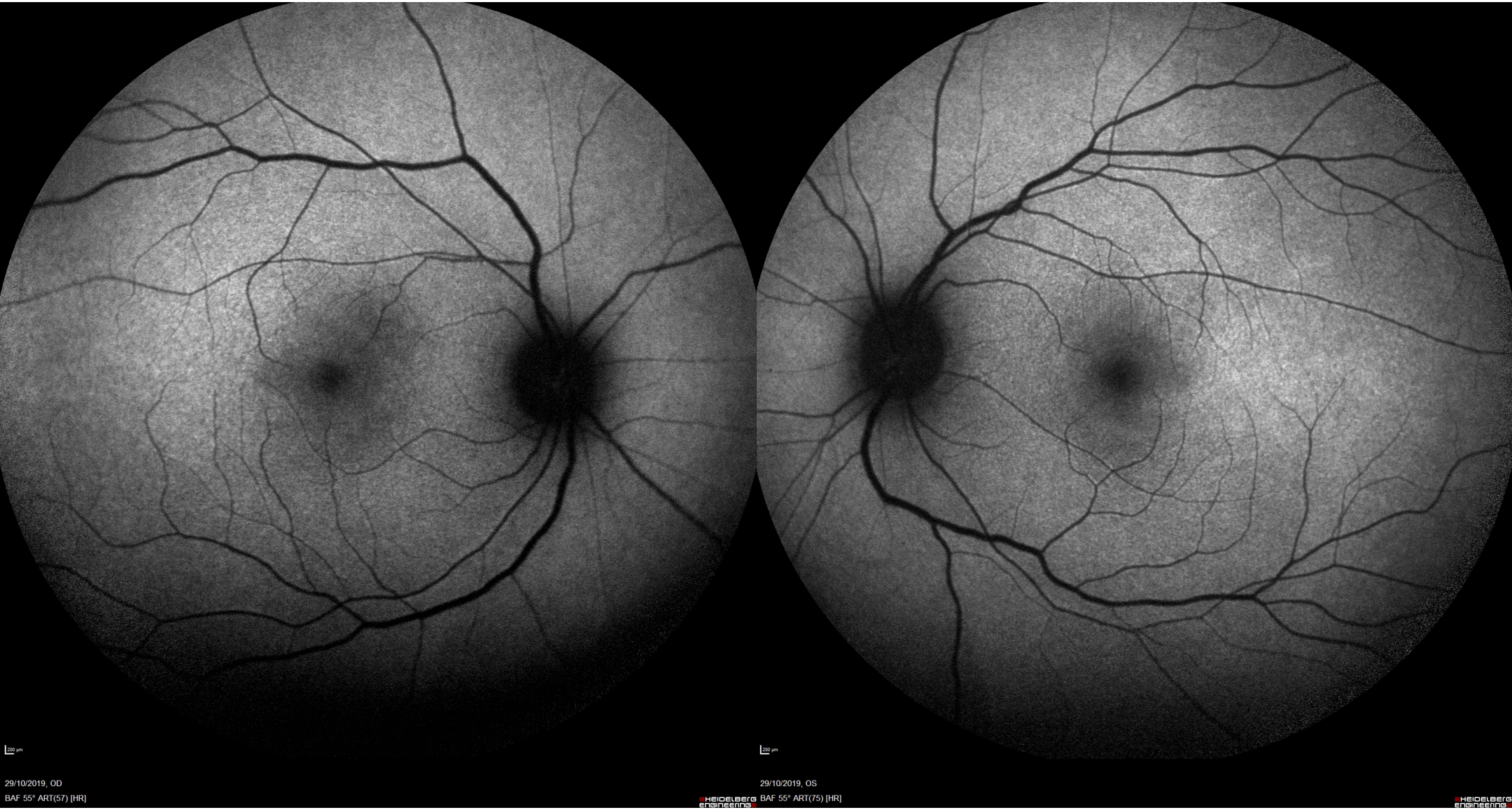
Traitement déjà effectué :

2 bolus IV de 500 mg corticoïdes, sans relais → Impression subjective de pause dans la dégradation visuelle

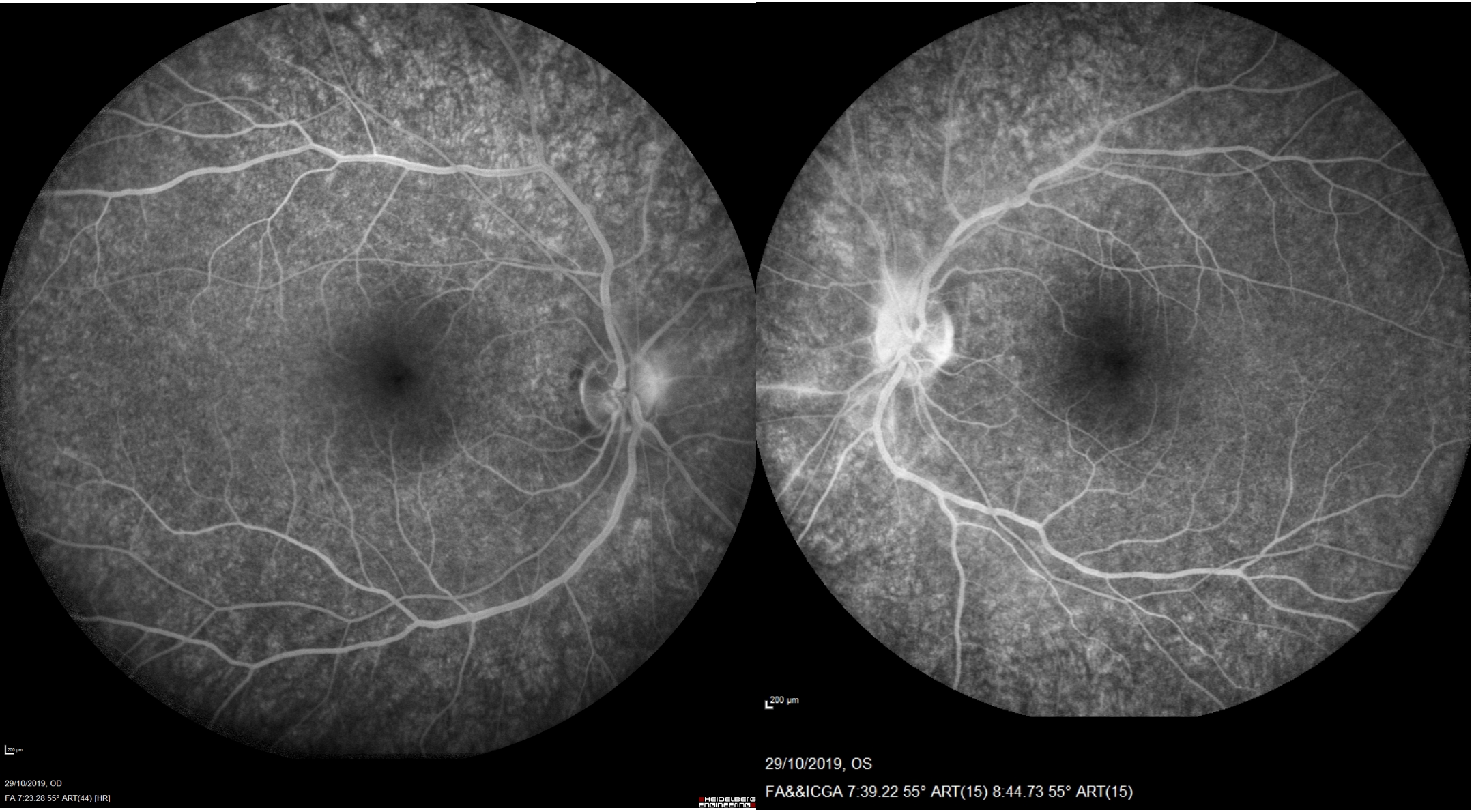
OCT le 29/10/19



Autofluorescence le 29/10/19



Angiographie fluorescéinique le 29/10/19



Quels autres examens complémentaires ?

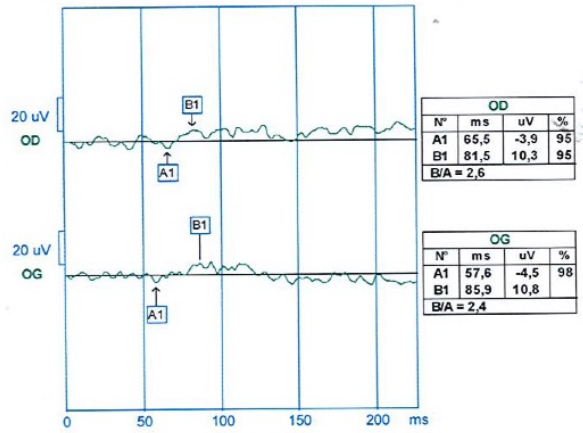
nom :
dossier : 8013812151
date naissa. : 31/10/2003

correction :
date examen : 30/10/2019
examen :

EXAMEN D'ELECTROPHYSIOLOGIE VISUELLE

ERG des bâtonnets (-25dB) 10mn 36s Val= 8 Rej= 0

Bl stimulé

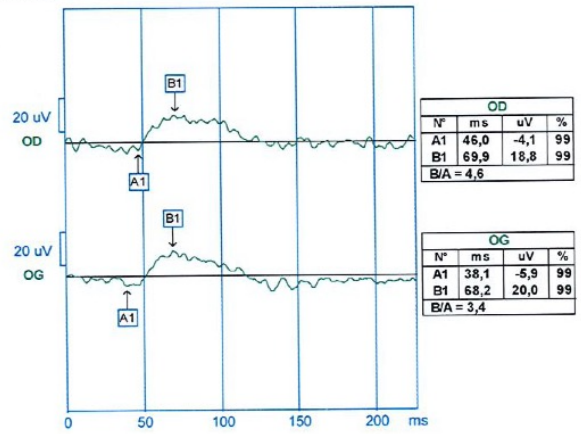


OD			
N°	ms	uV	%
A1	65,5	-3,9	95
B1	81,5	10,3	95
B/A = 2,6			

OG			
N°	ms	uV	%
A1	57,6	-4,5	98
B1	85,9	10,8	
B/A = 2,4			

ERG mixte: (-15dB) 11mn 47s Val= 8 Rej= 0

Bl stimulé

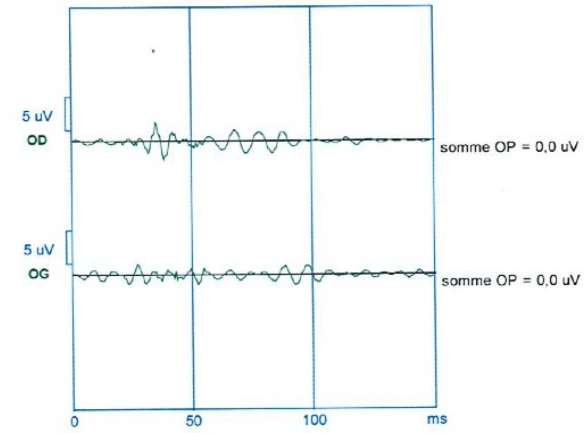


OD			
N°	ms	uV	%
A1	46,0	-4,1	99
B1	69,9	18,8	99
B/A = 4,6			

OG			
N°	ms	uV	%
A1	38,1	-5,9	99
B1	68,2	20,0	99
B/A = 3,4			

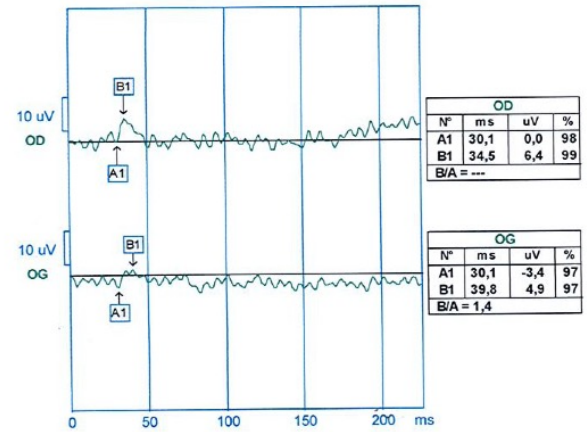
ERG potentiels oscillatoires 13mn 12s Val= 8 Rej= 0

Bl stimulé



ERG des cônes: flash blanc 20mn 24s Val= 17 Rej= 0

Bl stimulé

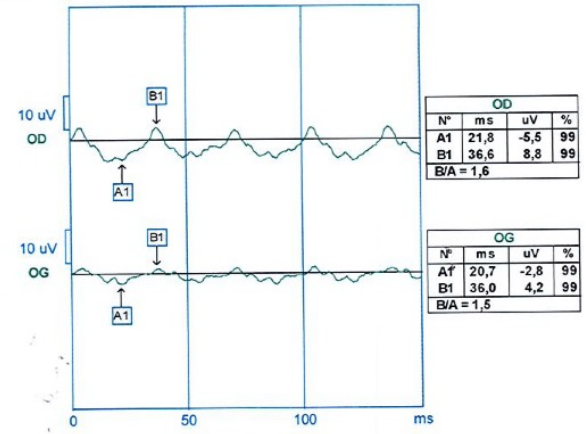


OD			
N°	ms	uV	%
A1	30,1	0,0	98
B1	34,5	6,4	99
B/A = ---			

OG			
N°	ms	uV	%
A1	30,1	-3,4	97
B1	39,8	4,9	97
B/A = 1,4			

ERG des cônes: flicker 30Hz 20mn 51s Val= 30 Rej= 0

Bl stimulé



OD			
N°	ms	uV	%
A1	21,8	-5,5	99
B1	36,6	8,8	99
B/A = 1,6			

OG			
N°	ms	uV	%
A1	20,7	-2,8	99
B1	36,0	4,2	99
B/A = 1,5			

CENTRE COCHIN AMBULATOIRE D'OPHTALMOLOGIE
27, rue du Fg Saint Jacques
75679 Paris cedex
Chef de service : Pr Antoine BREZIN
Responsable d'unité : Dr Dominique MONNET

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Groupe Hospitalier Cochin - Saint Vincent de Paul
Service d'Ophtalmologie
Pr Antoine BRÉZIN
Hôpital Cochin - 27 rue du Faubourg Saint Jacques - 75679 PARIS Cedex 14

(EC) ERG global (sc / fino)
Protocole ISCEV.
pupilles dilatées.
NB: Coopération OC pdt les examens

Moniteur Ophtalmologique
WIN8000F
Metrovision
4 rue des platanes
59840 Pérenchies
France
tel 33 (0)3 20 17 19 50
http://www.metrovision.fr

METROVISION

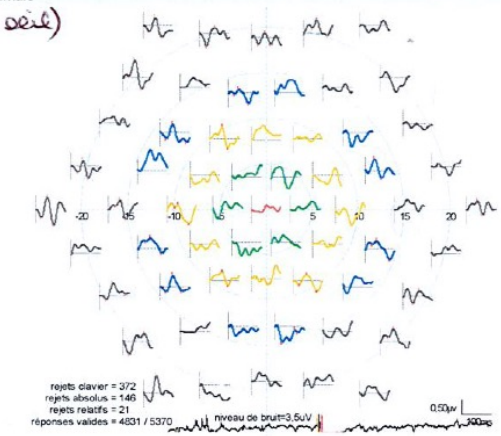
nom :
dossier : 8013812151
date naissa. : 31/10/2003

correction : +2.75
date examen : 30/10/2019
examen :

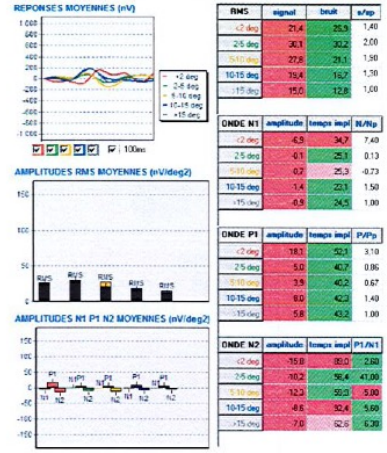
EXAMEN D'ELECTROPHYSIOLOGIE MULTIFOCALE

MERG61B — CARTE DES REPONSES LOCALES
OD stimulé

(1er oeil)



MERG61B — ANALYSE PAR ZONES (ANNEAUX)
OD stimulé



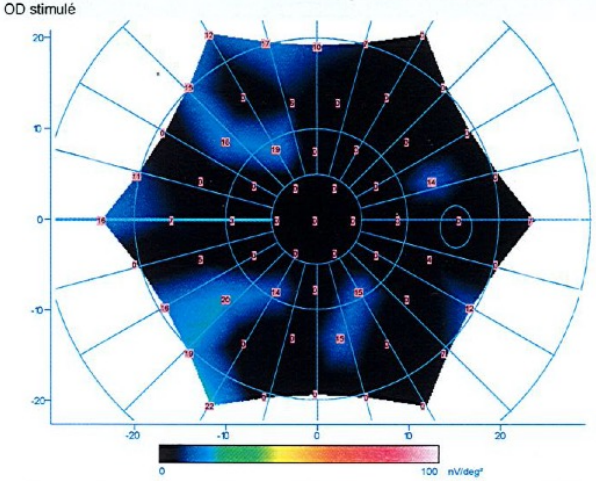
RMS	signal	bruit	s/sp
<2 deg	21,4	25,5	1,40
2-5 deg	30,1	30,2	2,00
5-10 deg	27,8	28,1	1,90
10-15 deg	19,4	18,7	1,30
>15 deg	15,0	12,8	1,00

ONDE N1	amplitude	temps impl	N/Np
<2 deg	-6,9	34,7	7,40
2-5 deg	-0,1	28,1	0,13
5-10 deg	0,2	25,3	-0,73
10-15 deg	1,4	23,1	1,50
>15 deg	-0,9	24,5	1,00

ONDE P1	amplitude	temps impl	P/Np
<2 deg	18,1	52,1	3,10
2-5 deg	5,0	40,7	0,86
5-10 deg	3,9	40,2	0,67
10-15 deg	8,0	42,2	1,40
>15 deg	5,8	43,2	1,00

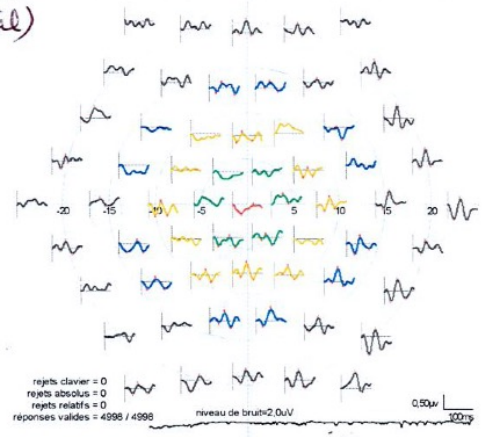
ONDE N2	amplitude	temps impl	P1/N1
<2 deg	-15,8	89,0	2,60
2-5 deg	-10,2	58,4	41,00
5-10 deg	-12,3	59,8	5,00
10-15 deg	-8,6	52,4	5,60
>15 deg	-7,0	52,6	6,20

MERG61B — CARTE AMPLITUDES ONDE P1
OD stimulé

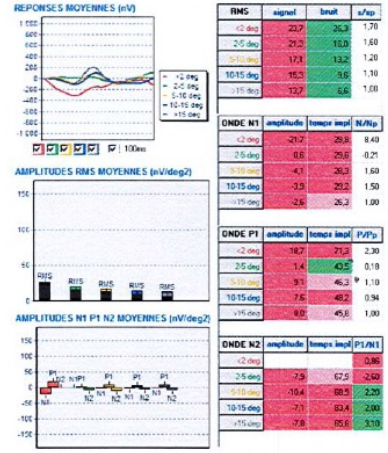


MERG61B — CARTE DES REPONSES LOCALES
OG stimulé

(2e oeil)



MERG61B — ANALYSE PAR ZONES (ANNEAUX)
OG stimulé



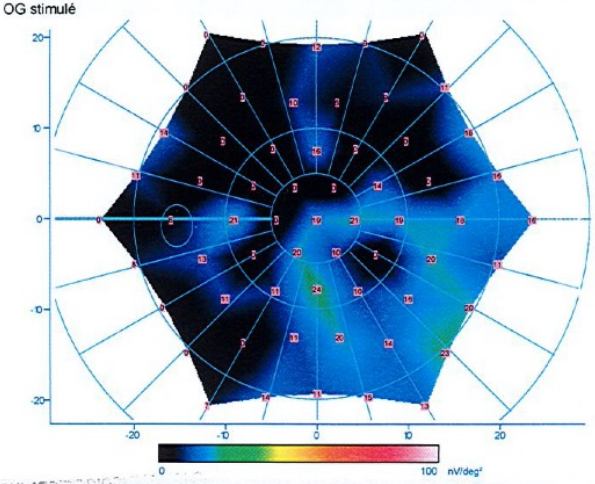
RMS	signal	bruit	s/sp
<2 deg	23,7	26,3	1,70
2-5 deg	21,2	18,0	1,60
5-10 deg	17,1	13,2	1,20
10-15 deg	15,5	9,6	1,10
>15 deg	12,7	6,6	1,00

ONDE N1	amplitude	temps impl	N/Np
<2 deg	-21,2	29,8	8,40
2-5 deg	0,6	29,6	-0,21
5-10 deg	-4,1	28,3	1,60
10-15 deg	-3,9	29,2	1,50
>15 deg	-2,6	26,2	1,00

ONDE P1	amplitude	temps impl	P/Np
<2 deg	18,7	71,3	2,30
2-5 deg	1,4	40,5	0,18
5-10 deg	9,1	46,3	1,10
10-15 deg	7,6	48,2	0,94
>15 deg	8,0	45,6	1,00

ONDE N2	amplitude	temps impl	P1/N1
<2 deg			0,86
2-5 deg	-7,9	67,5	-2,60
5-10 deg	-10,4	68,5	2,20
10-15 deg	-7,1	63,4	2,00
>15 deg	-7,8	65,6	3,10

MERG61B — CARTE AMPLITUDES ONDE P1
OG stimulé



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Groupe Hospitalier Cochin - Saint Vincent de Paul
Service d'Ophtalmologie
Pr Antoine BRÉZIN
Hôpital Cochin - 27 rue du Faubourg Saint Jacques - 75679 PARIS Cedex 14

(EC) ERG flash OD/OG
pupilles dilatées
coopération OD / fixation parfaite.
Lentilles : ERG jet utilisées OD/OG.

CENTRE COCHIN AMBULATOIRE D'OPH...
27, rue du Fg Saint Jacques
75679 Paris cedex ...
Chef de service : Pr Antoine ...
Responsable d'unité : Pr Dominique ...

Quelles hypothèses diagnostiques ?

Autoimmune Retinopathy

LANDON GRANGE, MONICA DALAL, ROBERT B. NUSSENBLATT, AND H. NIDA SEN

• PURPOSE: To provide a detailed review of current clinical guidelines for the diagnosis, work-up and treatment of autoimmune retinopathy and to preview briefly possible future therapies.

• DESIGN: Perspective based on literature review and clinical expertise.

• METHODS: Interpretation of current literature, relying on the authors' clinical experience.

• RESULTS: Autoimmune retinopathy is a rare immunologic disease characterized by the presence of circulating antiretinal antibodies along with electroretinographic and visual field abnormalities. An ophthalmic examination can be normal or show minimal findings. The diagnosis of autoimmune retinopathy is made difficult by diagnostic criteria that are both limited and nonstandardized. Currently, the diagnosis is made based on the demonstration of serum antiretinal antibodies and the presence of clinical manifestations (including abnormal electroretinographic findings). The mere presence of these antibodies is not diagnostic. Lack of an accepted gold standard for antiretinal antibodies detection and poor interlaboratory concordance make the diagnosis challenging. There are anecdotal reports of immunosuppressive therapy in autoimmune retinopathy; however, the response to treatment is variable, with more favorable results achieved in paraneoplastic retinopathy, particularly cancer-associated retinopathy, with a combination of chemotherapy and immunosuppression. Whether an earlier attempt to treat nonparaneoplastic autoimmune retinopathy would be more beneficial is unknown. Early treatment attempts are limited by lack of sensitive and specific assays and definitive clinical criteria.

• CONCLUSIONS: Little is known about the clinical course, prognosis and treatment of autoimmune retinopathy. Additional studies should examine the specificity and pathogenicity of antiretinal antibodies and screen for biomarkers, and they should be conducted concurrently with studies seeking to identify appropriate treatment. (Am J Ophthalmol 2014;157:266–272. Published by Elsevier Inc.)

Autoimmune retinopathy

Curr Opin Ophthalmol 2013, 24:598 – 605

Didar U. Comlekoglu, Ian A. Thompson, and H. Nida Sen

Purpose of review

Autoimmune retinopathy (AIR) is an immune-mediated disorder characterized by progressive visual loss, abnormal electroretinographic and visual field findings in the presence of circulating anti-retinal antibodies. This review highlights advances made toward understanding the pathophysiology, clinical manifestations, and trends in the management of AIR.

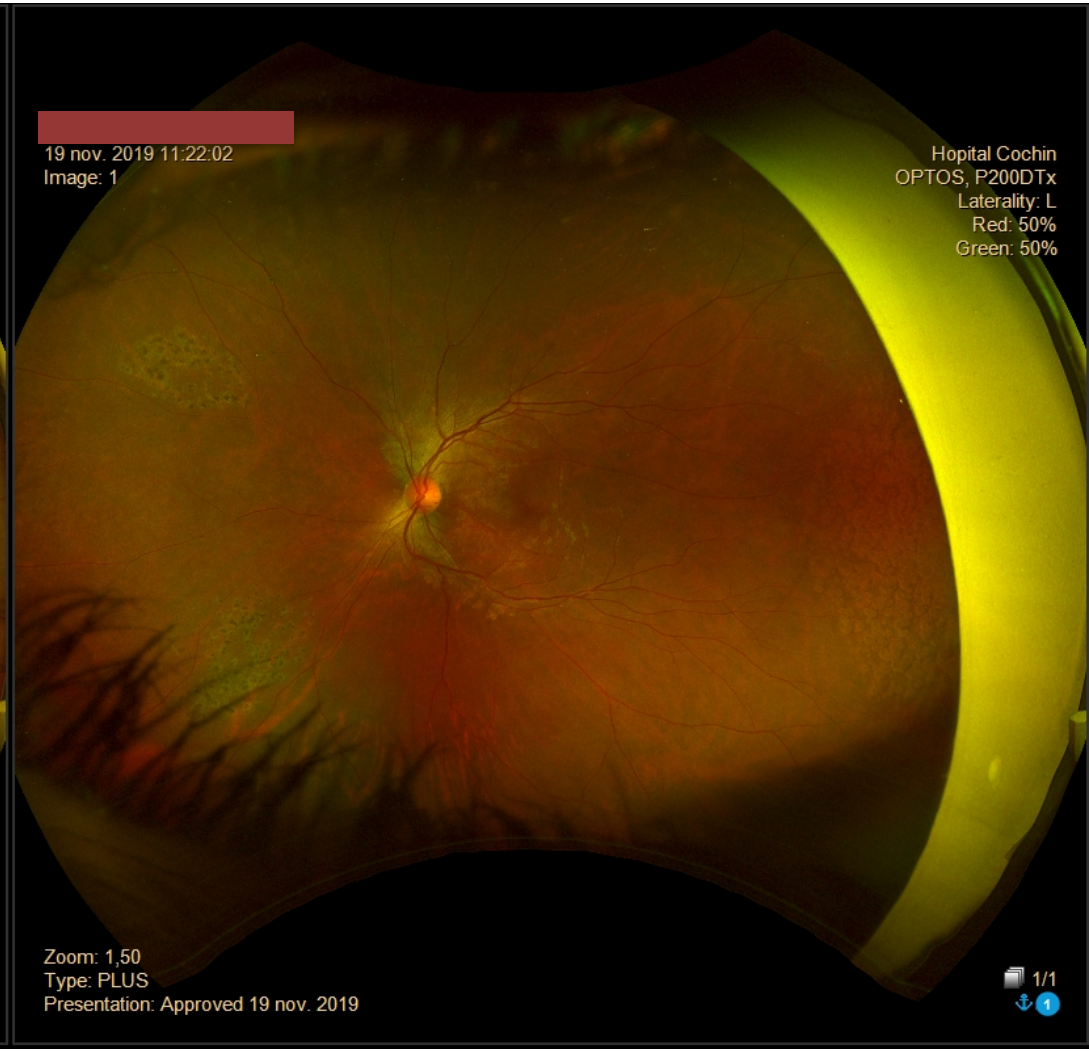
Recent findings

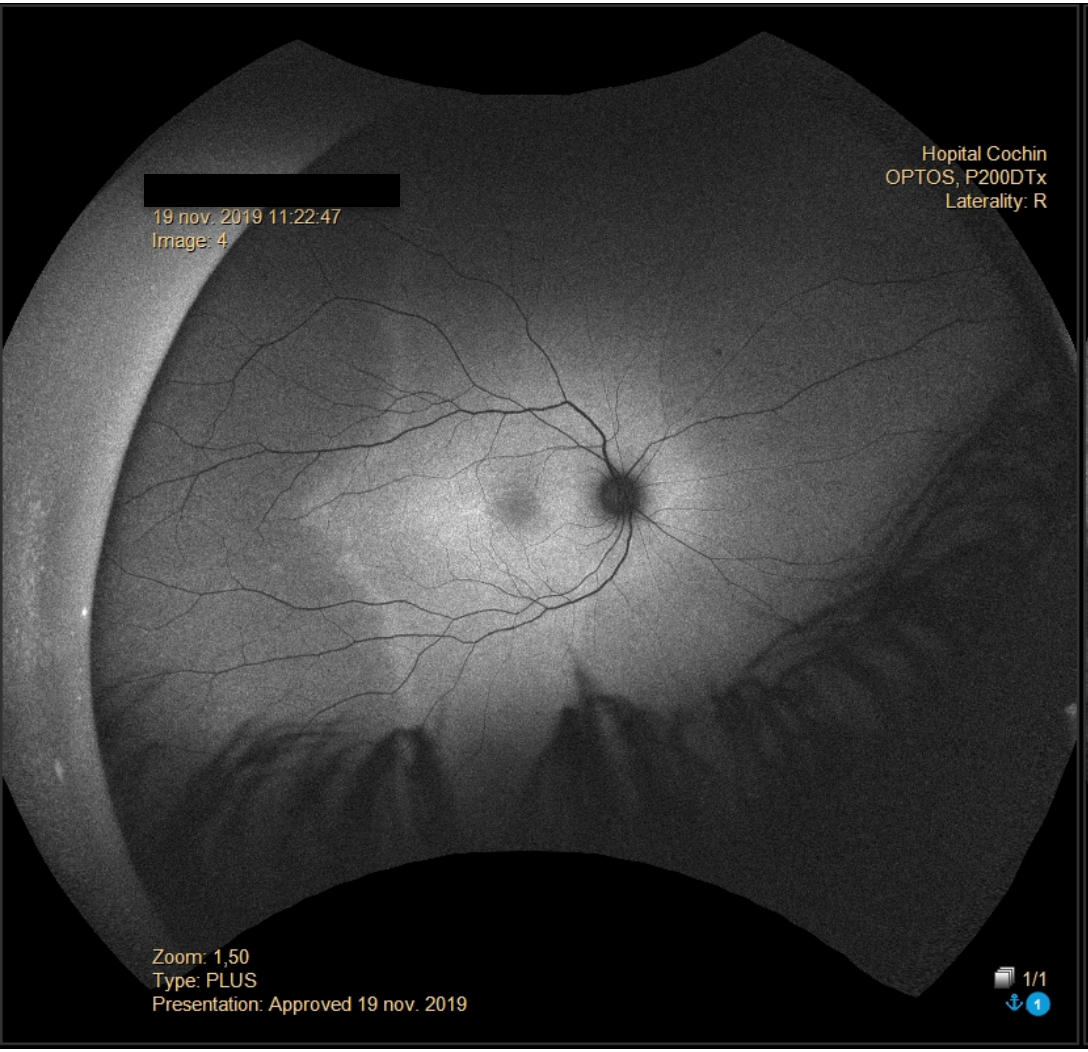
The pathophysiology of AIR is likely antibody-mediated. AIR serum autoantibodies are variable in their size and retinal tissue they target and can also be present in healthy controls and multiple autoimmune diseases. Rarely, AIR may be associated with dysregulated self-tolerance mechanisms in the thymus. Despite progress in research, our understanding of AIR remains incomplete. Lack of standardized methods for anti-retinal antibody testing continues to challenge the interpretation of seropositivity. Conventional immunosuppressives have been further studied, and promising immunomodulatory therapies, such as targeted B-cell therapy, have been introduced. Newer imaging modalities such as fundus autofluorescence and spectral domain optical coherence tomography may be helpful in diagnosis, monitoring progression of disease and response to treatment.

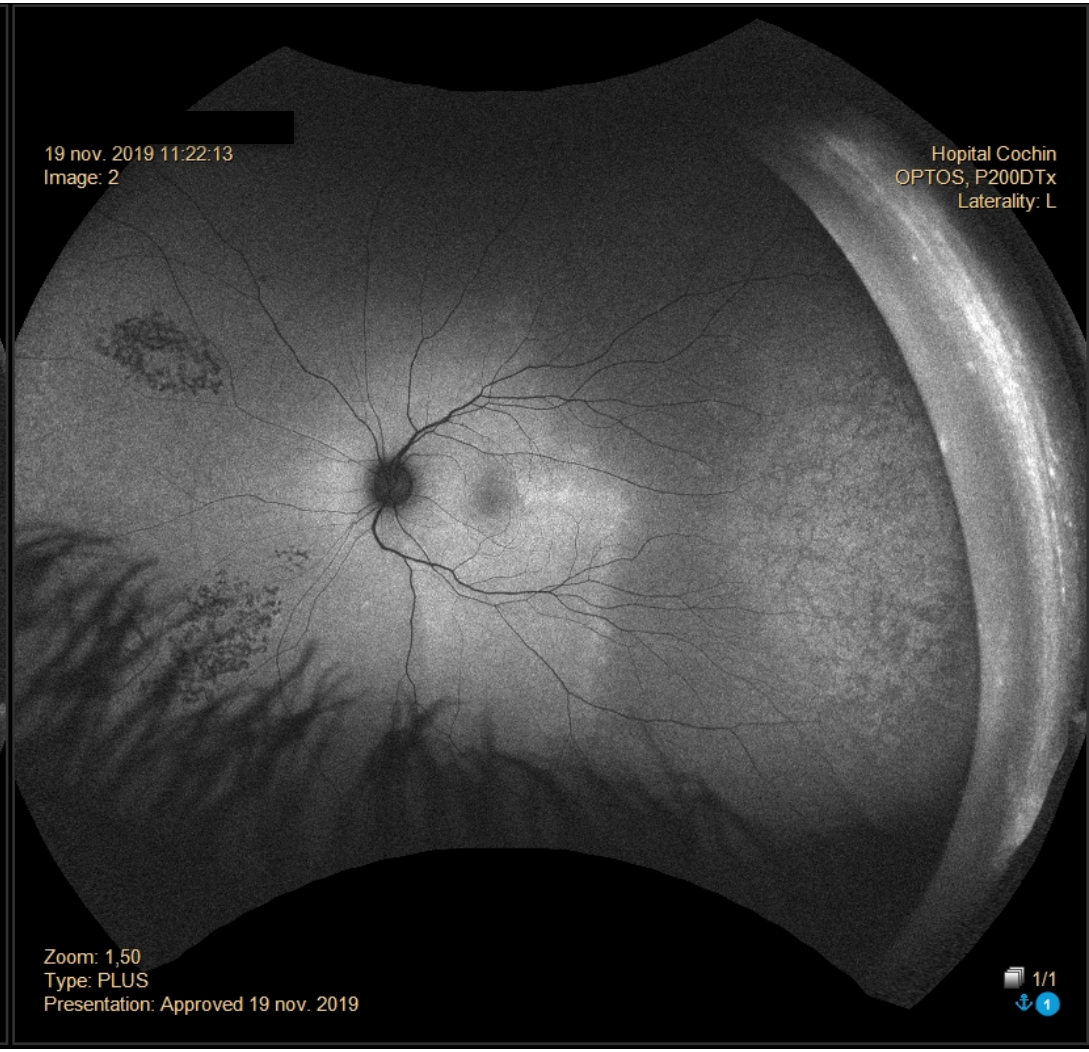
Summary

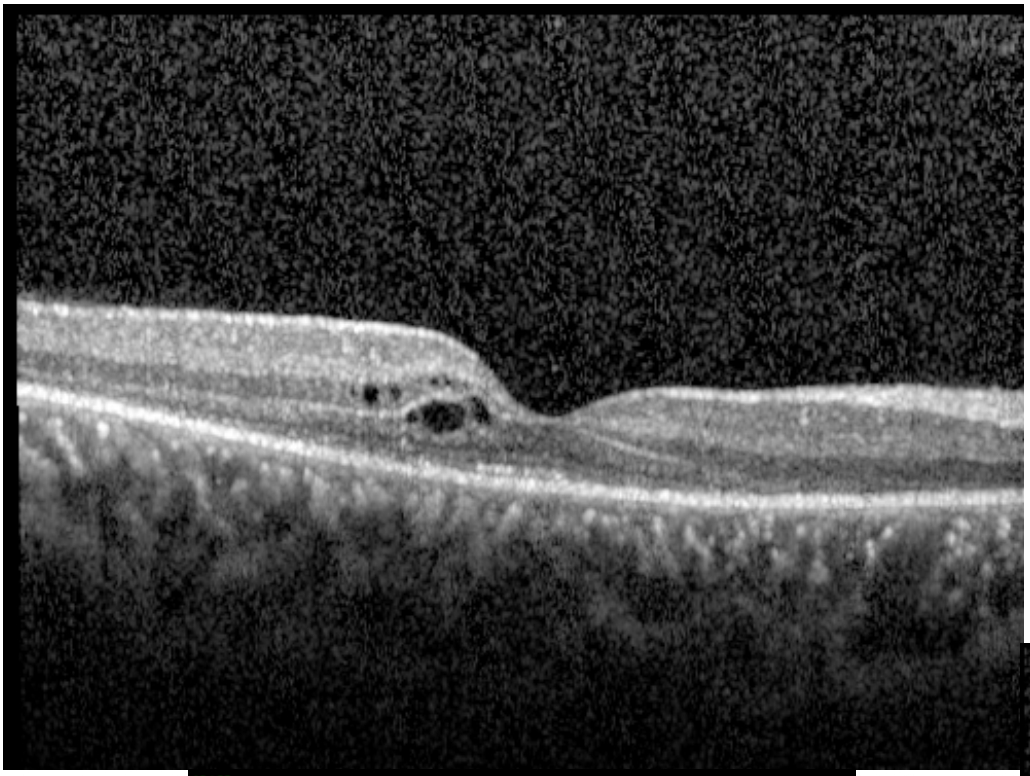
AIR is a rare but vision-threatening disease whose pathogenesis is poorly defined. Lack of standardized clinical or laboratory criteria further complicates the diagnosis and management. Despite recent progress, further basic science research into the autoimmune process is needed. Prospective controlled clinical trials with immunomodulatory therapy can help define future treatment paradigms

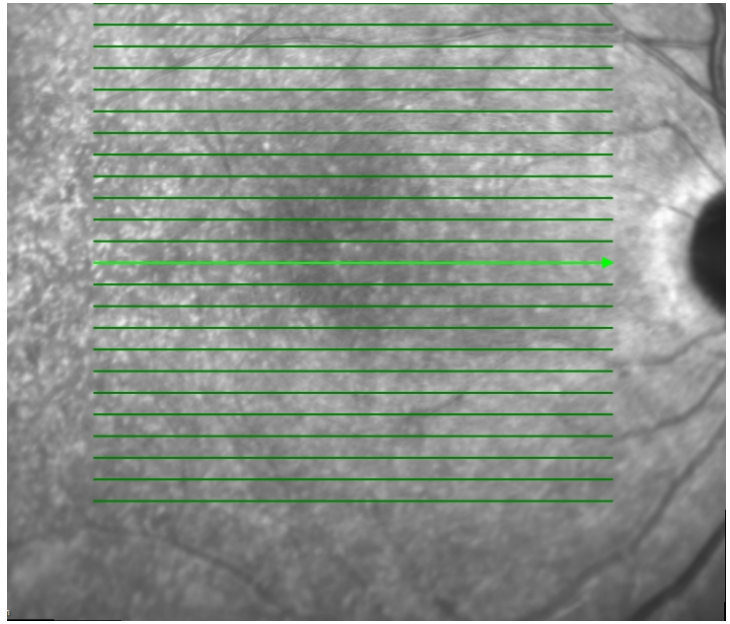

[redacted]
19 nov. 2019 11:22:37
Image: 3
Hopital Cochin
OPTOS, P200DTx
Laterality: R
Red: 50%
Green: 50%
Zoom: 1,50
Type: PLUS
Presentation: Approved 19 nov. 2019
1/1
1

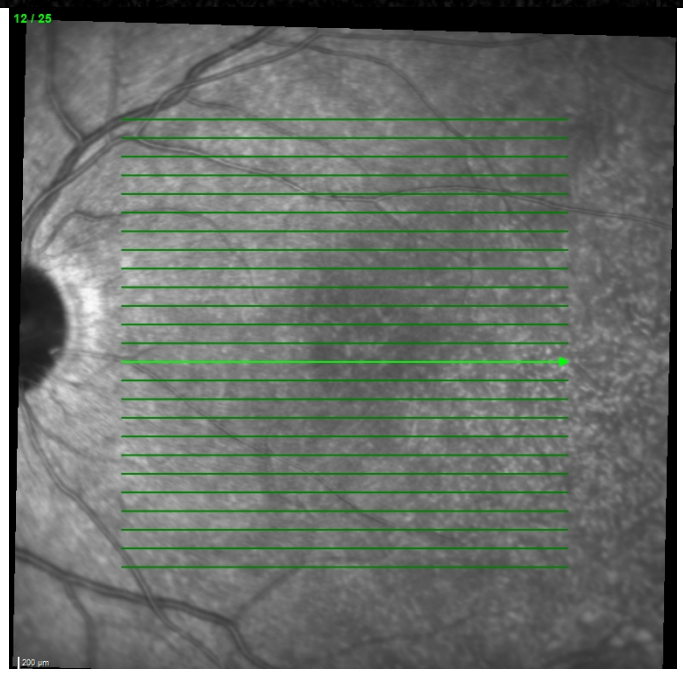

[redacted]
19 nov. 2019 11:22:02
Image: 1
Hopital Cochin
OPTOS, P200DTx
Laterality: L
Red: 50%
Green: 50%
Zoom: 1,50
Type: PLUS
Presentation: Approved 19 nov. 2019
1/1
1

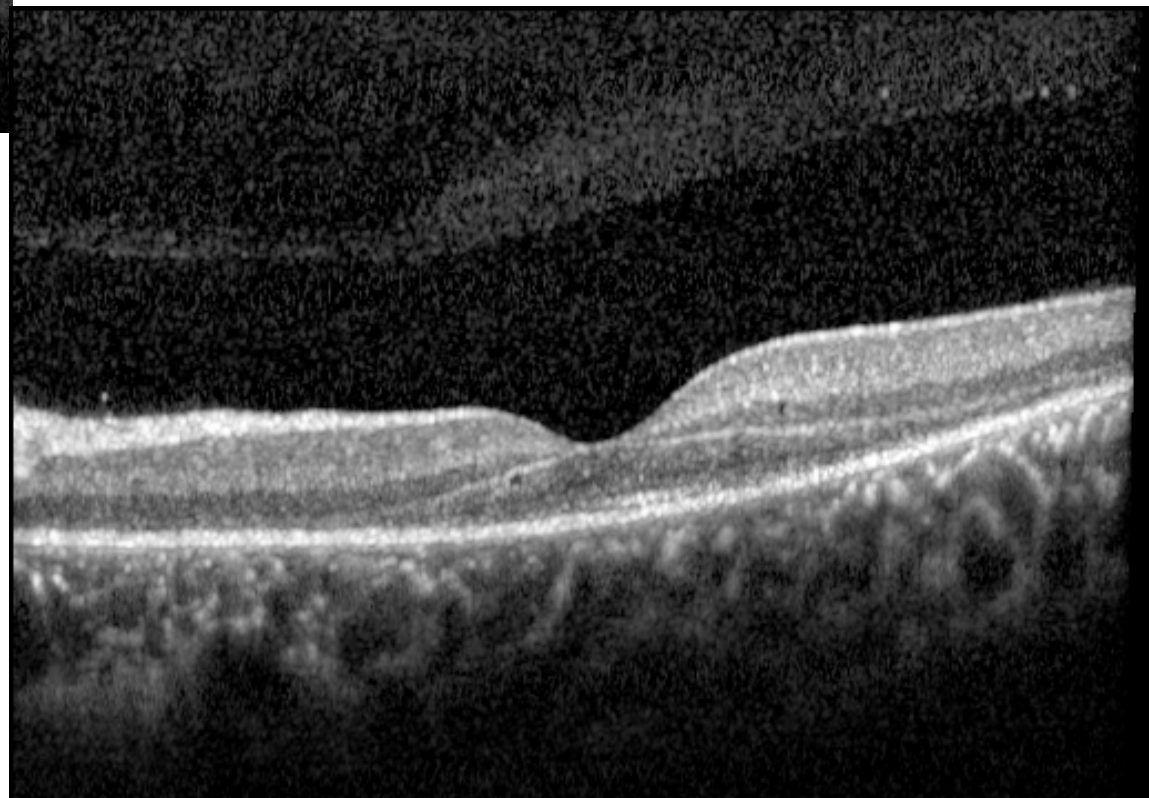

[redacted]
19 nov. 2019 11:22:47
Image: 4
Hopital Cochin
OPTOS, P200DTx
Laterality: R
Zoom: 1,50
Type: PLUS
Presentation: Approved 19 nov. 2019
1/1
1


[redacted]
19 nov. 2019 11:22:13
Image: 2
Hopital Cochin
OPTOS, P200DTx
Laterality: L
Zoom: 1,50
Type: PLUS
Presentation: Approved 19 nov. 2019
1/1
1





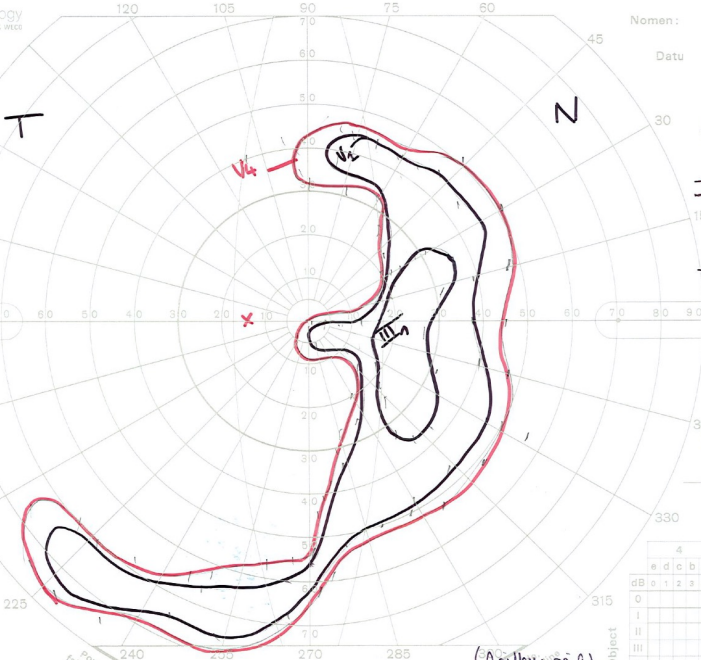

12 / 25
200 µm



Luneau Technology

Nomen :

Datu



le 19.11.19

- TA exclue au V4
- Racquette nasale de vision qui comprend un îlot central inf.
- II1 non perçu

AV = 5/10c
= coop ☺
└ T. bonne fixation
Examen fiable

(Meilleur œil)
2e œil testé

OS / OD

Relat. Intens.

		dB
1	0,0315	15
2	0,100	10
3	0,315	5
4	1,00	0
a	0,40	4
b	0,50	3
c	0,63	2
d	0,80	1
e	1,00	0

Object: mm² — 0 = V/16, I = 1/4, II = 1, III = 4, IV = 16, V = 64

Für Seitenwechsel Zeiger hier durchführen

Correctio: SC — sph ○ — cyl — °

Refractio:

Visus:

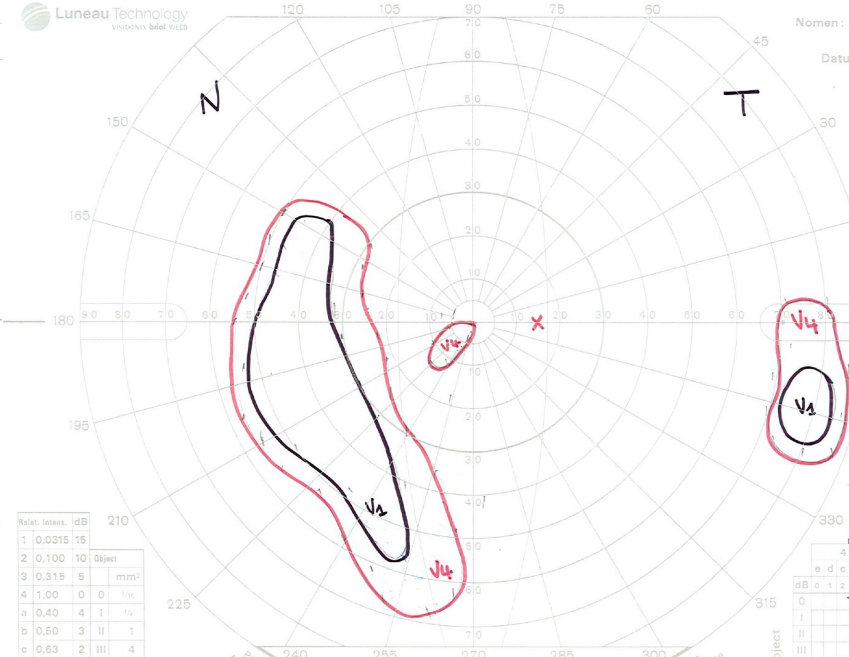
LUNEAU SAS - 2, rue Roger Bonnet - 27340 Pont de l'Arche

Tél. 15.10.21

Luneau Technology

Nomen :

Datu



le 19.11.19

- TA exclue au V4
- Ilot temporal de vision, ilot central et racquette nasale au V4 - V1
- III1 non perçu

[fixation ☺
coopération ☺

AV = 4/10c F, devrait voir le V1 ou III1 au centre ? 1er CV gelé
→ Test fiable ✓

1er œil

OS / OD

Relat. Intens.

		dB
1	0,0315	15
2	0,100	10
3	0,315	5
4	1,00	0
a	0,40	4
b	0,50	3
c	0,63	2
d	0,80	1
e	1,00	0

Object: mm² — 0 = V/16, I = 1/4, II = 1, III = 4, IV = 16, V = 64

Für Seitenwechsel Zeiger hier durchführen

Correctio: SC — sph ○ — cyl — °

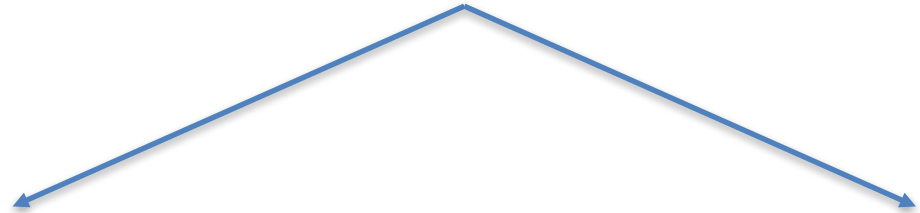
Refractio:

Visus:

LUNEAU SAS - 2, rue Roger Bonnet - 27340 Pont de l'Arche

Tél. 15.10.21

Rétinopathies auto-immunes



Paranéoplasiques

CAR
MAR
BDUMP

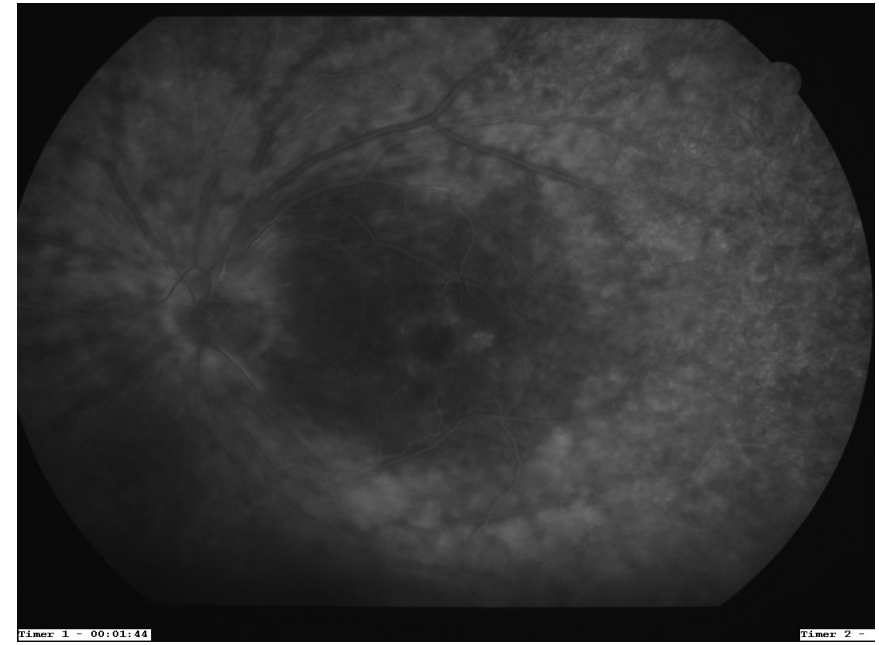
AIR

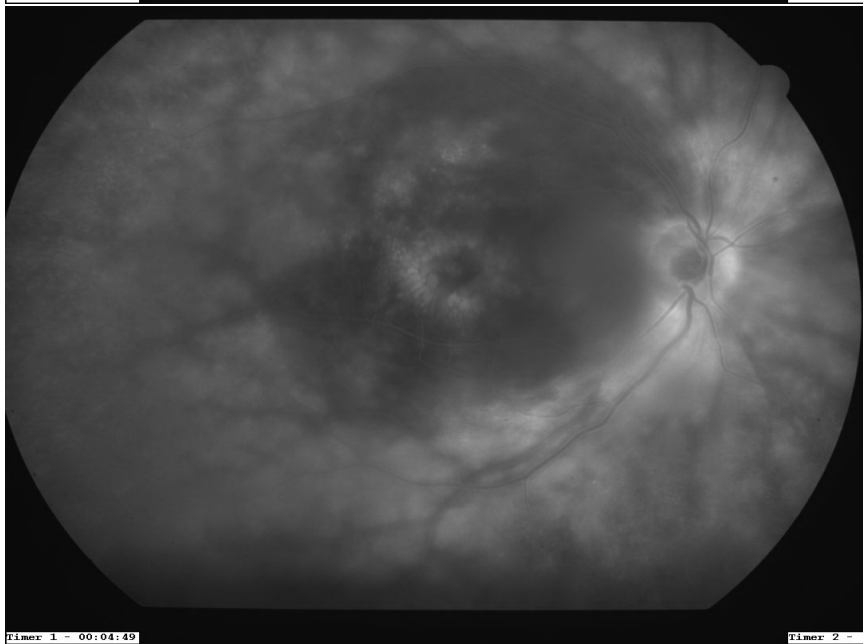
Monsieur L. 71 ans

BAV depuis 9 mois

0,3 – 0,2

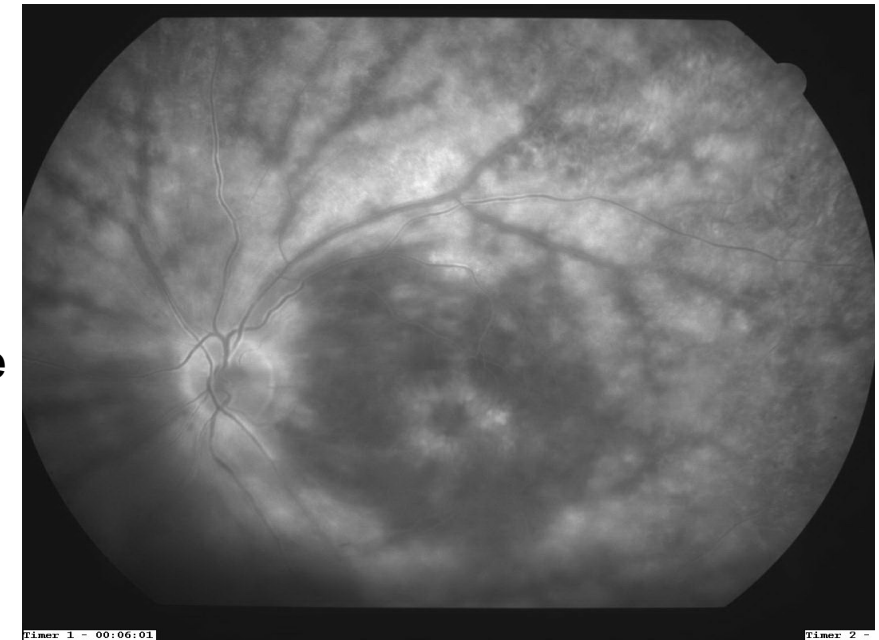
Hyalite 1-2+

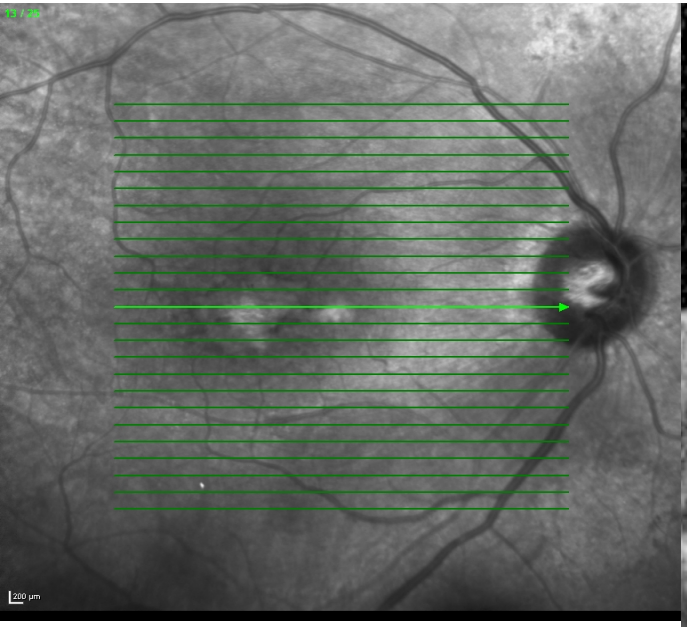


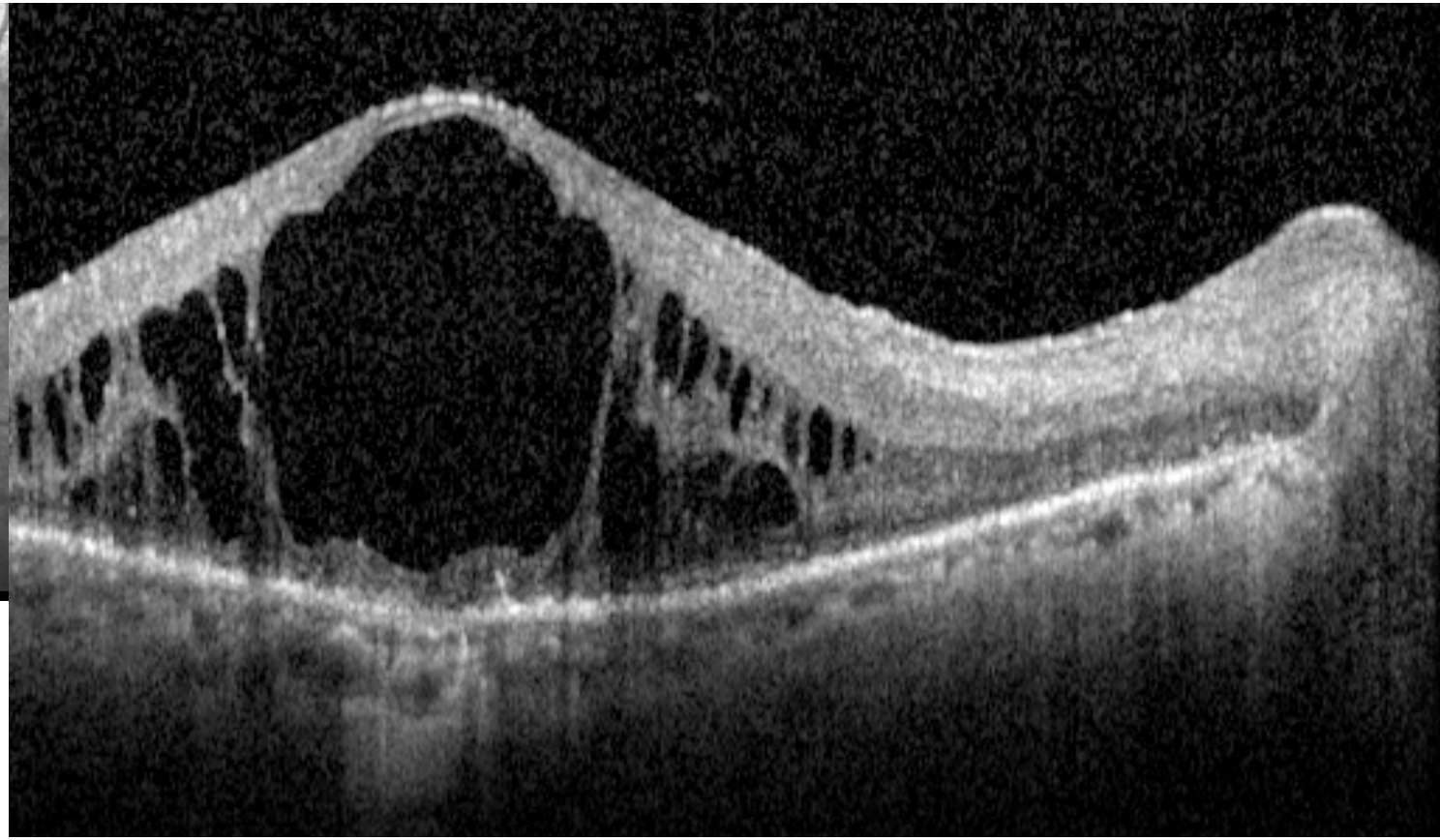


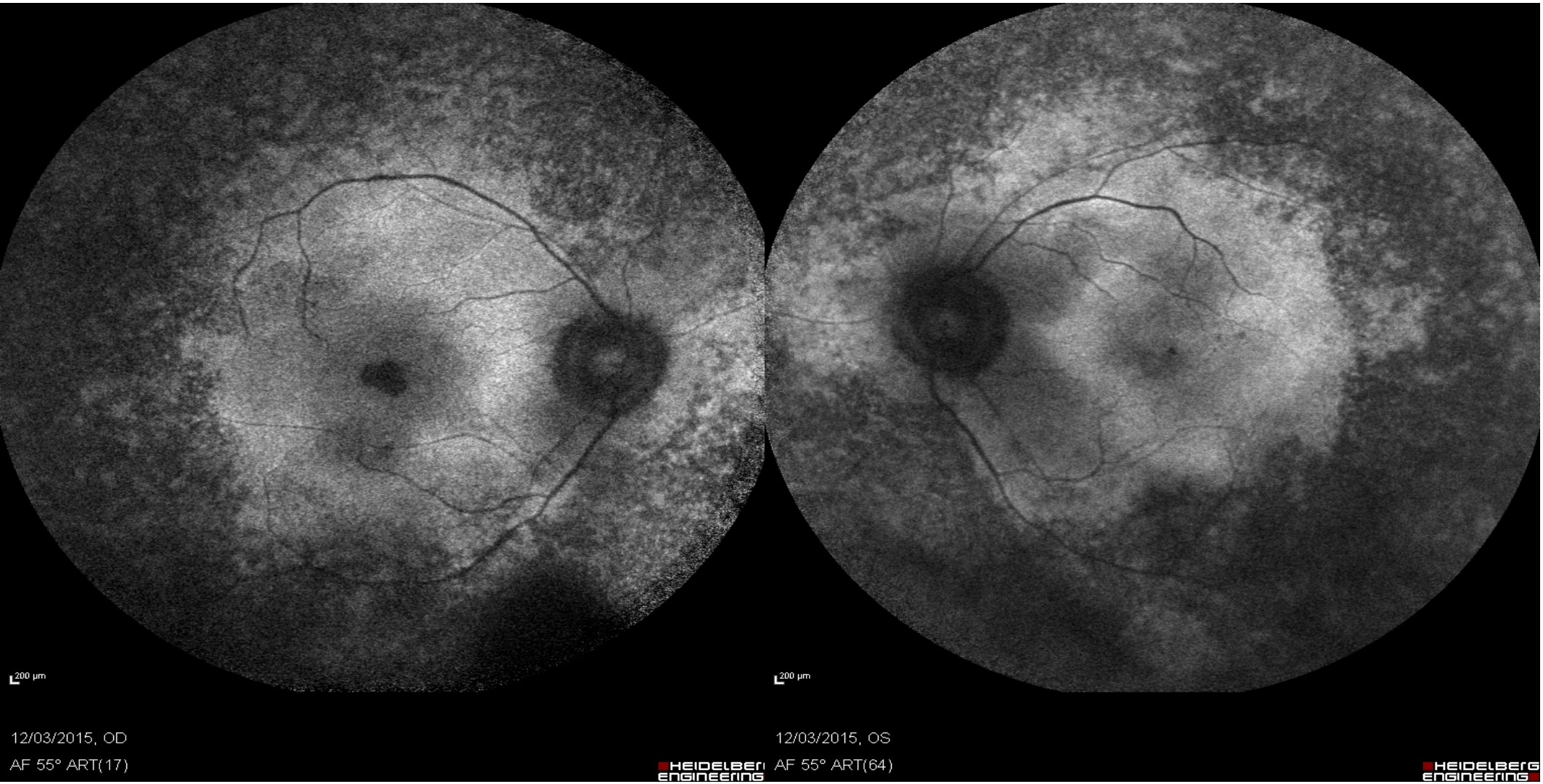
Œdème maculaire bilatéral

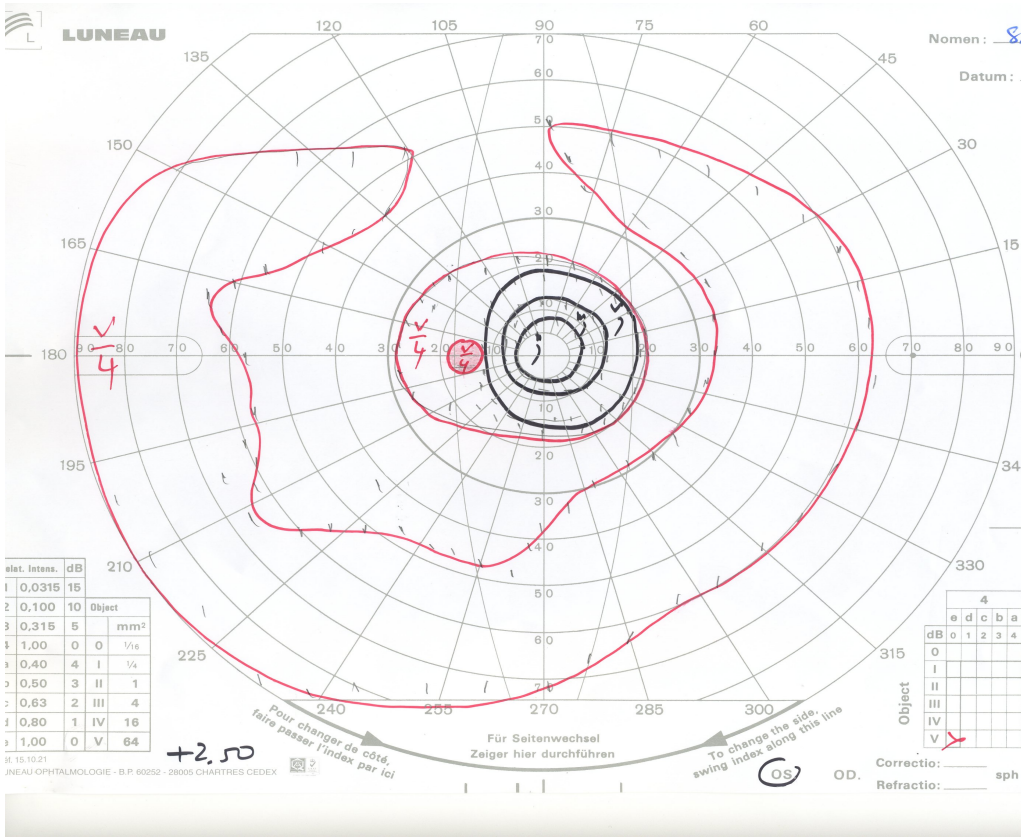
malgré prednisone *per os* 60➔10mg/j

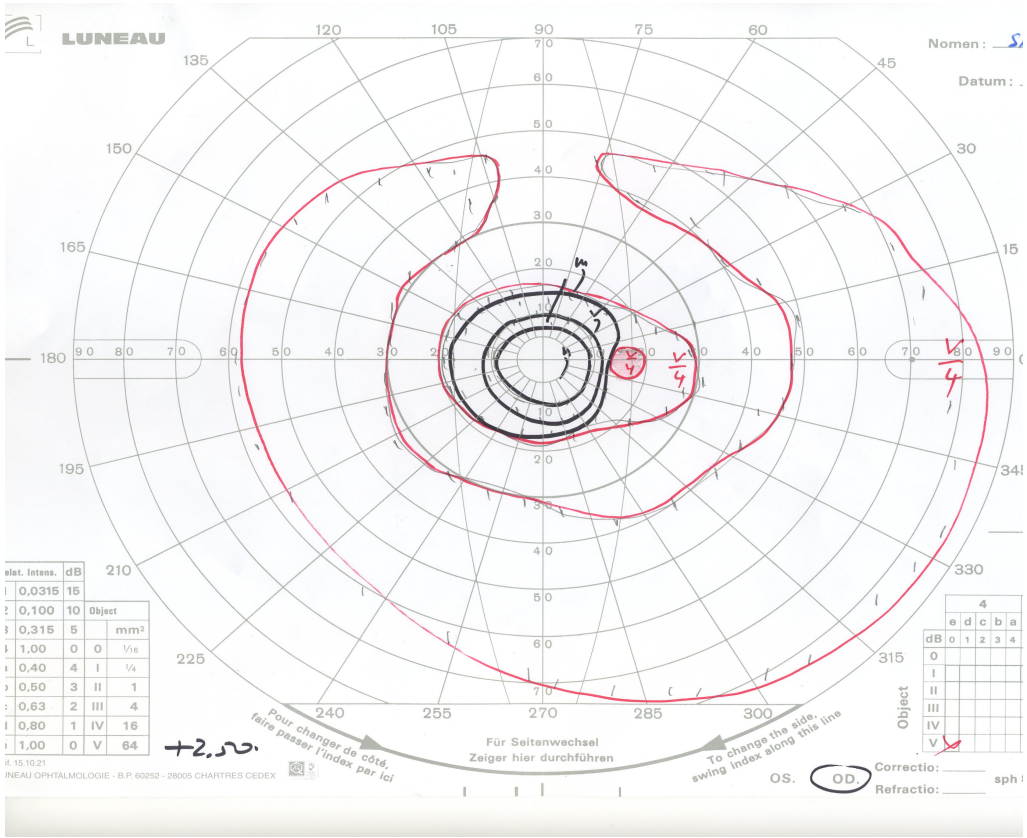



200 µm




200 µm
12/03/2015, OD
AF 55° ART(17)
HEIDELBERG ENGINEERING
200 µm
12/03/2015, OS
AF 55° ART(64)
HEIDELBERG ENGINEERING


LUNEAU
Nomen:
Datum:
+2,50
OS.
OD.
Correctio:
Refractio:
sph
Object
Für Seitenwechsel
Zeiger hier durchführen
Pour changer de côté,
faire passer l'index par ici
To change the side,
swing index along this line
LUNEAU OPHTALMOLOGIE - B.P. 60252 - 28005 CHARTRES CEDEX


LUNEAU
Nomen:
Datum:
+2,50
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OD.
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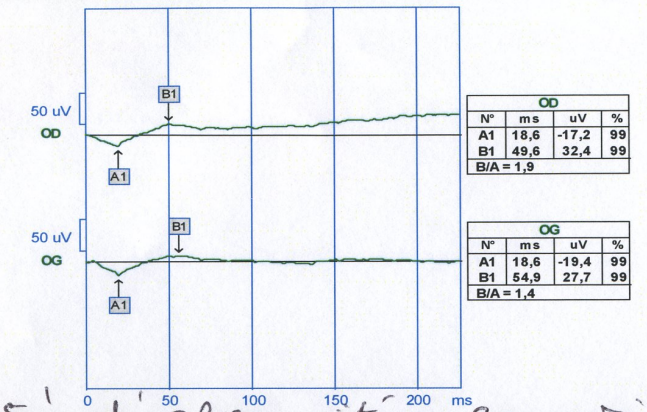
Refractio:
sph
Object
Für Seitenwechsel
Zeiger hier durchführen
Pour changer de côté,
faire passer l'index par ici
To change the side,
swing index along this line
LUNEAU OPHTALMOLOGIE - B.P. 60252 - 28005 CHARTRES CEDEX

PHOTOPIQUE

EXAMEN D'ELECTROPHYSIOLOGIE VISUELLE

ERG des cônes: flash blanc — 3mn 7s — Val= 30 — Rej= 0

BI stimulé

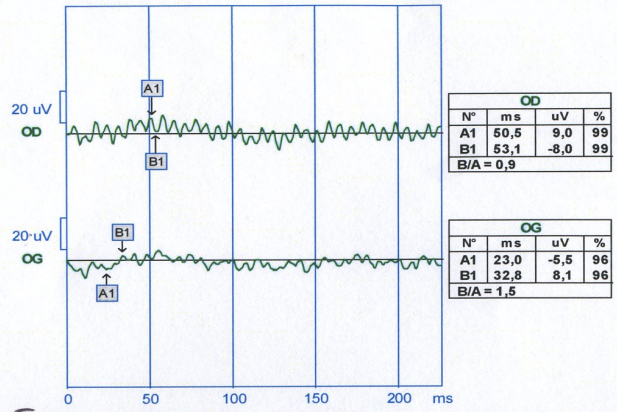


OD			
N°	ms	uV	%
A1	18,6	-17,2	99
B1	49,6	32,4	99
B/A = 1,9			

OG			
N°	ms	uV	%
A1	18,6	-19,4	99
B1	54,9	27,7	99
B/A = 1,4			

ERG des cônes: flash rouge — 5mn 3s — Val= 30 — Rej= 0

BI stimulé

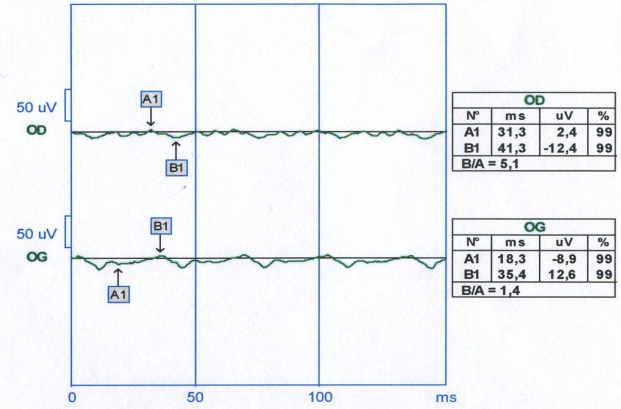


OD			
N°	ms	uV	%
A1	50,5	9,0	99
B1	53,1	-8,0	99
B/A = 0,9			

OG			
N°	ms	uV	%
A1	23,0	-5,5	96
B1	32,8	8,1	96
B/A = 1,5			

ERG des cônes: flicker 30Hz — 5mn 33s — Val= 30 — Rej= 0

BI stimulé



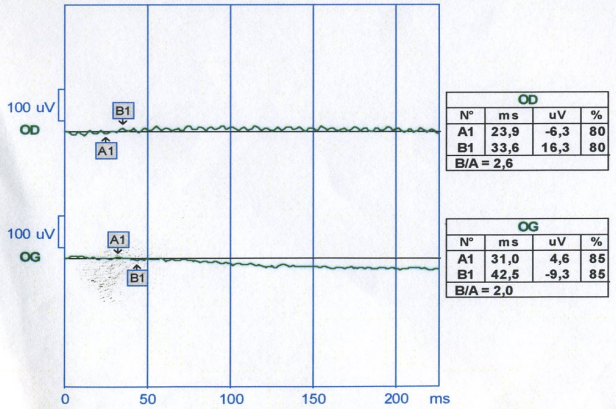
OD			
N°	ms	uV	%
A1	31,3	2,4	99
B1	41,3	-12,4	99
B/A = 5,1			

OG			
N°	ms	uV	%
A1	18,3	-8,9	99
B1	35,4	12,6	99
B/A = 1,4			

S i d'obscurité : SCOTOPIQUE

ERG des bâtonnets (-25dB) — 12mn 45s — Val= 8 — Rej= 0

BI stimulé

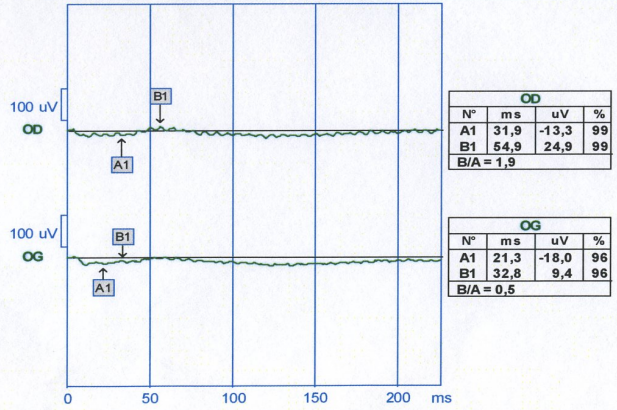


OD			
N°	ms	uV	%
A1	23,9	-6,3	80
B1	33,6	16,3	80
B/A = 2,6			

OG			
N°	ms	uV	%
A1	31,0	4,6	85
B1	42,5	-9,3	85
B/A = 2,0			

ERG mixte: cônes +bâtonnets — 14mn 55s — Val= 8 — Rej= 0

BI stimulé

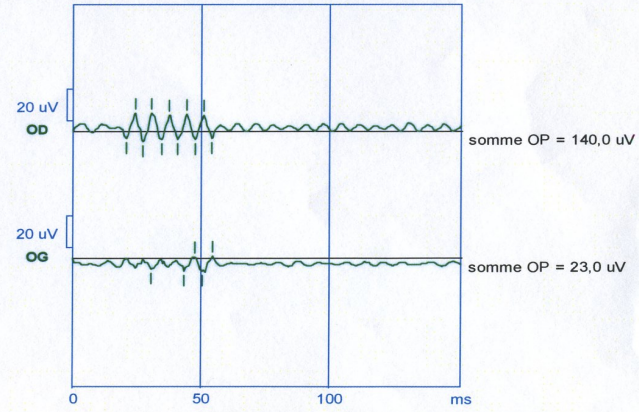


OD			
N°	ms	uV	%
A1	31,9	-13,3	99
B1	54,9	24,9	99
B/A = 1,9			

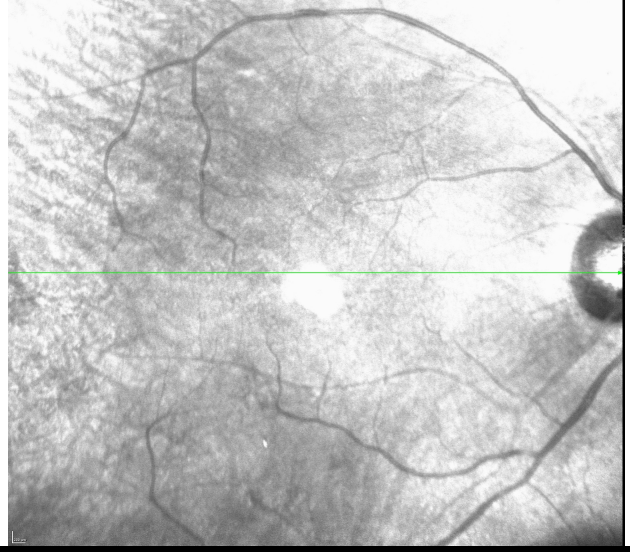
OG			
N°	ms	uV	%
A1	21,3	-18,0	96
B1	32,8	9,4	96
B/A = 0,5			

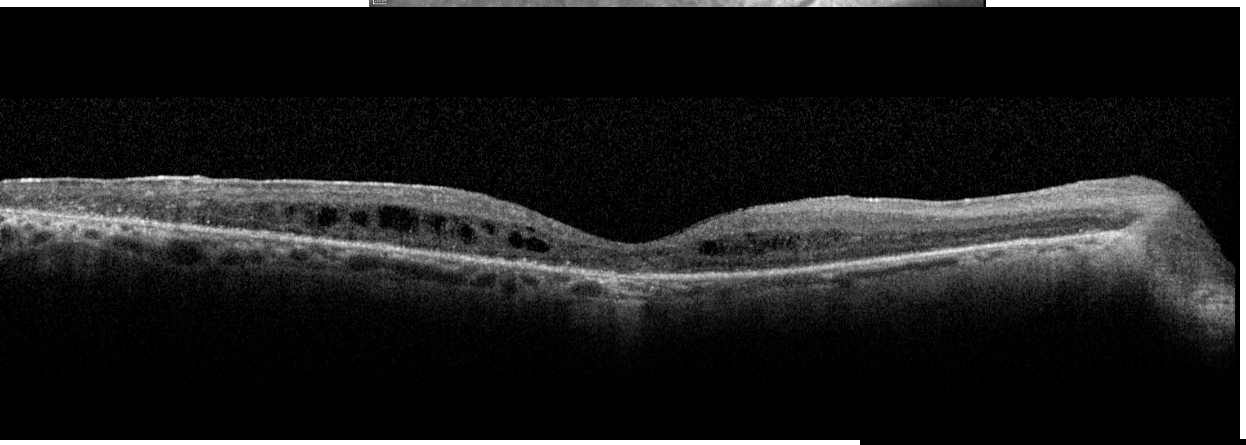
ERG potentiels oscillatoires — 16mn 21s — Val= 8 — Rej= 0

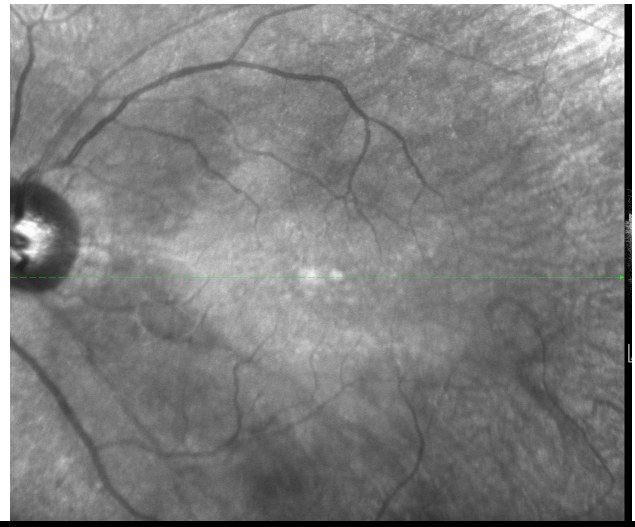
BI stimulé

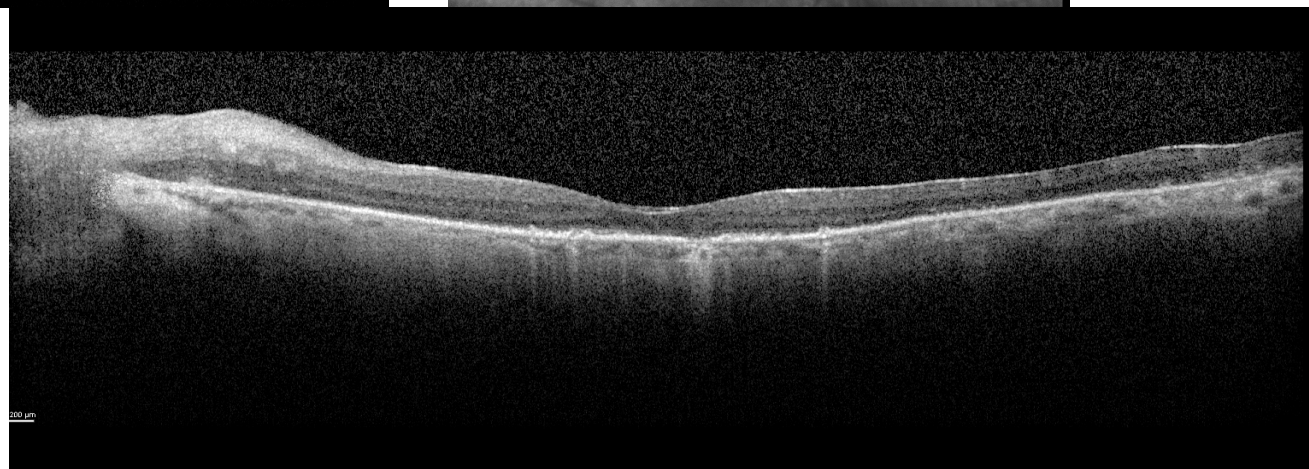


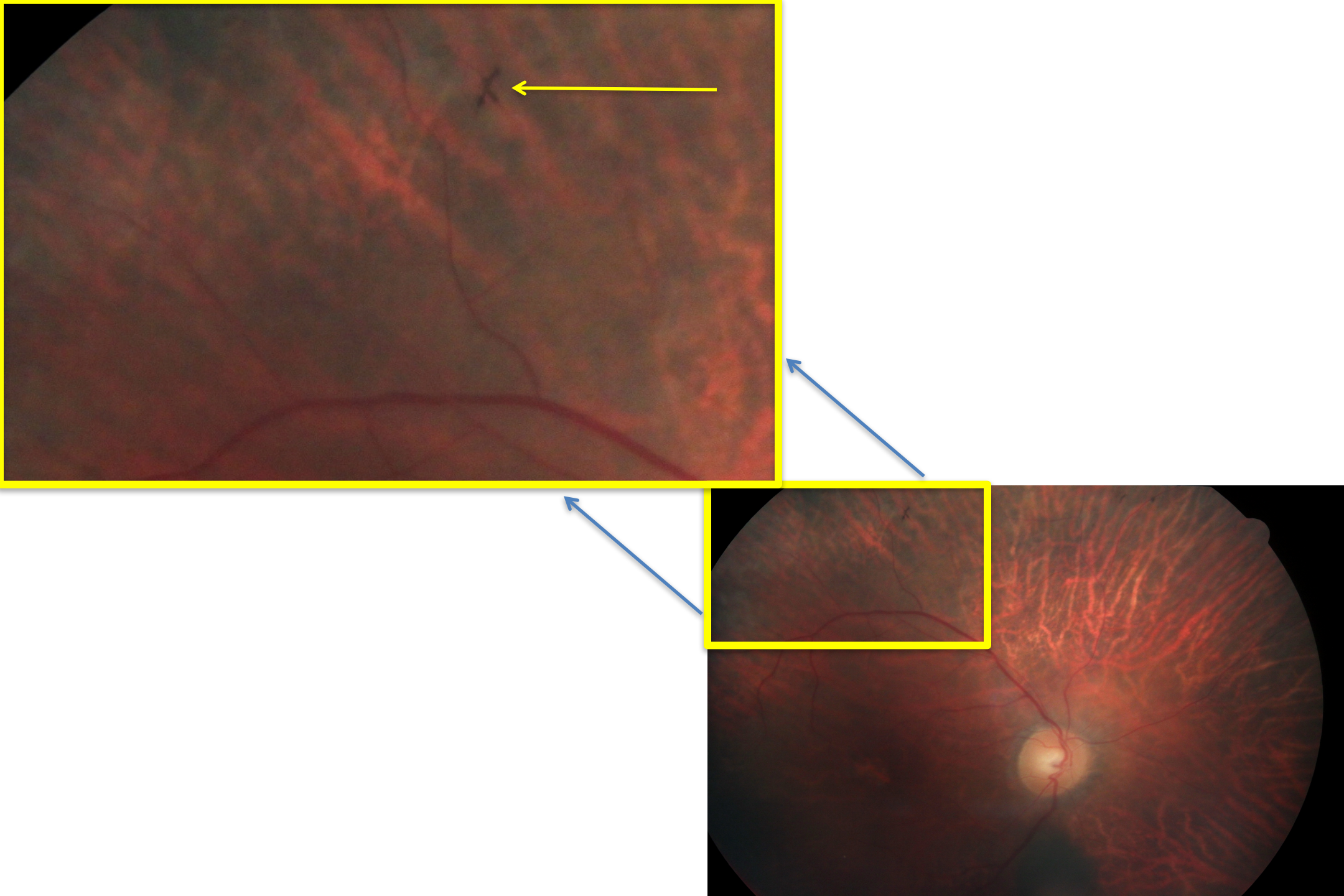
Cinq ans plus tard











Hoogewoud et al. BMC Ophthalmology (2018) 18:285
https://doi.org/10.1186/s12886-018-0948-2

BMC Ophthalmology

RESEARCH ARTICLE Open Access

Cancer-associated retinopathy preceding the diagnosis of cancer

Florence Hoogewoud[1*], Pauline Butori[1], Philippe Blanche[2] and Antoine P. Brézin[1]

Abstract

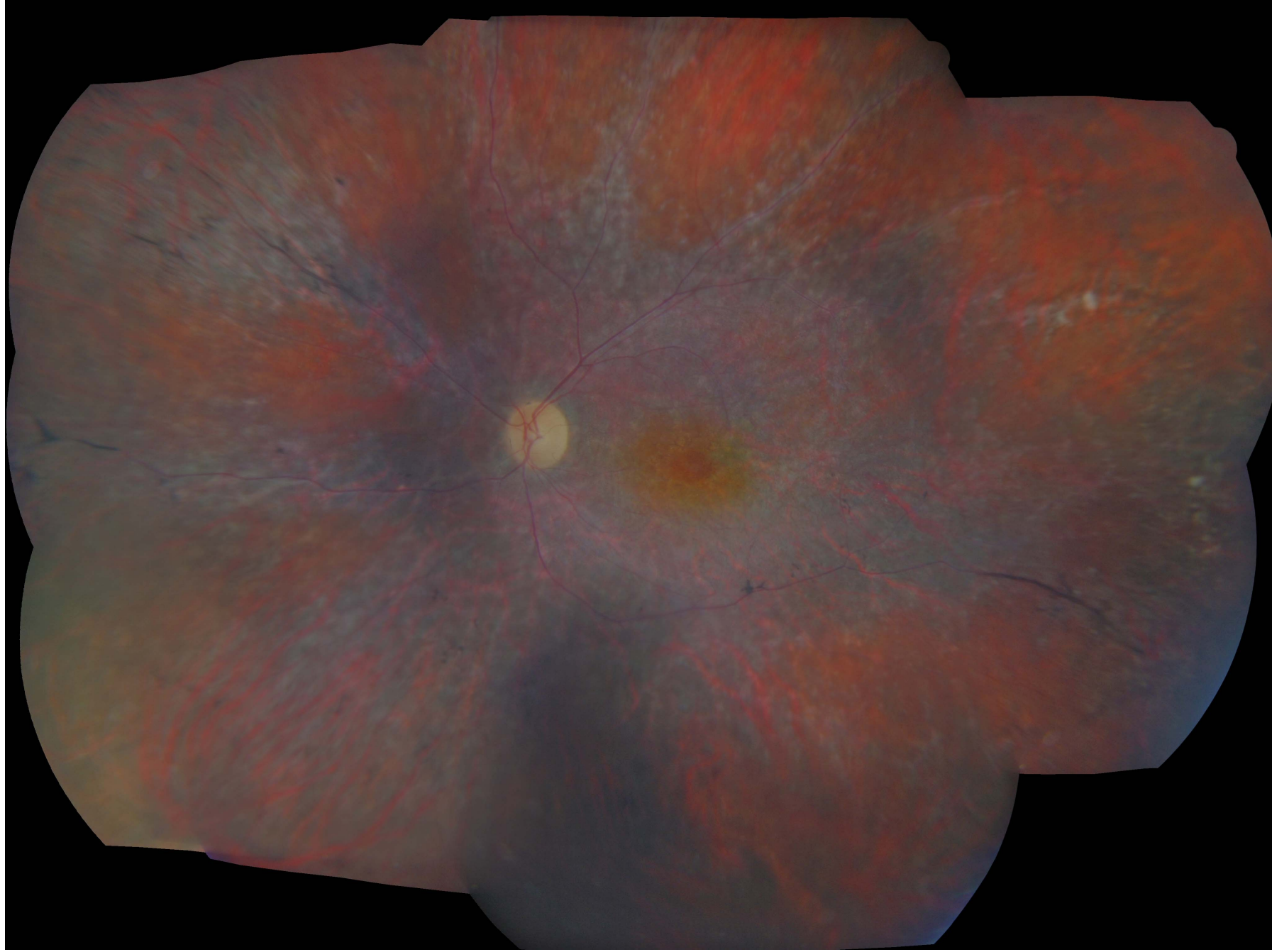
Background: The early diagnosis of cancer is of crucial importance and a key prognostic factor. Cancer-associated retinopathy (CAR) can be symptomatic prior to other manifestations directly related to malignant tumors. The aim of this study was to show that, in selected cases, ophthalmic findings are consistent enough with the diagnosis of CAR to trigger investigations aimed at detecting a previously unknown malignancy.

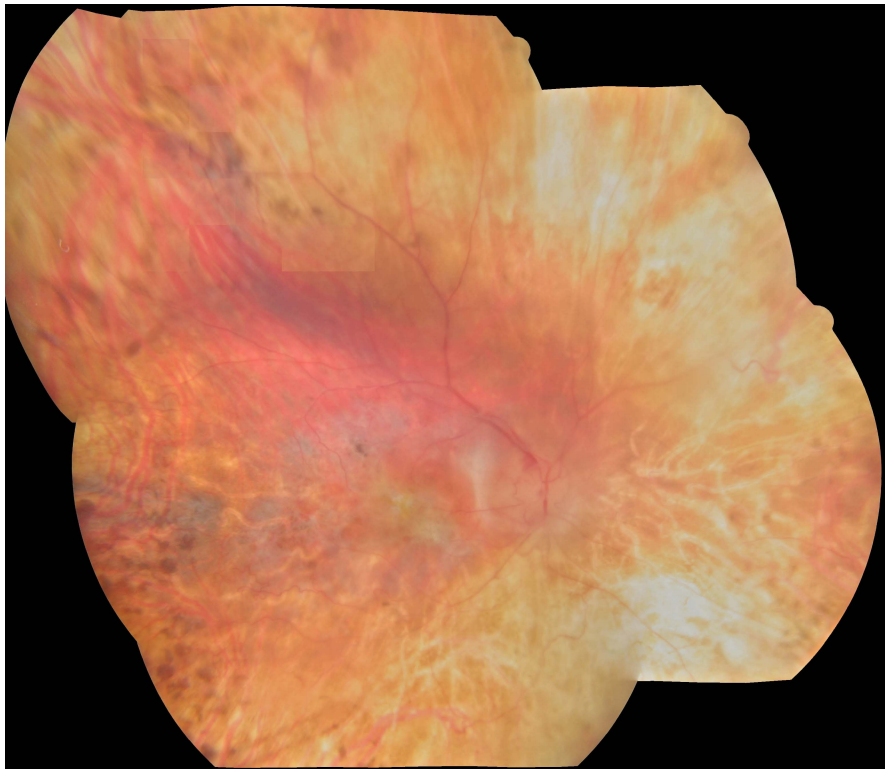
Methods: This was a monocentric retrospective case series performed in a tertiary referral center. Patients with a diagnosis of CAR were included. Diagnosis was based on the clinical presentation, the visual field and electroretinogram alterations. The clinical presentation, visual field testing and electroretinographic results were analyzed as well as the malignancies identified following the diagnosis of CAR. Follow-up data was collected.

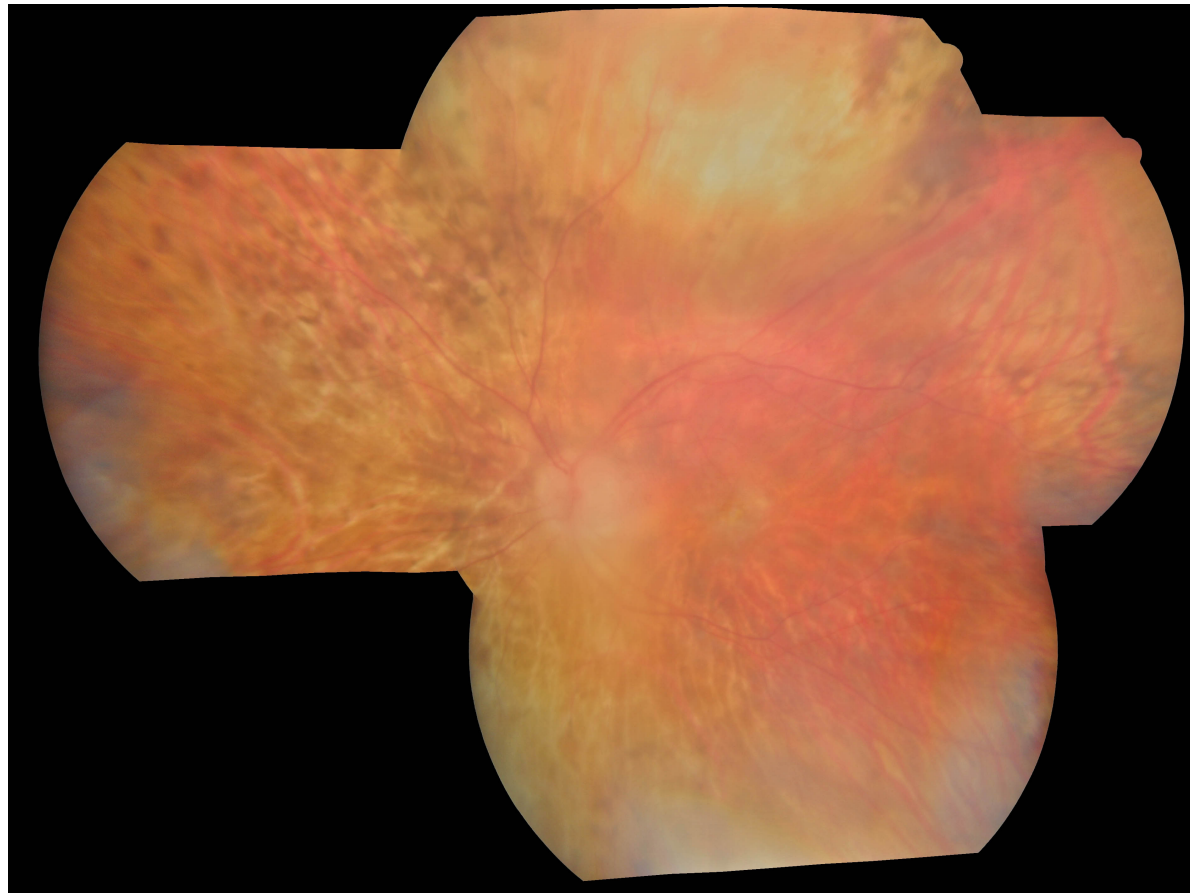
Results: Four patients (two men, two women, median age 65.5 years) were included. All patients presented with posterior segment inflammation at initial presentation as well as advanced visual field loss and an extinguished electroretinogram. The best corrected decimal visual acuity was 0.8 or better in both eyes of three patients and decreased to 0.3 OD and 0.2 OS in one patient due to a bilateral macular edema. No patient had a previously known history of cancer. Once the diagnosis of CAR was made, investigations aimed at identifying a malignant tumors subsequently led to the diagnosis of two cases of small cell lung tumors, of one prostate carcinoma and of a uterine sarcoma. The treatment of CAR included plasmapheresis, systemic corticosteroids, azathioprine, cyclosporine and periocular or intraocular corticosteroid injections. In all cases the intraocular inflammation resolved, but pigment mottling, diffuse retinal atrophy, optic disc pallor and arterial narrowing were among manifestations observed during the follow-up of the patients.

Conclusion: In selected patients, findings suggestive of CAR can be useful for the early detection of a cancer.

Keywords: Cancer-associated retinopathy, Uveitis, Paraneoplastic retinopathy, Cancer







Melanoma Associated Retinopathy (M.A.R.)

Cas clinique

Patiente de 79 ans

Adressée pour BAV bilatérale rapidement progressive associée à des douleurs oculaires et des céphalées plutôt temporales.

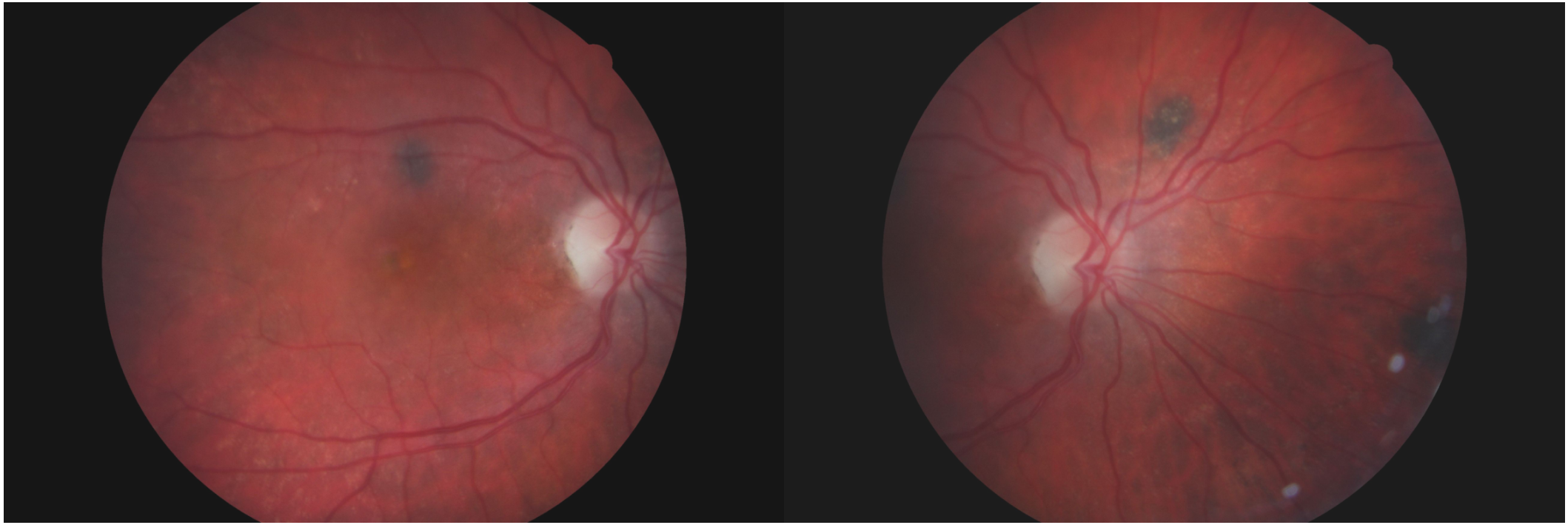
ATCD ophtalmologiques :

Chirurgie de la cataracte bilatérale non compliquée (2012)

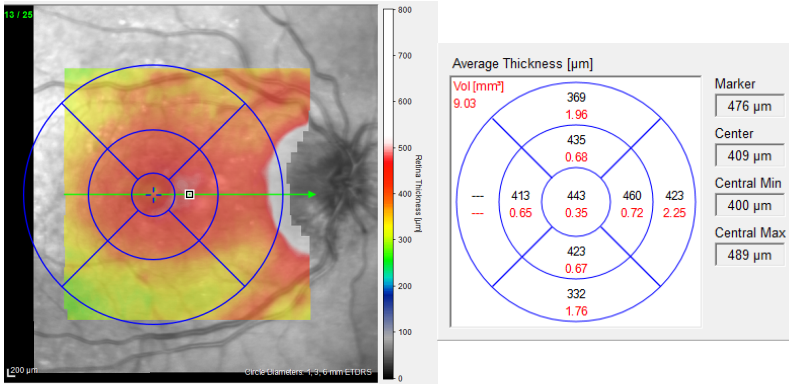
Examen OPH initial

OD		OG
1,5/10	AV	1/10
12	PIO	11
PRD granulomateux Tyndall x Pas de SIC ICP Pannus sup + inf sur blépharite	LAF	PRD granulomateux Tyndall ½ x Pas de SIC ICP Pannus sup + inf sur blépharite
16 ph/ ms	Flare	88 ph / ms
Œdème papillaire avec papille pâle, Sans hémorragie Pas de hyalite Pas de foyer	FO	Papille normale Pas de hyalite Pas de foyer

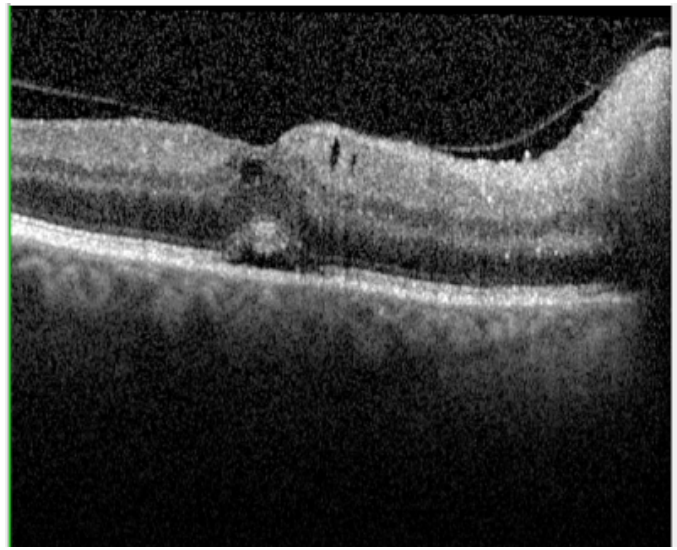
OD



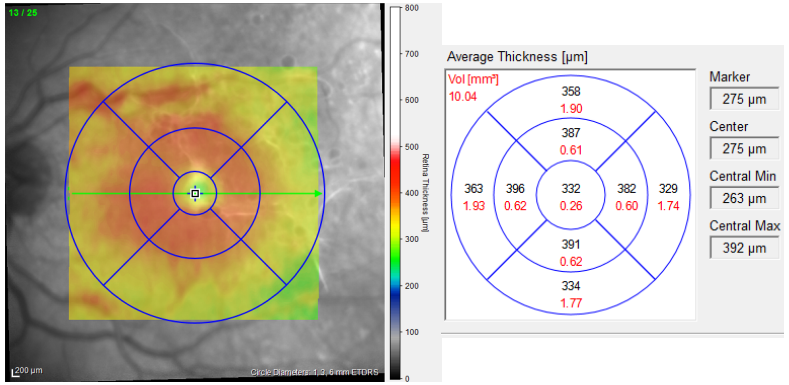
OD

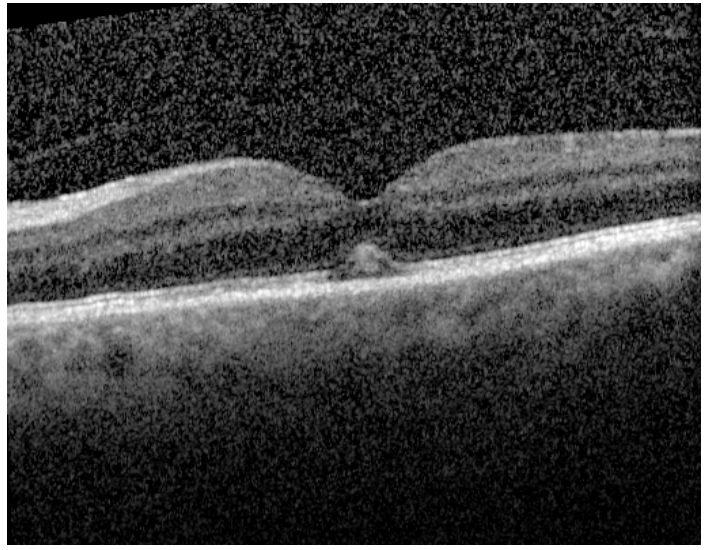

13 / 25
Circle Diameters 1.3, 6 mm ETDRS
Retina Thickness [µm]
800
700
600
500
400
300
200
100
0
Average Thickness [µm]
Vol [mm³]
9.03
369
1.96
435
0.68

413
0.65
443
0.35
460
0.72
423
2.25
423
0.67
332
1.76
Marker
476 µm
Center
409 µm
Central Min
400 µm
Central Max
489 µm



OG


13 / 25
Circle Diameters 1.3, 6 mm ETDRS
Retina Thickness [µm]
800
700
600
500
400
300
200
100
0
Average Thickness [µm]
Vol [mm³]
10.04
358
1.90
387
0.61
363
1.93
396
0.62
332
0.26
382
0.60
329
1.74
391
0.62
334
1.77
Marker
275 µm
Center
275 µm
Central Min
263 µm
Central Max
392 µm



Uvéite antérieure aigue bilatérale compliquée d'œdème maculaire et d'œdème papillaire à droite

- Hypothèses :
  - NOIAA dans le cadre d'une maladie de Horton ?
  - Uvéopapillite infectieuse (Lyme ?) ou inflammatoire

- Bilan biologique et examens complémentaires : négatifs, sauf VS = 120
- Angiographie :

OD= œdème papillaire et œdème maculaire, pas de vascularite

OG= Initialement pas d'anomalie retrouvée

Traitement initial : corticothérapie (maladie de Horton ?)

Médecine interne

ATCD

- Arthrose
- Dyslipidémie
- Ostéoporose
- Cancer du col de l'utérus en 2011
  - Hystérectomie totale
  - Chimiothérapie

Hernie discale opérée

Bilan uvéo-papillite bilatérale

OPH => NOIAA, Uvéo-papillite infectieuse

Pas de signe clinique pour un Horton

BAT négative

vs 120 mais CRP 20 mg/L

Bilan infectieux négatif

ECA négative, BGSA normale

PL normale

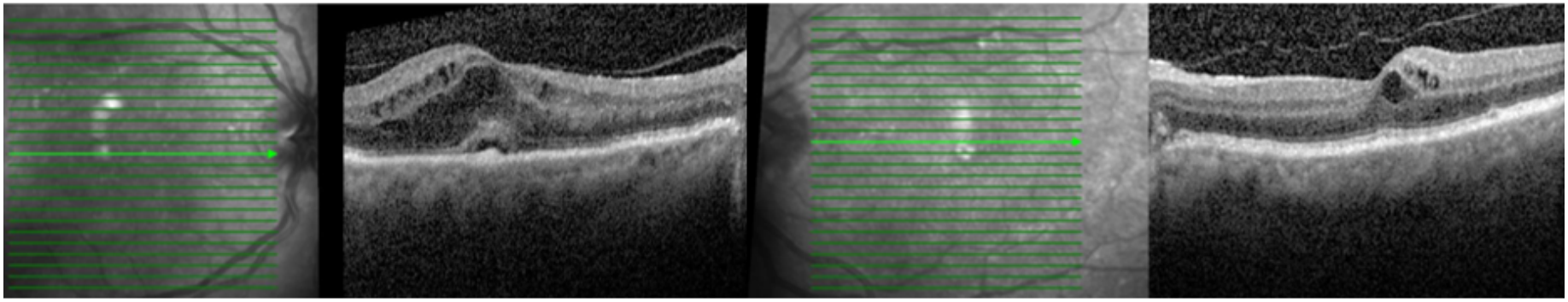
TDM thoracique normal

=> CTC 1 mg/kg (NOIAA, Horton ?) avec amélioration rapide

Evolution

- Récidives de poussées inflammatoires avec œdème maculaire ODG répondant aux injections sous-ténoniennes de Kénacort puis intra-vitréennes d'Ozurdex

-



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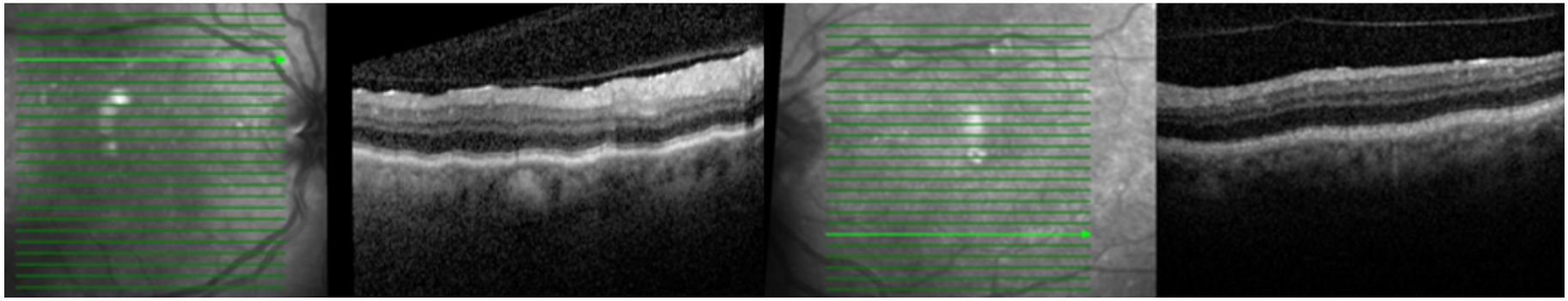
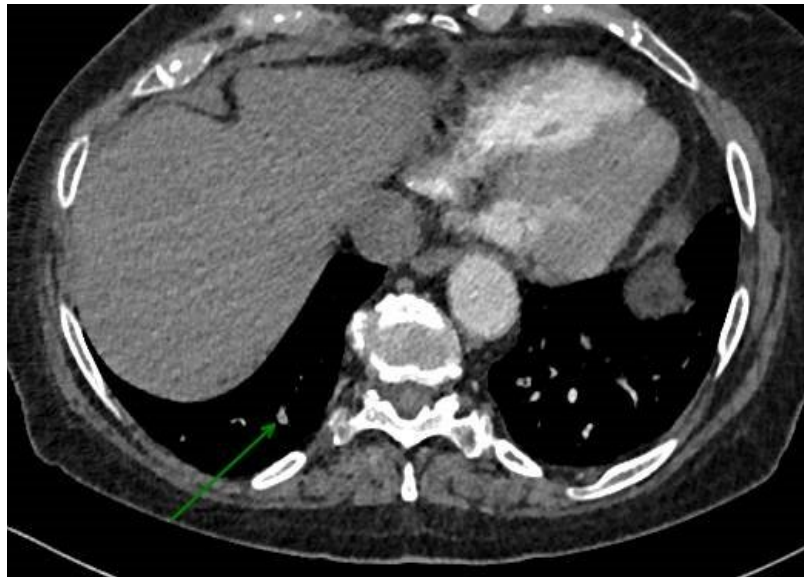
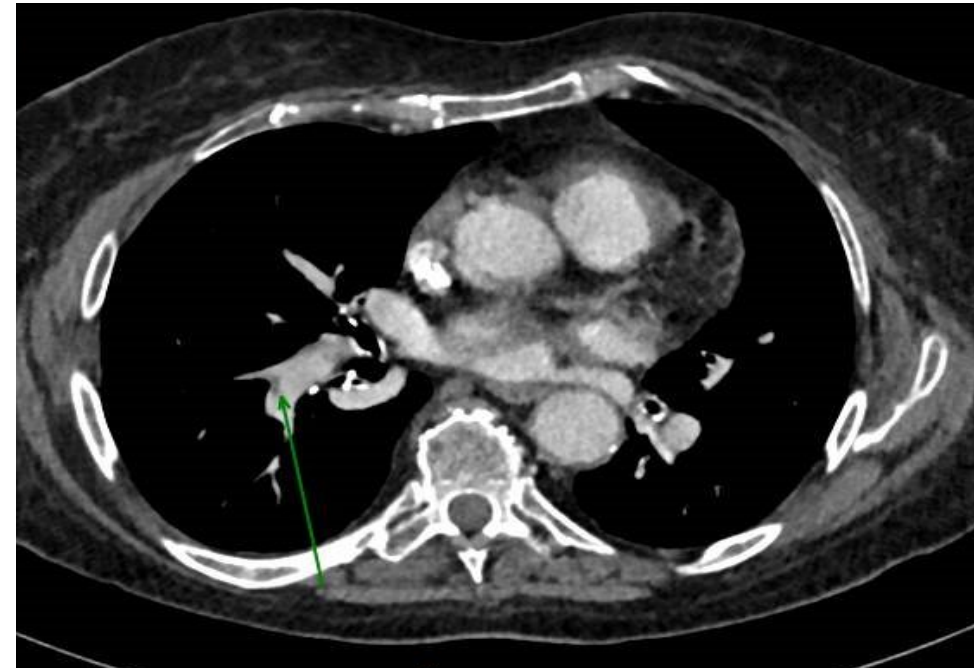


6 mois plus tard

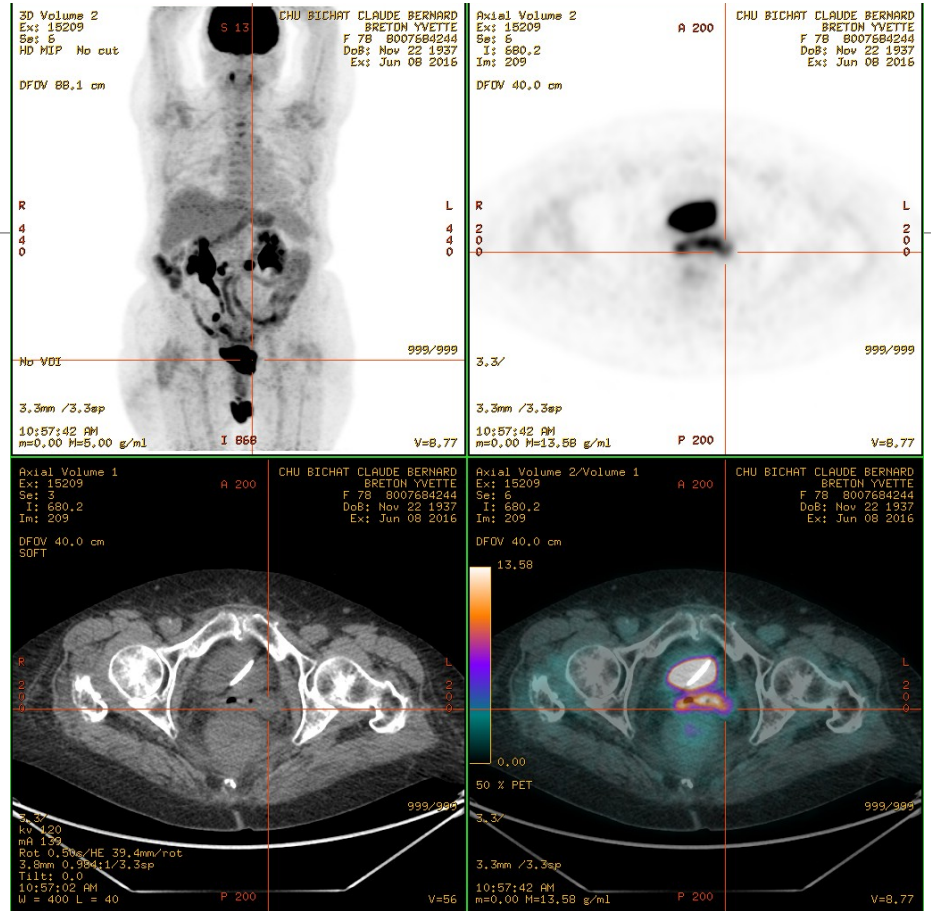
- Altération état général -10 kg
- Syndrome inflammatoire biologique
- Dyspnée
- Angio-TDM : embolie pulmonaire





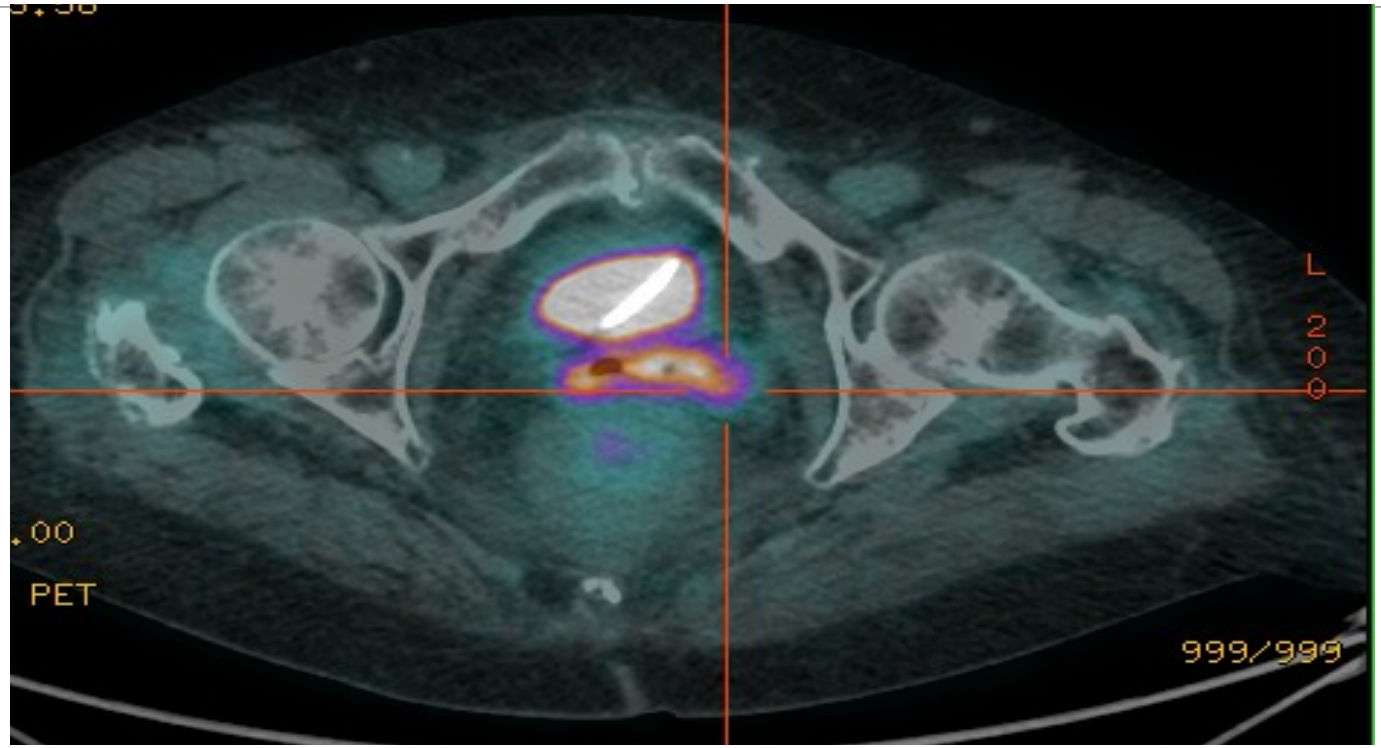
Néoplasie ?

PET-TDM

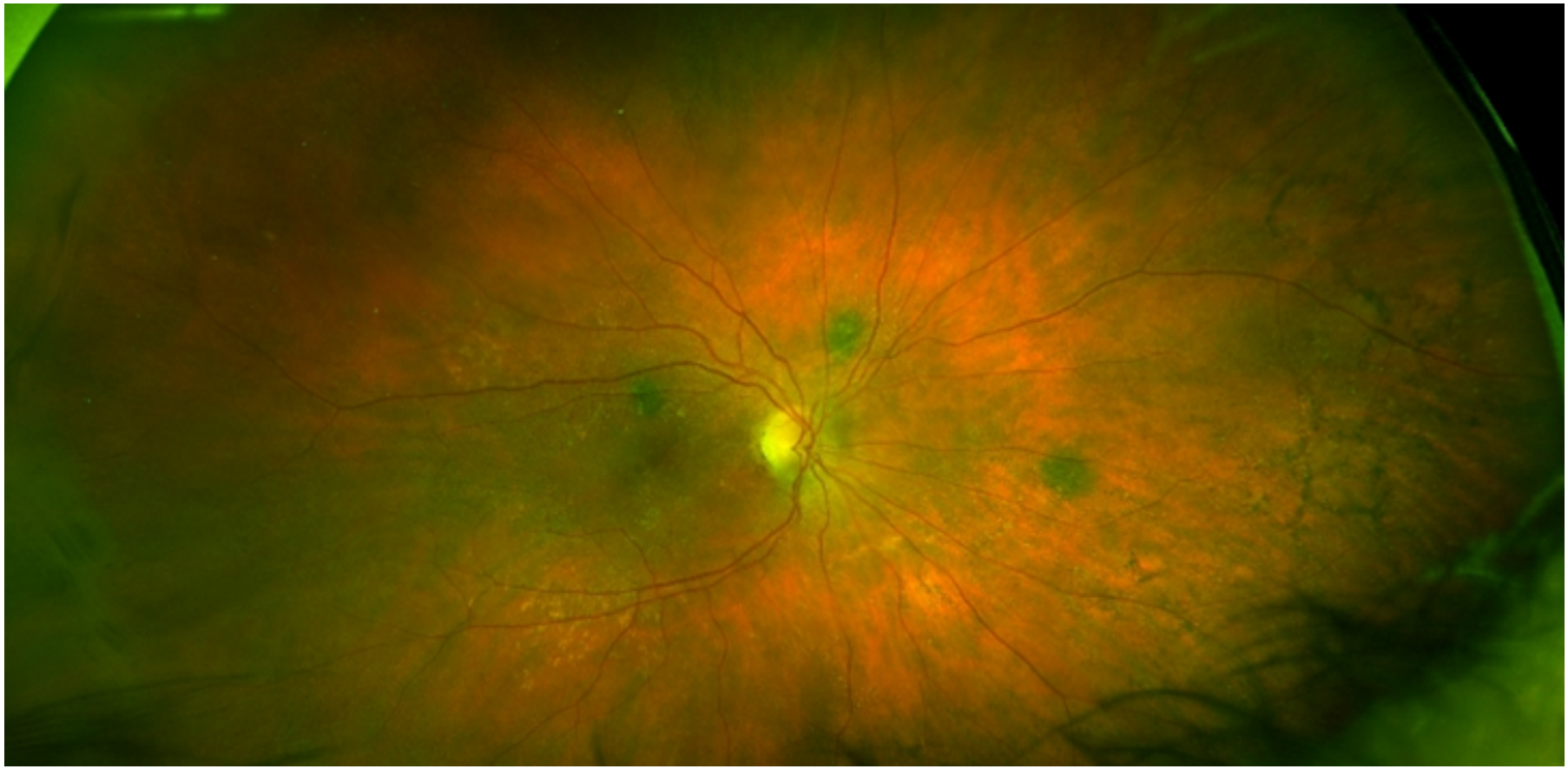


Hypermétabolisme partie proximale du vagin (SUVmax15) et ADP latéro-aortiques (SUVmax 9) et bifurcation iliaque (SUVmax 11,5)

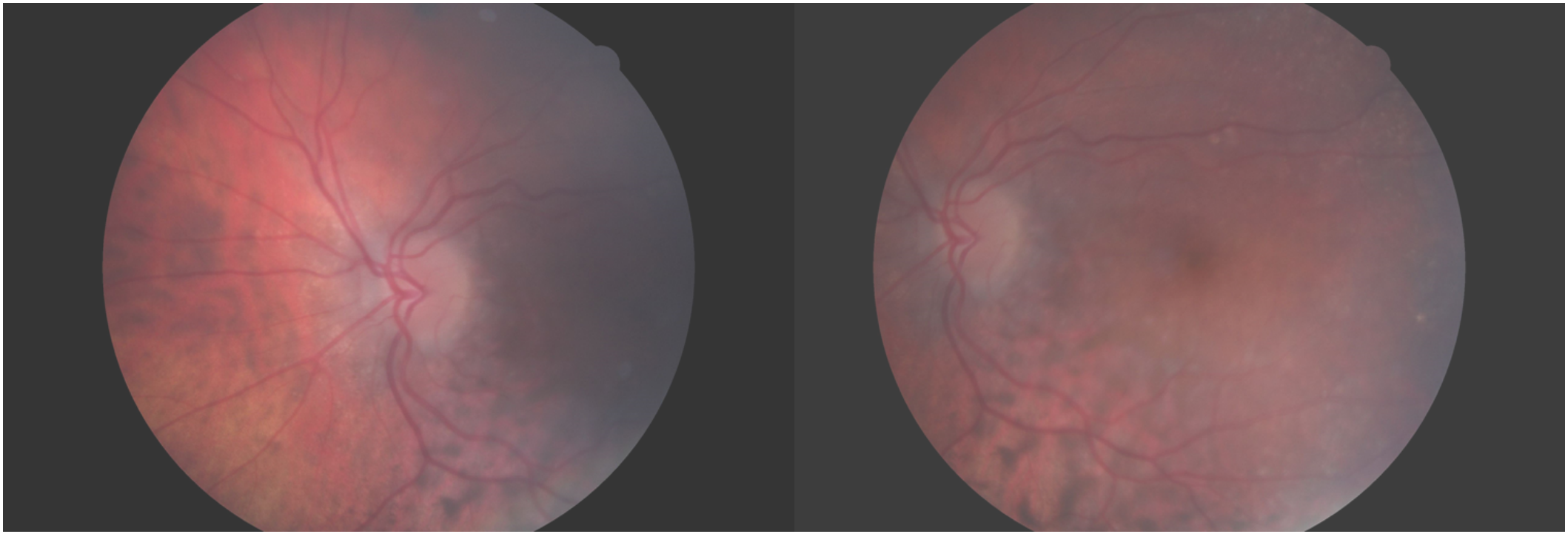
Biopsie vaginale

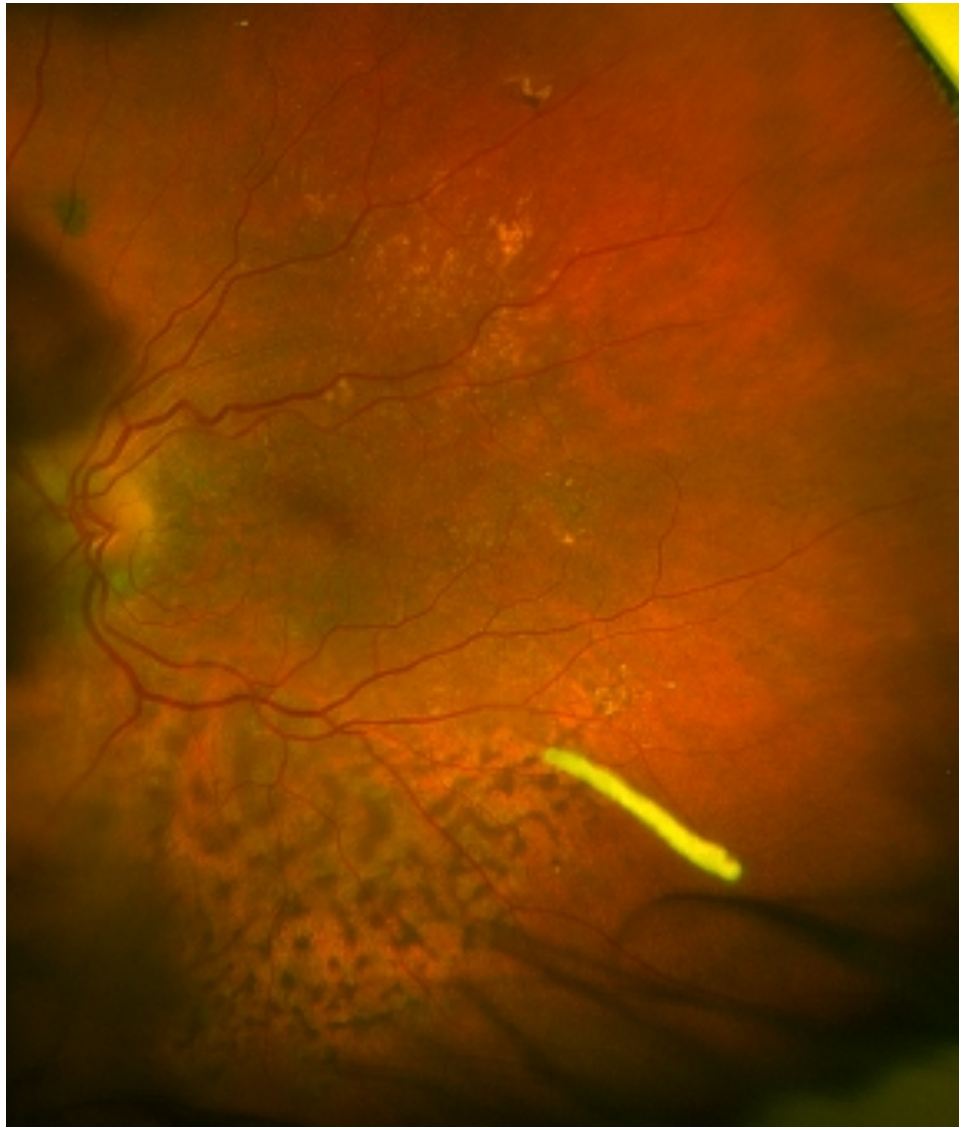


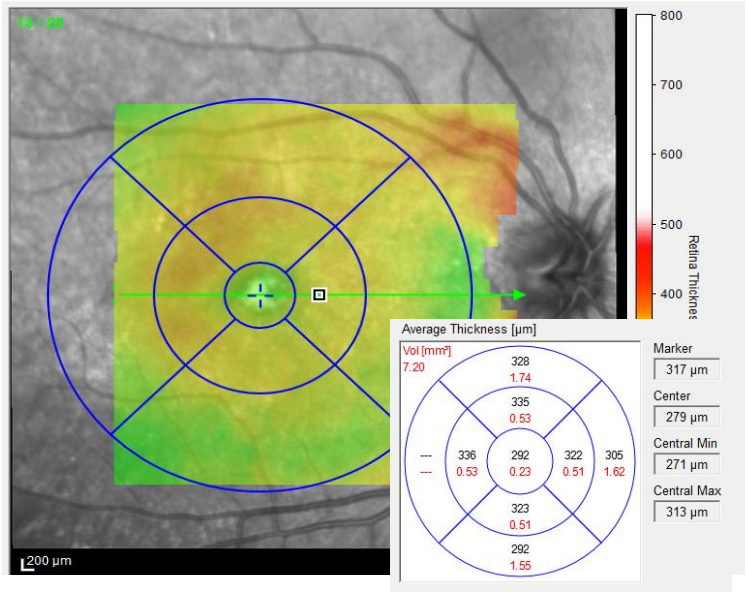
Récidive d’un carcinome du col utérin



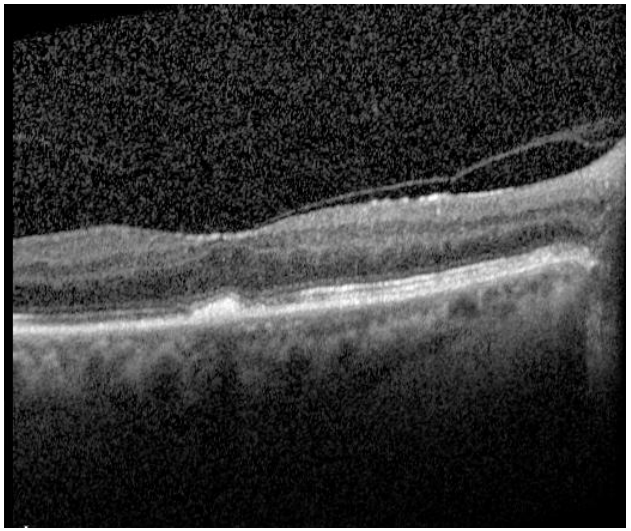
OG

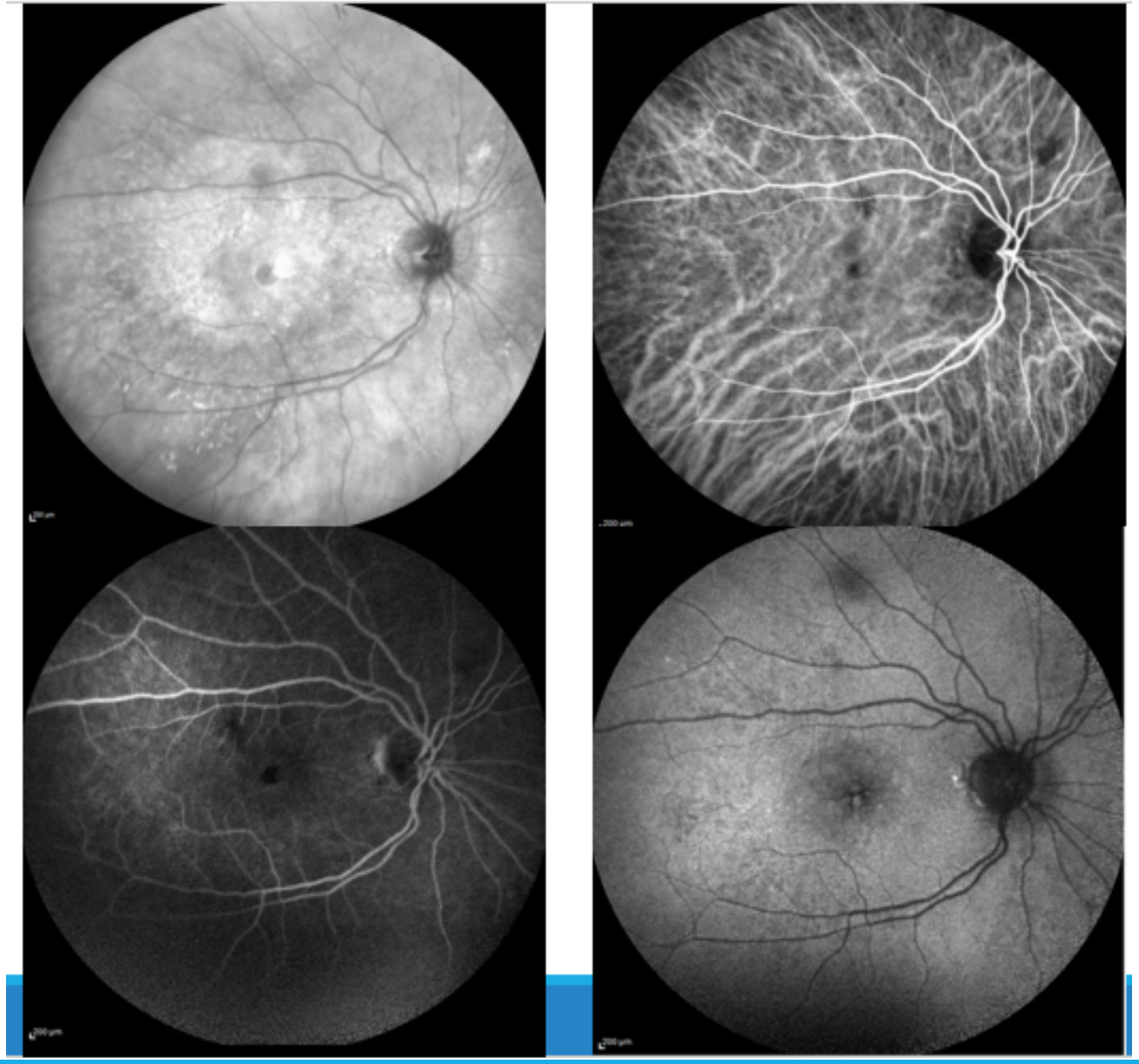


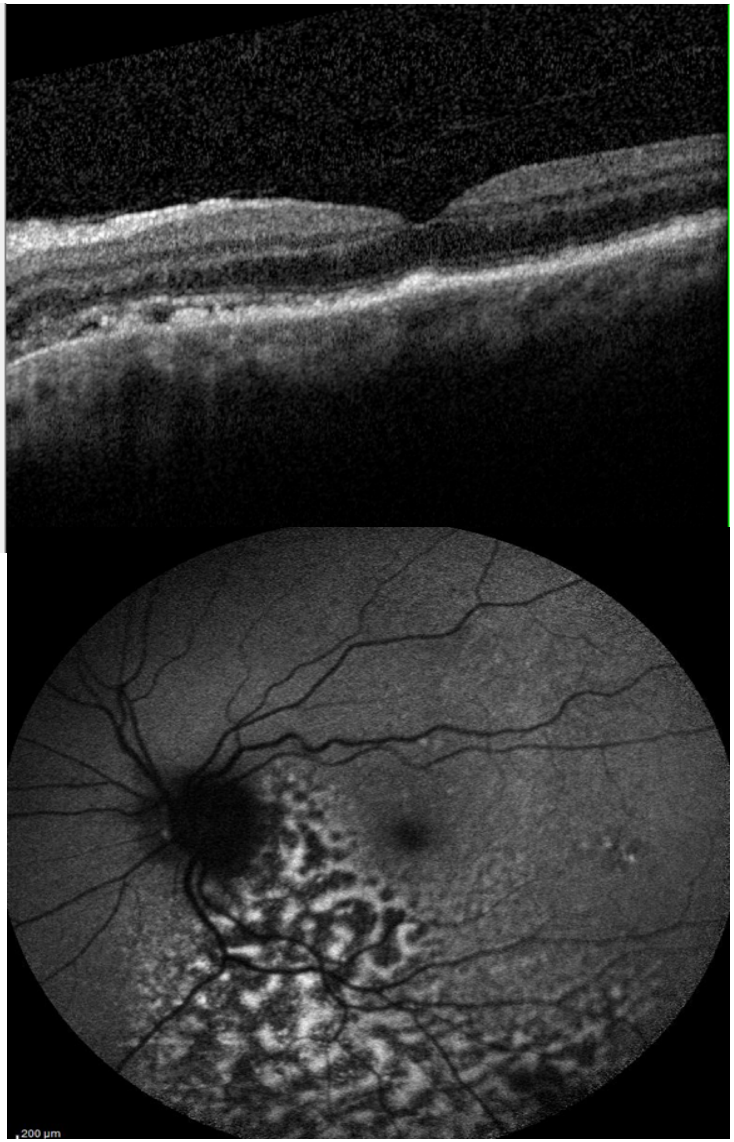


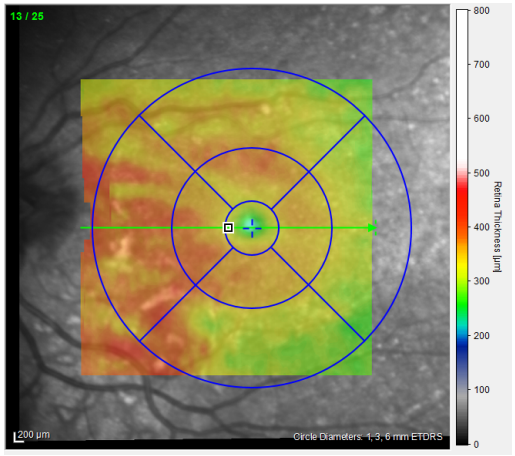

800
700
600
500
400
Retina Thickness
200 µm
Average Thickness [µm]
Vol [mm³]
7.20
328
1.74
335
0.53

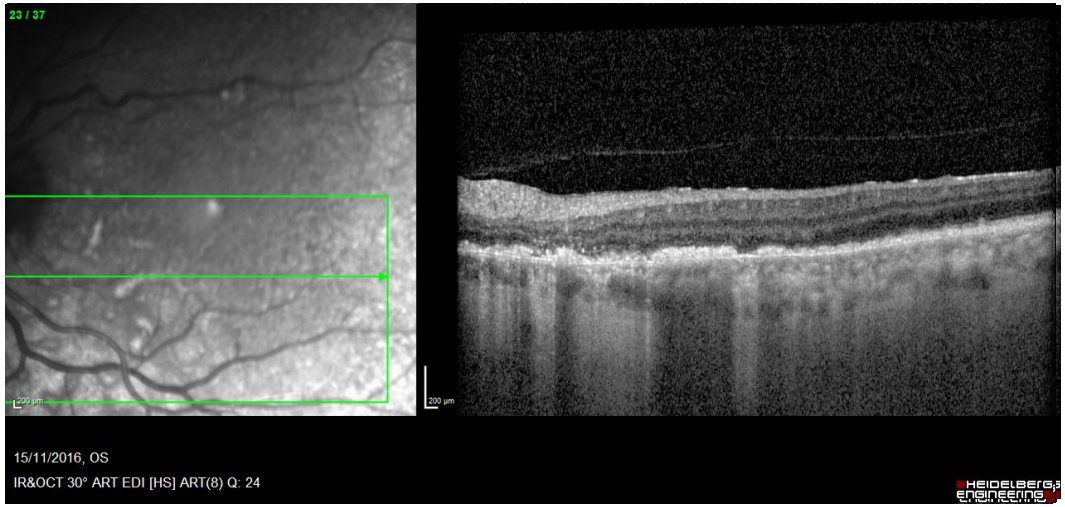
336
0.53
292
0.23
322
0.51
305
1.62
323
0.51
292
1.55
Marker
317 µm
Center
279 µm
Central Min
271 µm
Central Max
313 µm



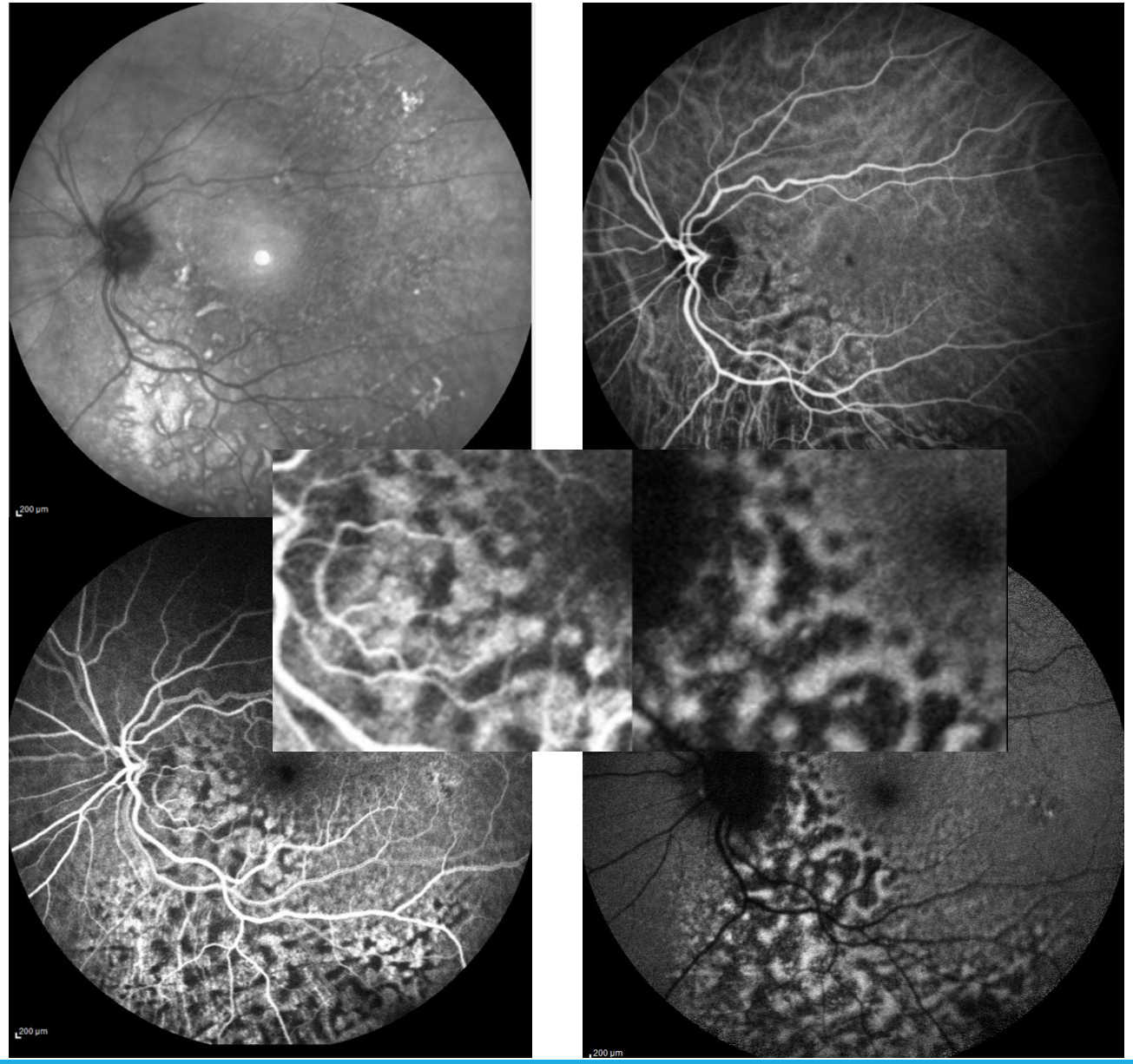



200 µm


13 / 25
800
700
600
500
400
300
200
100
0
Retina Thickness [µm]
200 µm
Circle Diameters: 1; 3; 6 mm ETDRS
Average Thickness [µm]
Vol [mm³]
9.57
332
1.76
357
0.56
376
1.99
369
0.58
304
0.24
347
0.55
305
1.62
359
0.56
323
1.71
Marker
315 µm
Center
225 µm
Central Min
220 µm
Central Max
360 µm


23 / 37
200 µm
200 µm
15/11/2016, OS
IR&OCT 30° ART EDI [HS] ART(8) Q: 24
HEIDELBERG ENGINEERING

OG



Quelles sont vos nouvelles hypothèses diagnostiques ?

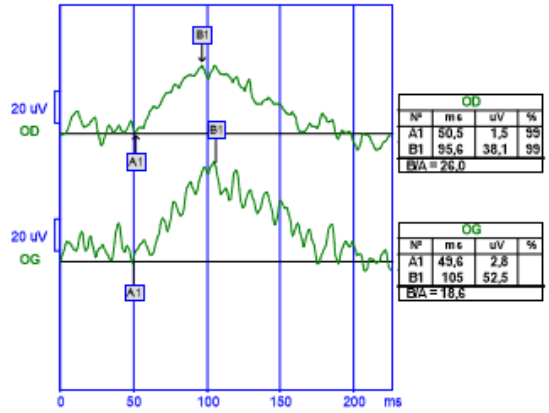
Lymphome ?

Cancer Associated Retinopathy ?

Métastase ?

EXAMEN D'ELECTROPHYSIOLOGIE VISUELLE

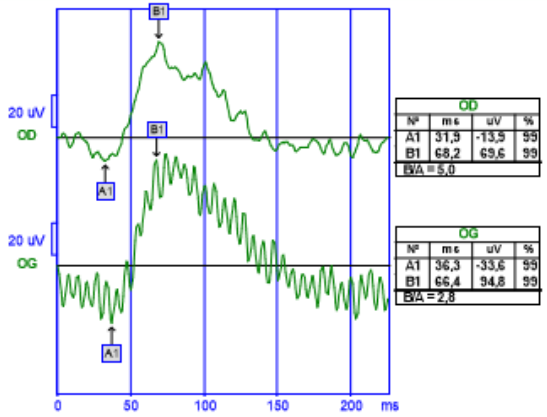
ERG des bâtonnets (-25dB) 11mn 12s Val= 8 Rej= 0
Bi stimulé



OD			
N°	ms	uV	%
A1	50,5	1,5	99
B1	55,6	38,1	99
BVA = 26,0			

OG			
N°	ms	uV	%
A1	49,6	2,8	
B1	105	52,5	
BVA = 18,6			

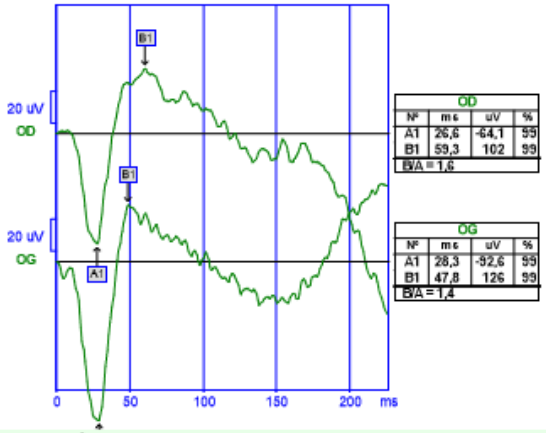
ERG mixte: (-15dB) 12mn 8s Val= 8 Rej= 0
Bi stimulé



OD			
N°	ms	uV	%
A1	31,9	-13,9	99
B1	68,2	69,6	99
BVA = 5,0			

OG			
N°	ms	uV	%
A1	36,3	-33,6	99
B1	66,4	34,8	99
BVA = 2,8			

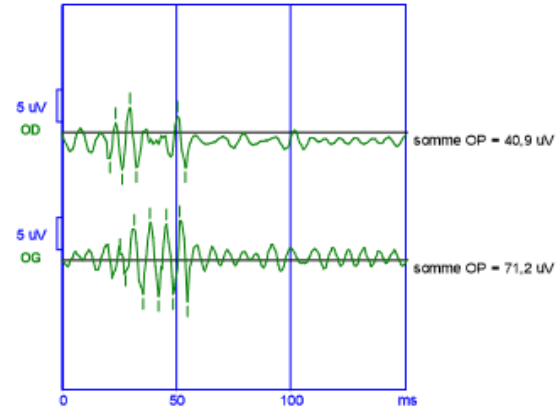
ERG mixte: cônes +bâtonnets 13mn 58s Val= 8 Rej= 0
Bi stimulé



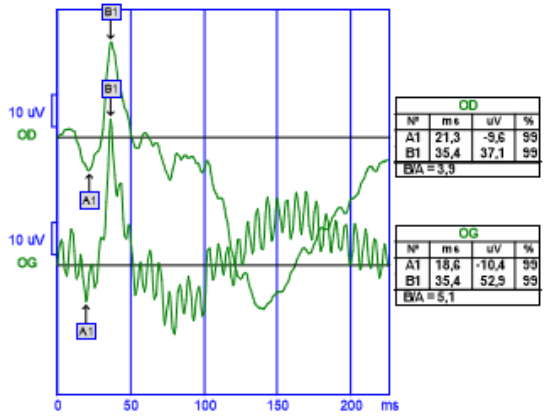
OD			
N°	ms	uV	%
A1	26,6	-64,1	99
B1	59,3	102	99
BVA = 1,6			

OG			
N°	ms	uV	%
A1	28,3	-92,6	99
B1	47,8	126	99
BVA = 1,4			

ERG potentiels oscillatoires 15mn 15s Val= 8 Rej= 0
Bi stimulé



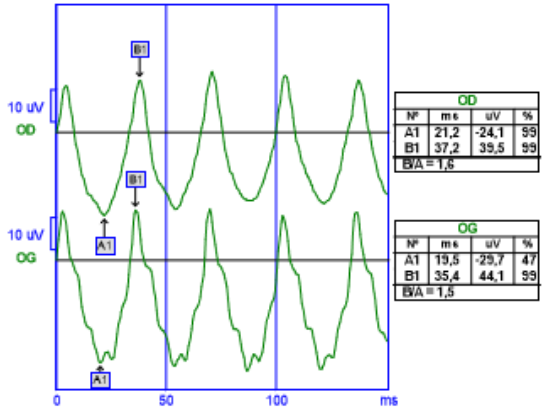
ERG des cônes: flash blanc 22mn 40s Val= 30 Rej= 0
Bi stimulé



OD			
N°	ms	uV	%
A1	21,3	-9,6	99
B1	35,4	37,1	99
BVA = 3,9			

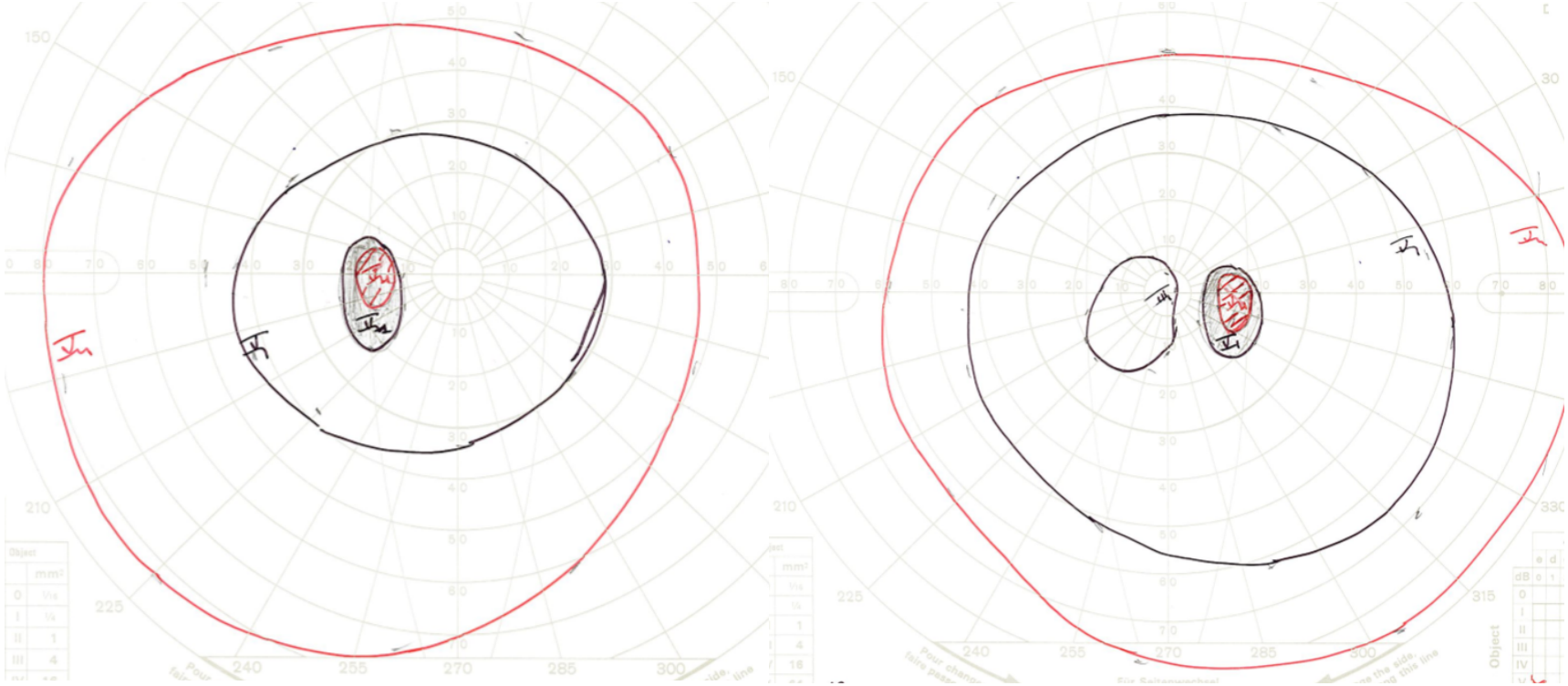
OG			
N°	ms	uV	%
A1	18,6	-10,4	99
B1	35,4	52,9	99
BVA = 5,1			

ERG des cônes: flicker 30Hz 23mn 13s Val= 30 Rej= 0
Bi stimulé



OD			
N°	ms	uV	%
A1	21,2	-24,1	99
B1	37,2	35,5	99
BVA = 1,6			

OG			
N°	ms	uV	%
A1	19,5	-25,7	47
B1	35,4	44,1	99
BVA = 1,5			

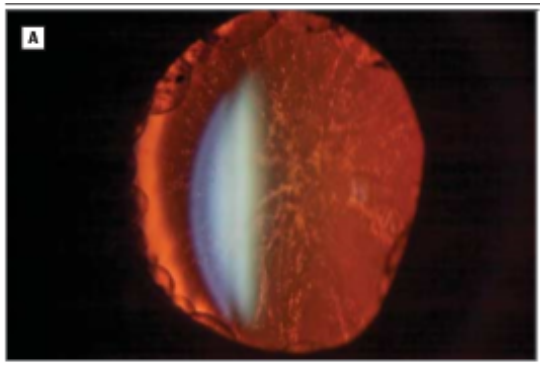


- Echographie B : Pas de masse choroïdienne, augmentation épaisseur choroïdienne

DIAGNOSTIC ?

Bilateral Diffuse Uveal Melanocytic Proliferation « BDUMP »

- Syndrome paranéoplasique pouvant précéder le cancer de 3-12 mois
- BAV bilatérale rapidement progressive
- Prolifération de mélanocytes au niveau de la choroïde ; provoquant une déstructuration de l'EP et de la rétine externe
- Cataracte sous capsulaire postérieure d'évolution rapide
- Au FO : lésions tachetées, multiples, orange, polygonale : « *giraffe pattern* »

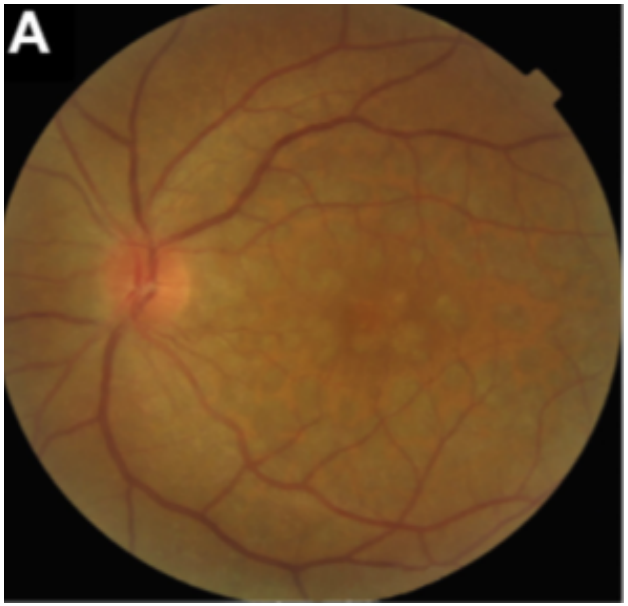


naevi

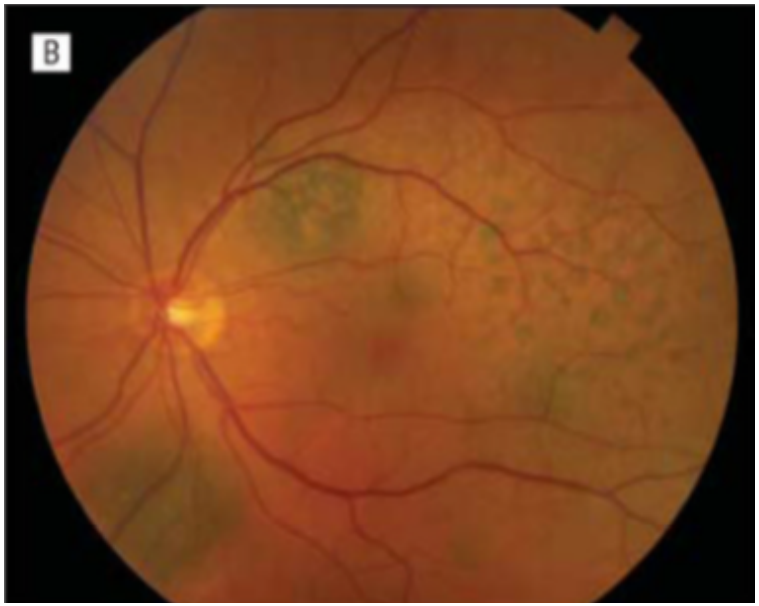
DSR

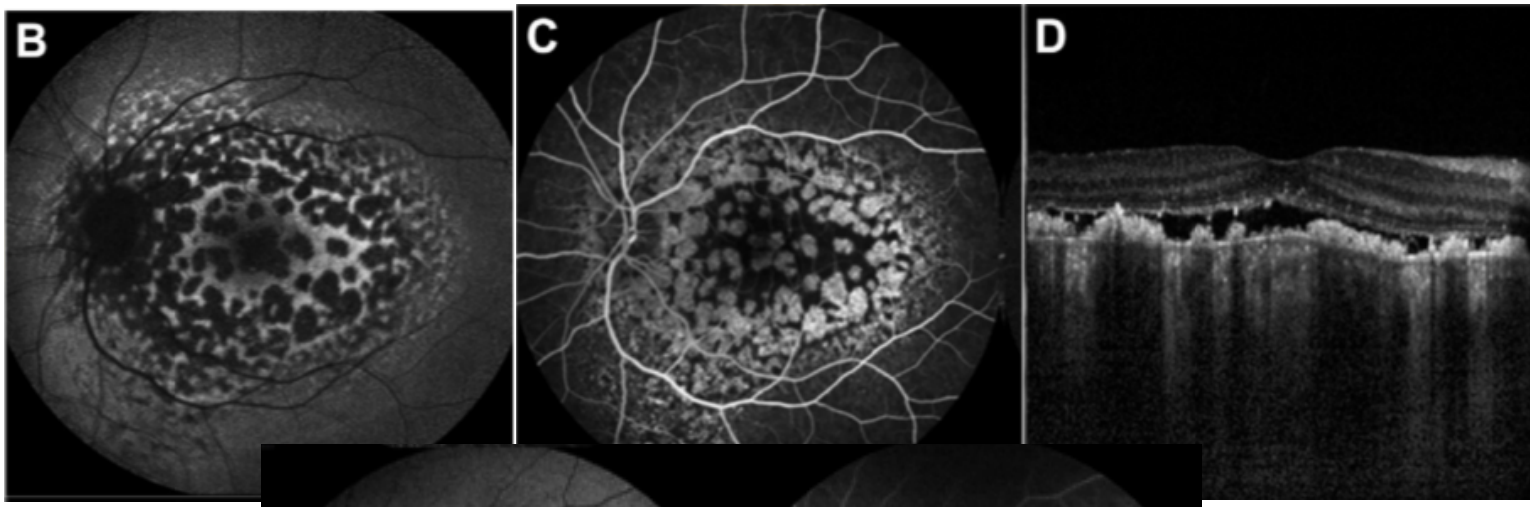
JAMA Ophthalmol, 2014

Ophthalmology, 2016



Arch ophthalmol,2011

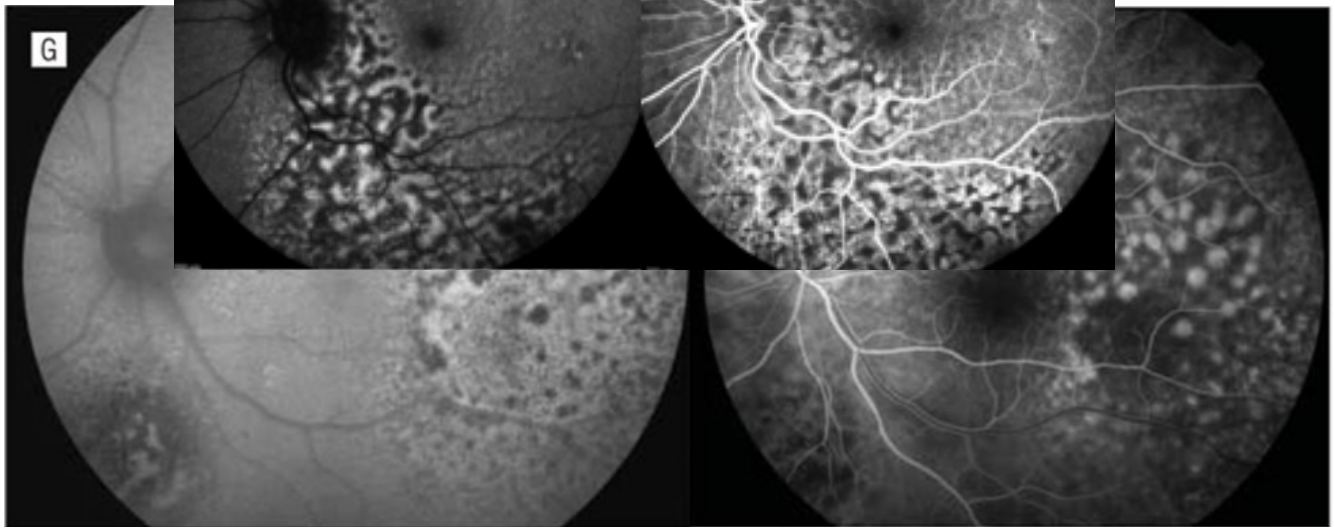




Ophthalmology,2016

Hypoau n angiographie

OCT: atteinte de l'EP : zo n + atteinte de la rétine externe



Arch Ophtalmol, 2011

Physiopathologie : Inconnue !

A FACTOR FOUND IN THE IgG FRACTION OF SERUM OF PATIENTS WITH PARANEOPLASTIC BILATERAL DIFFUSE UVEAL MELANOCYTIC PROLIFERATION CAUSES PROLIFERATION OF CULTURED HUMAN MELANOCYTES

RETINA 32:1959–1966, 2012

Thérapeutique :

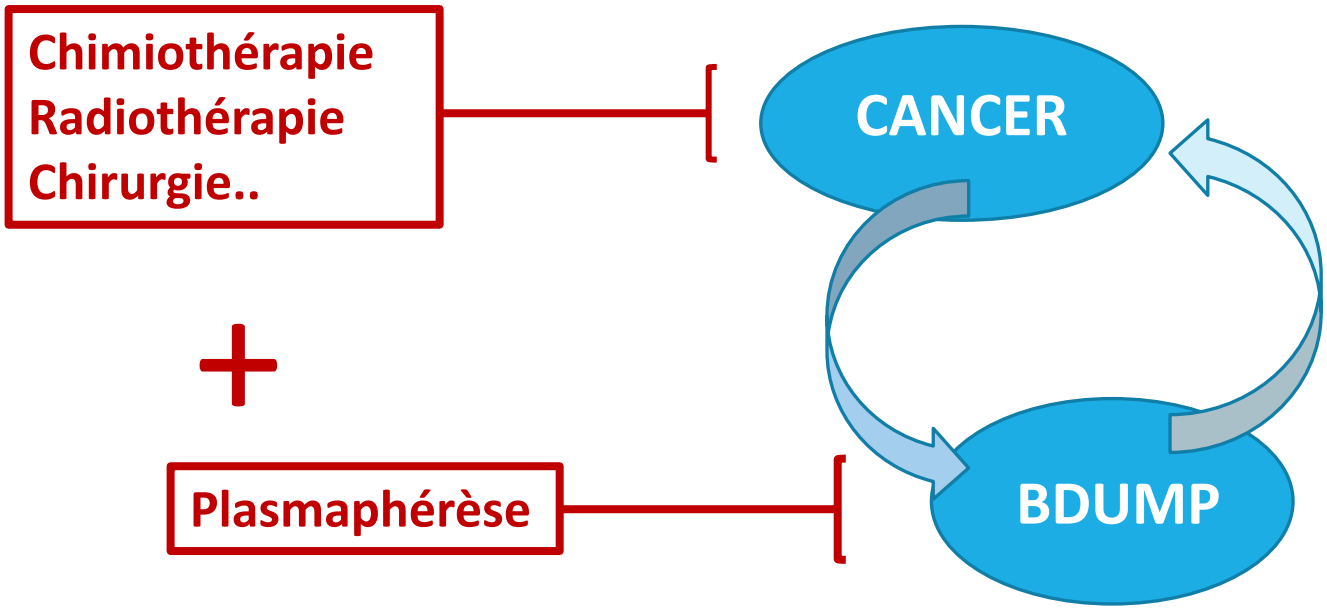
Early diagnosis and successful treatment of paraneoplastic melanocytic proliferation

Br J Ophthalmol 2015

Bilateral diffuse uveal melanocytic proliferation: Case report and literature review

Kristian Klemp,[1] Jens Folke Kiilgaard,[1] Steffen Heegaard,[1,2] Tove Nørgaard,[3] Mette Klarskov Andersen[4] and Jan Ulrik Prause[2]

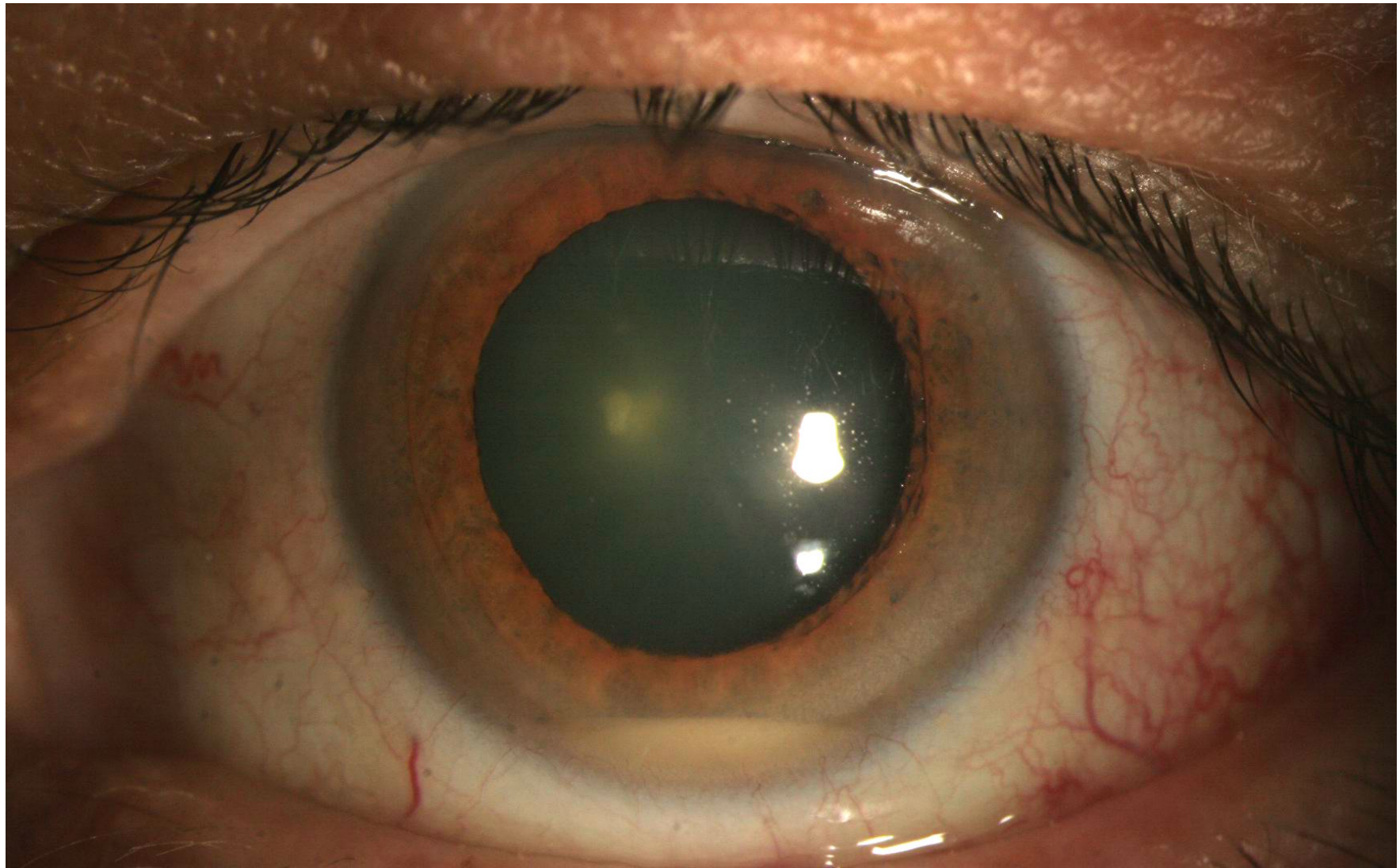
Bilateral diffuse uveal melanocytic proliferation (BDUMP) is a rare paraneoplastic intraocular disease that causes progressive visual loss in patients driven by an IgG factor associated with an underlying malignancy. Characteristic ocular findings include exudative retinal detachment, rapid cataract formation and uveal melanocytic tumours. The awareness and documentation of BDUMP has increased during the past decade, and the increasing amount of data collected demonstrates the effect of treatment with plasmapheresis and the value of diagnostic tools in BDUMP such as genetic and immunologic investigations. The literature of BDUMP has not been reviewed since 2003, and there is a growing need for an updated review on diagnosis and management of BDUMP. We review the literature and report a case of BDUMP with a white ciliary body tumour, iris rubeosis, increased iris pigmentation and cataract.

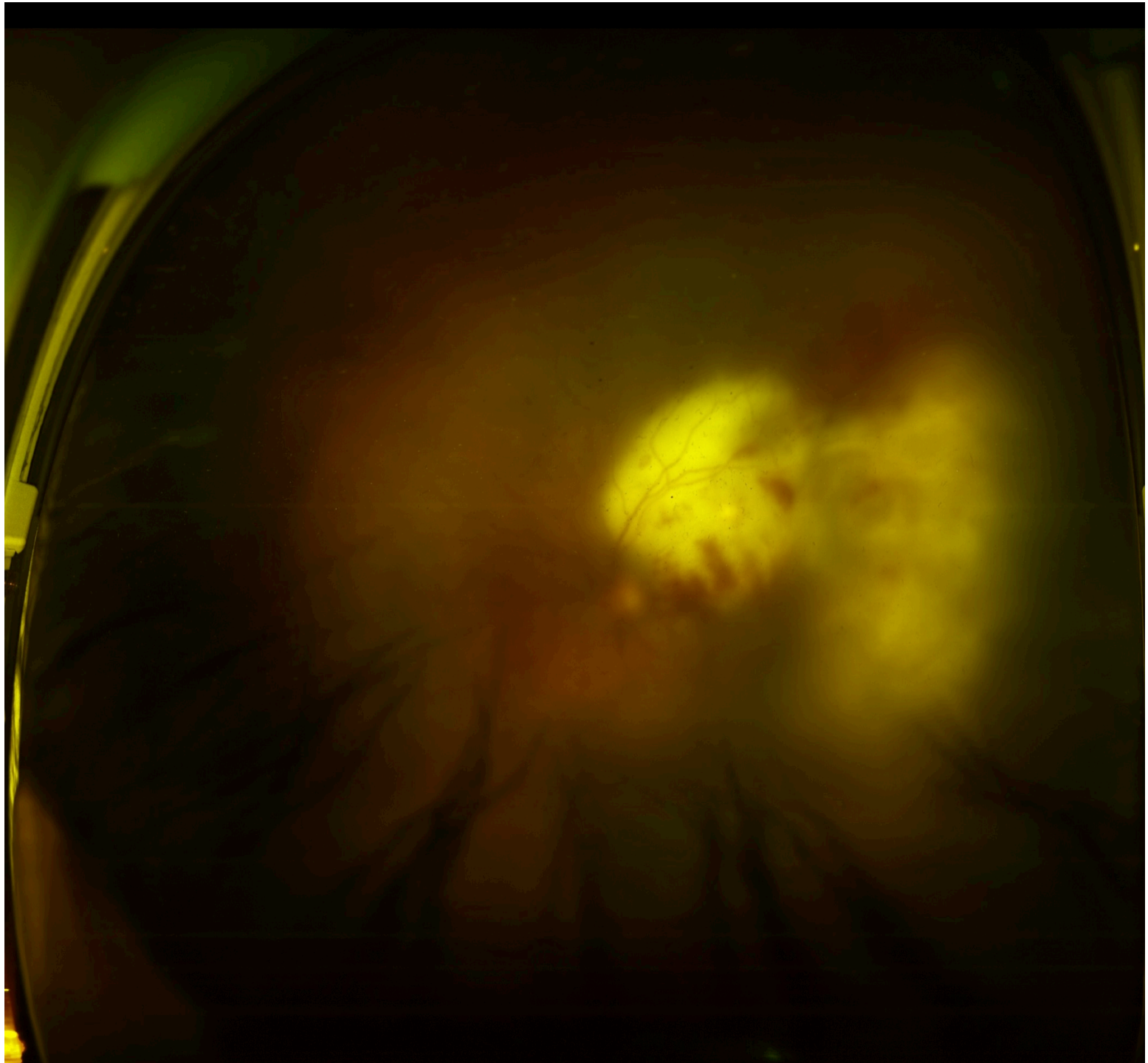


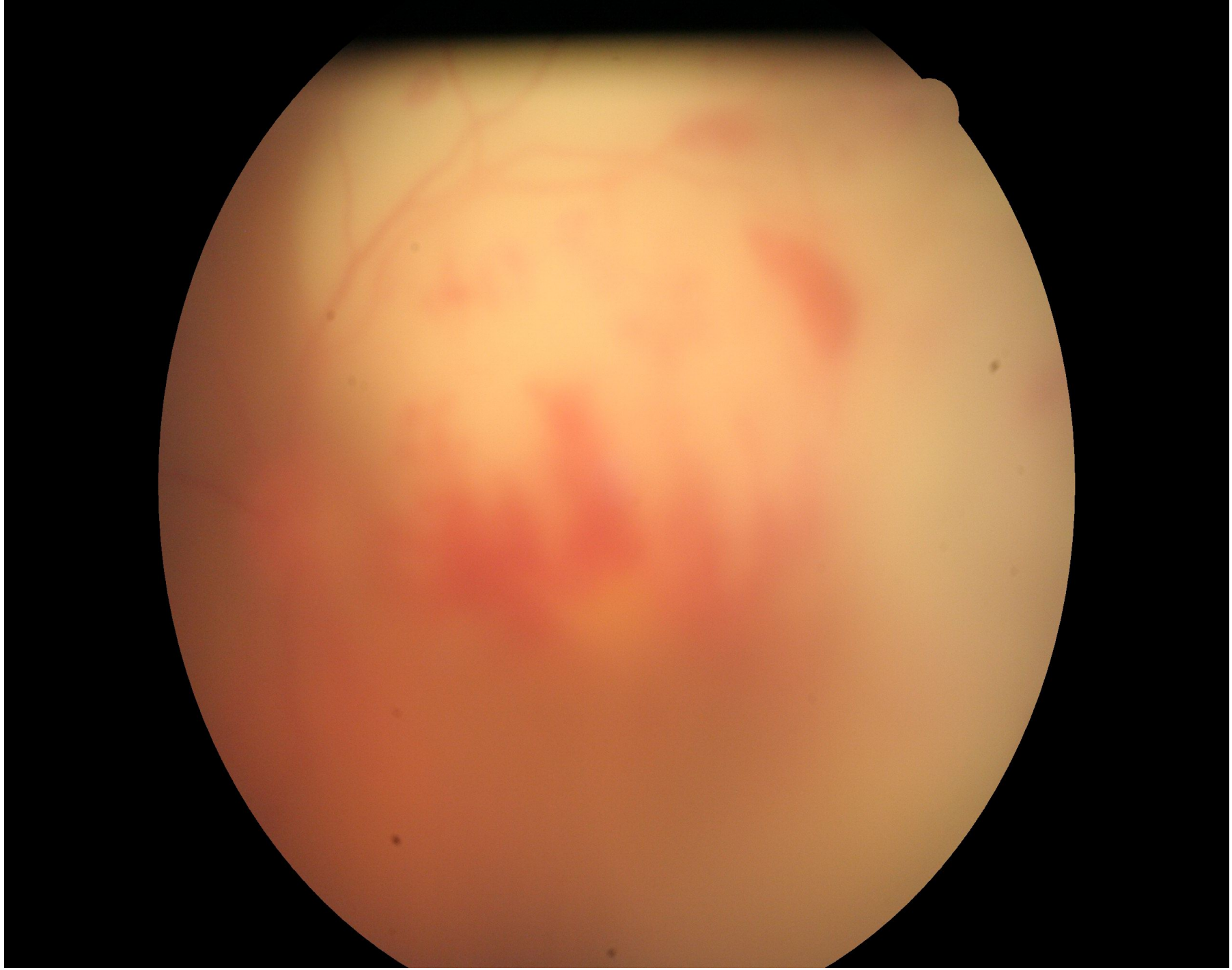
Diagnostic

- Mauvais pronostic, médiane de SURVIE 15,6 MOIS

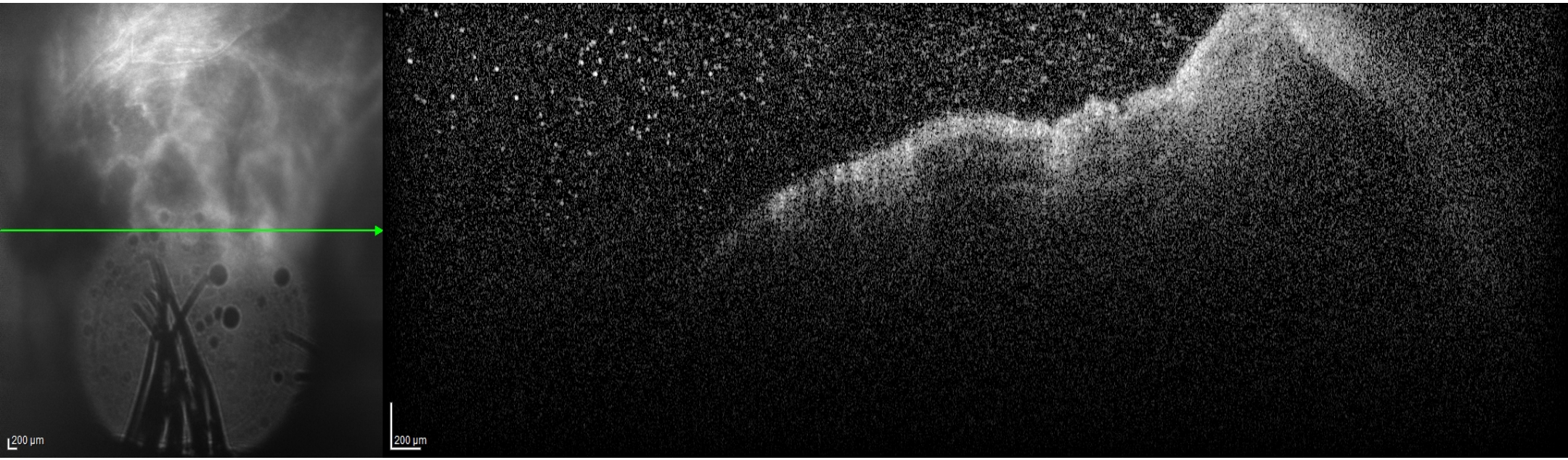
Monsieur C. 54 ans
Baisse d’acuité visuelle installée en quelques jours du côté gauche
AV OD 1,0 OG VBLM







OCT



Mr C., 54 ans

- **Contexte fébrile**
  - **Importante altération de l'état général**
  - **Syndrome inflammatoire biologique**

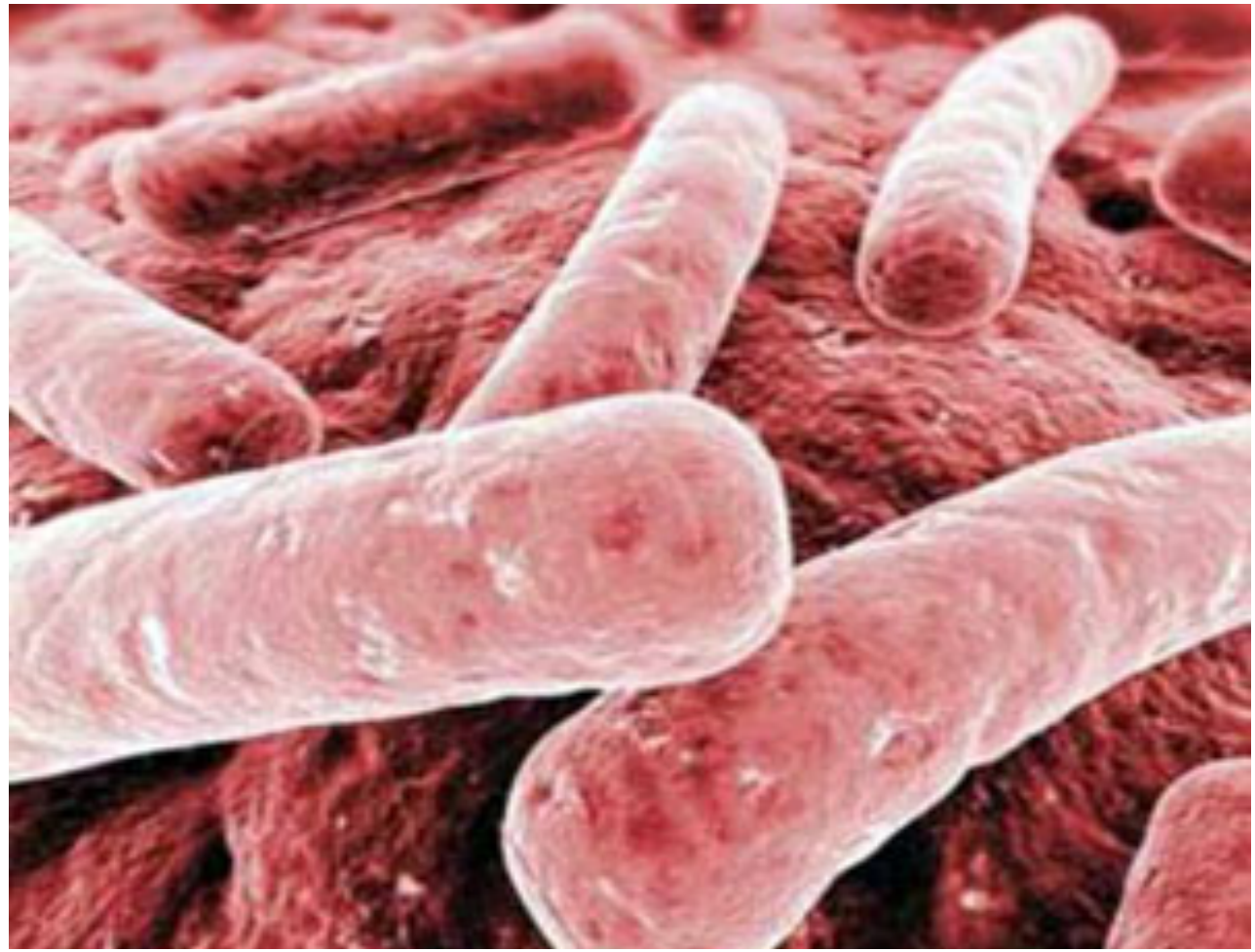
- Antécédents
  - Cardiopathie ischémique, pose de *stents*
  - Hypertension artérielle
  - Diabète de type 2

- Mode de vie
  - Patient d'origine Française
  - Gendarme, père de 2 enfants en bonne santé
  - Pas de voyage à l'étranger

Hypothèses diagnostiques ?

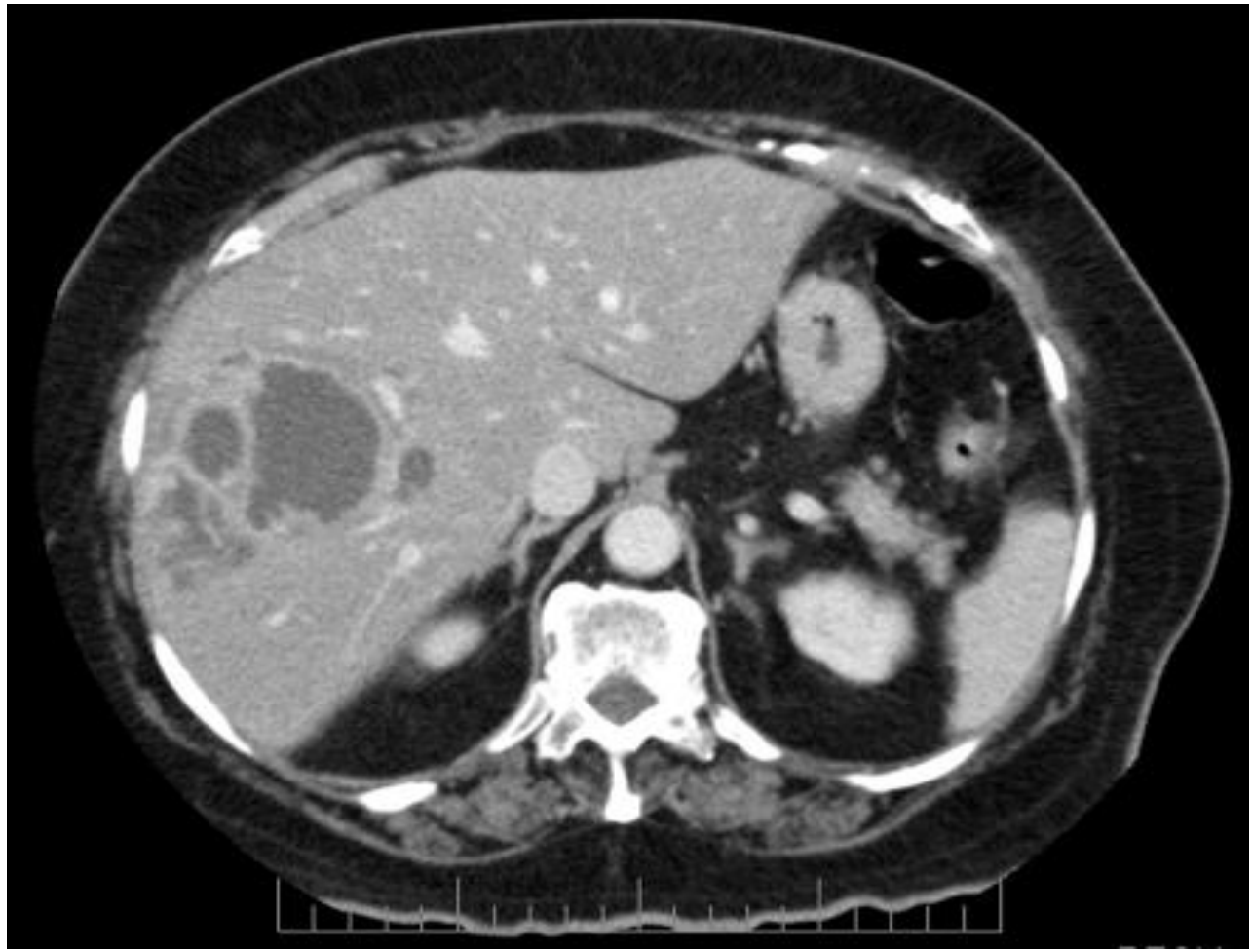
Explorations complémentaires

- **ECBU et Hémocultures positifs à Klebsiella pneumoniae**



Explorations complémentaires

- **ECBU et hémocultures positifs à Klebsiella pneumoniae**
- **TDM thoraco-abdomino-pelvien : multiples abcès hépatiques**



L'endophtalmie à Klebsielle: vers une pandémie ?

	Durée	Lieu	Nb yeux	Klebsielles	Abcès hépatique	Diabète
Lim et al.	2005-2011	Corée	57	43%	39,5%	46,5%
Lee et al.	1996-2010	Corée	97	48,4%	25%	42,5%
Chung et al.	1993-2006	Corée	18	38,9%	/	50%
Ang et al.	1986-2007	Singapore	113	54%	77,5%	55,7%
Chen et al.	1992-2002	Taiwan	74	61%	/	/
Jackson et al.	1984-2001	Angleterre	19	5%	100%	100%
Okada et al.	1980-1990	USA	28	3,6%	/	/

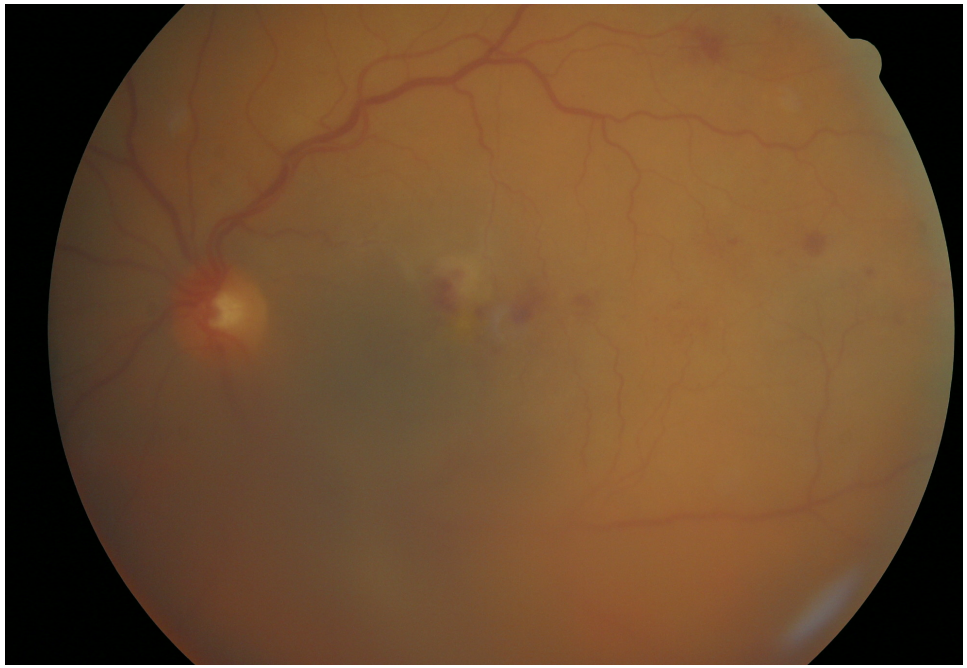
Klebsiella pneumoniae hypervirulente

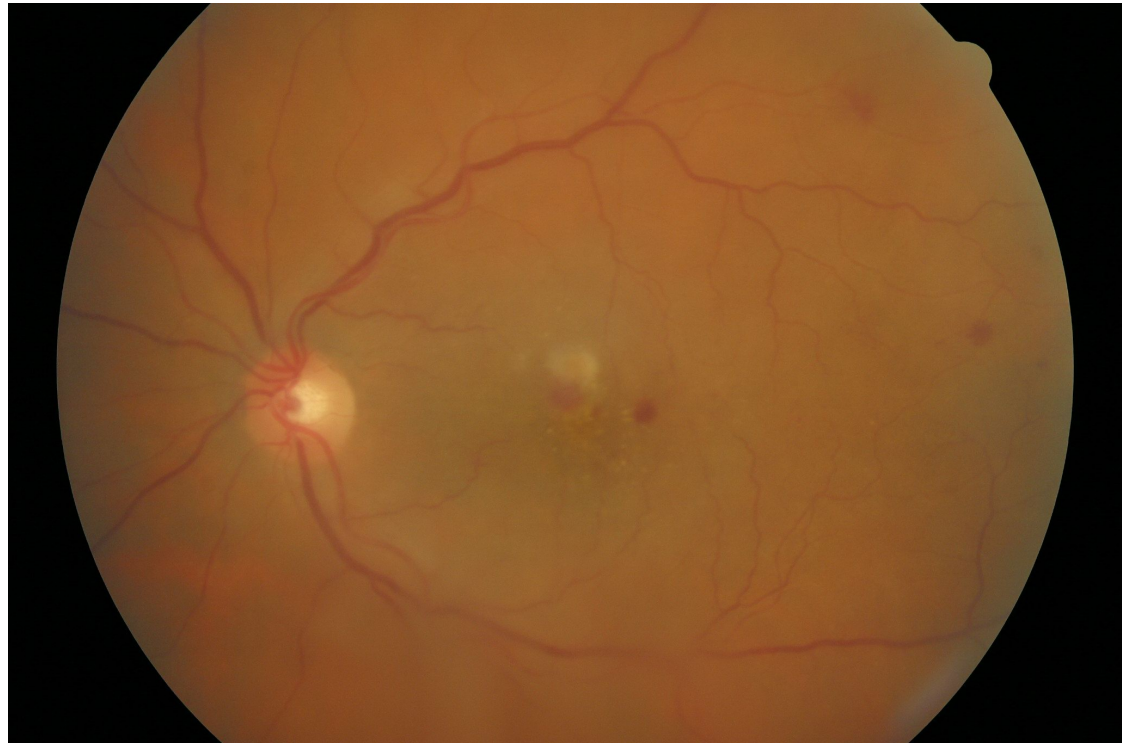
Fréquent en Asie du Sud-Est

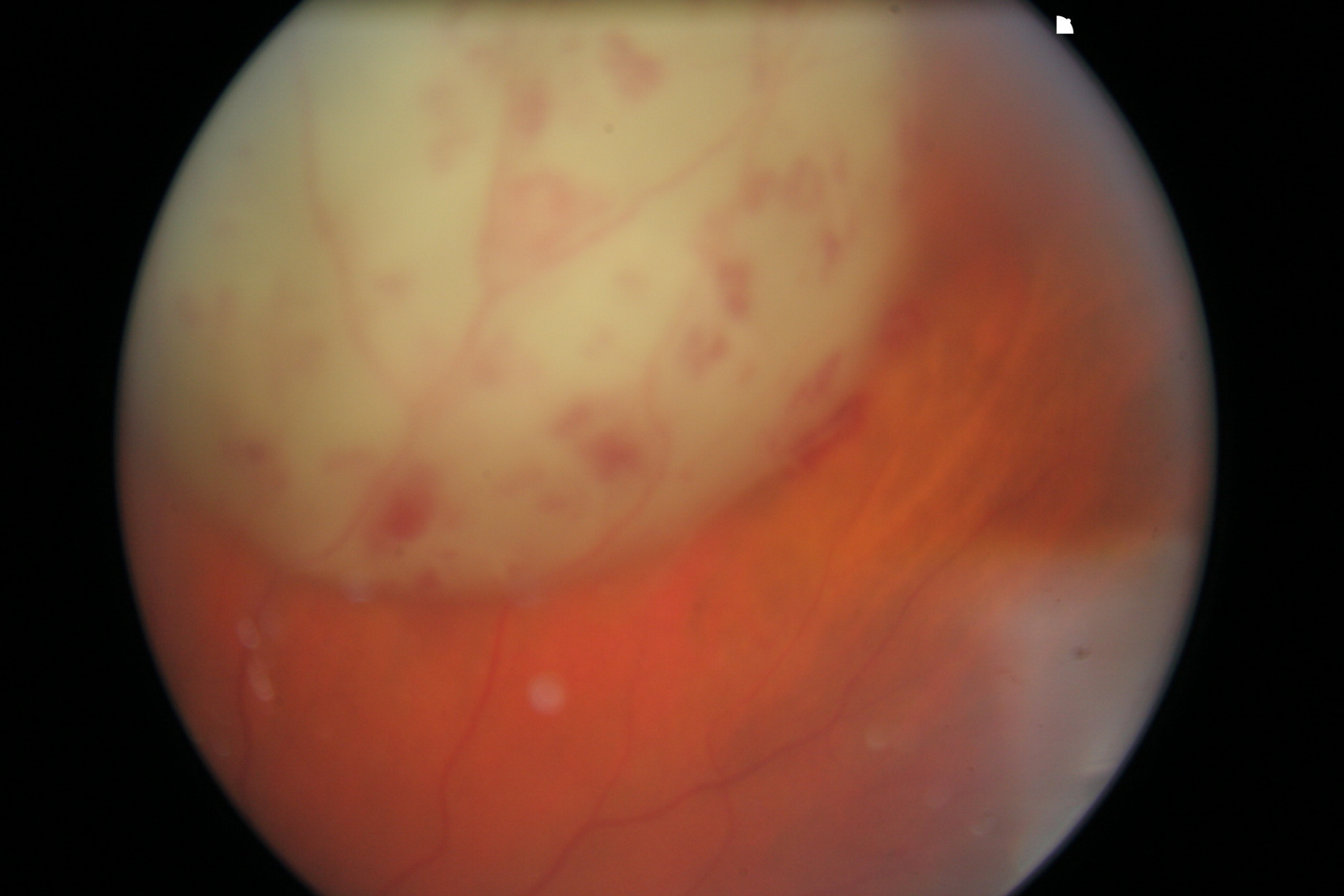
Rôle suspecté des pousses de soja

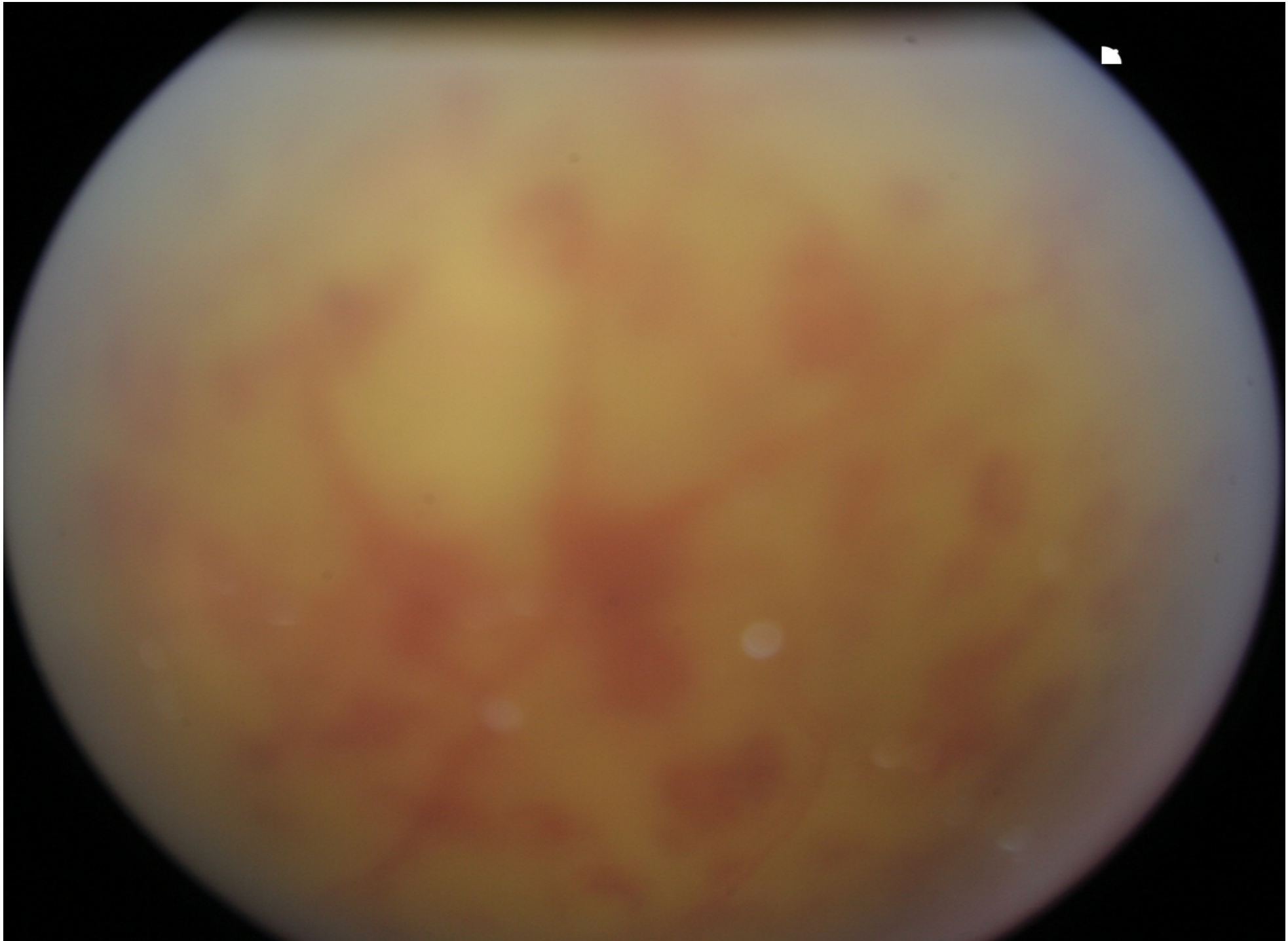
Quelques autres cas..

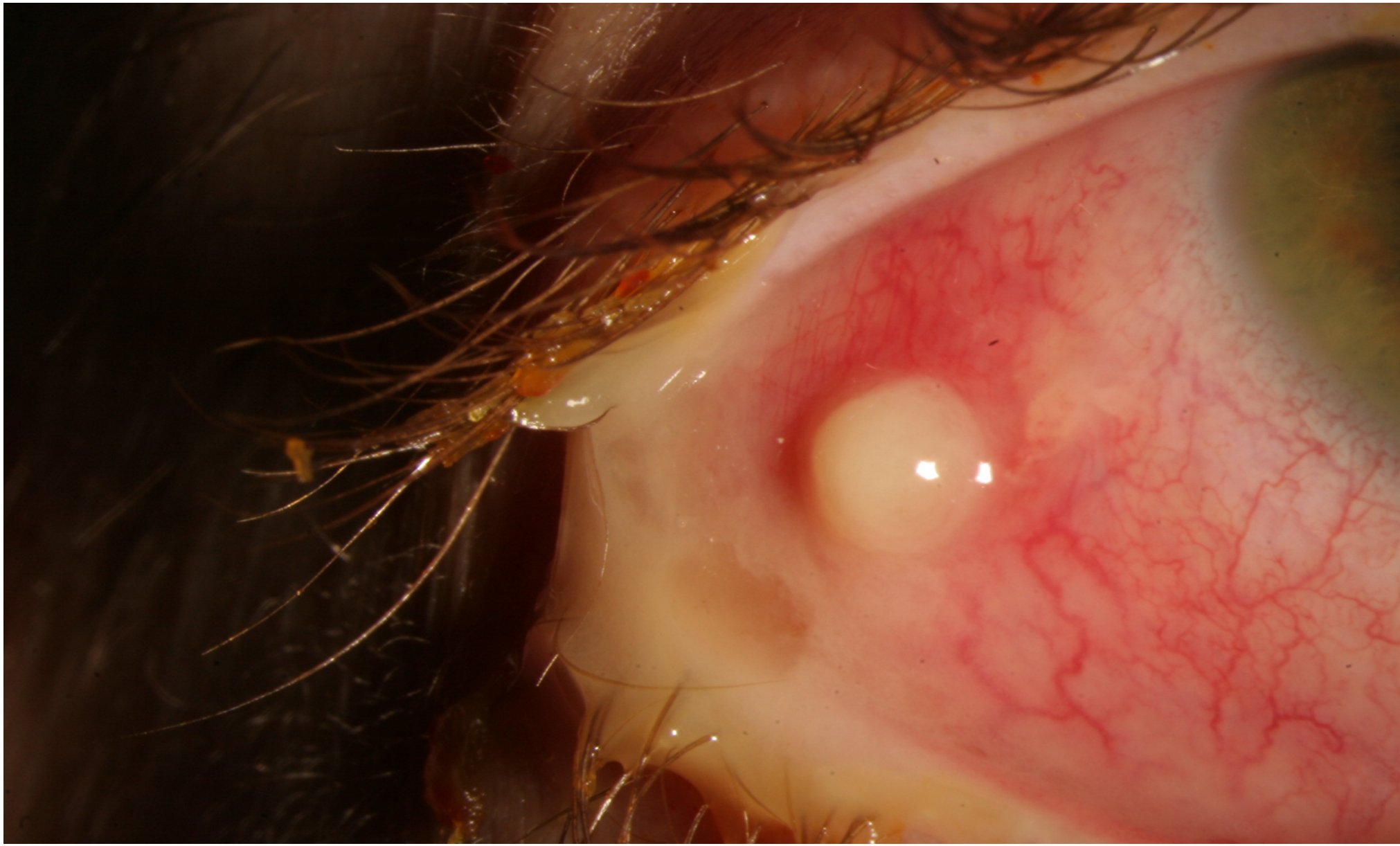
Endocardite Infectieuse











Mme J…, 28 ans

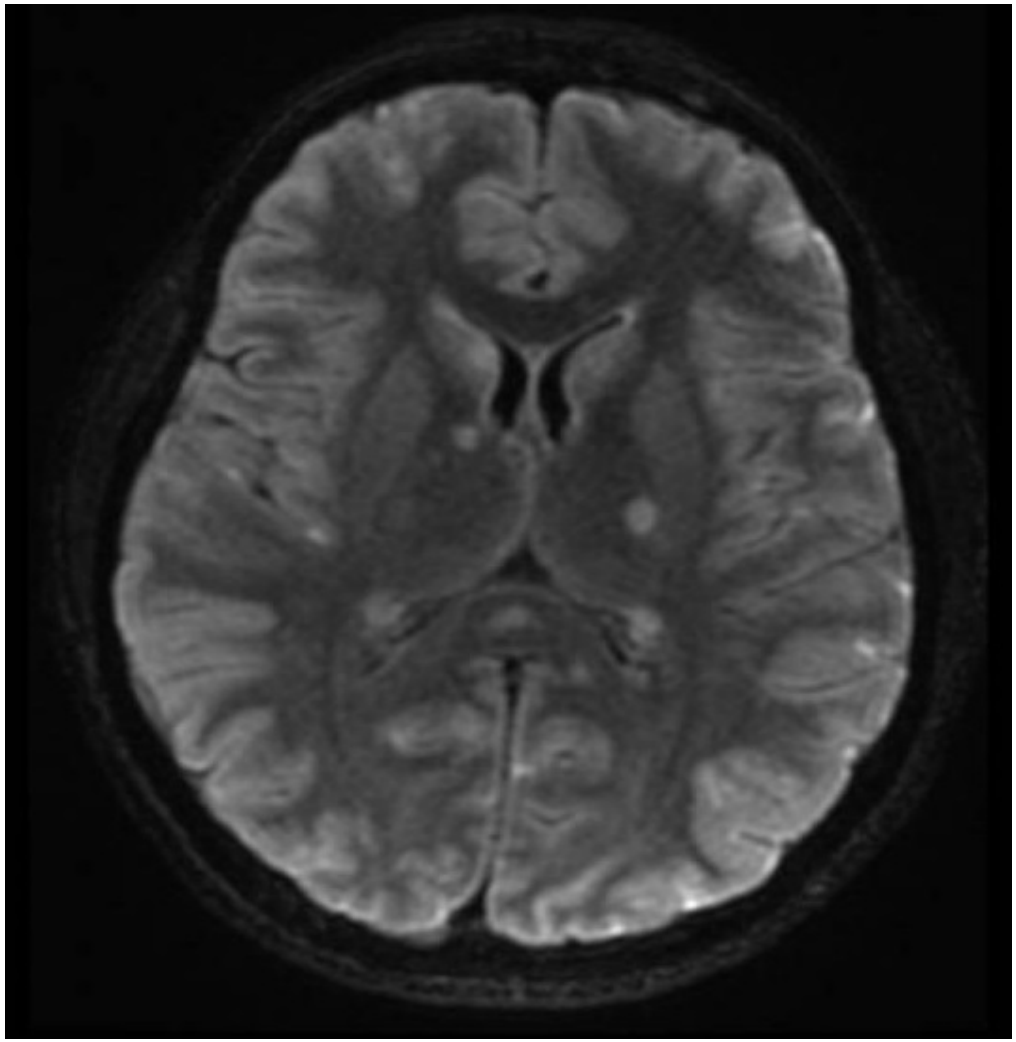
- **Antécédents**
  - **Migraines ophtalmiques**
  - **G2P1 IVG**
  - **ATCD familial de SPA**
- **Mode de vie**
  - **Educatrice spécialisée**
  - **Vit avec son compagnon et son fils de 2 ans**
  - **Tabagisme actif (<10 PA)**
- **Traitements**
  - **Contraception oestro-progestative**

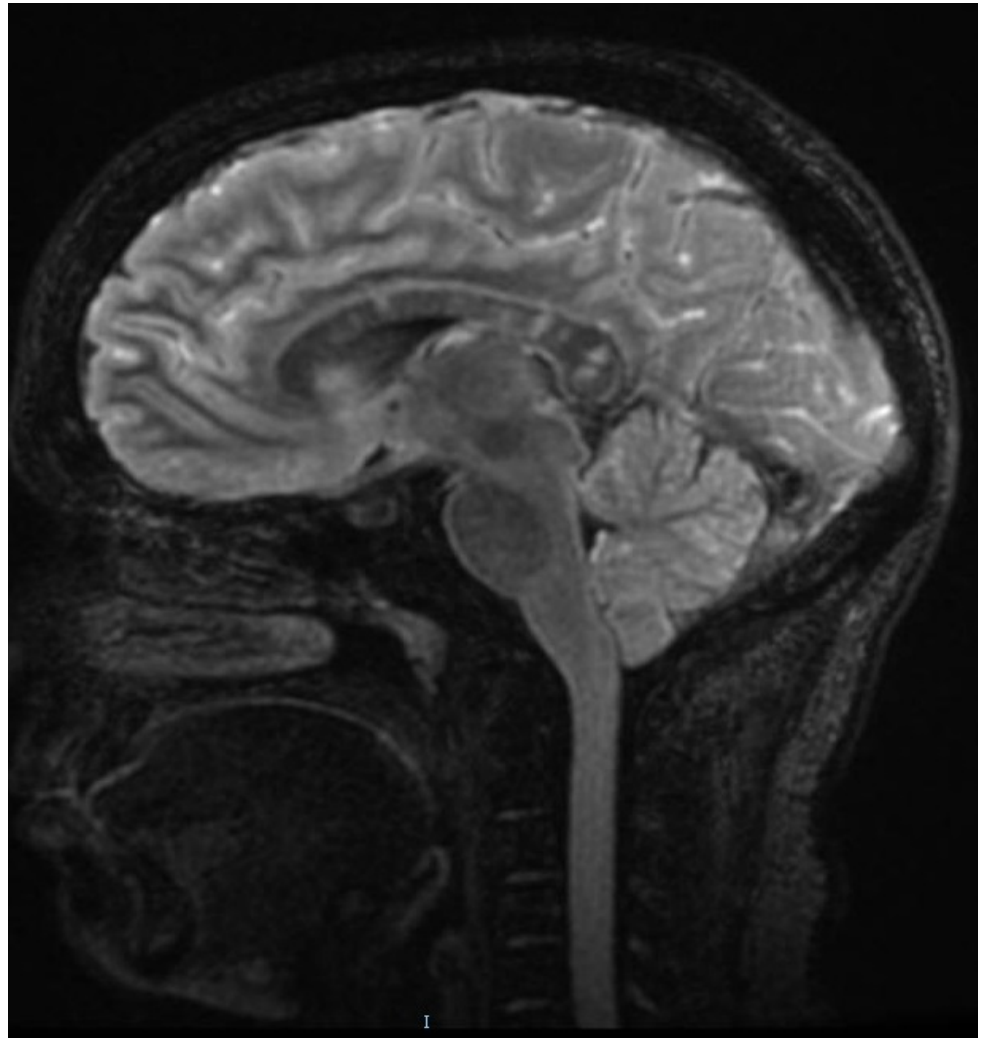
Histoire de la maladie : Mars 2016

- **Vertiges**
- **Vomissements**
- **Troubles de la marche et de l'équilibre**
- **Nystagmus, Romberg**
- **ROT vifs, signe de Romberg**
- **Flou visuel**

- **IRM cérébrale : lésions d'aggravation rapide**

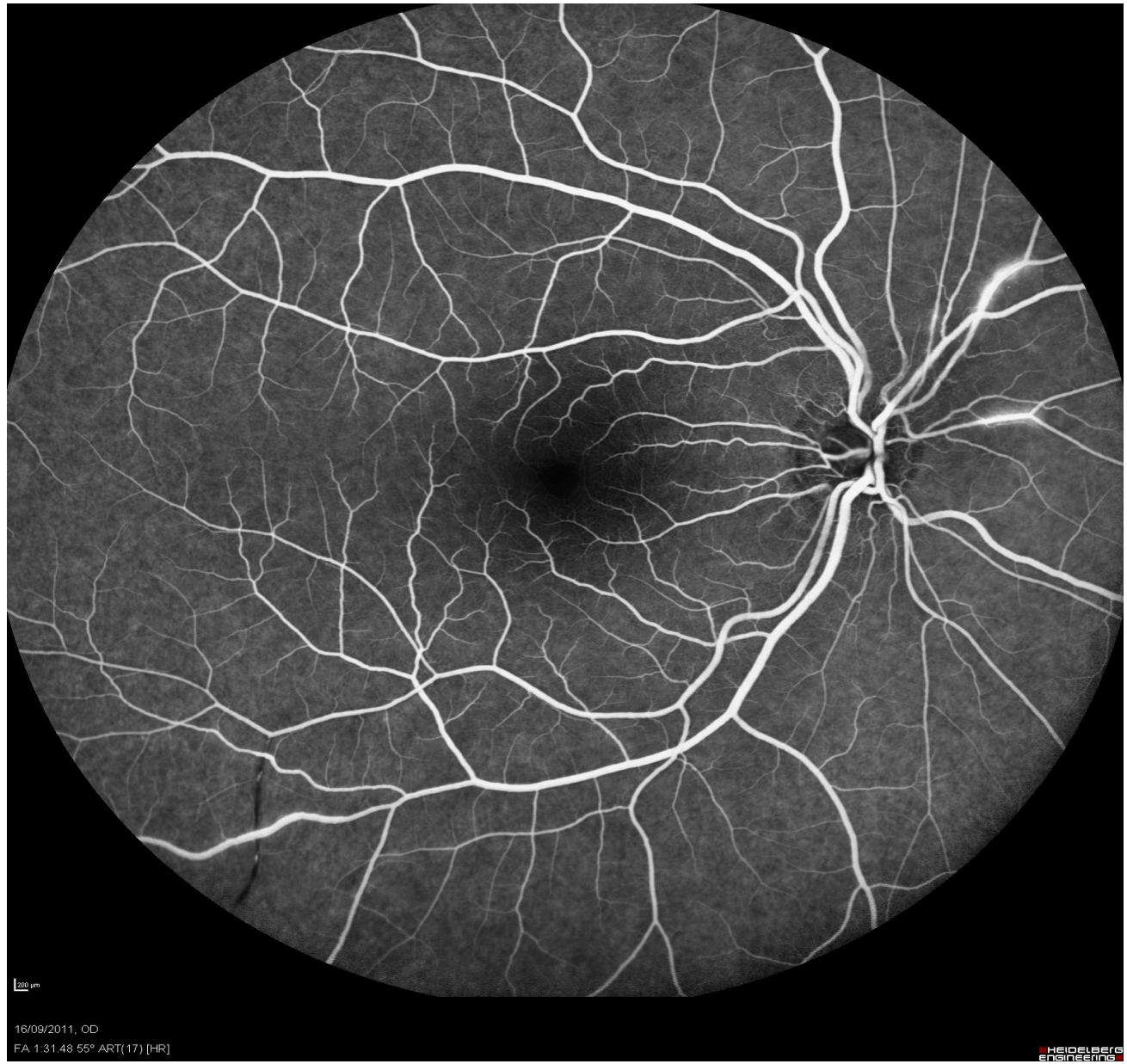
IRM cérébrale

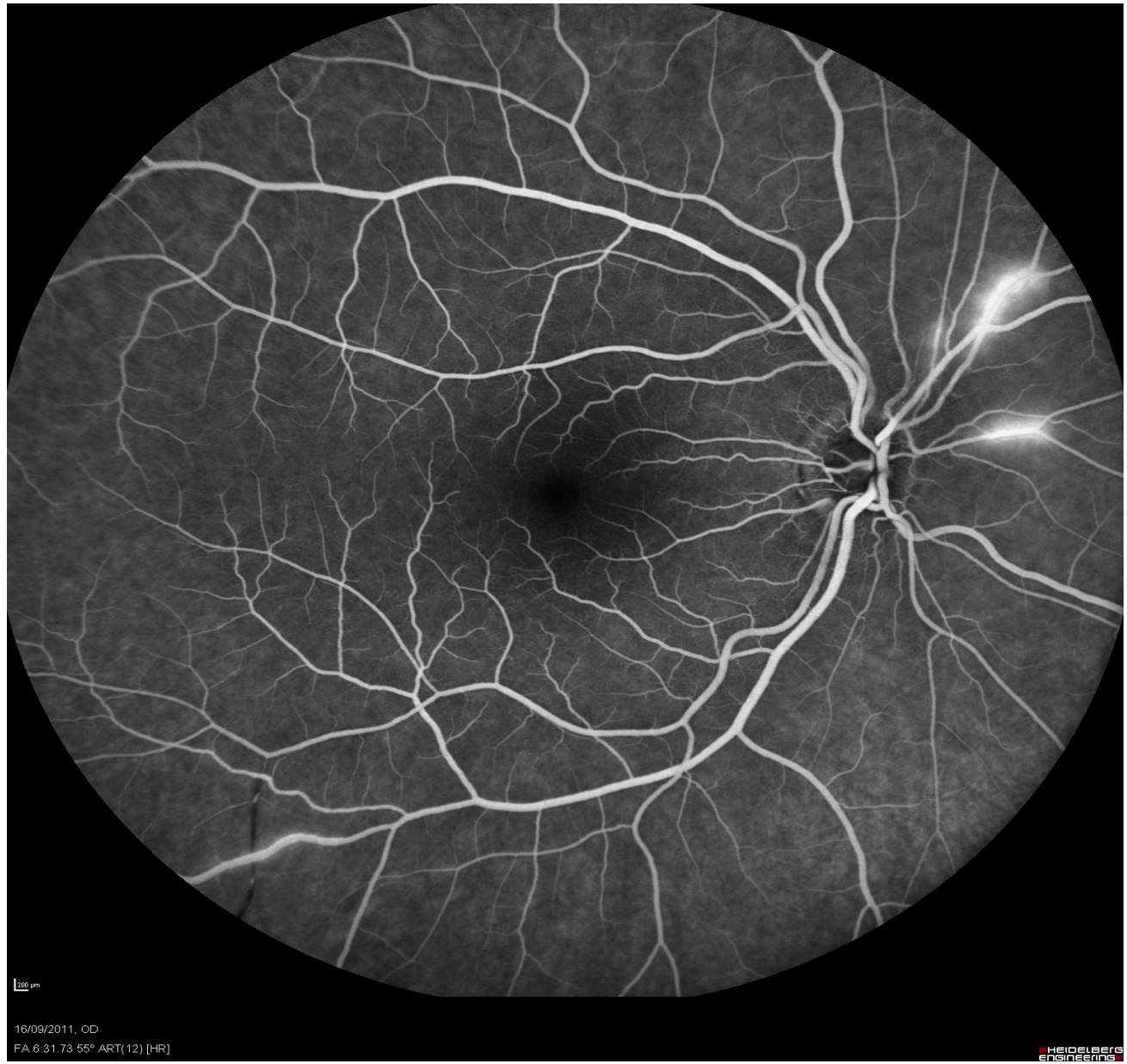




Explorations

- **ANCA, FAN neg**
- **CRP normale**
- **ECA normale, BGSA négative**
- **TDM TAP normal**
- **EEG : encéphalopathie légère**
- **Ponction Lombaire**
  - **9 éléments/mm3 dont 98% de Lympho, 3 GR/mm3**
  - **Culture négative**


200 µm
16/09/2011, OD
FA 1:31.48 55° ART(17) [HR]
HEIDELBERG ENGINEERING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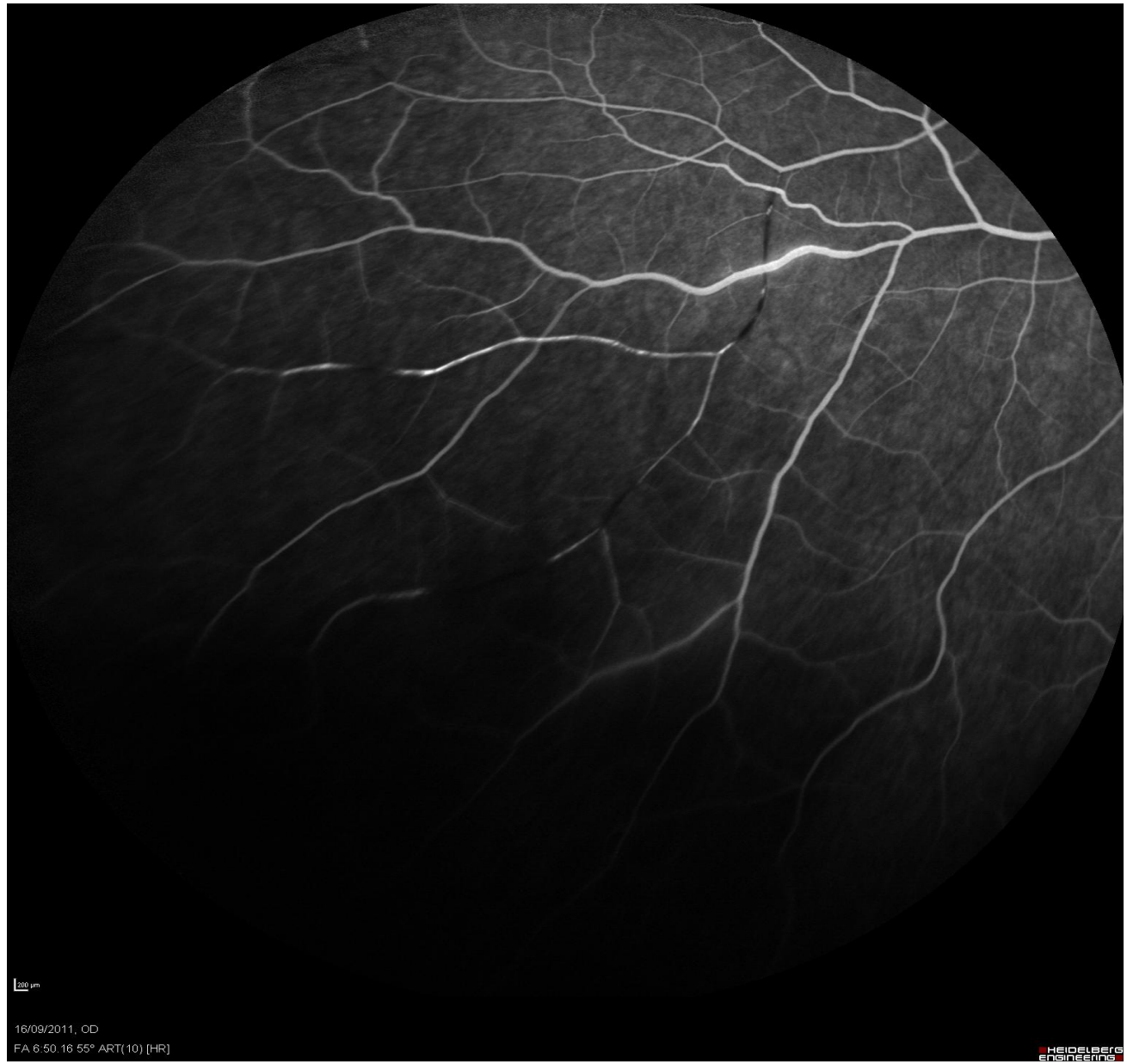
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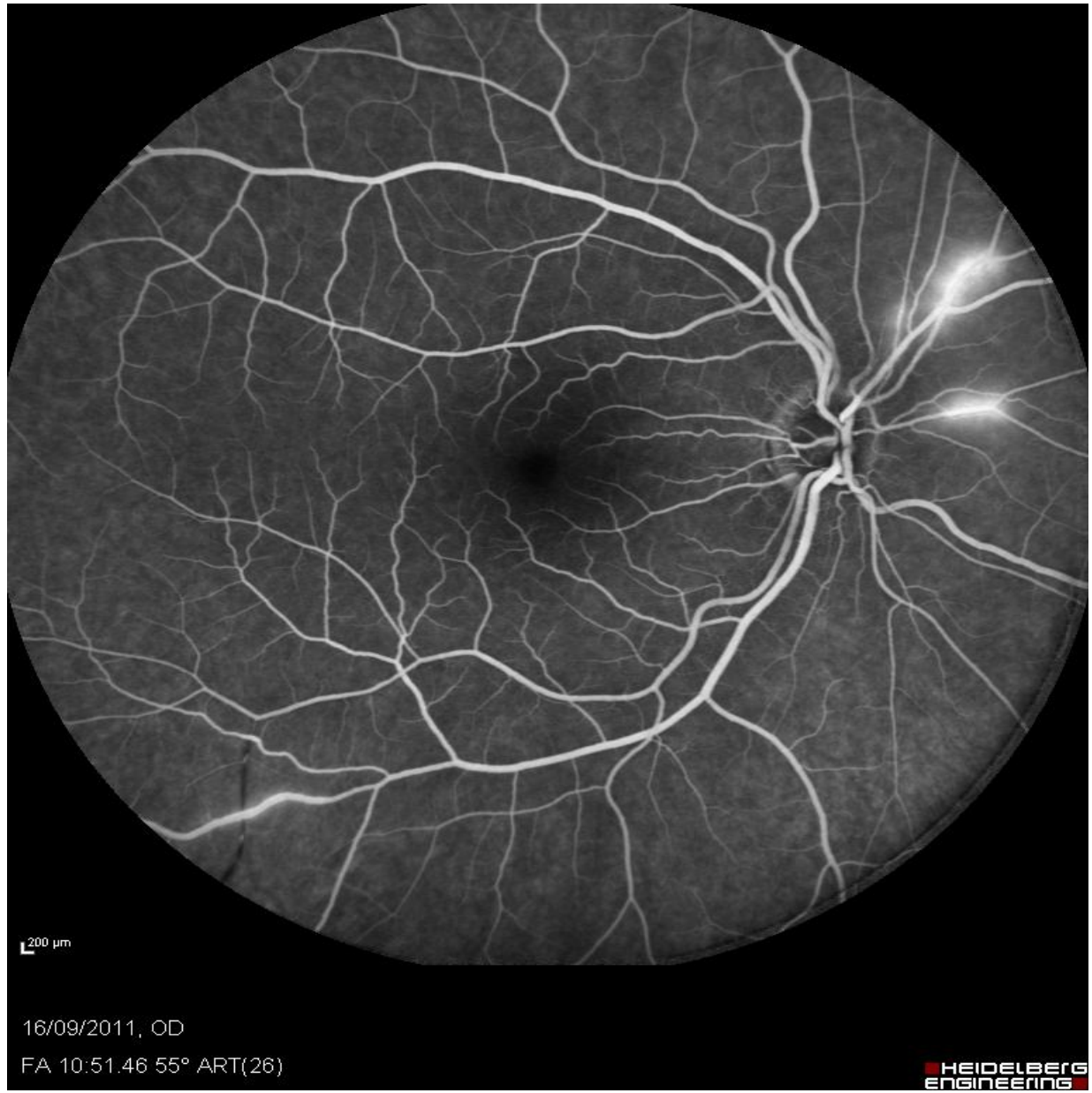
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16/09/2011, OD
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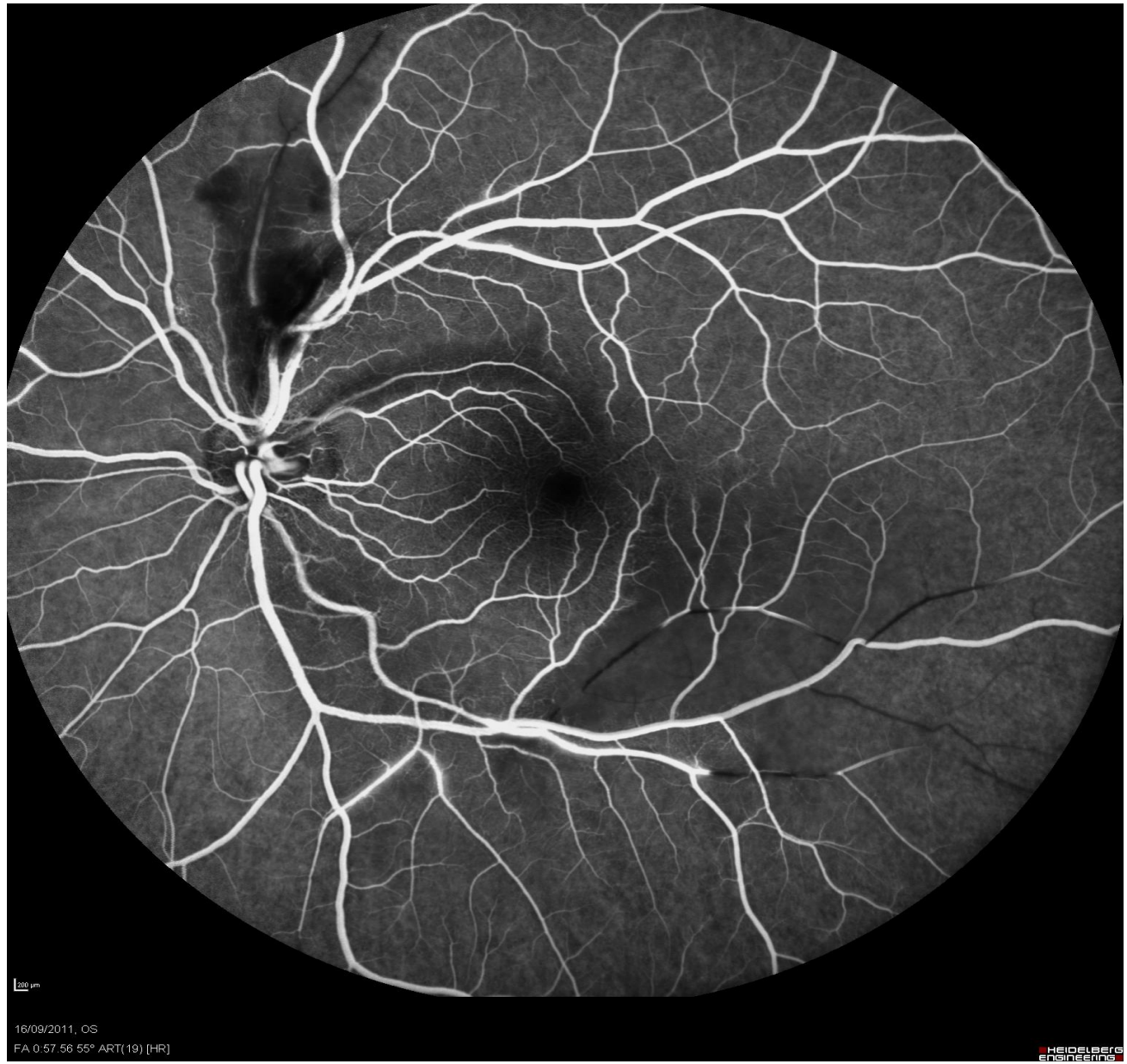
HEIDELBERG ENGINEERING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16/09/2011, OS
FA 0:57:56 55° ART(19) [HR]
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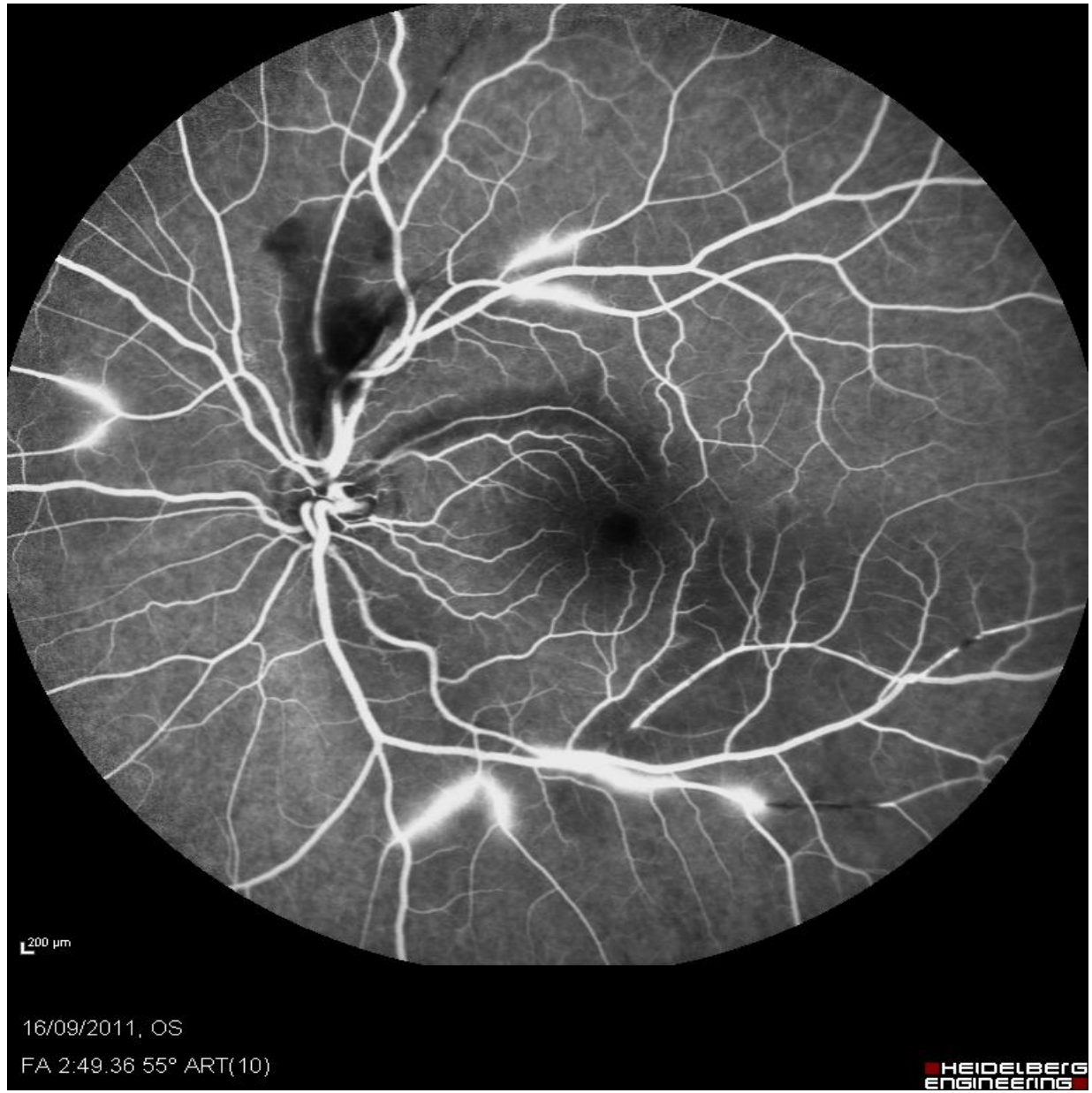
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FA 2:49.36 55° ART(10)
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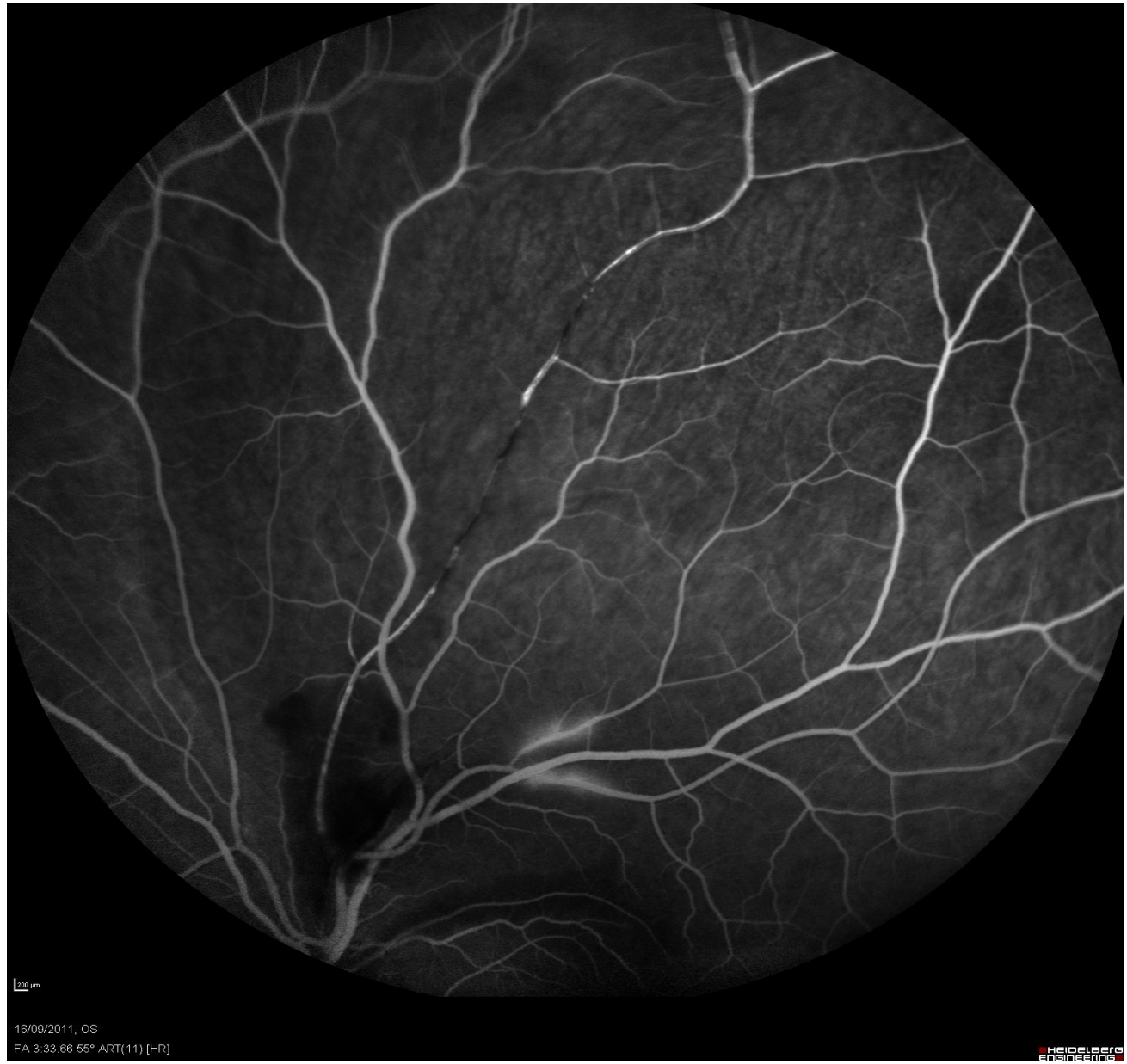
200 µm
16/09/2011, OS
FA 3.33.66 55° ART(11) [HR]
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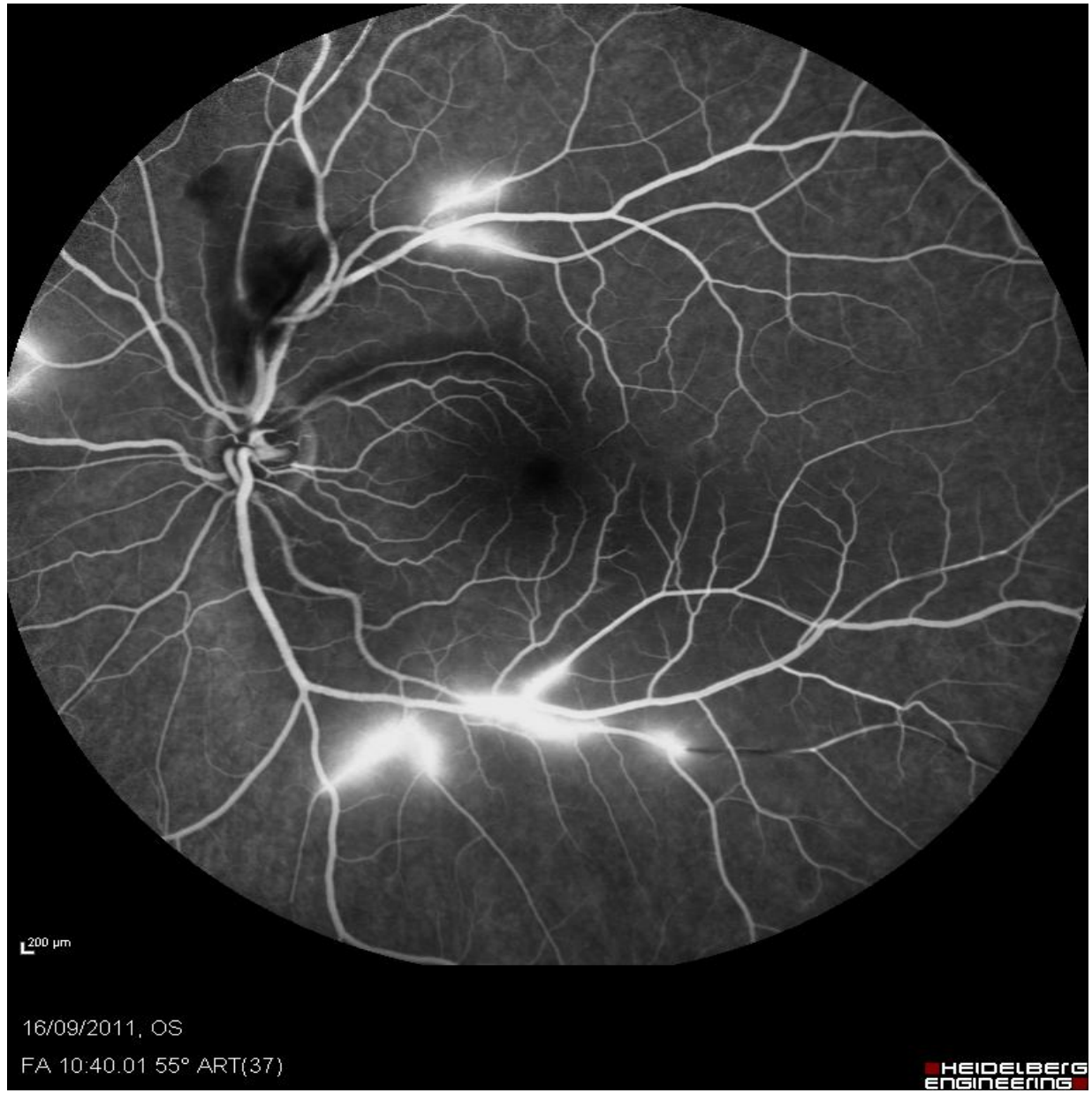
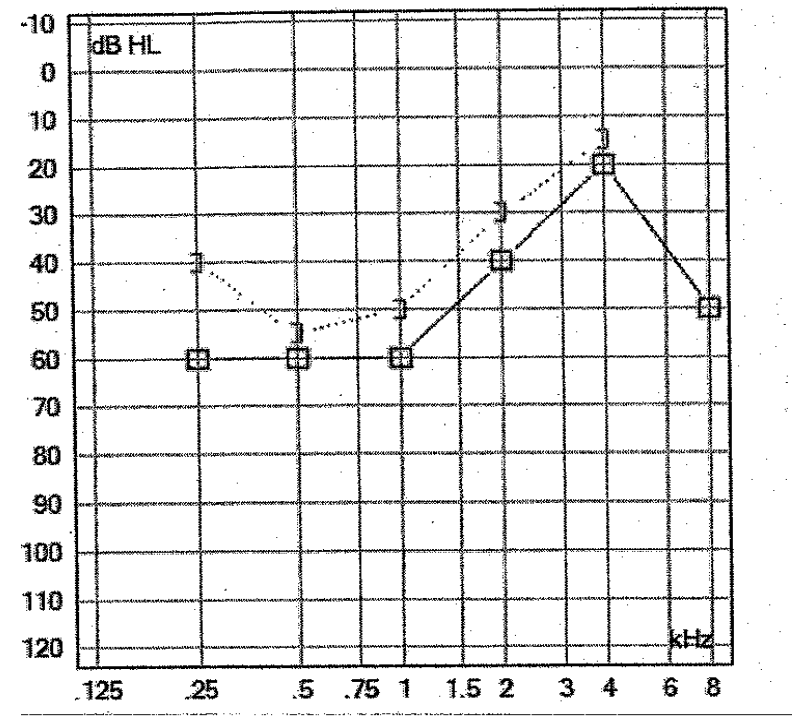

200 µm
16/09/2011, OS
FA 10:40.01 55° ART(37)
HEIDELBERG ENGINEERING

Evolution (1)

- **5 bolus de methylprednislone et relais CTC 1mg/kg et antiaggrégants plaquettaires**
- **Apparition le 17/05 de**
  - **Hypoacousie droite de survenue brutale**
  - **Céphalées**
  - **Vomissements**
  - **Babinski**



Audiogramme : surdité de perception

Evolution (2)

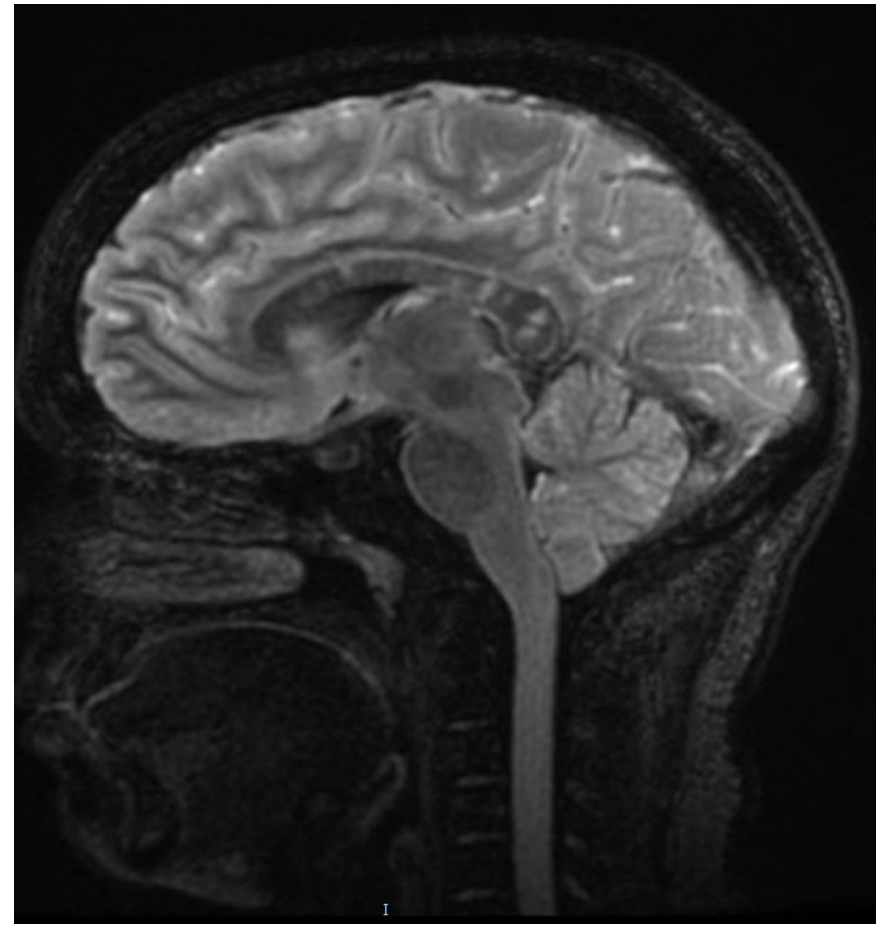
- **Anticoagulation efficace**
- **Stabilisation puis plusieurs rechutes OPH**
  - **6 perfusions d'Endoxan**
  - **Relais Azathioprine puis Methotrexate**
  - **IgIV**

Sd de Susac

- **SICRET : Small Infarctions of Cochlear Retinal and Encephalic Tissues**
- **Femme 80% cas, âge médian 30 ans (9- 69 ans)**
- **Physiopathologie inconnue**
  - **Pas de vascularite à l'histo**
  - **Lésions vasculaires endothéliales**
  - **Origine auto-immune probable**

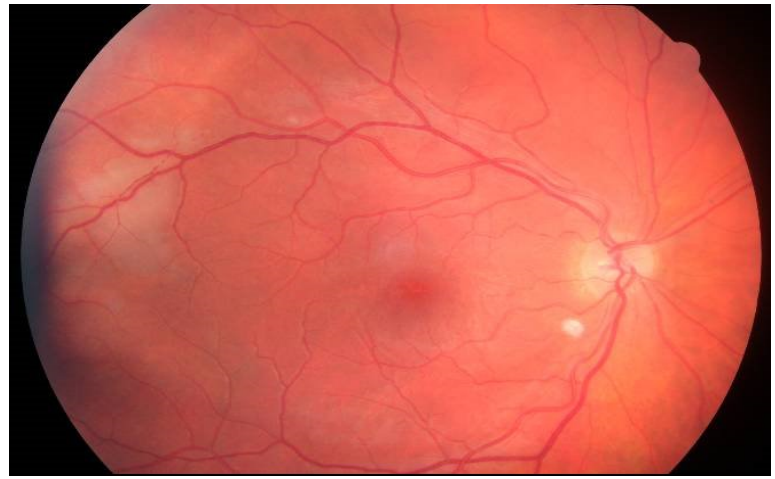
Atteinte neuro centrale

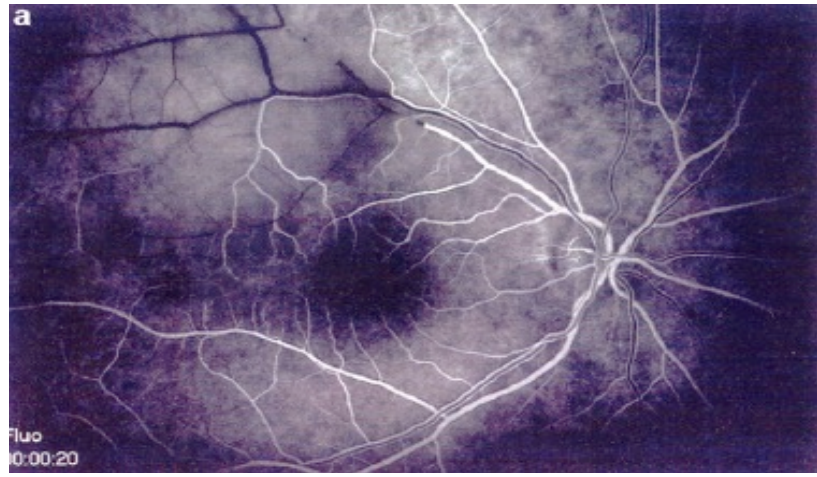
- **Céphalée (migraine +/- aura)**
- **Encéphalopathie, tbles psychiatriques (BDA,…)**
- **Sd cérébelleux, Sd Pyramidal, atteinte PC, comitialité**
- **Méningite lymphocytaire très modérée, protéinorachie élevée**



Rétinopathie

- **Occlusions bilatérales et multiples des branches de l'artère centrale de la rétine**
- **Fuites capillaires à l'angio**

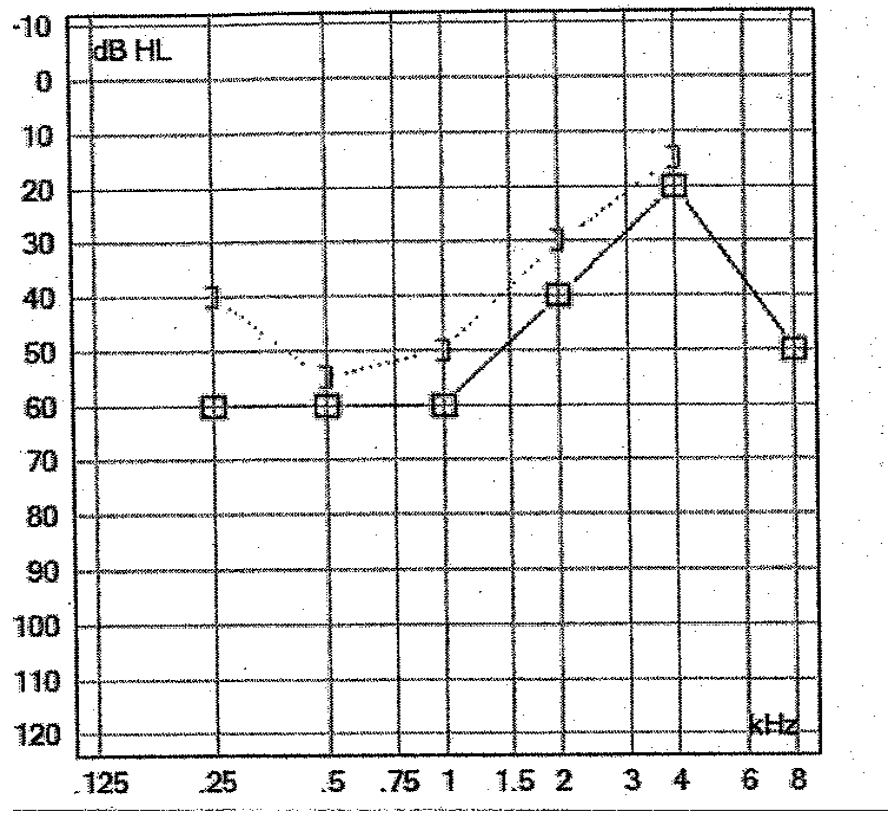




Papo, Rev Med Interne 2012

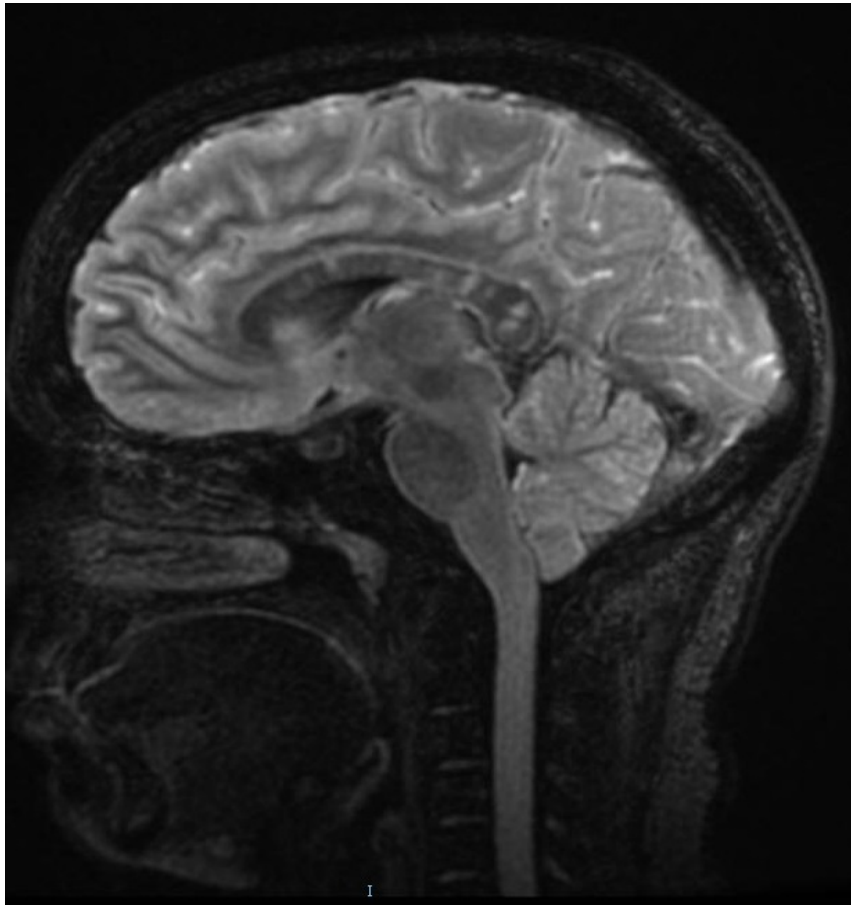
Atteinte cochléo-vestibulaire

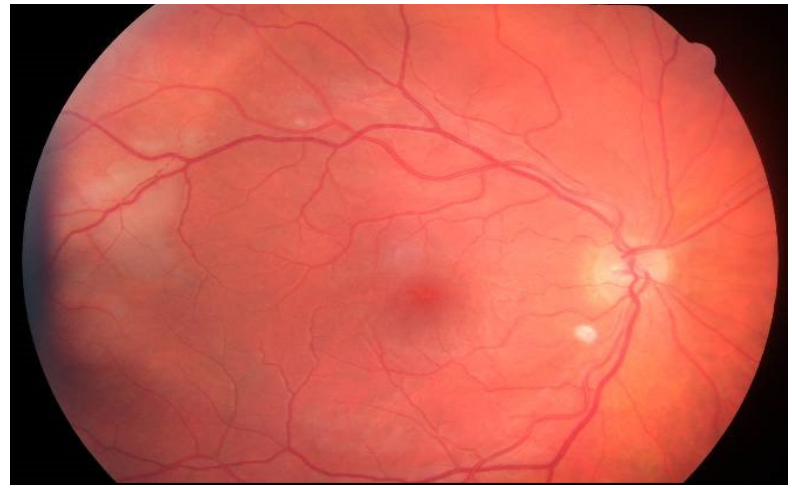
- **Hypoacousie brutale avec surdité de perception prédominant sur les fréquence basses**
- **Vertige rotatoire**
- **Ataxie**
- **Nystagmus**

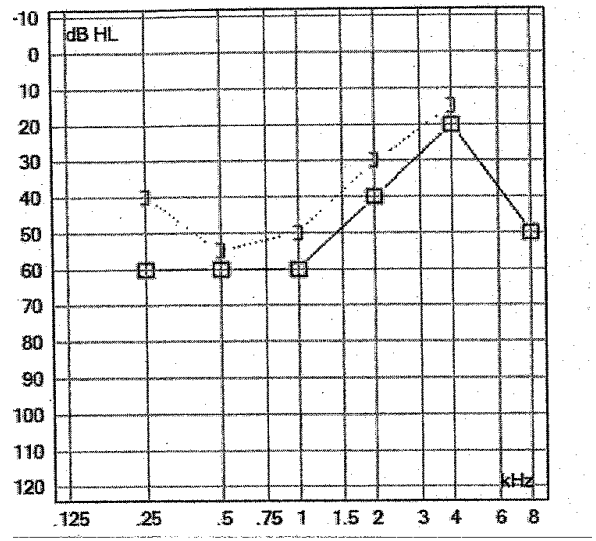


- **Phase « chaude » avec plusieurs poussées pendant 2-3 ans**
- **Puis « phase froide »**
  - **Rémission atteinte cérébrale et ORL (séquelles)**
  - **Fuites capillaires à l'angio sans nouvelles occlusions artériolaires**
- **Traitement**
  1) **CTC (bolus au début) +AAP**
  2) **Anticoagulation efficace**
  3) **ENDOXAN**
  4) **IgIV ou RITUXIMAB**

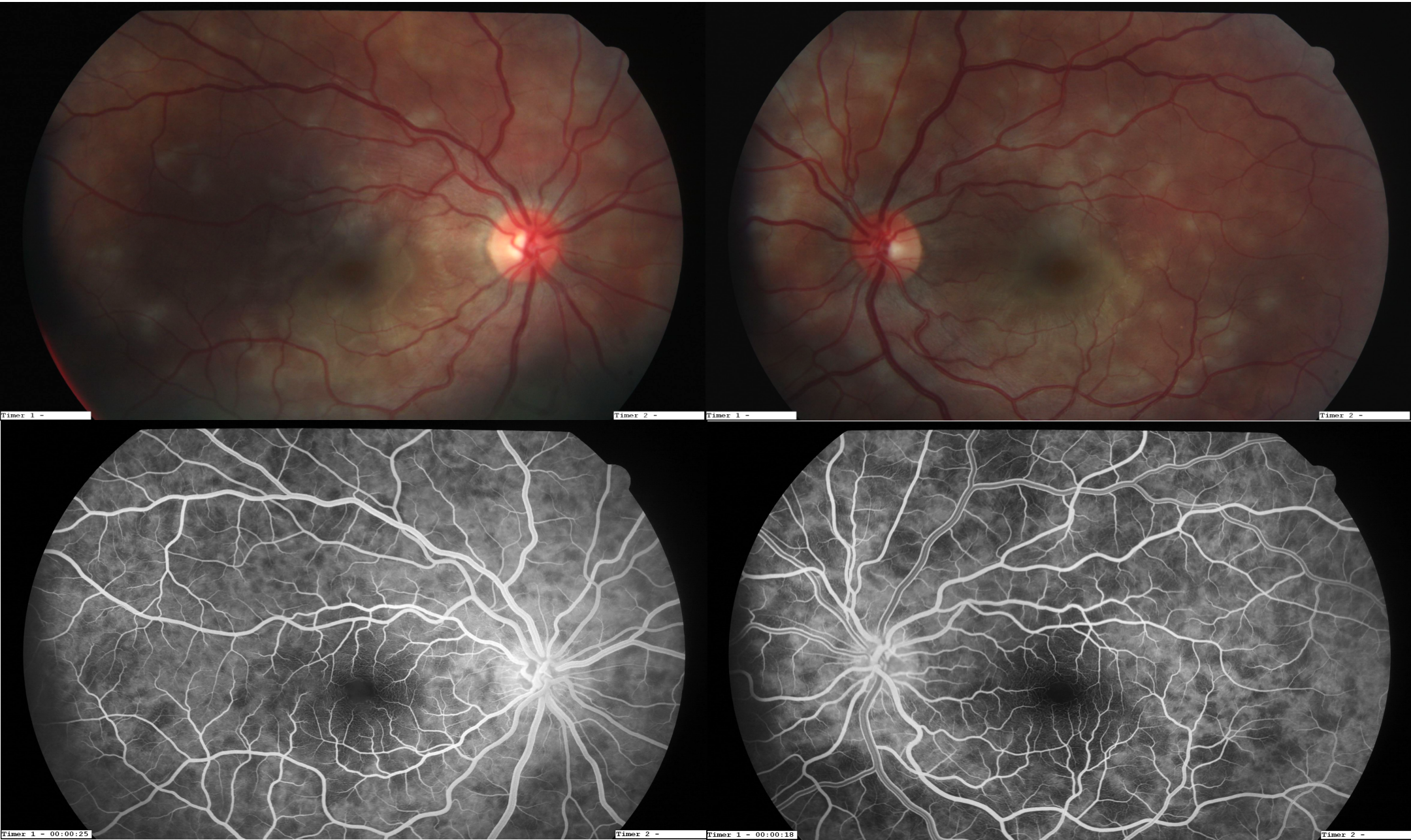
Triade du Sd de Susac







Mlle F., 23 ans, BAV OD le 16/11/13 ; BAV OG le 18/11/13 – Examen le 21/11/13 : VBLM ODG



Antécédents médicaux : RAS
Sept jours auparavant, épisode « pseudo-grippal » (fièvre – céphalées – myalgies), spontanément résolutif

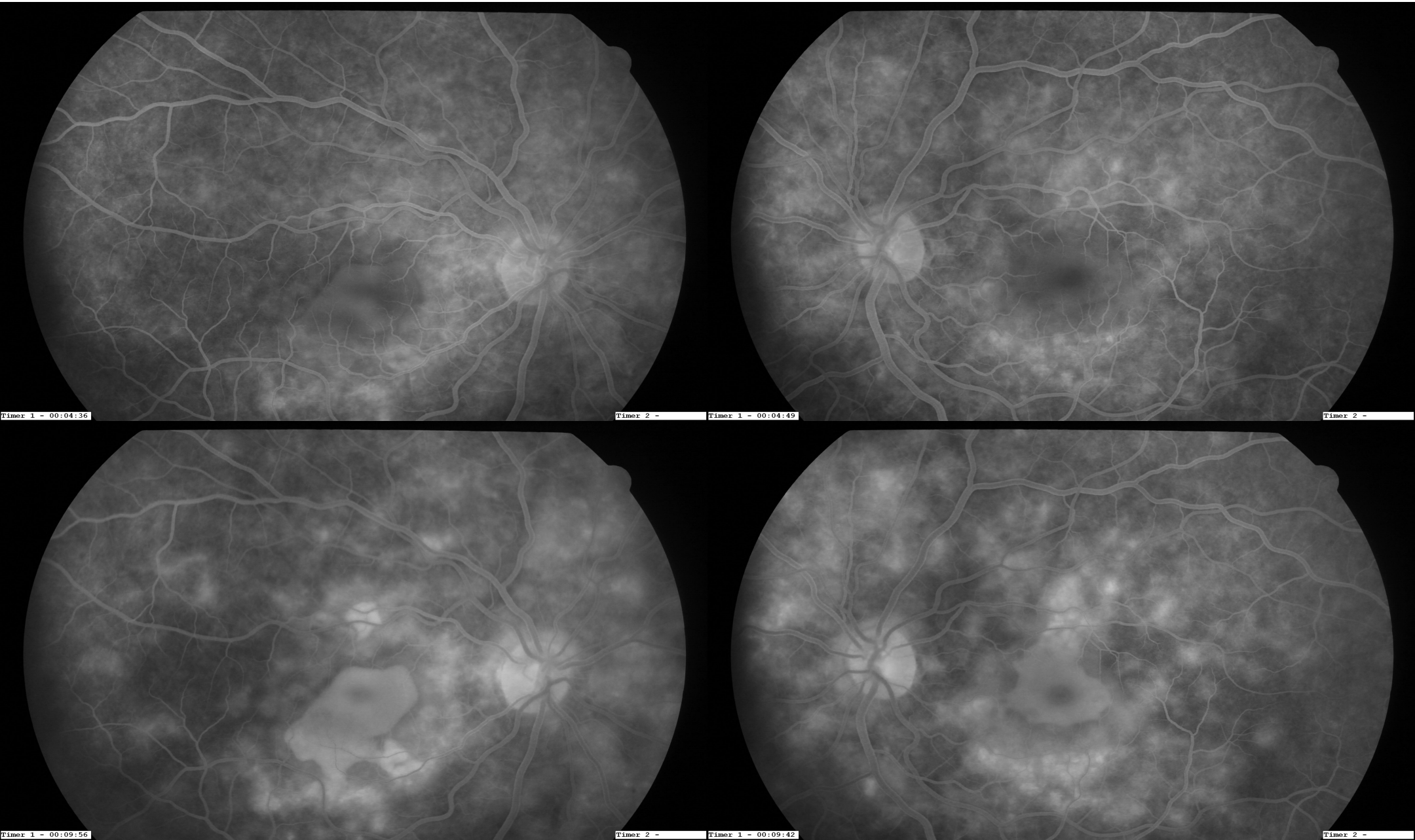
Antécédents ophtalmologiques : RAS

Examen :

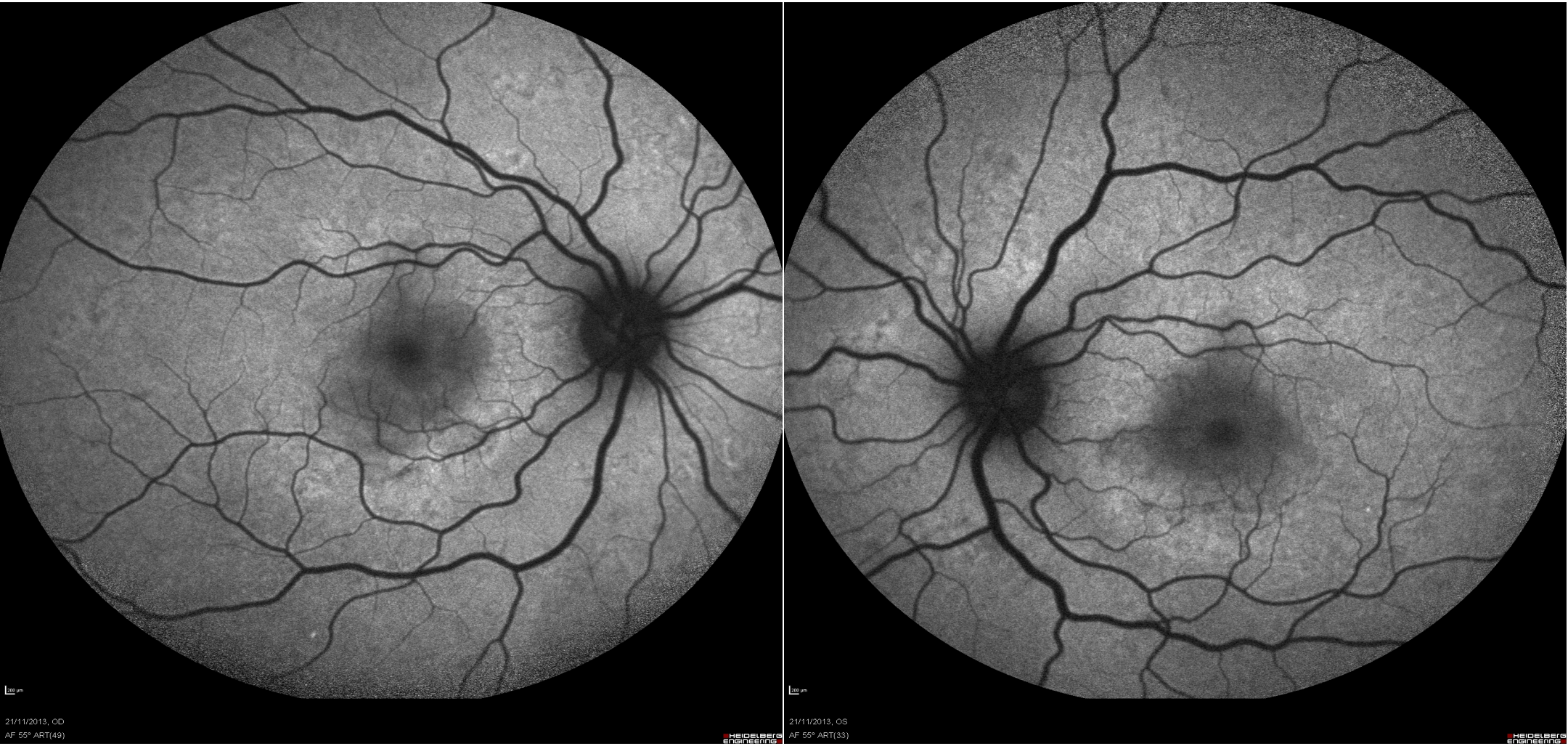
OD	OG
Œil blanc	Œil blanc
Tyndall cellulaire 1+	Tyndall cellulaire 1+
Flare 1+	Flare 1+
Pas de synéchie	Pas de synéchie
Pas de hyalite	Pas de hyalite

Cf. imagerie du fond d'œil

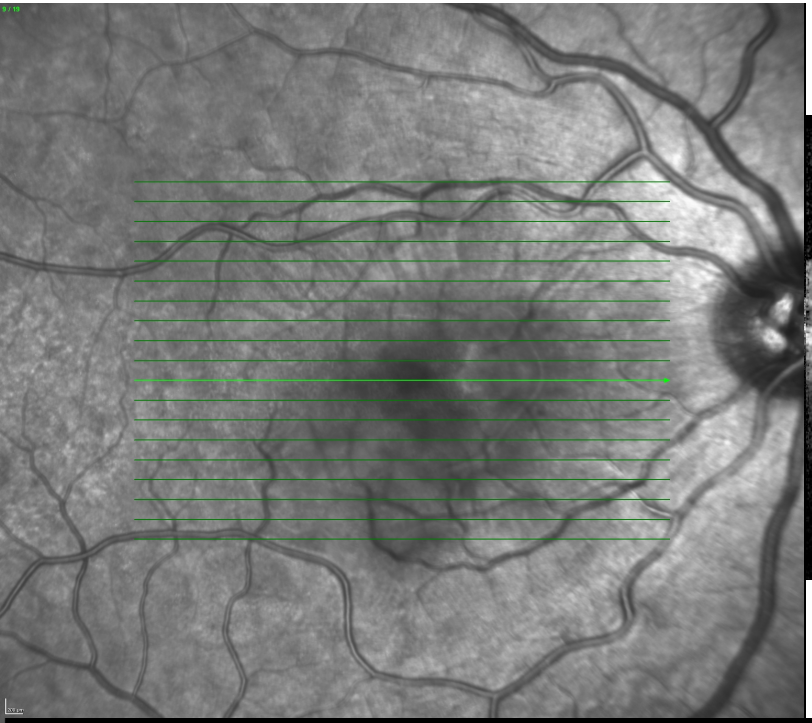
Examen initial - angiographie fluorescéinique

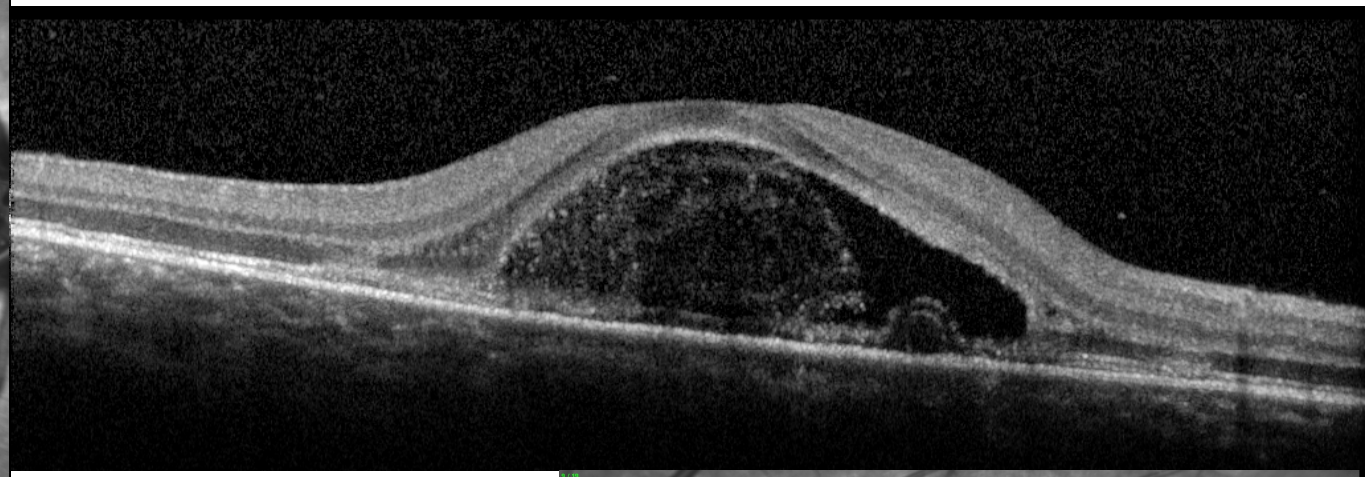


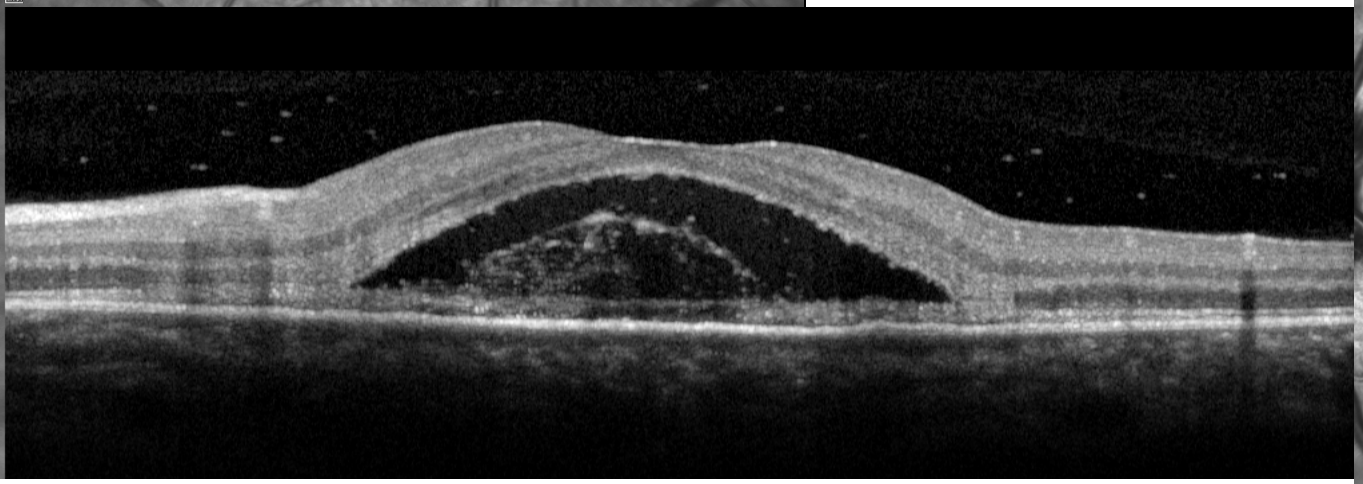
Examen initial - autofluorescence

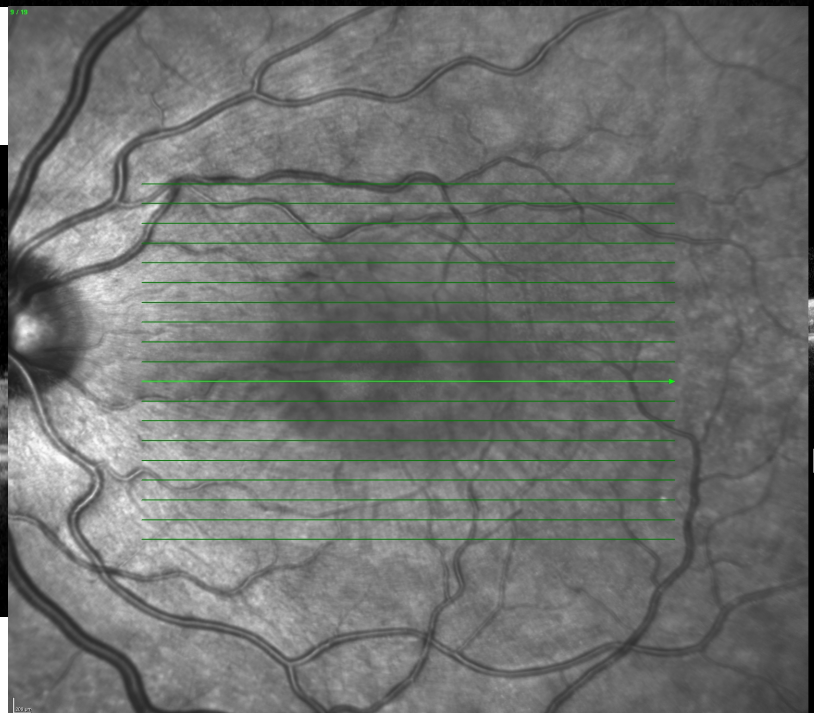


Examen initial - OCT









Hospitalisation en médecine interne

Syndrome inflammatoire biologique : CRP 32 mg/l
Hypergammaglobulinémie polyclonale (16,7 g/l)

LCR : protides 0,53 g/l, glucose 3,7 mM/l,
121 éléments nucléés /mm^3 (100% lymphocytes)

RAS par ailleurs

Question N° 1 : **Votre première hypothèse diagnostique est :**

A. Syndrome des taches blanches multiples évanescentes (*Multiple Evanescent White Dot Syndrome – MEWDS*)

B. Choroïdite multifocale

C. Choroïdite ponctuée interne

D. Epithéliopathie en plaques

E. Choriorétinopathie de type birdshot

Vous retenez le diagnostic d'**épithéliopathie en plaques**

Question N° 2 : **Parmi les éléments ci-dessous, lequel pourrait être un élément atypique dans ce contexte diagnostique**

A. Sexe féminin

B. Méningite lymphocytaire

C. Hypofluorescence aux temps précoces, hyperfluorescence aux temps tardifs

D. Décollements séreux rétiniens

E. Aucune des réponses ci-dessus : toutes les manifestations sont typiques

Question N° 3 : **Quelle est votre décision thérapeutique initiale**

A. Abstention

B. Prednisone *per os* 1 mg/kg/j

C. Corticothérapie par 3 bolus IV de methylprednisolone (500 mg/j)

D. Corticothérapie par 3 bolus IV et immunosuppresseur (Mycophénolate mofétil 2g/j)

E. Corticothérapie par 3 bolus IV et injections intravitréennes bilatérales d'anti-VEGF

Question N° 4 : **Après avoir pris votre décision thérapeutique, dans quel délai attendez-vous une acuité visuelle ≥ 2/10 ?**

A. Environ 1 semaine

B. Environ 1 mois

C. Environ 3 mois

D. Environ 1 an

E. Jamais

Question N° 5 : **La famille de la patiente, dont sa tante médecin généraliste, vous demande un entretien pour faire le point sur le pronostic à long terme. Votre pronostic est :**

A. Orientation vers un établissement spécialisé - Apprentissage du Braille recommandé

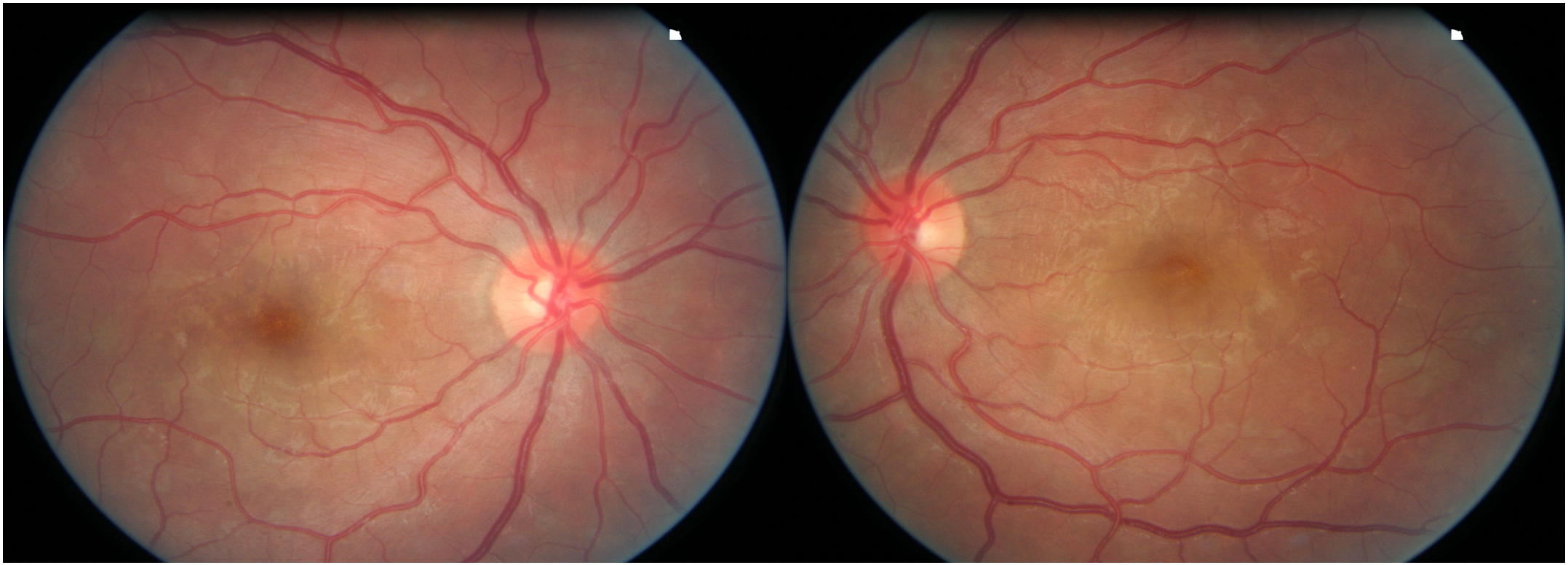
B. Amélioration fonctionnelle, mais AV finale incompatible avec la conduite automobile

C. Pronostic très variable, aucune prédiction possible

D. Récupération quasi-totale d'une vision normale, mais risque de rechute

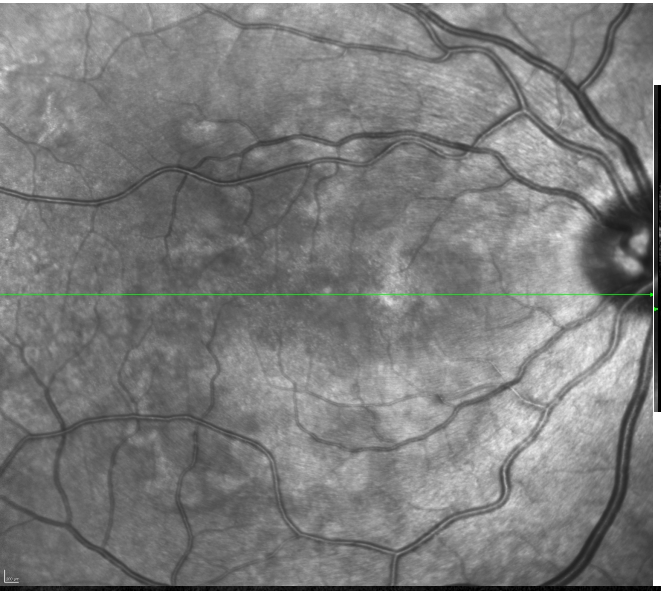
E. Récupération quasi-totale d'une vision normale, sans risque de rechute

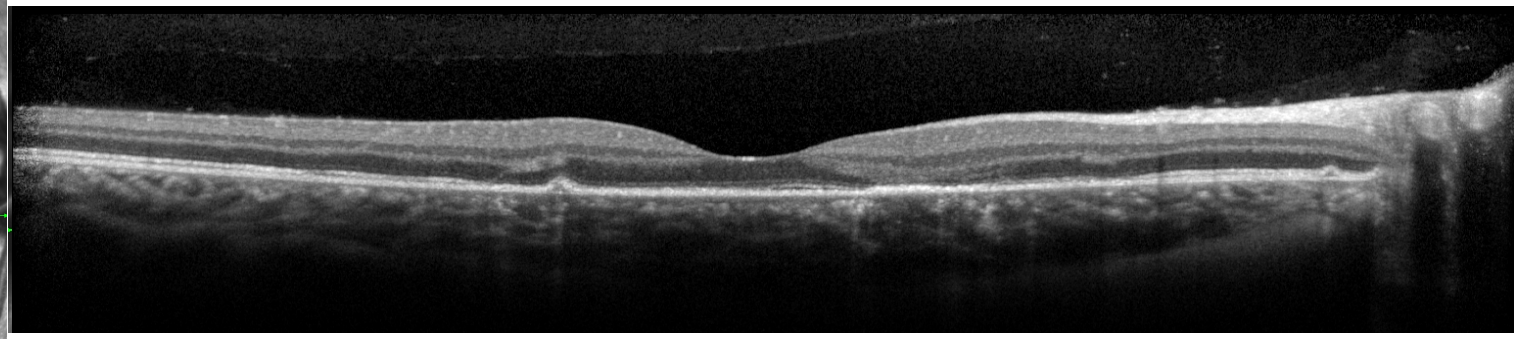
Fond d’œil à J7

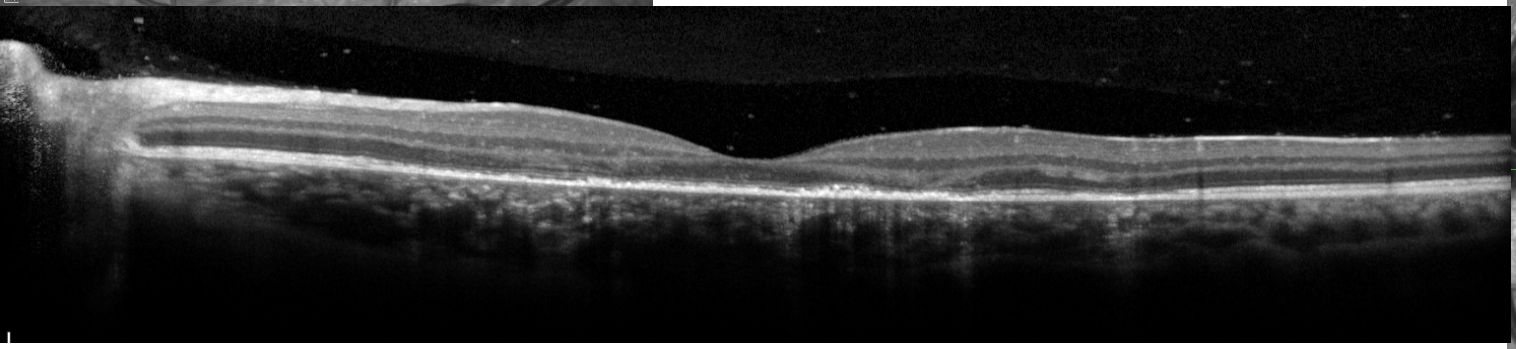


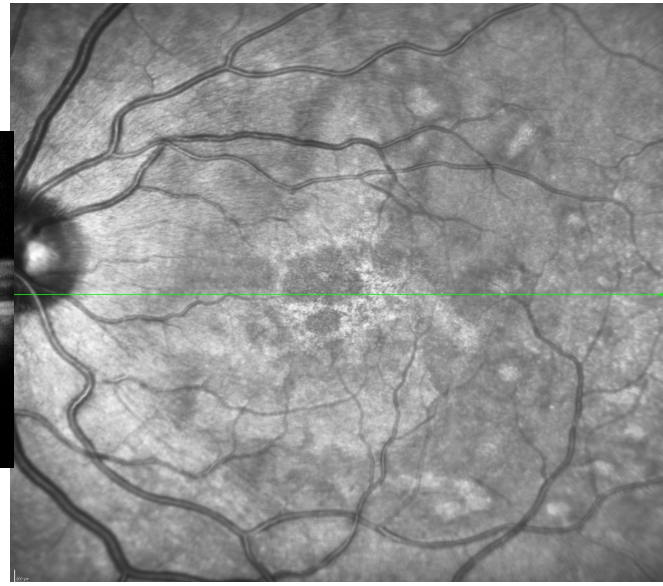
OCT à J7

AV : 4/10 ODG

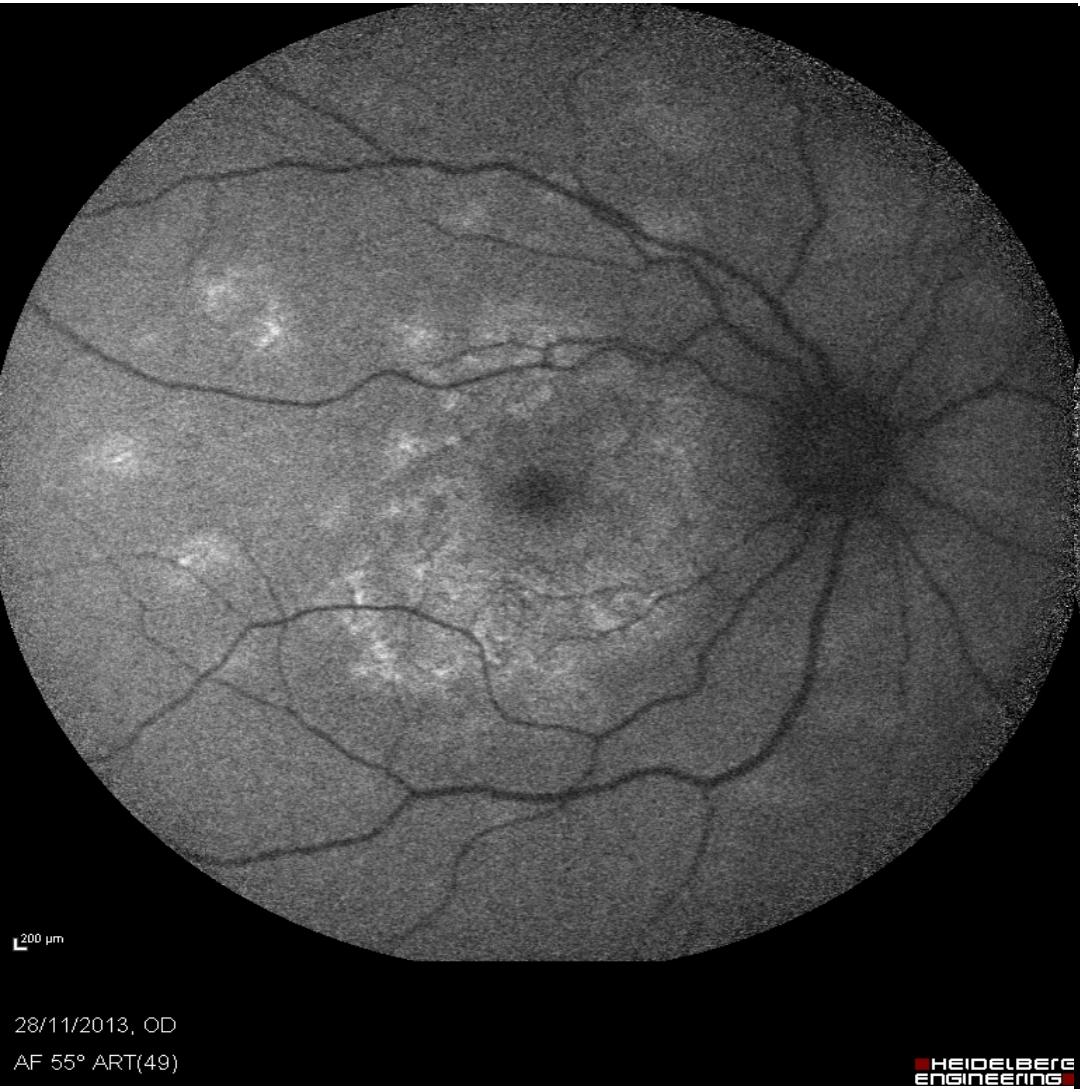


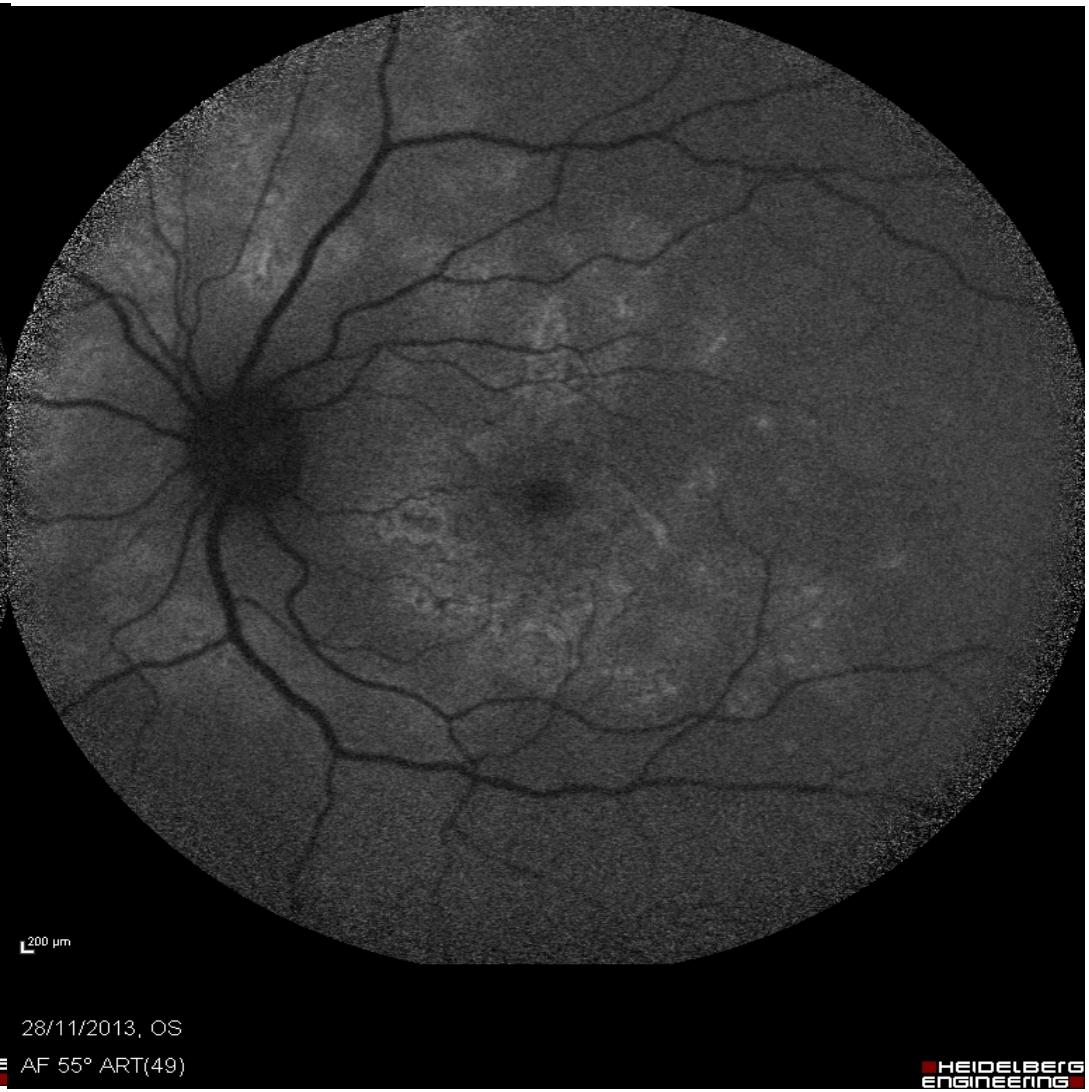




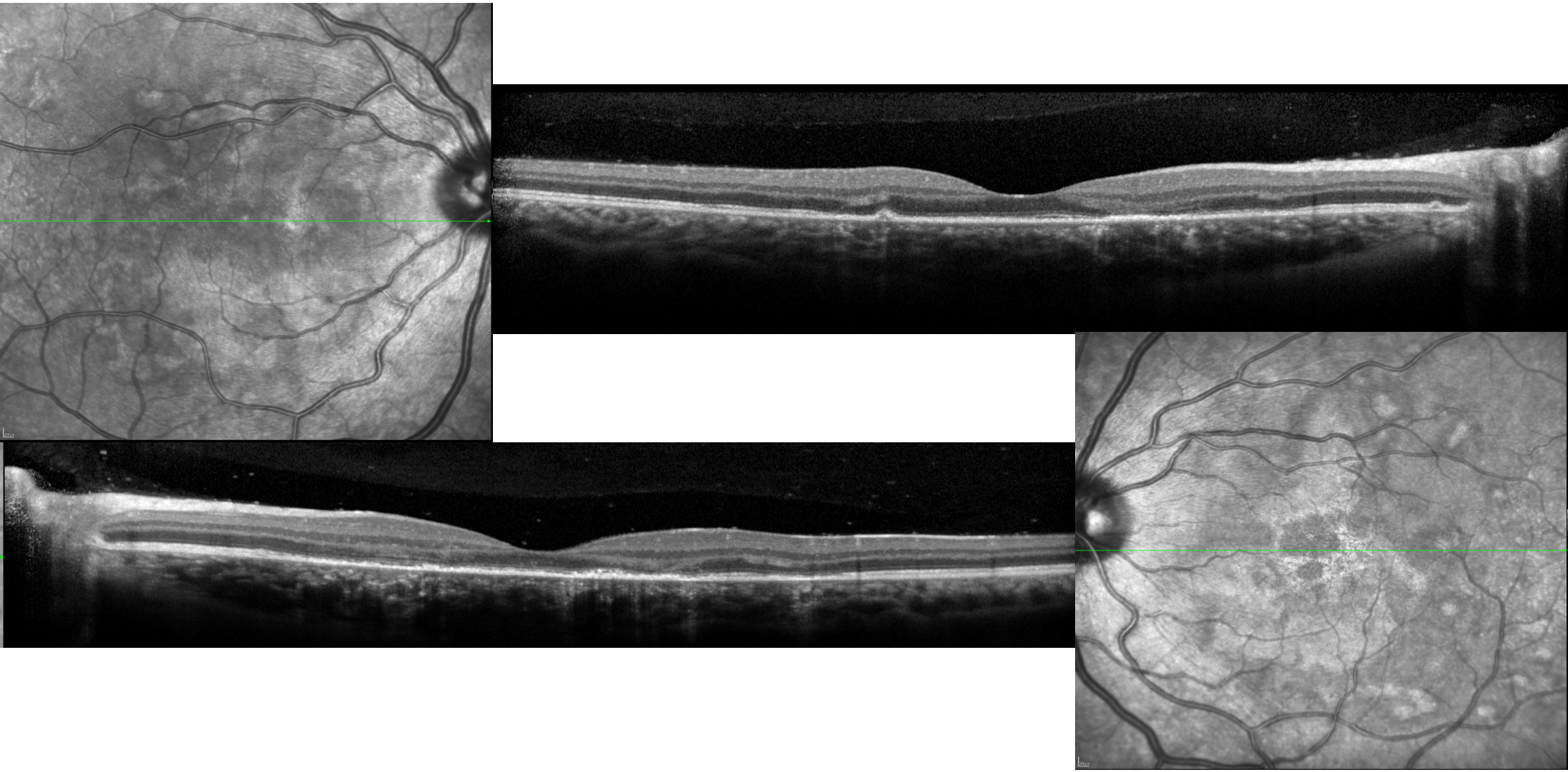


Autofluorescence à J7





OCT à J7



Champ visuel à 1 mois

Test de seuil central 30-2

Contrôle de fixation: Tache aveugle
Cible de fixation: Central
Pertes de fixation: 0/16
Erreurs faux pos.: 0/9
Erreurs faux nég.: 0/8
Durée du test: 08:12

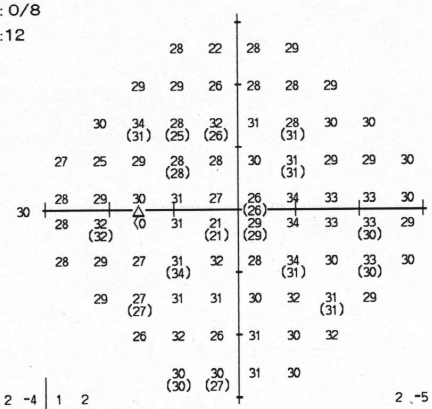
Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: FASTPAC

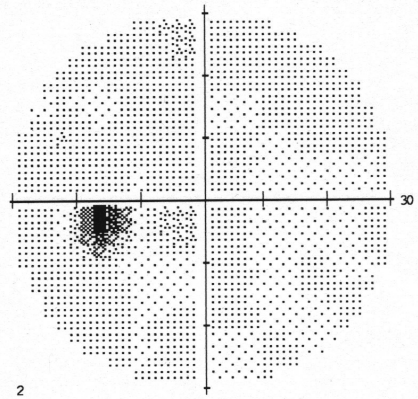
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

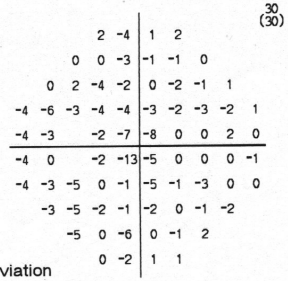
Date: 13-12-2013
Heure: 10:21
L'âge: 23

Fovéa: 31 dB ■

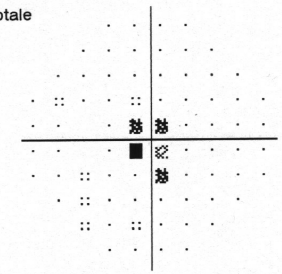
(testée 3 x)

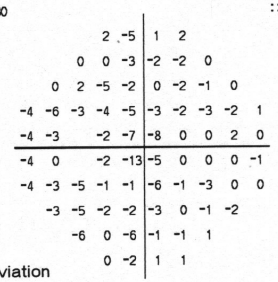




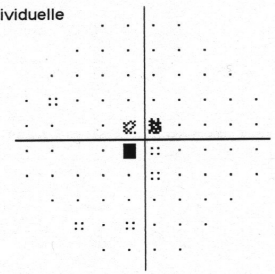


Déviation otale





Déviation ndividuelle



MD -2.01 dB
PSD 3.09 dB
SF 1.29 dB
CPSD 2.73 dB P < 5%

:: < 5%
▒ < 2%
▓ < 1%
■ < 0.5%

HOPITAL COCHIN
SERVICE D'OPHTALMOLOGIE
DPT D'ORTHOPTIE
75014 PARIS
TEL 01 58 41 22 17

Test de seuil central 30-2

Contrôle de fixation: Tache aveugle
Cible de fixation: Central
Pertes de fixation: 0/15
Erreurs faux pos.: 0/9
Erreurs faux nég.: 0/8
Durée du test: 07:55

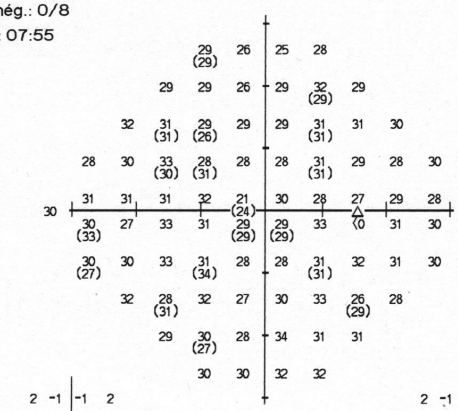
Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: FASTPAC

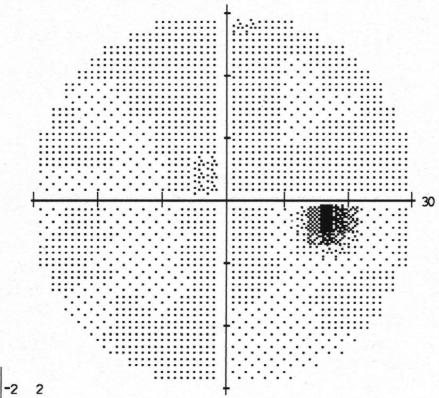
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

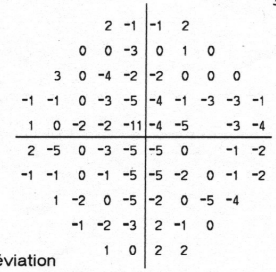
Date: 13-12-2013
Heure: 10:09
L'âge: 23

Fovéa: 36 dB

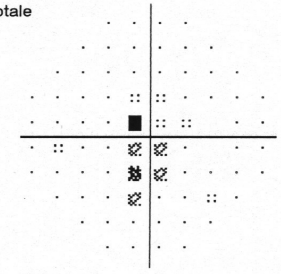
(testée 3 x)

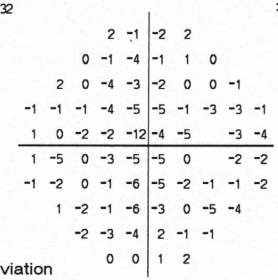




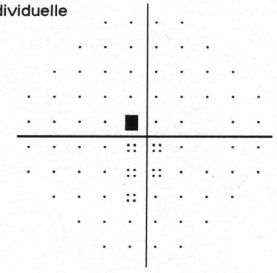


Déviation otale





Déviation ndividuelle



MD -1.98 dB
PSD 2.75 dB
SF 1.48 dB
CPSD 2.18 dB P < 10%

:: < 5%
▒ < 2%
▓ < 1%
■ < 0.5%

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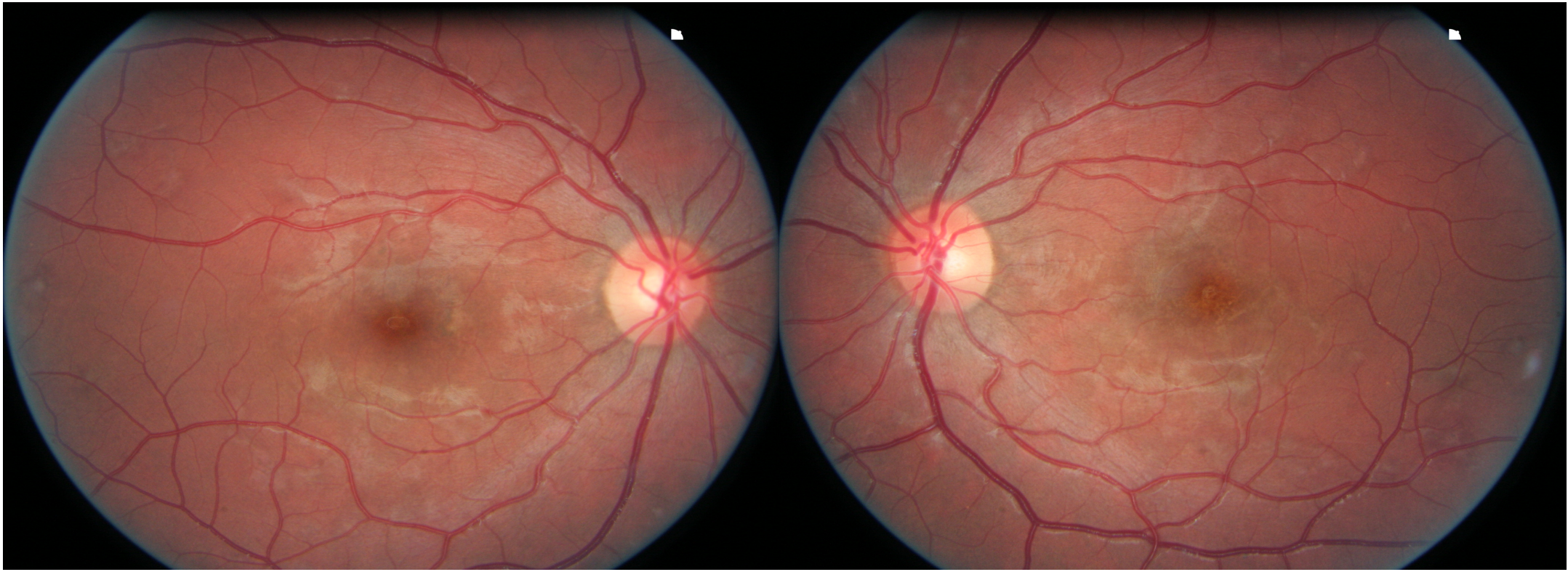
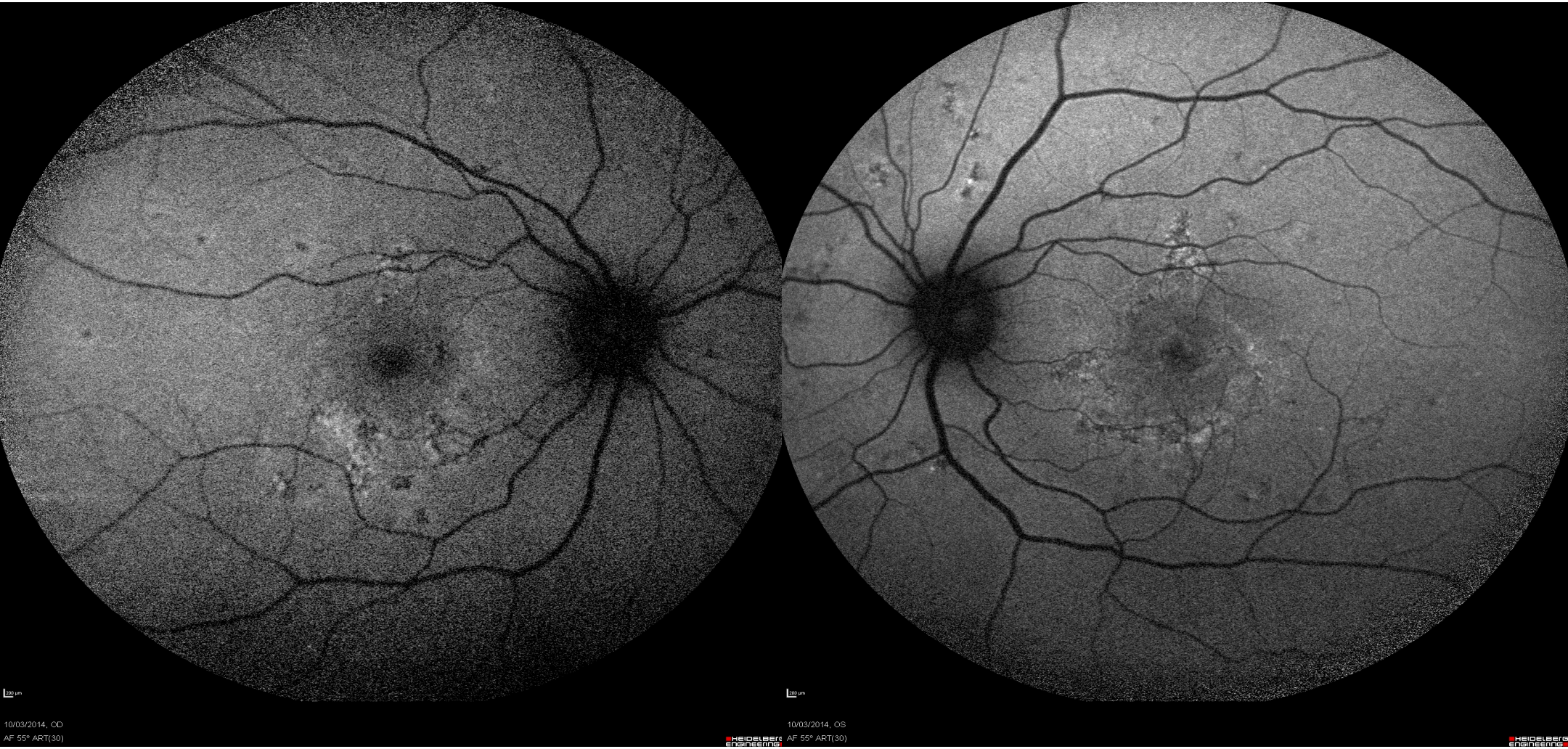
Fond d’œil à M4

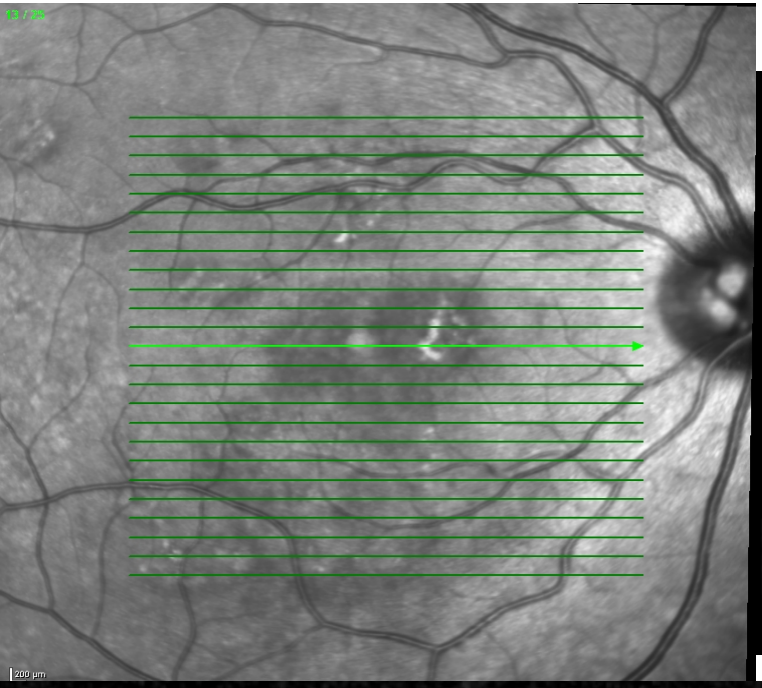


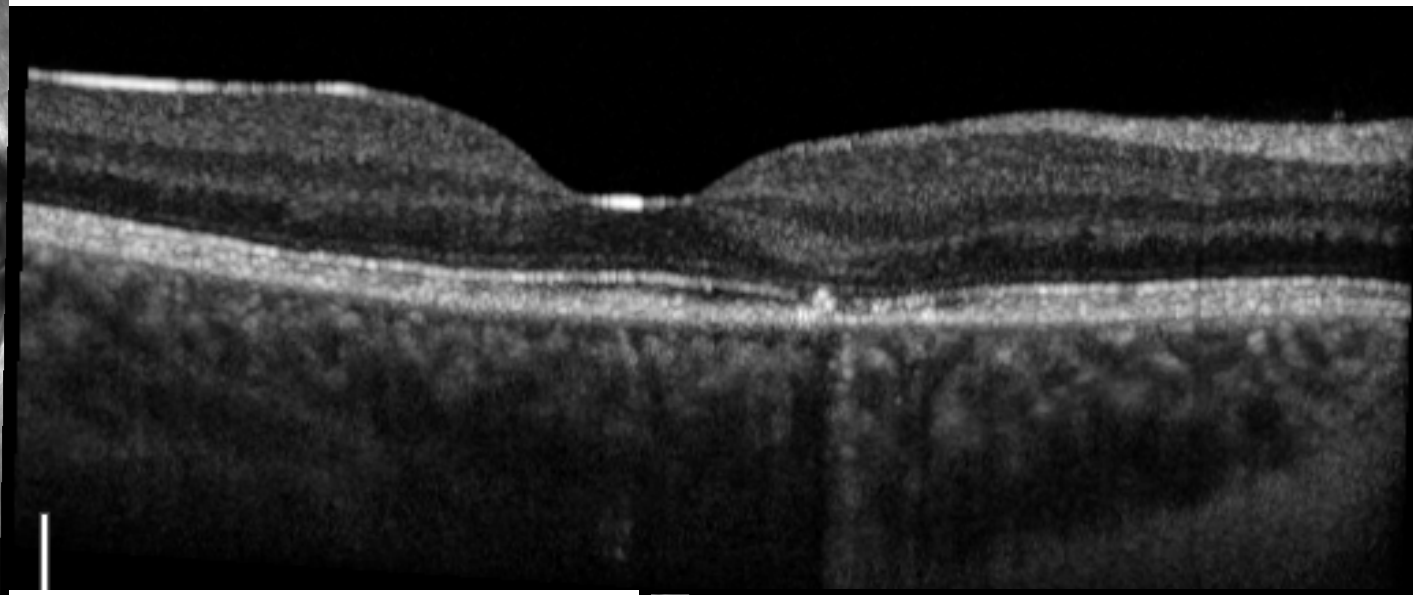
Autofluorescence à M4

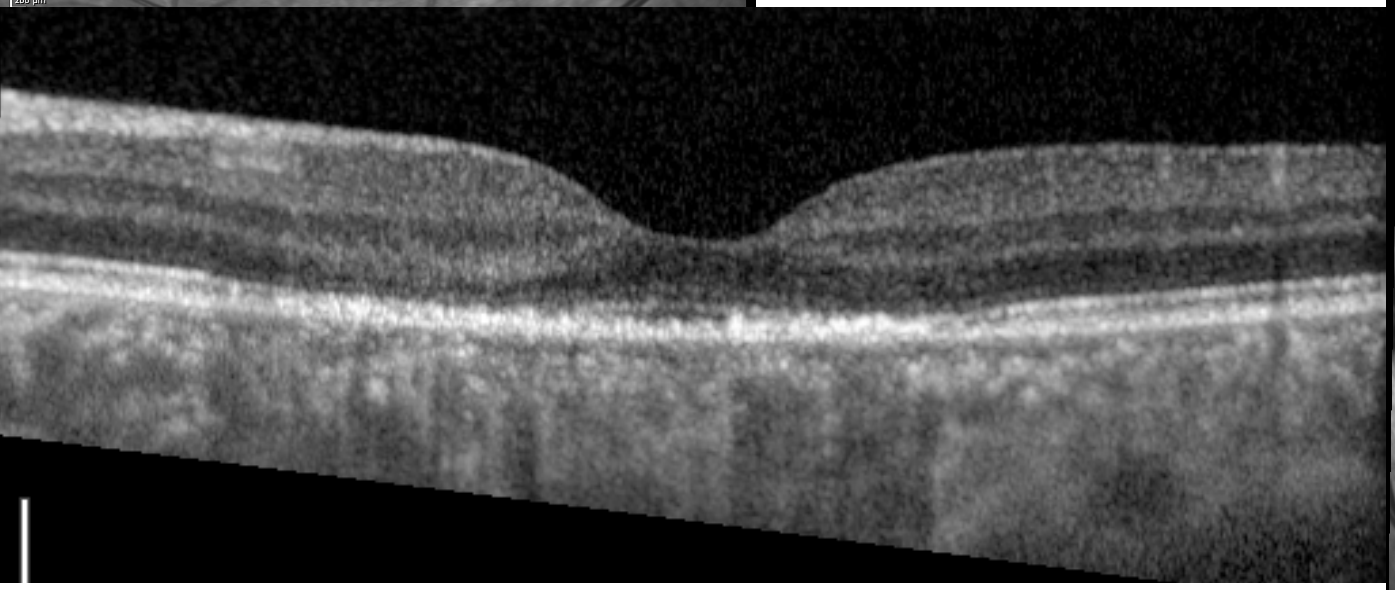


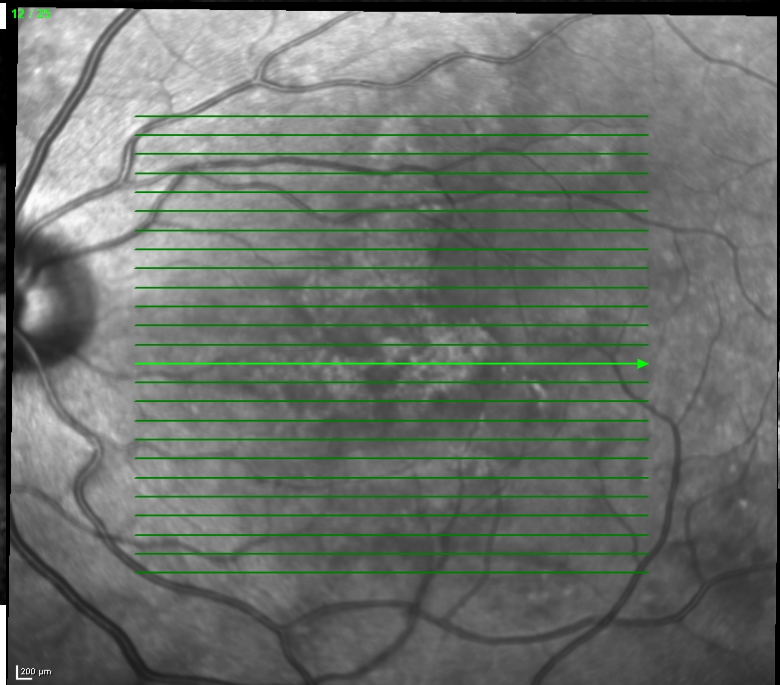
OCT à M4

AV OD : 10/10 – AV OG 9/10f









Champ visuel à M4

Test de seuil central 30-2

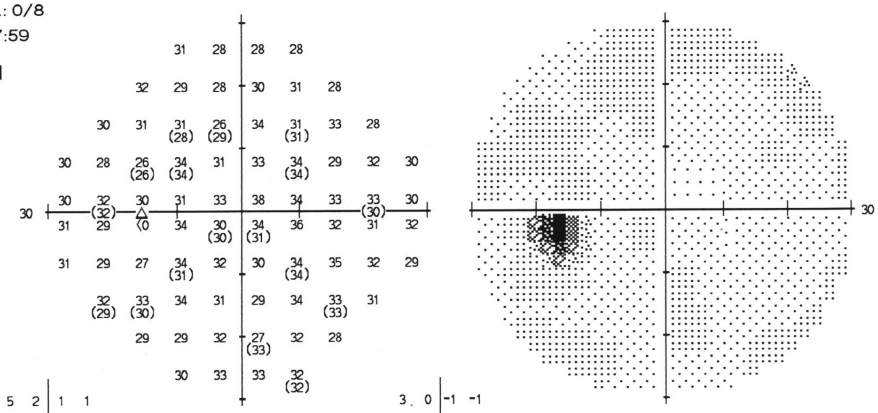
Contrôle de fixation: Tache aveugle
Cible de fixation: Central
Pertes de fixation: 0/16
Erreurs faux pos.: 0/8
Erreurs faux nég.: 0/8
Durée du test: 07:59

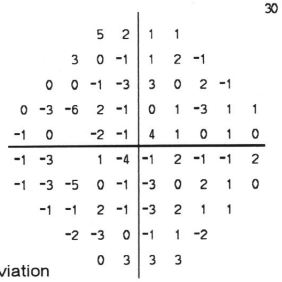
Fovéa: 31 dB

Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: FASTPAC

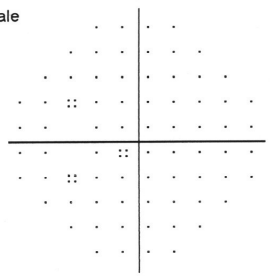
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

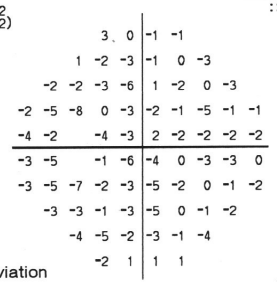
Date: 04-03-2014
Heure: 11:05
L'âge: 23



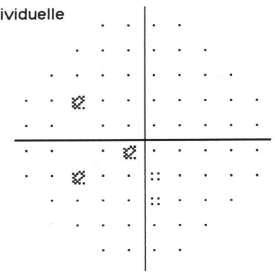


Déviation
otale





Déviation
ndividuelle



MD -0.13 dB
PSD 2.24 dB
SF 1.35 dB
CPSD 1.63 dB

:: < 5%
▨ < 2%
▩ < 1%
■ < 0.5%

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Test de seuil central 30-2

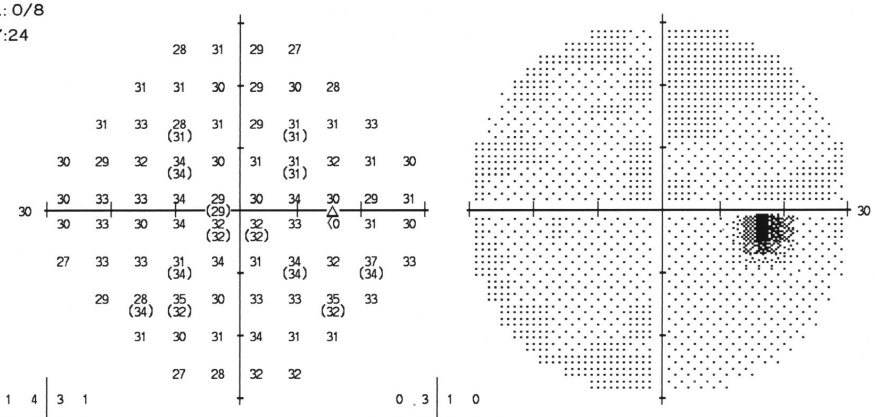
Contrôle de fixation: Tache aveugle
Cible de fixation: Central
Pertes de fixation: 0/15
Erreurs faux pos.: 0/8
Erreurs faux nég.: 0/8
Durée du test: 07:24

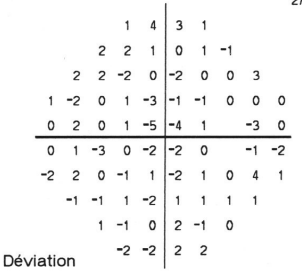
Fovéa: 34 dB

Stimulus: III, Blanc
Fond: 31.5 ASB
Stratégie: FASTPAC

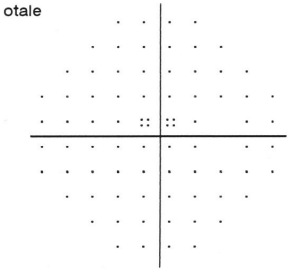
Diamètre de la pupille:
Acuité visuelle:
RX: DS DC X

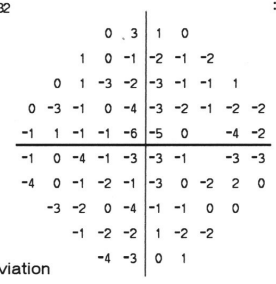
Date: 04-03-2014
Heure: 10:55
L'âge: 23



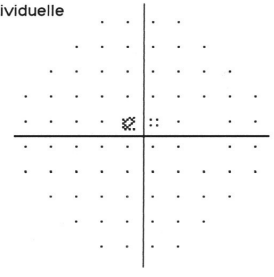


Déviation
otale





Déviation
ndividuelle



MD -0.21 dB
PSD 1.92 dB
SF 1.76 dB
CPSD 0.00 dB

:: < 5%
▨ < 2%
▩ < 1%
■ < 0.5%

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BMC Ophthalmology

RESEARCH ARTICLE Open Access

Untreated Acute Posterior Multifocal Placoid Pigment Epitheliopathy (APMPPE): a case series

Olivia Xerri, Sawsen Salah*, Dominique Monnet and Antoine P. Brézin

Abstract

Background: Acute Posterior Multifocal Placoid Pigment Epitheliopathy (APMPPE) is a rare inflammatory eye disease that affects the Retinal Pigment Epithelium and outer retina. The purpose of this study was to describe its presentations, as well as its prognosis in a series of untreated patients.

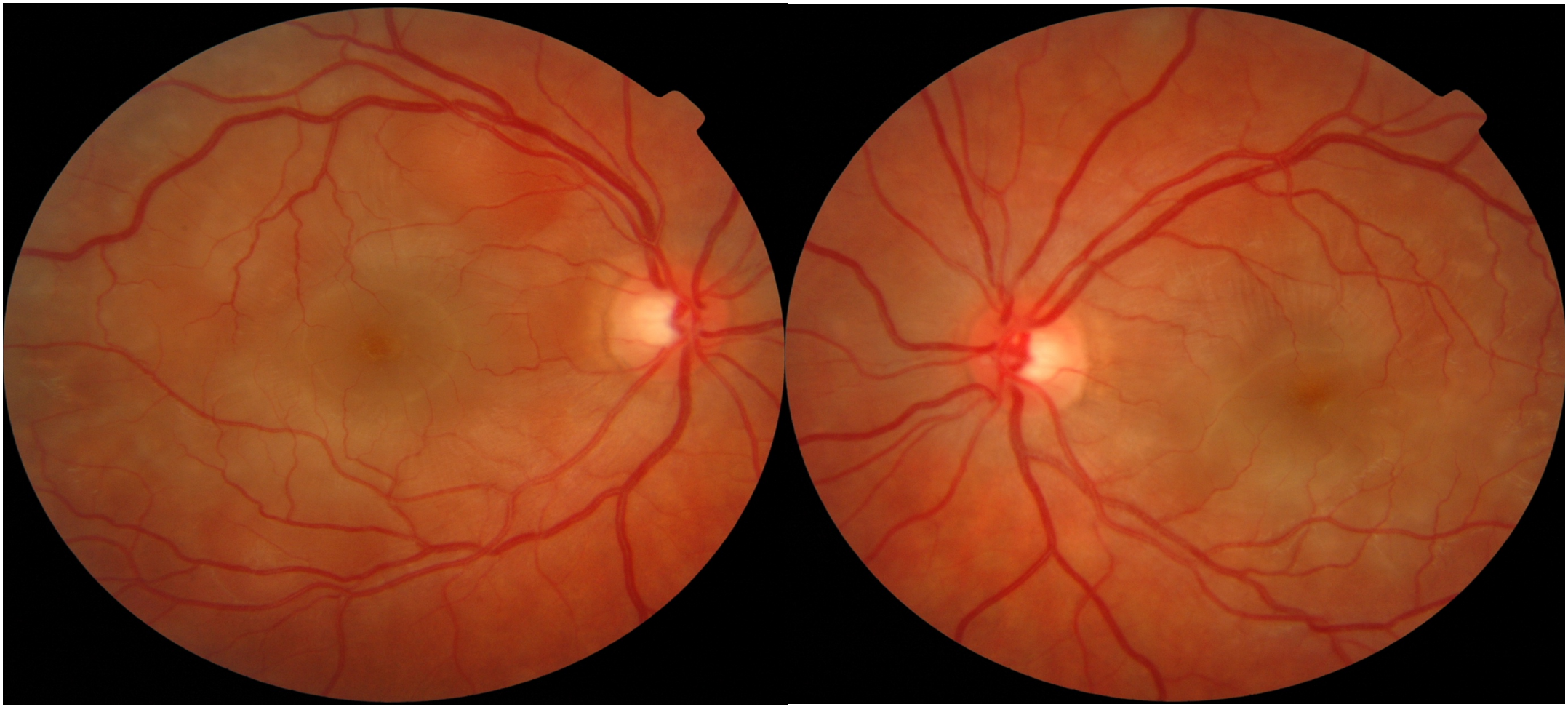
Methods: Records of patients seen in the department of Ophthalmology at Cochin University Hospital, Paris, between April 2002 and June 2015 were retrospectively studied. Patients were included if they presented with the typical findings of APMPPE characterized by whitish or yellowish bilateral placoid lesions, a typical pattern of early hypofluorescence and late hyperfluorescence on fluorescein angiography. Only untreated patients who had been followed for at least 1 month were included.

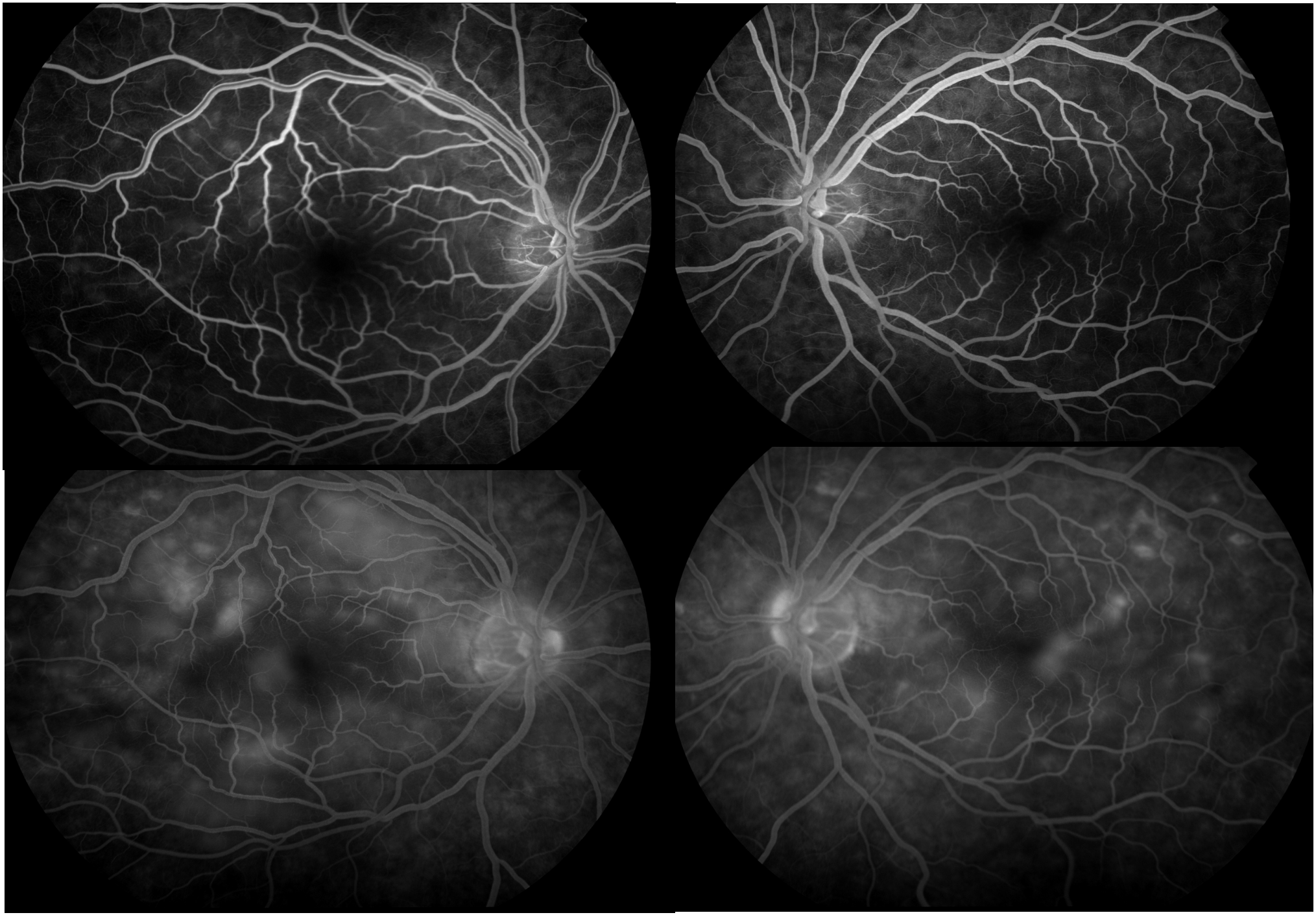
Results: Out of 22 patients' records with a diagnosis of APMPPE, 10 patients (9 women, 1 man), with a mean age of 24.5 ± 4.2 years, fulfilled the study criteria with a diagnosis of typical untreated APMPPE. Prodromal symptoms were reported in 7/10 patients. Macular lesions were observed in 18/20 eyes. Sub-retinal fluid was seen at presentation in 3 eyes. Initial mean BCVA was 0.56 ± 0.81 LogMAR [− 0.10 to 2.30]. In 9 out of 10 cases, the time interval between manifestations in the first affected eye and the fellow eye was less than 3 days. After 1 month, BCVA had improved to 0.05 ± 0.089 LogMAR [0–0.3], with a decimal BCVA ≥0.8 in 17/20 eyes.

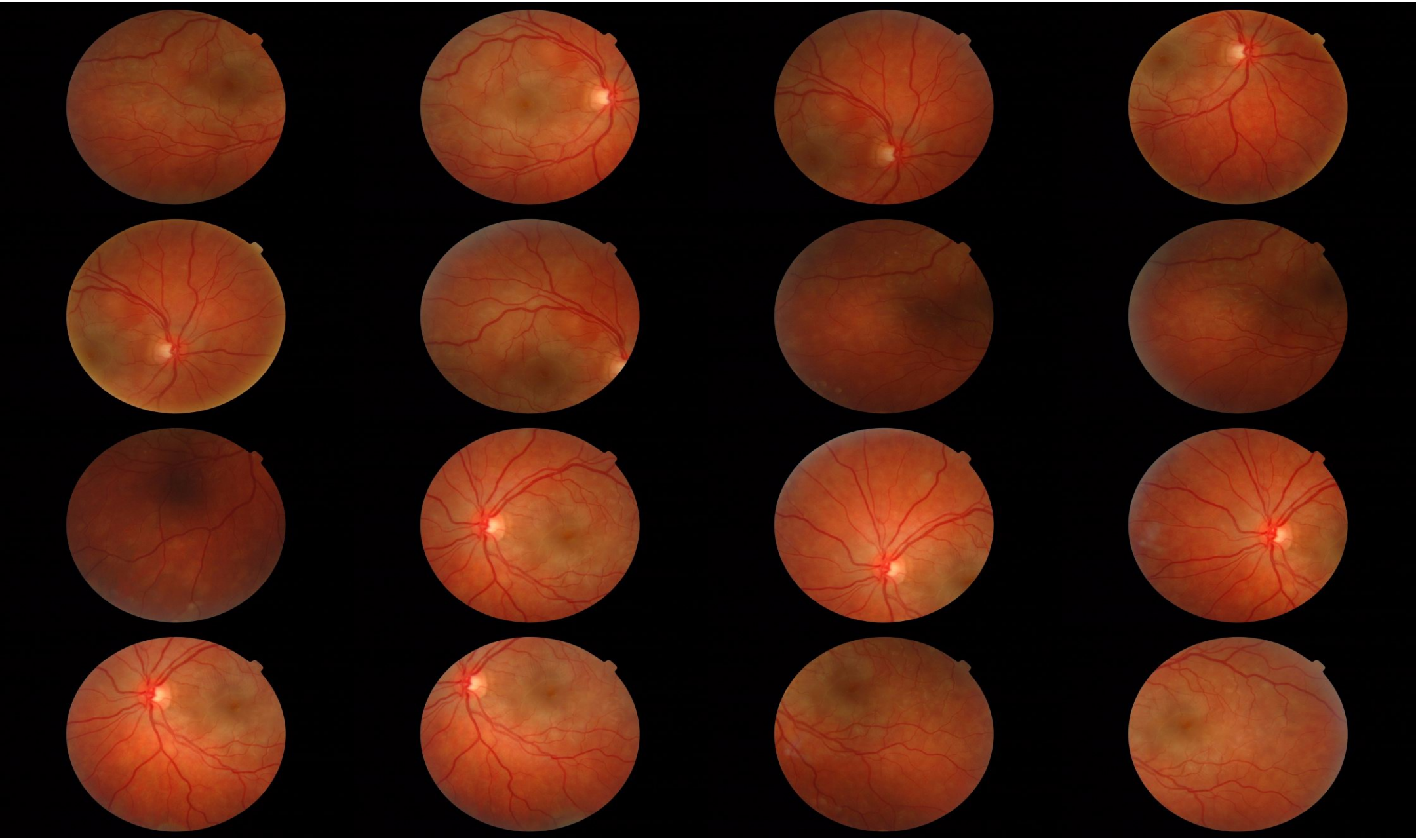
Conclusions: In these 10 cases of untreated APMPPE, a favorable outcome was observed.

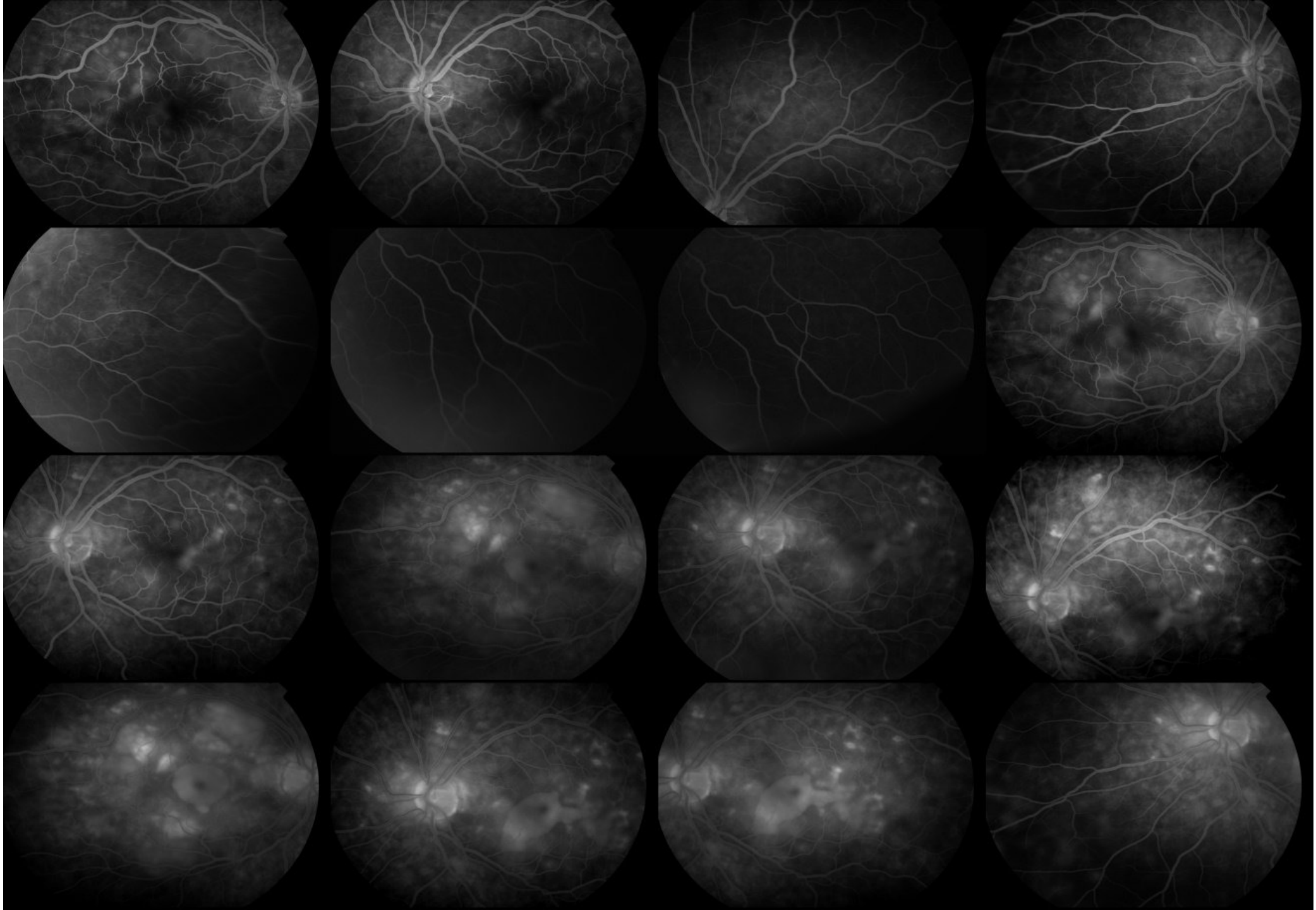
Keywords: Acute posterior multifocal placoid pigment epitheliopathy, Inflammatory disease, Posterior uveitis, Retina, Retinal pigment epithelium

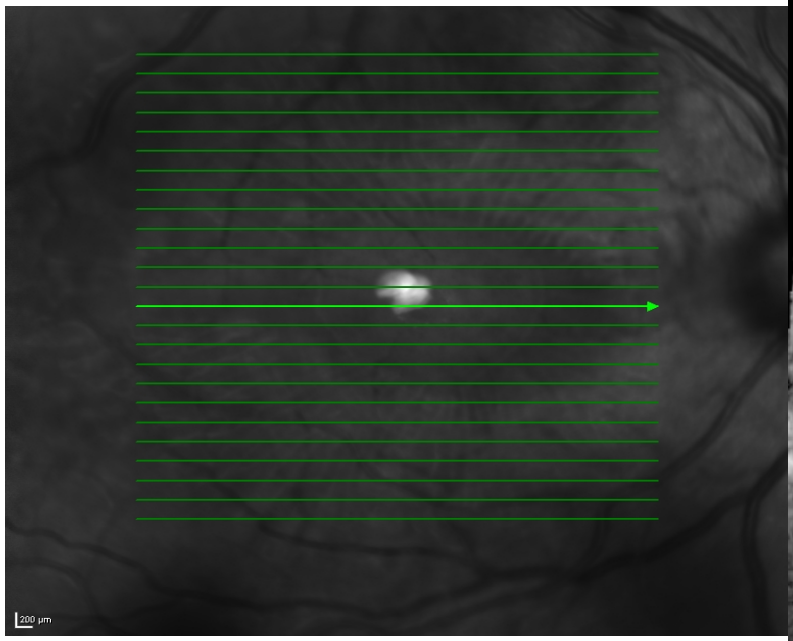
Quelques autres cas ….







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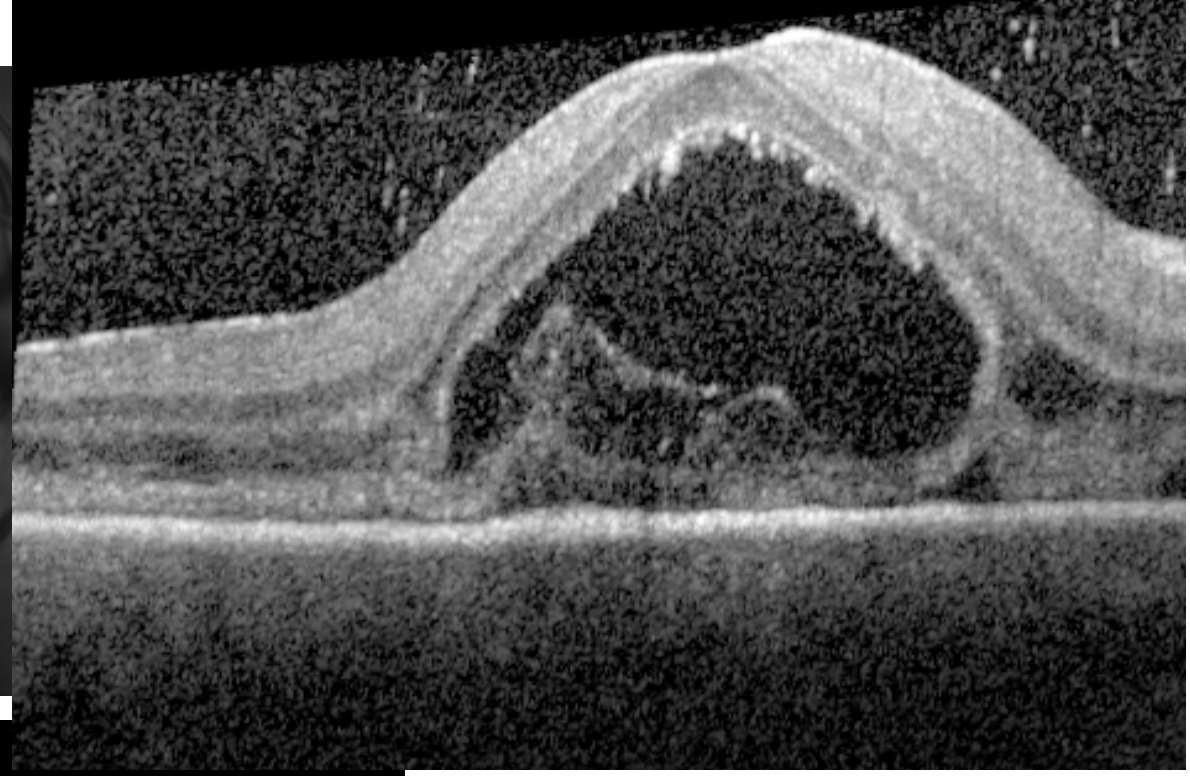
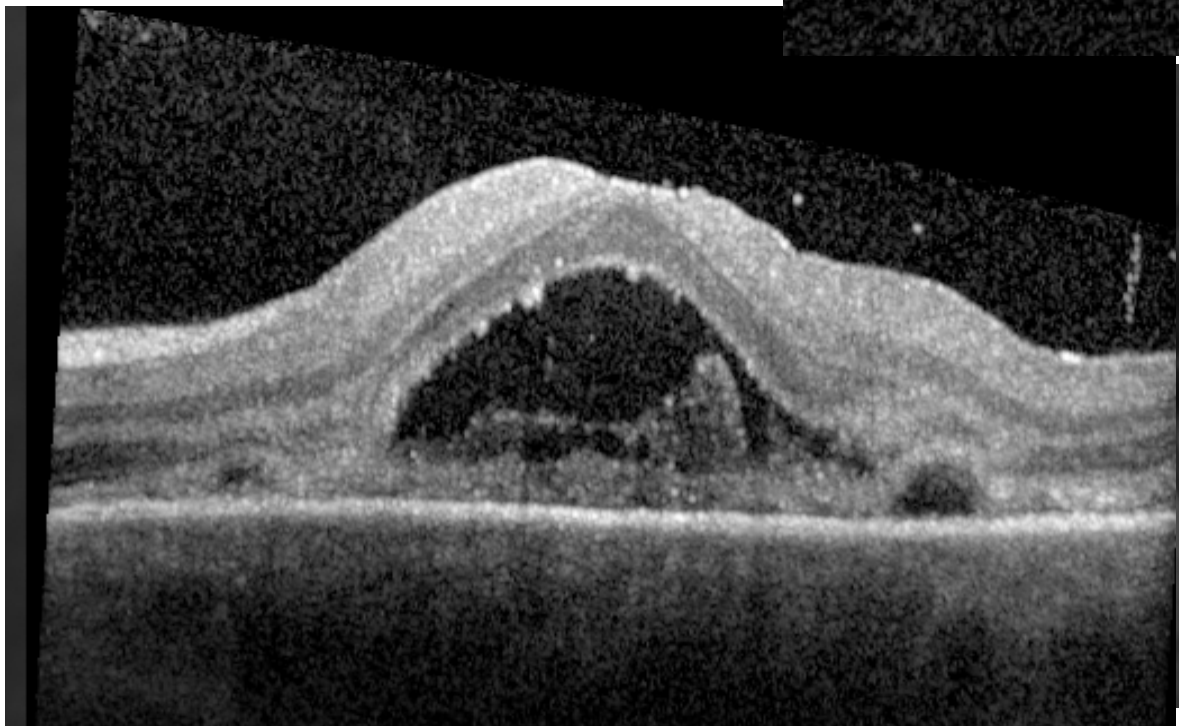
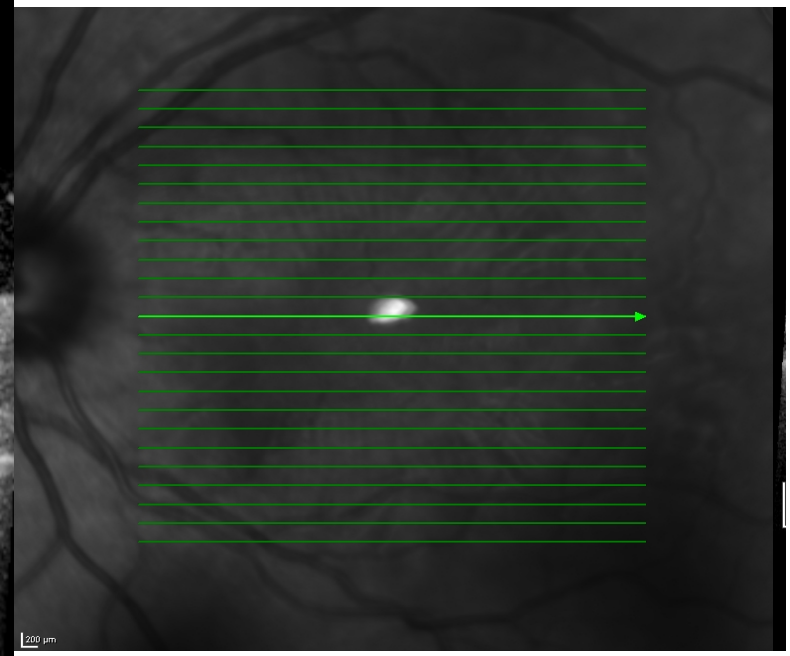
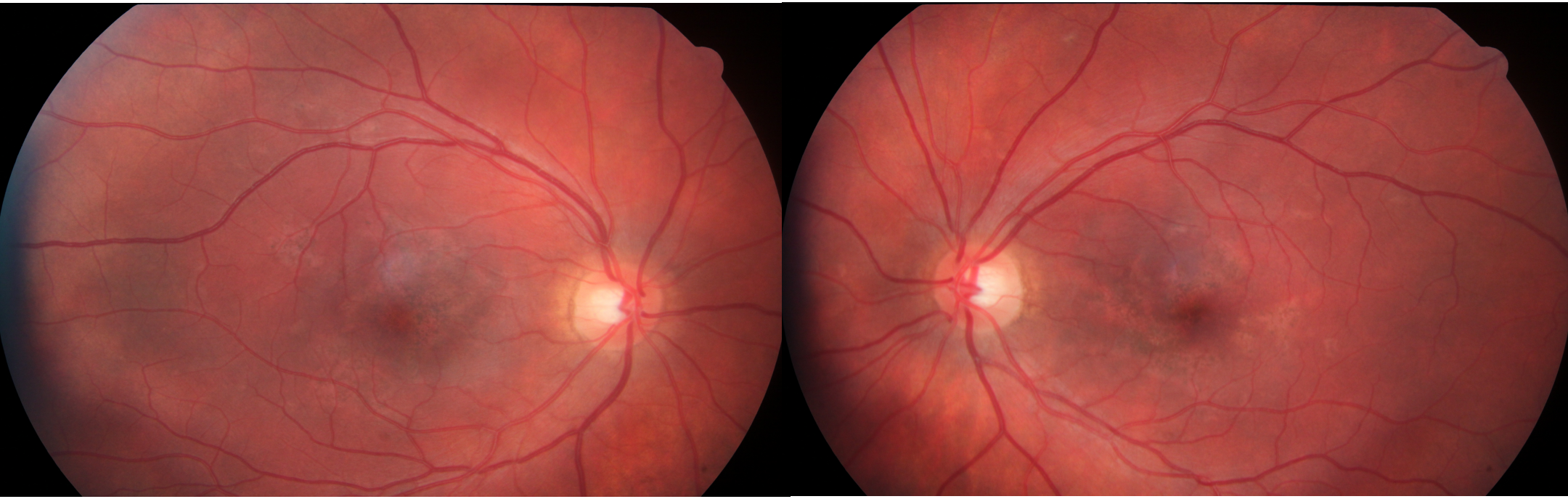
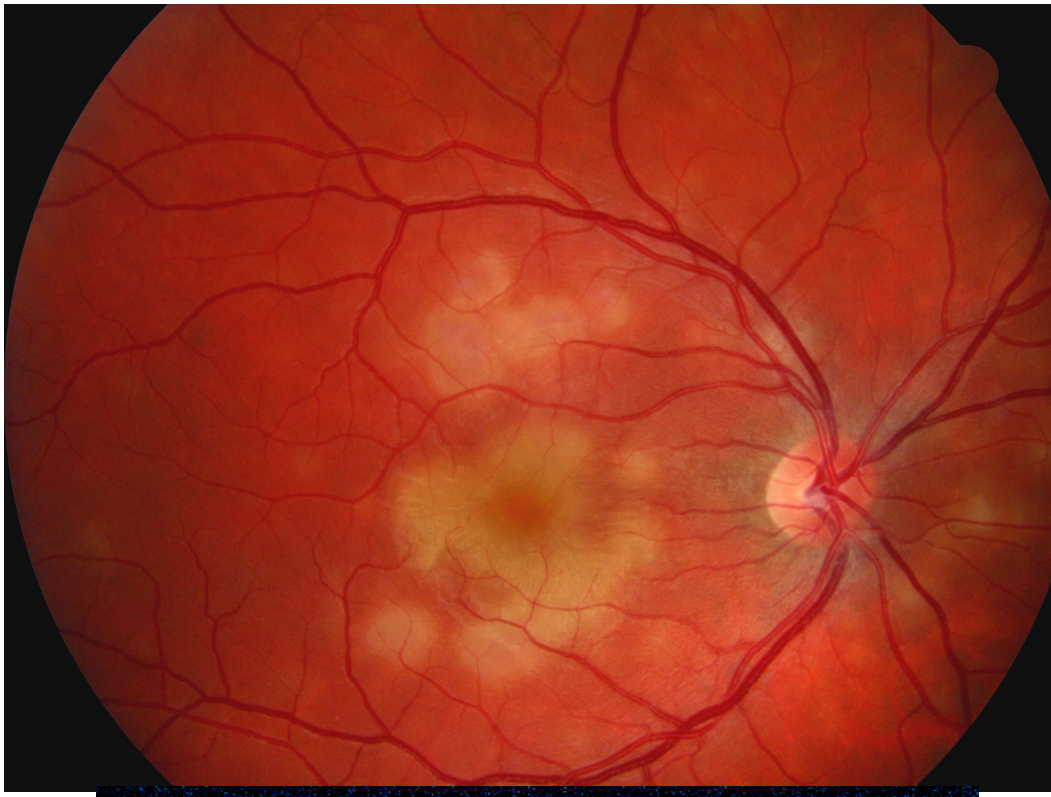


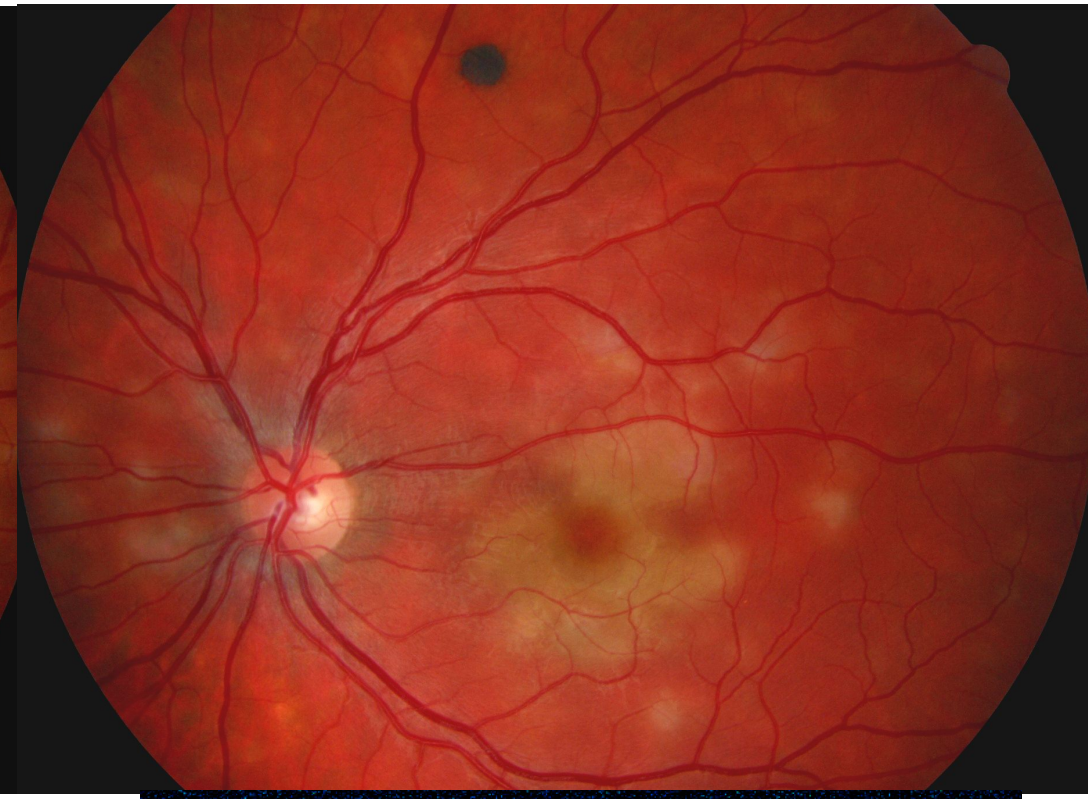


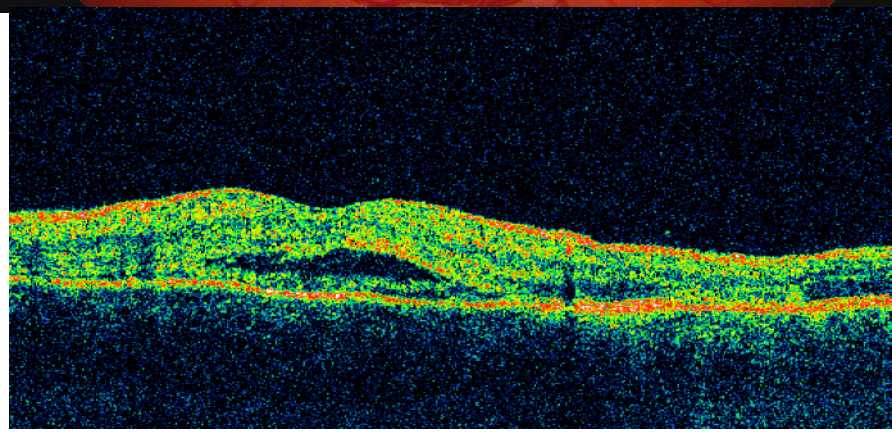

200 µm

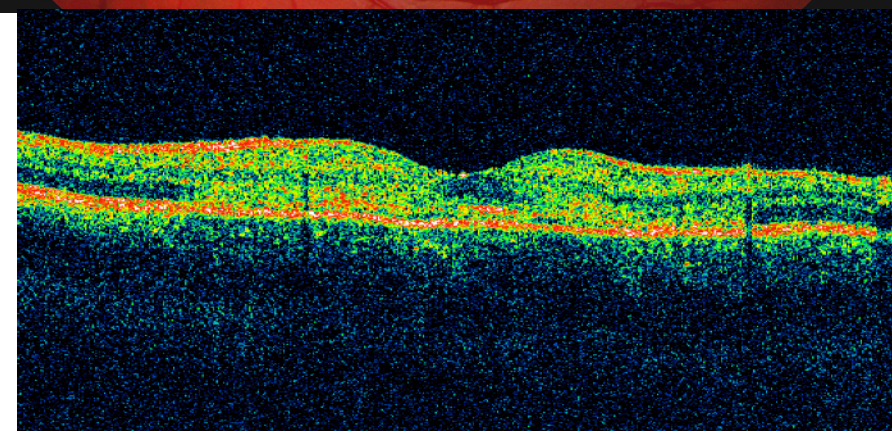
3 mois plus tard





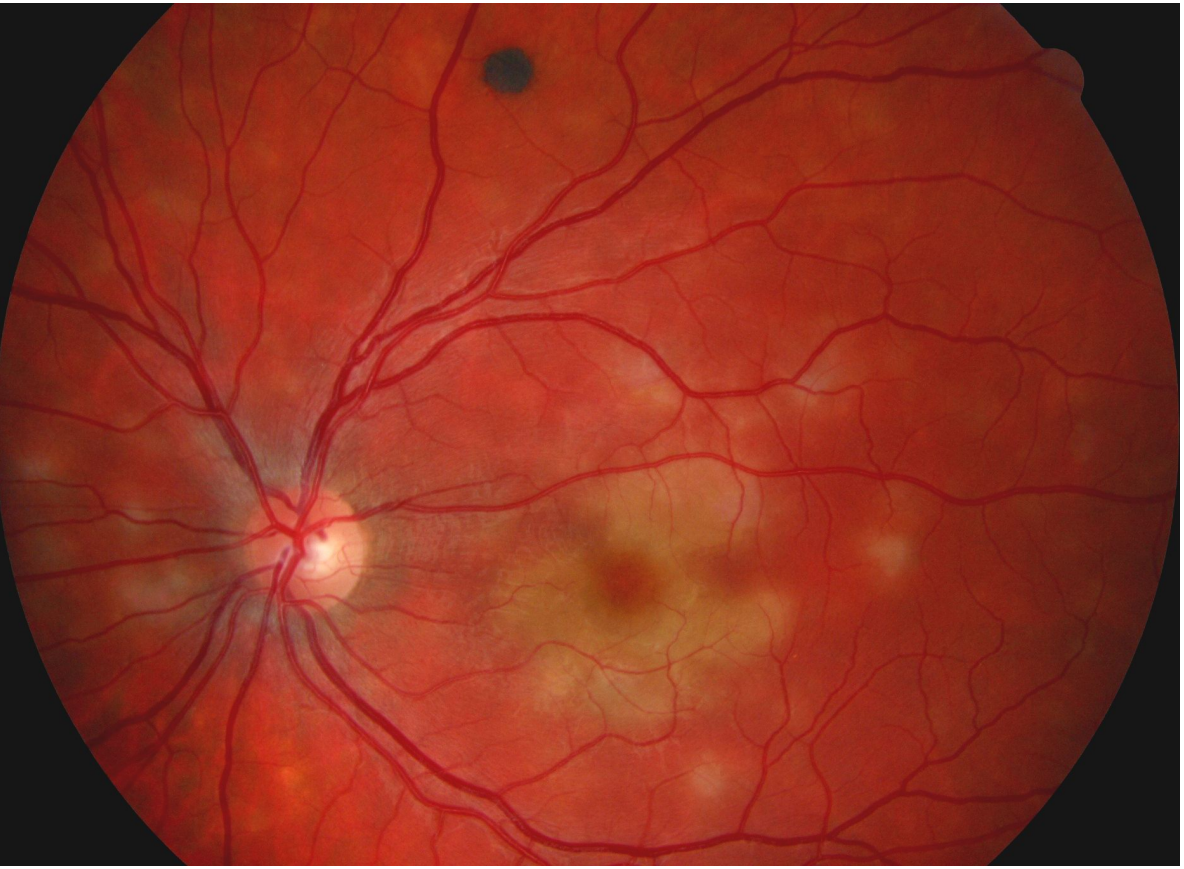


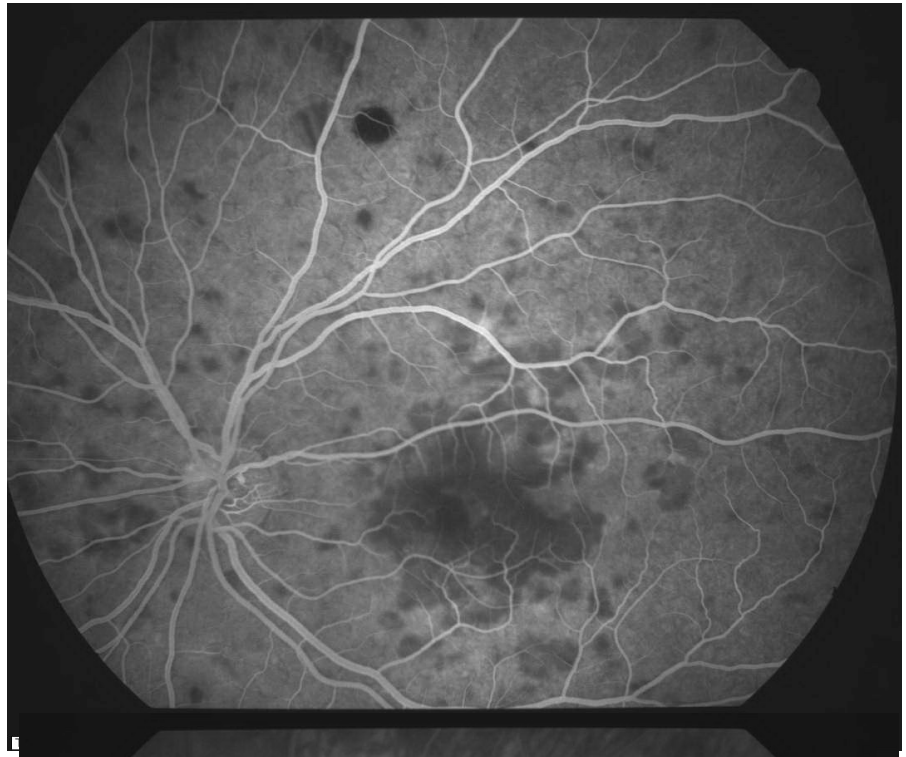


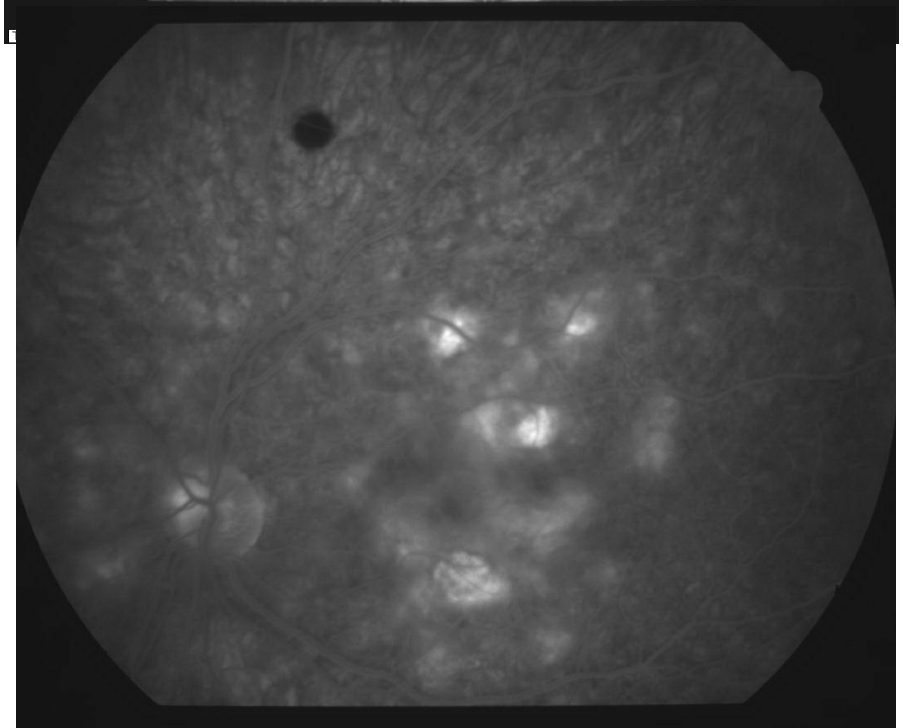


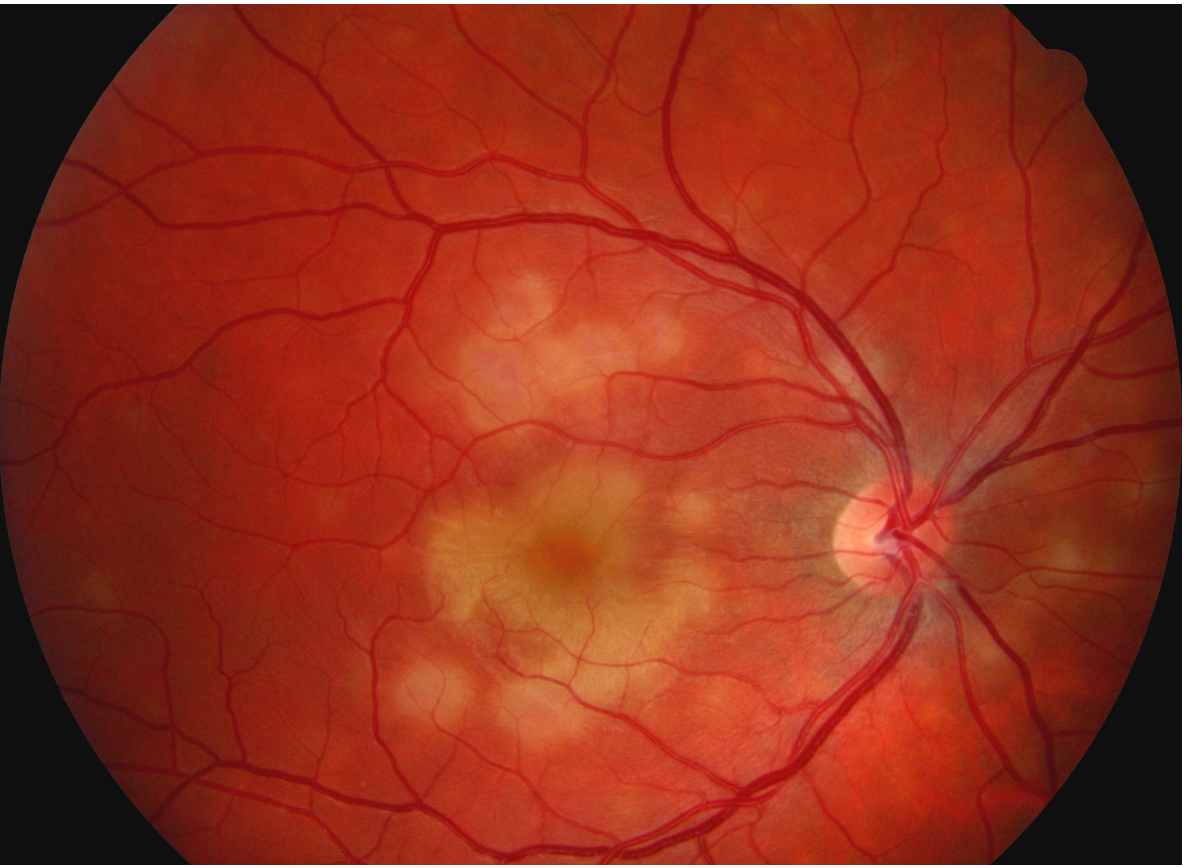
AV 1/20

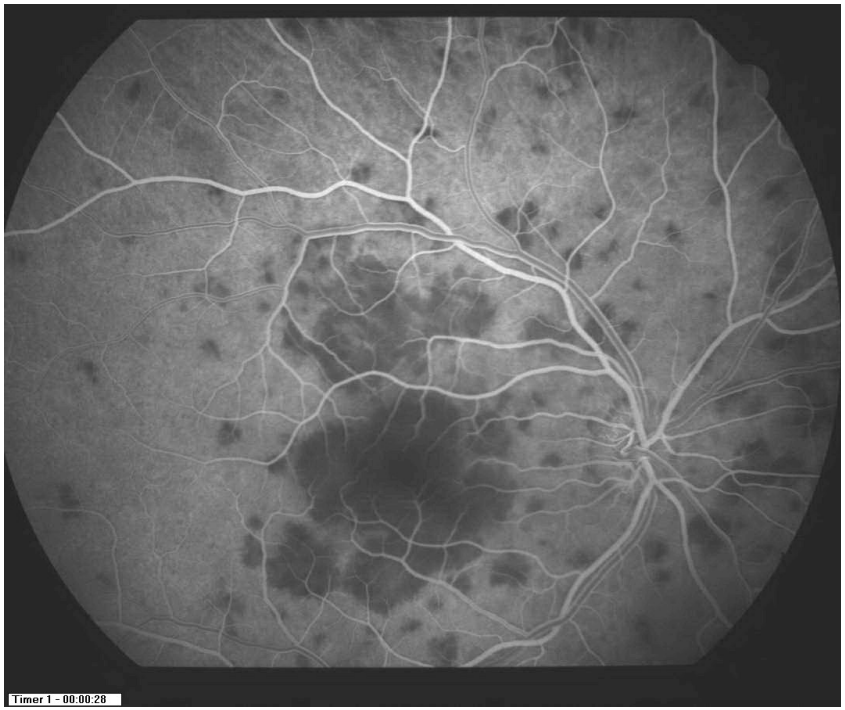
AV 4/10







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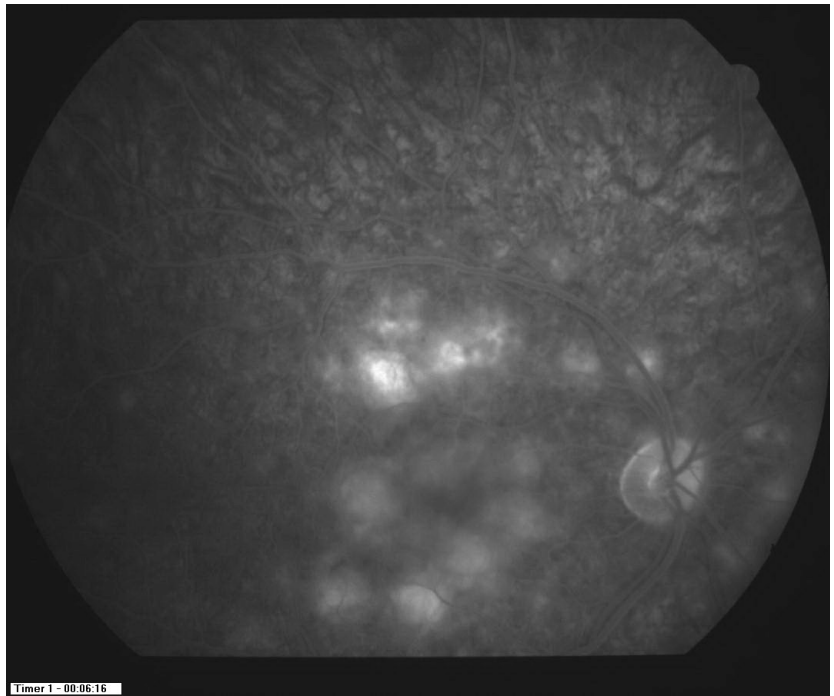
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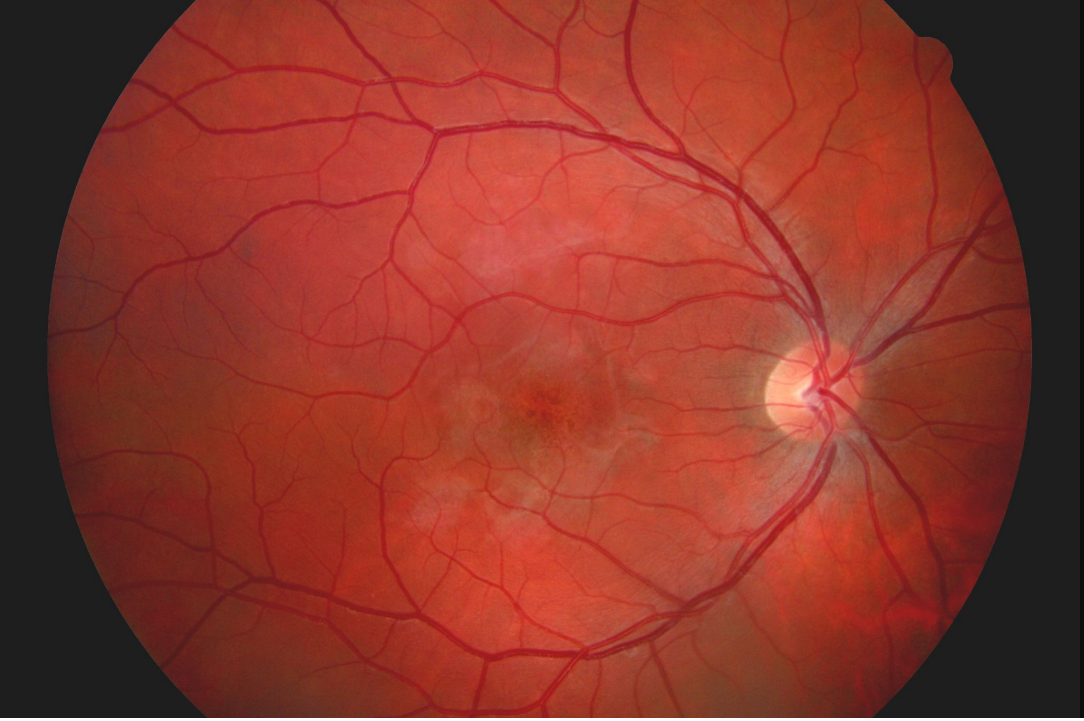

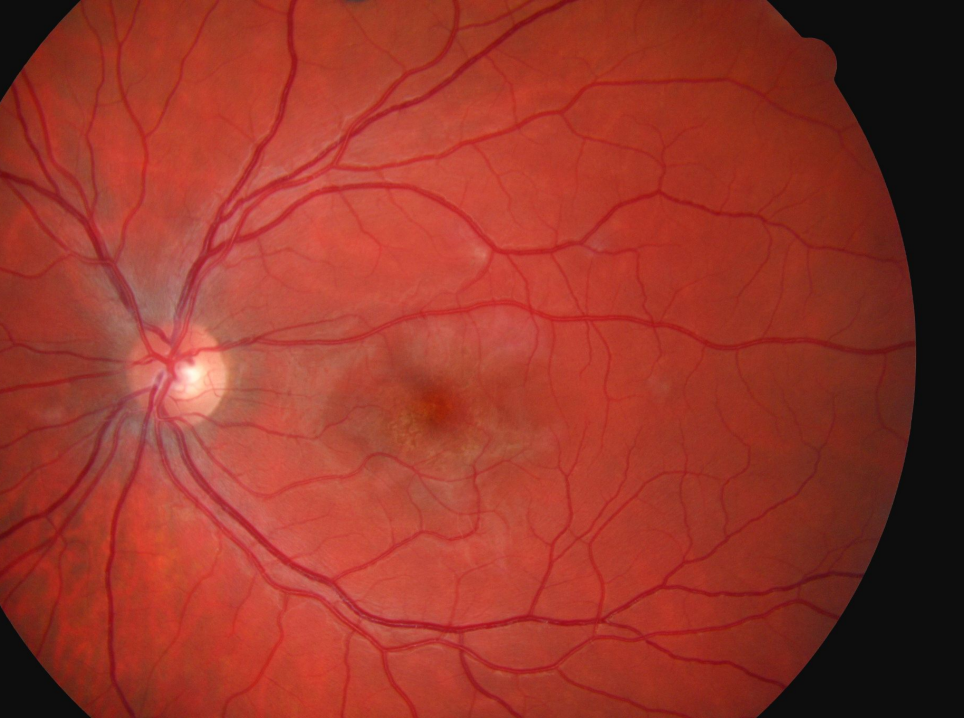

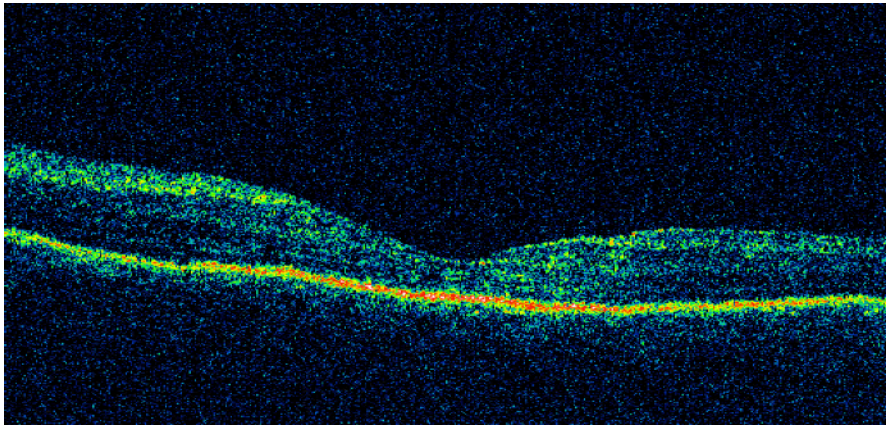

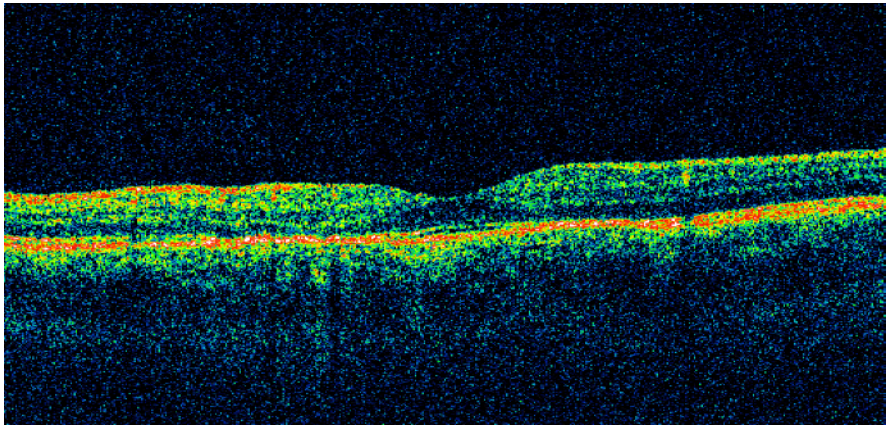

AV 10/10

AV 10/10