Hôpital Lariboisière
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AP-HP.Nord

Université Paris Cité

HÔPITAL FONDATION
Adolphe de ROTHSCHILD
LA RÉFÉRENCE TÊTE ET COU

ASPECTS DE L'ISCHÉMIE CHOROÏDIENNE AIGÜE

Alain Gaudric

Service d'Ophtalmologie
Hôpital Lariboisière

Aucun conflit d'intérêt

L'ischémie peut être sectorielle ou multifocale

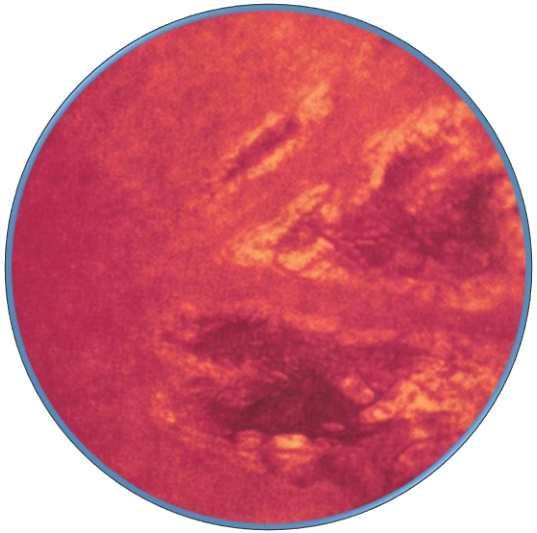
Ischémie choroïdienne en secteur

- ISCHÉMIE CHOROÏDIENNE EN SECTEUR
  - Maladie de Horton
- ISCHÉMIE CHOROÏDIENNE MULTIFOCALE
  - Toxémie gravidique
  - Rétinopathie hypertensive maligne
  - Maladie de Harada
  - Expérimentation animale
  - Épithéliopathie en plaques
  - Choroidite serpigineuse

Ischémie choroïdienne en secteur

SYNDROME TRIANGULAIRE

P. Amalric Bull Soc Oph Fr , 1963



ACUTE CHOROIDAL ISCHÆMIA

P. AMALRIC (Albi, France)

SINCE 1958, we have been publishing papers on the clinical investigation of the choroidal vessels.

In 1963, the long posterior ciliary artery was easily visualized with direct ophthalmoscopy or diaphanoscopy, but we had to wait for new techniques to be discovered to be able to visualize the short posterior ciliary arteries. The advent of fluorescein angiography and retinography with monochromatic light allowed a far better and more precise investigation of the choroid. We do not want to mention again the anatomical details, but we would like to state that it is possible to compare Prof. Ashton's drawing after neoprene impregnation with our clinical studies realized after several verifications.

Now, we know that the short posterior ciliary arteries appear inside the globe, all around an oval area, defined by the papillo-macular region. From there, they spread like spokes towards the periphery and they become more and more autonomous; anastomosed at their origin, they are no more connected at their terminal dividing. They participate there to the formation of the great arterial circle of the iris.

Anastomoses are numerous between these different vessels, particularly in the central parts of fundus.

Moreover, we must say that at the capillary level the choroid forms an important vascular network richly connected, which is for many people a sufficient element to prevent any system-atization of the vascular lesions of this area.

But this anatomical concept cannot have an absolute strict-ness for the clinician.

I think we must accept a concept of functional anatomy before discussing the clinical problem.

Well known to the clinicians, in the last century, it explained some typical choroidal appearances.

Modern techniques allow us to-day to establish a clinical and angiographic distinction between the appearances of choroidal ischæmia due either to a localized arteriolo-capillary obliteration

305

P. Amalric 1923-1999

Amalric P. Acute Choroidal Ischemia. Trans Ophthalmol Soc U K. 1971;91:305-322.

Communications

Occlusion of the posterior ciliary artery

I. Effects on choroidal circulation

SOHAN SINGH HAYREH AND JILLIANE A. B. BAINES
Department of Ophthalmology, University of Edinburgh

The literature contains no reference to the effects *in vivo* of occlusion of the posterior ciliar artery (PCA) on the choroidal circulation. It has simply been assumed that occlusio of one or more PCAs is not likely to produce any filling defect in the choroid because th choroidal vascular bed has been described as being one continuous bed, with no segmenta distribution (Nicholls, 1938; Vilstrup, 1952; Wybar, 1954a, b; Correia, 1957; Scullica 1957; Ruskell, 1961; Ring and Fujino, 1967). Our experimental studies involvin occlusion of the PCAs in rhesus monkeys have, on the contrary, revealed that such a assumption is entirely incorrect and that the distribution of the PCAs is, in fact, segmenta These studies have also led to many interesting observations which are reported below.

There is a good deal of confusion as to the nomenclature, number, origin, and distribu tion of the PCAs. One of us has helped to clarify these subjects (Hayreh, 1962, 196 1970, 1971). Briefly, the ophthalmic artery in humans gives out one (in 3 per cent. two (in 48 per cent.), or three (in 39 per cent.) PCAs. Each artery divides into multipl branches before piercing the sclera, medial (by the medial PCA) or lateral (by the later PCA) to the optic nerve. Of these branches, two small ones (one on the medial and th other on the lateral side) are called the long PCAs, while the rest are the short PCAs.

Material

The study was carried out in 85 rhesus monkey eyes.

Methods

By lateral orbitotomy, the PCAs were cauterized near their site of entry into the eyeball, leaving small arterial stump close to the globe as follows:

Lateral PCAs (LPCAs) in 31 eyes
Medial PCAs (MPCAs) in 17 eyes
All PCAs (APCAs) in 37 eyes

The Table (overleaf) shows the follow-up period after PCA occlusion in 85 eyes of rhesus monke The choroidal circulation in these eyes was assessed by repeated intravenous fluorescence angi graphy (IVFA).

Received for publication January 13, 1972
Address for reprints: Department of Ophthalmology, Princess Alexandra Eye Pavilion, Chalmers Street, Edinburgh, EH3 9HA
This project was supported by a grant from The Medical Research Council

Occlusion of the posterior ciliary artery

II. Chorio-retinal lesions

SOHAN SINGH HAYREH AND JILLIANE A. B. BAINES
Department of Ophthalmology, University of Edinburgh

The clinical picture of central retinal artery occlusion is well-known. Little is known, however, about the clinical picture of occlusion of the posterior ciliary arteries (PCAs). We have, therefore, carried out experimental occlusion of the various PCAs individually or together in rhesus monkeys. The effects of such occlusions have been investigated in the choroid, pigment epithelium (PE), and retina of these monkeys over a period of time, ophthalmoscopically, by intravenous fluorescence fundus angiography (IVFA), and (after death) by histology. The study has revealed fundus lesions the nature of which has hitherto been obscure.

There are usually two to three PCAs, arising from the ophthalmic artery, which supply the posterior half of the choroid up to the equator of the eye. These are designated medial (MPCA) and lateral (LPCA), depending upon their relationship to the optic nerve near their site of entry into the sclera. A detailed account of the anatomy of the PCAs is given elsewhere (Hayreh, 1962, 1970).

The effects of occlusion of the various PCAs on the choroida been described in detail (Hayreh and Baines, 1972). Briefly, th in the choroid was seen to be segmental, so that occlusion of perfusion of the choroid in that region during the transit of the a very late and sluggish patchy filling of the choroid *via* the place; this improved with time, so that in 2 to 4 weeks the restored.

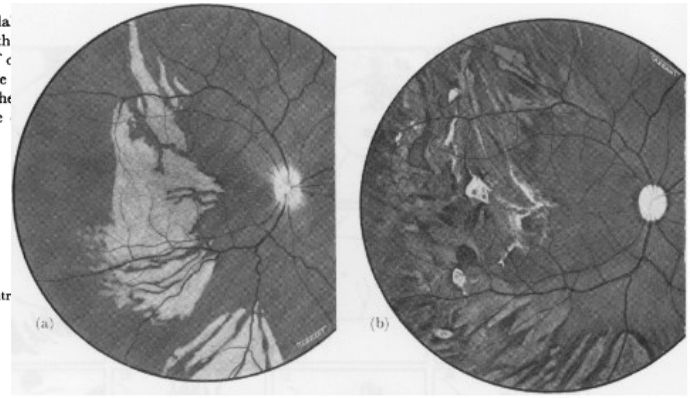
Material

The study was carried out in 85 rhesus monkey eyes.

Methods

By lateral orbitotomy, the PCAs were cauterized near their site of entr small arterial stump close to the globe as follows:

Lateral PCAs (LPCAs) in 31 eyes.
Medial PCAs (MPCAs) in 17 eyes.
All PCAs (APCAs) in 37 eyes.

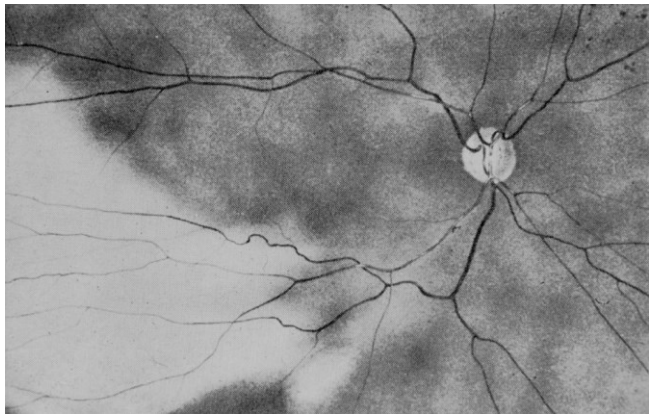


Received for publication January 17, 1971
Address for reprints: Department of Ophthalmology, Princess Alexandra Eye Pavilion, Chalmers Street, Edinburgh EH3 9HA
This project was supported by a grant from the Medical Research Council

SS Hayreh

Ischémie choroïdienne en secteur

- La forme avec nécrose blanche aigüe de l'EP est rare



Foulds, W. S et al (1971). "Clinical and pathological aspects of choroidal ischaemia." Trans Ophthalmol Soc U K 91: 323-341.

Spolaore, R., A. Gaudric, et al (1984). "Acute sectorial choroidal ischemia." Am J Ophthalmol 98(6): 707-716.

CHOROIDAL ISCHEMIA

A. Gaudric, M.D., and G. Coscas, M.D.
Paris, France
and
A. C. Bird, M.D.
London, England

Nine patients, seven women and two men, ranging in age from 21 to 86 years, had generalized vascular disease and significant deficits in choroidal perfusion but no retinal vascular changes. Because these perfusion abnormalities can be identified only by fluorescein angiography and the fundus appears to be normal, choroidal ischemia may be more common than has been realized. Infarction of the choroid, retinal pigment epithelium, and outer retina are usually unassociated with retinal detachment; even ischemia severe enough to induce retinal detachment seldom produces significant atrophy.

Choroidal ischemia has received less attention than retinal vascular disease. Published reports describe two well-defined and distinct manifestations of reduced choroidal blood flow in patients with generalized vascular disease. Large triangular infarcts may occur in the acute phase in the form of areas of opacification of the retinal pigment epithelium and outer sensory retina[1,2] or, later, in the form of well-defined regions of scarring.[3,4] These lesions have been ascribed to obstruction of short posterior ciliary arteries and have their counterparts in experimental arterial obstruction in animals.[5] The second form of disease typically occurs in accelerated hypertension in which there is multifocal opacification of the outer retina and serous detachment of the sensory retina. It has been suggested that there is widespread obstruction of choroidal arterioles in such cases.

During the last decade there has been progressively more support for the theory that choroidal vascular disease may be responsible for retinal pigment epithelial ischemia in the young in the absence of obvious vascular disease elsewhere,[6-8] appearing as multifocal opacification of the retinal pigment epithelium[9] or as retinal detachment,[10] although the evidence for ischemia in such cases is circumstantial.

We examined a series of nine patients with documented generalized vascular disorders and a wide variety of manifestations of presumed choroidal vascular disease.

Subjects and Methods

Nine patients with generalized vascular disease had well-documented disturbances of choroidal circulation in one eye (four patients) or both eyes (five patients) without significant retinal vascular dis-

Accepted for publication May 24, 1982.
From the Centre Hospitalier Intercommunal, Paris, France (Drs. Gaudric and Coscas), and the Institute of Ophthalmology, Moorfields Eye Hospital, London, England (Dr. Bird).
Reprint requests to A. C. Bird, M.D., Institute of Ophthalmology, Moorfields Eye Hospital, City Road, London EC1V 2PD, England.

ACUTE SECTORIAL CHOROIDAL ISCHEMIA

Roberto Spolaore, M.D.
Conegliano, Italy
and
Alain Gaudric, M.D., Gabriel Coscas, M.D., and Jean De Margerie, M.D.
Créteil, France

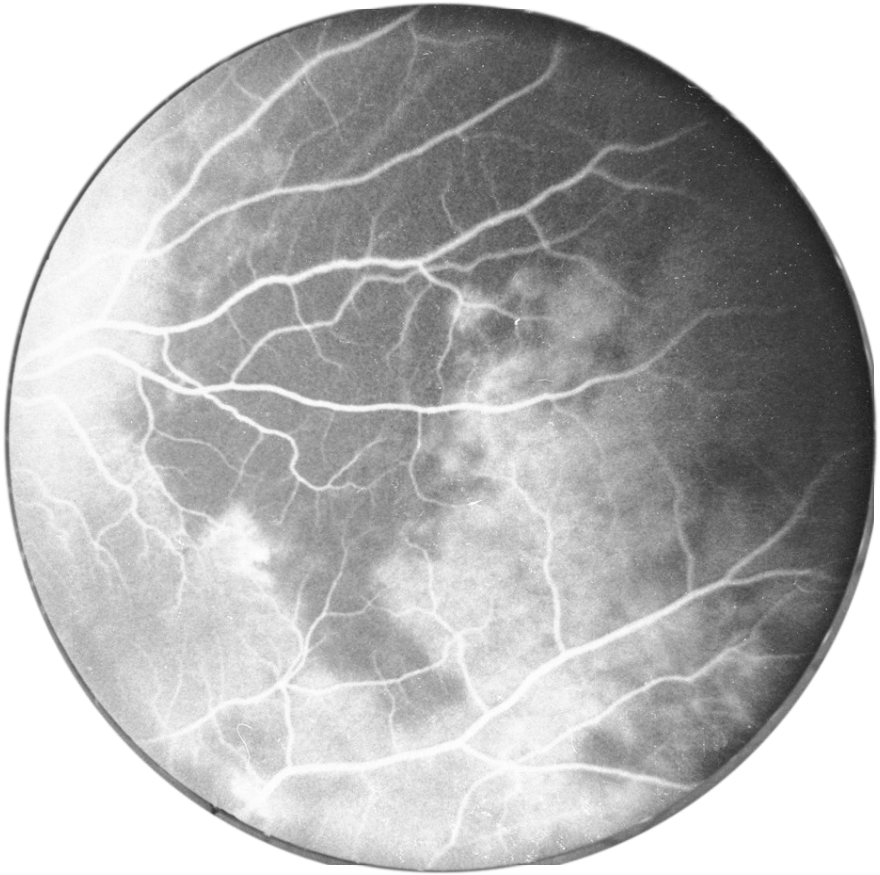
Four eyes (of three patients) with sectorial choroidal ischemia by temporal arteritis or carotid obstruction underwent fluorescein angiography during and after the acute phase. In all cases hypoperfusion affected the whole temporal choroid; however, disturbance of retinal pigment epithelium was much less extensive. In two cases the retinal pigment epithelium became necrotic and scarred within a limited area that was typically triangular in shape; in the third case scarring was limited to some pigmented streaks, and in the fourth case, retinal pigment epithelium remained normal. The triangular scar that appeared after episodes of choroidal hypoperfusion did not seem to be a true reflection of the extent of the choroidal artery obstruction in the acute phase. Our observations suggest that the deficient choroidal perfusion involved more than the choroidal artery supplying the triangular area of pigment epithelium disturbance. Several mechanisms (for example, retrograde venous filling, restoration of perfusion) reduce the deleterious effects of ischemia. Thus the necrosis of the retinal pigment epithelium would appear in only those sectors where ischemia was particularly severe or prolonged.

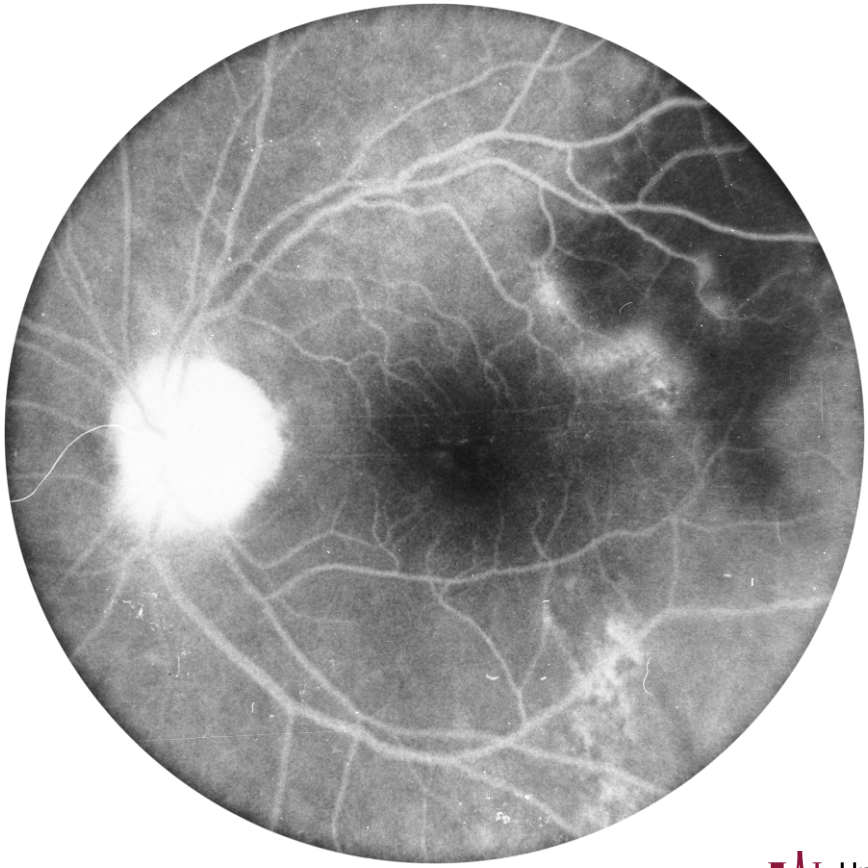
The triangular syndrome described by Amalric[1,2] is a retinochoroidal scar induced by sectorial choroidal ischemia. The late sequelae are now well recognized as part of many vascular diseases[3] but the acute phase of sectorial choroidal hypoperfusion has rarely been observed.[4-10] The few available observations and the experimental study by Goldbaum and associates[8] supported the view that the triangular scar corresponds to the nonperfused territory of a posterior ciliary artery. However, Hayreh and Baines[11,12] showed experimentally that an extensive sector of choroidal ischemia could lead to scarring of the retinal pigment epithelium over a smaller area, or even to absence of scarring.[12] Several clinical observations[4,7,14] have shown that obvious choroidal ischemia could be transient and apparently not affect the retinal pigment epithelium.

We studied three cases (four eyes) with choroidal ischemia during and after the acute phase. Fluorescein angiography disclosed that the area of hypoperfusion of the choroid noted during the acute phase is always much more extensive

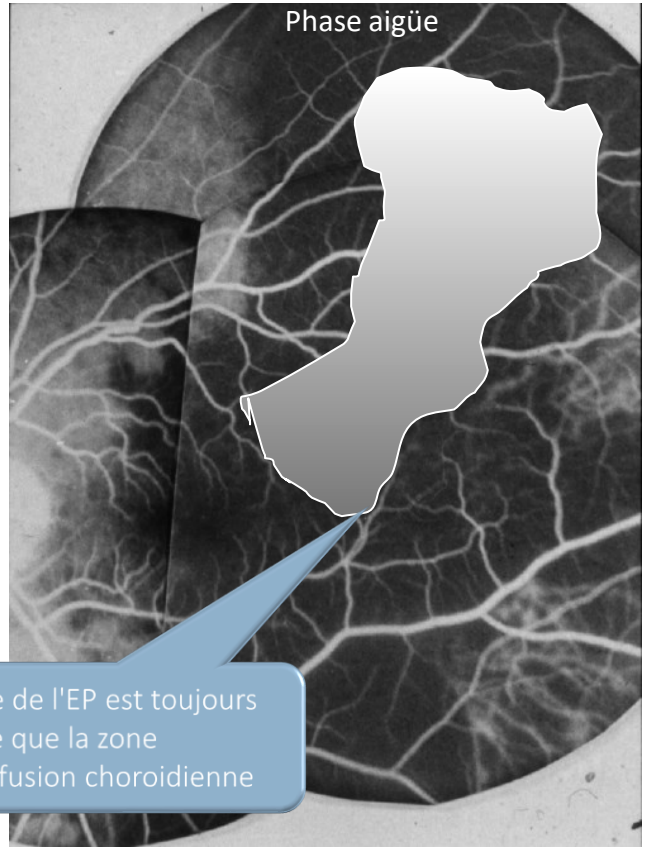
Accepted for publication Aug. 8, 1984.
From the Department of Ophthalmology, Ospedale Generale Provinciale de Conegliano, Conegliano, Italy (Dr. Spolaore); and the Clinique Ophtalmologique Universitaire de Créteil, Université Paris XII, Créteil, France (Drs. Gaudric, Coscas, and De Margerie).
Reprint requests to Alain Gaudric, M.D., Clinique Ophtalmologique Universitaire, 40, Av. de Verdun 94000 Créteil, France.

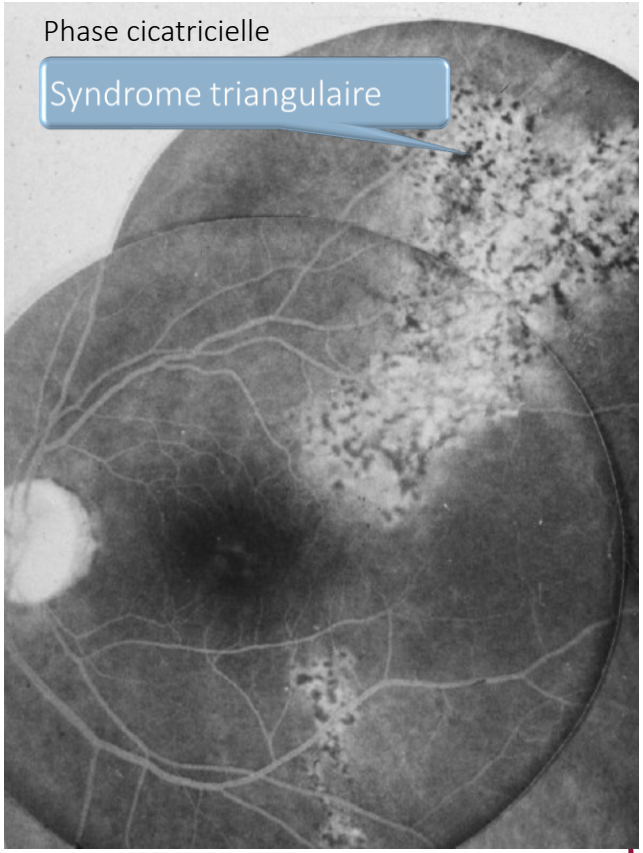
Ischémie choroïdienne en secteur





Ischémie choroïdienne en secteur



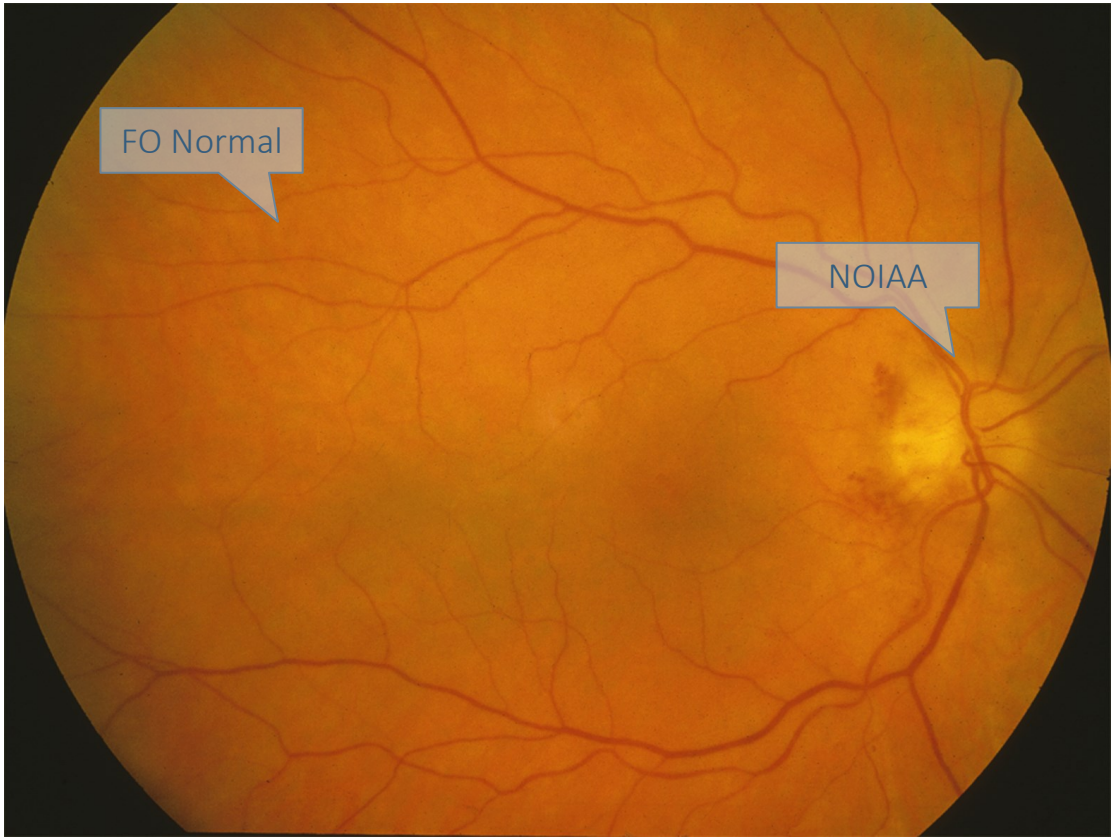


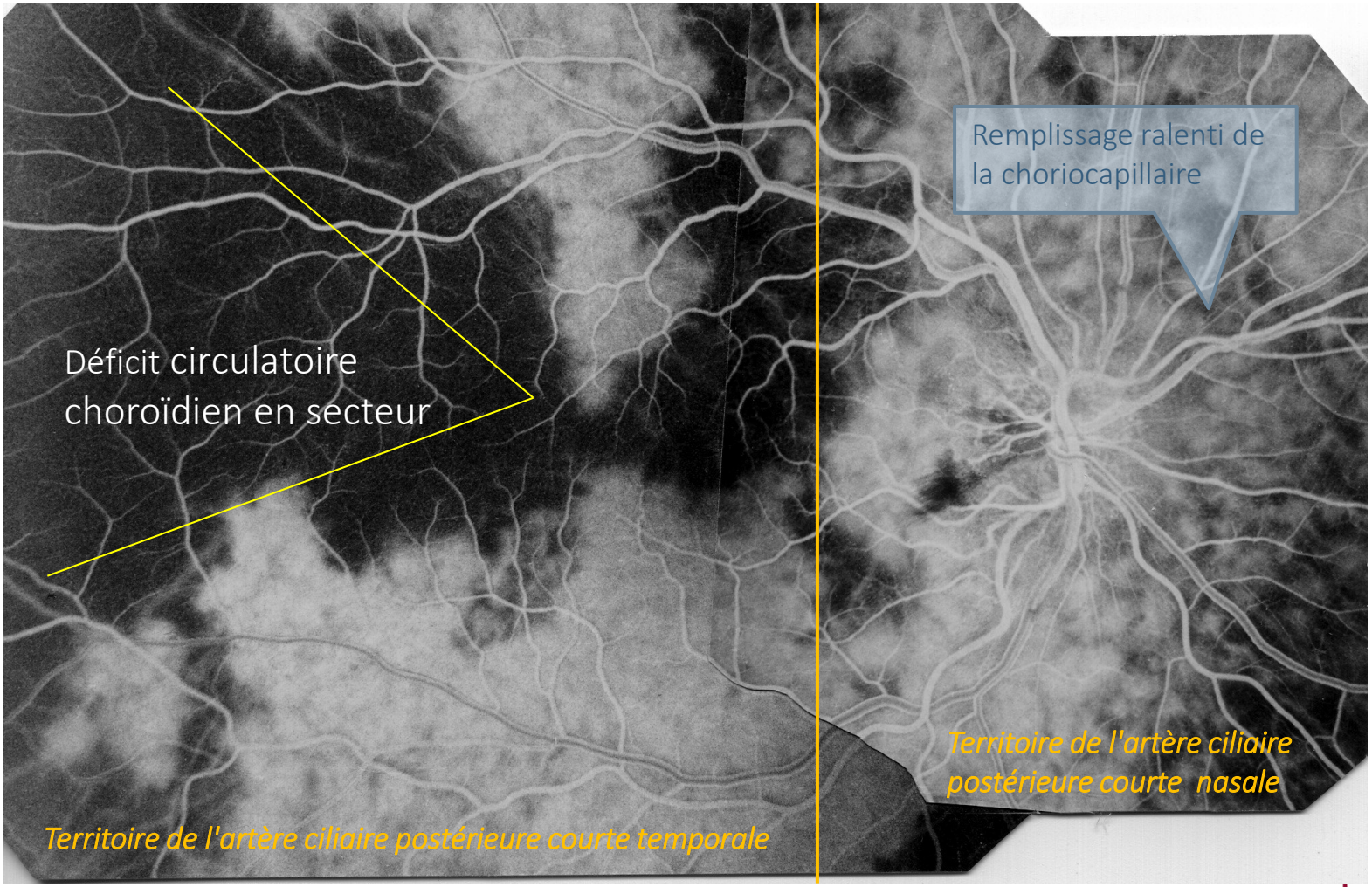
Ischémie choroïdienne en secteur

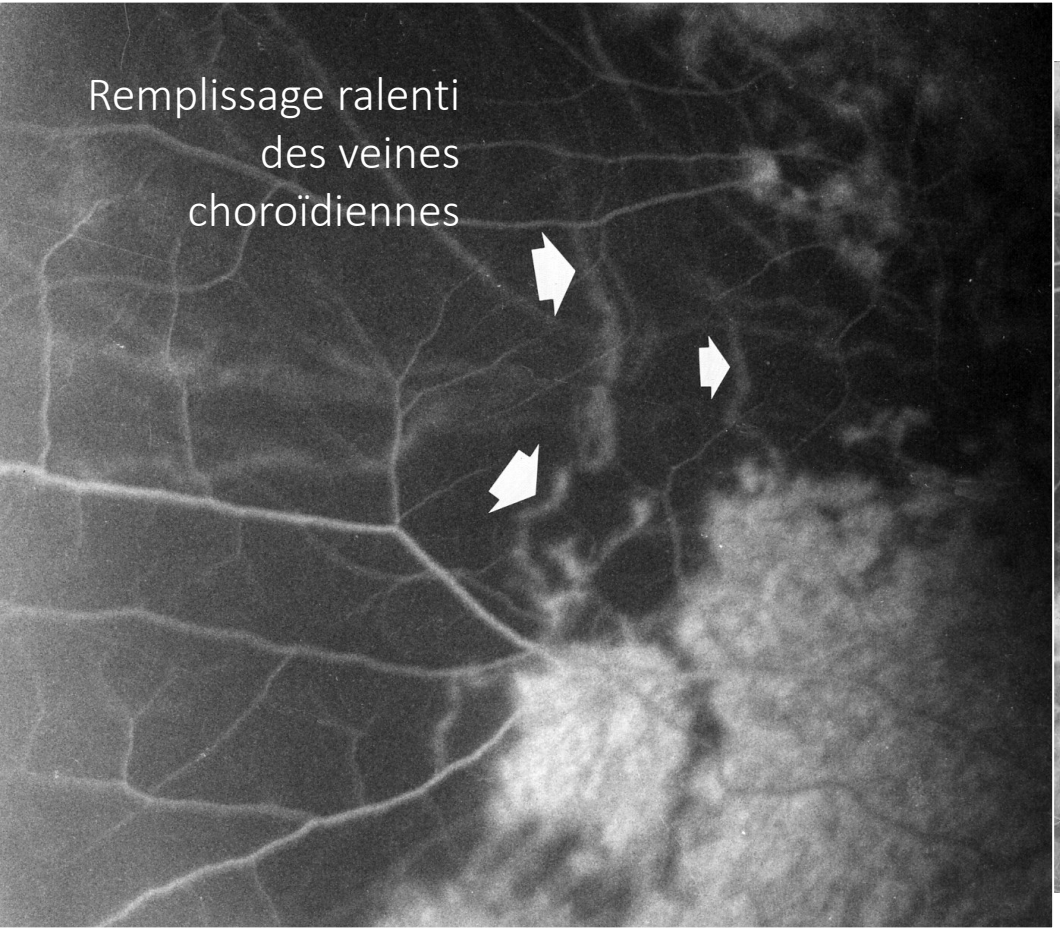
- L'EP supporte assez longtemps l'hypoperfusion choroïdienne

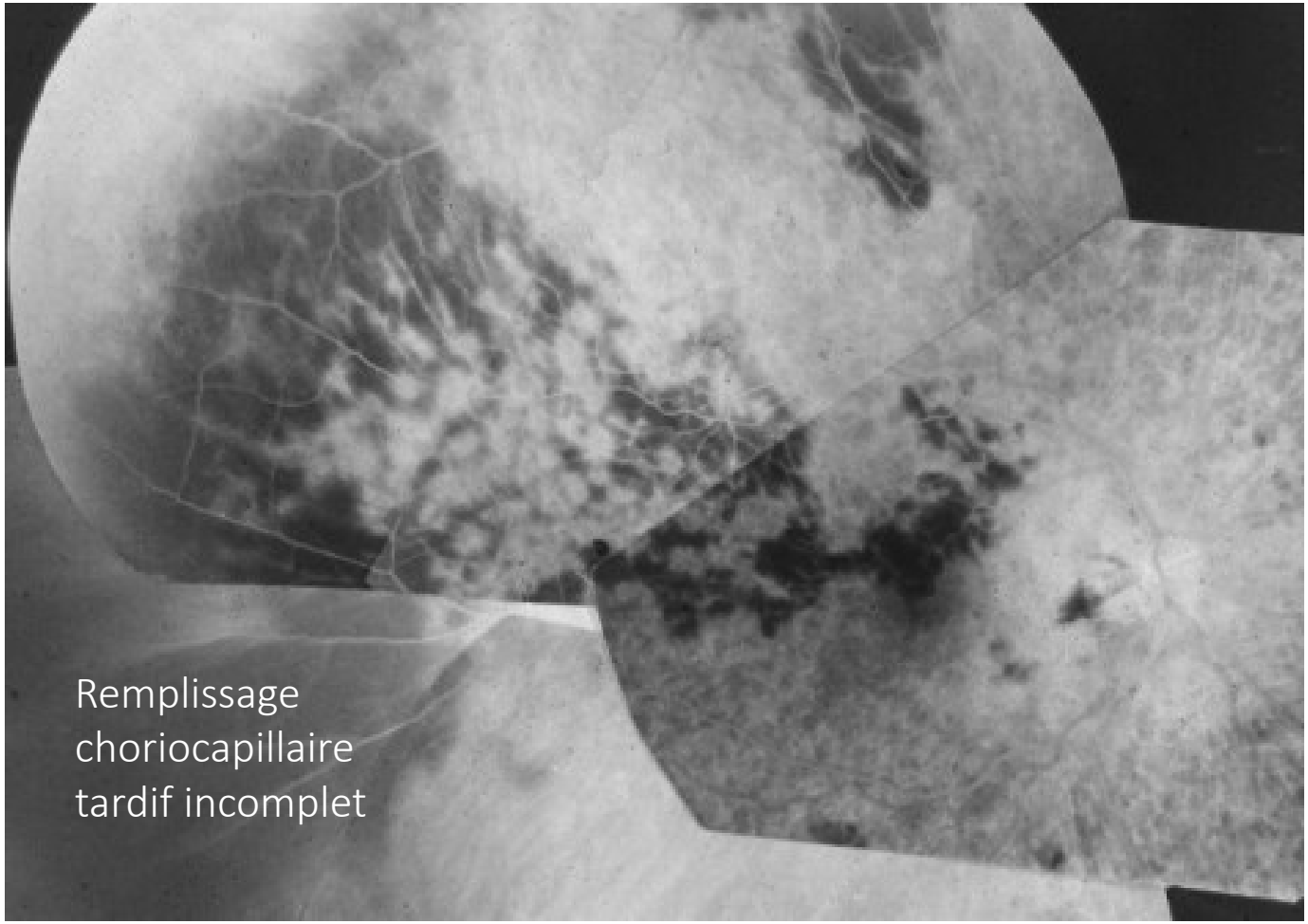
Homme 77 ans AV : 0.05

NOIA



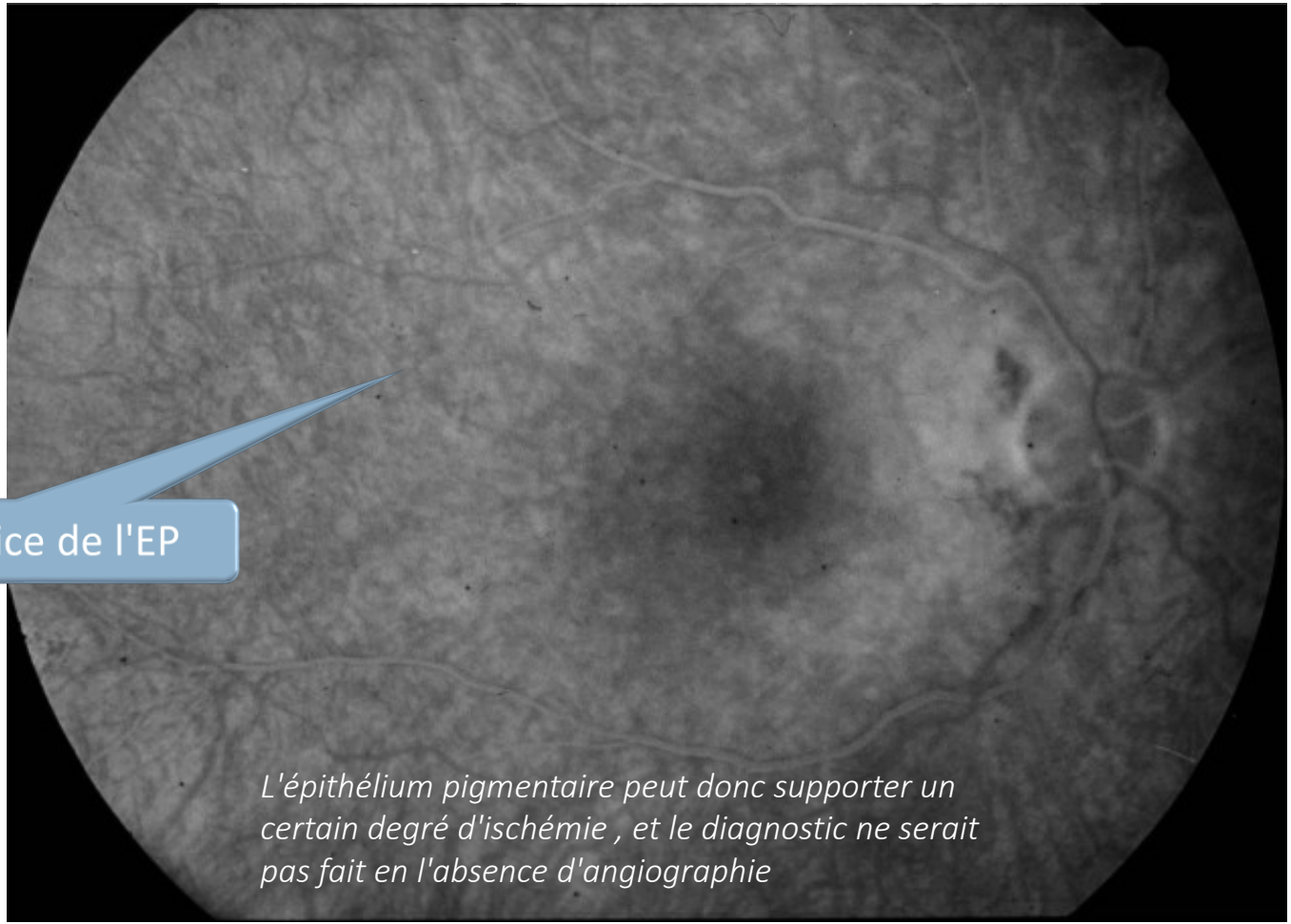

Remplissage ralenti de la choriocapillaire
Déficit circulatoire choroïdien en secteur
Territoire de l'artère ciliaire postérieure courte nasale
Territoire de l'artère ciliaire postérieure courte temporale


Remplissage ralenti
des veines
choroïdiennes

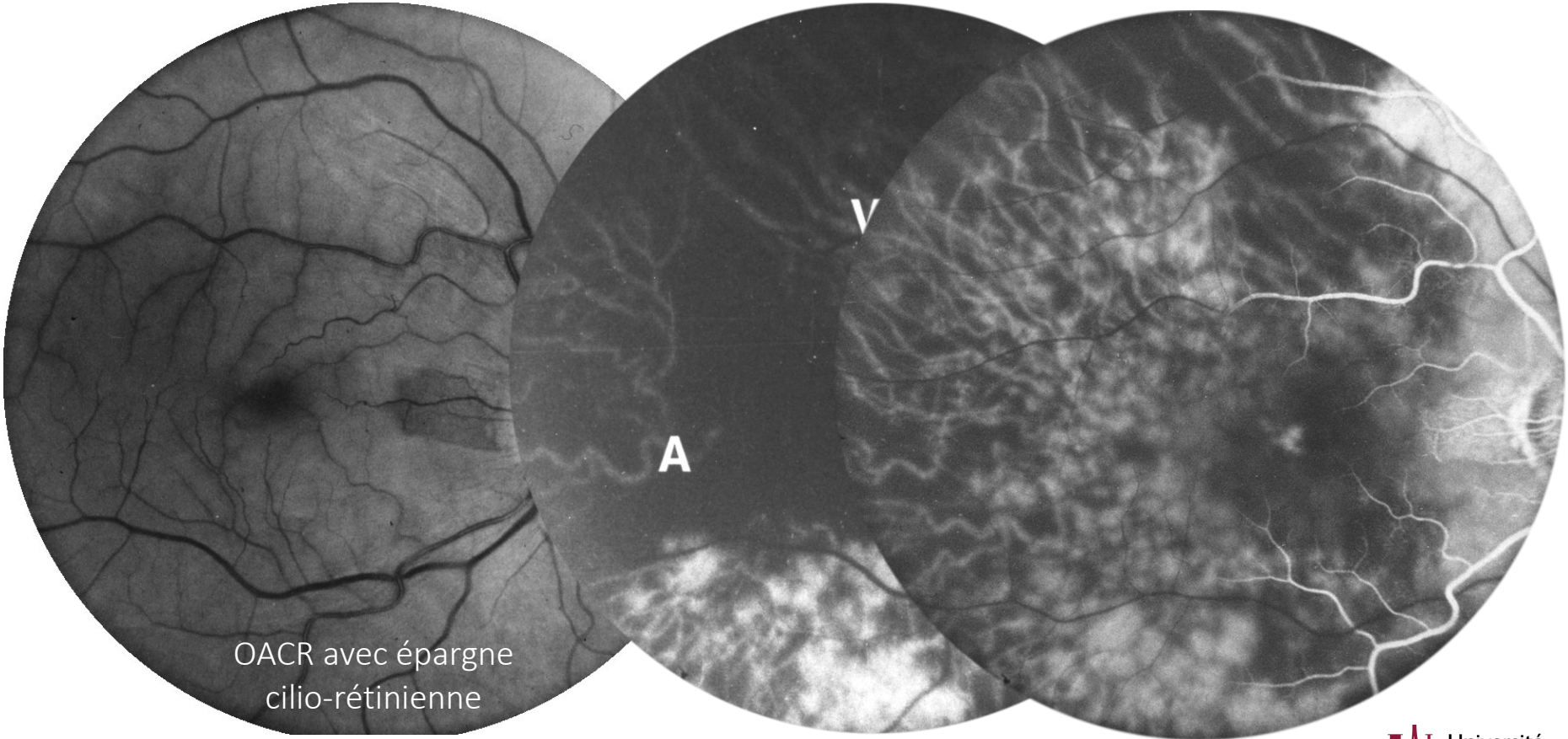

Remplissage
choriocapillaire
tardif incomplet

3 semaines après début du traitement

(Prednisolone IV puis prednisone 1mg/kg per os)

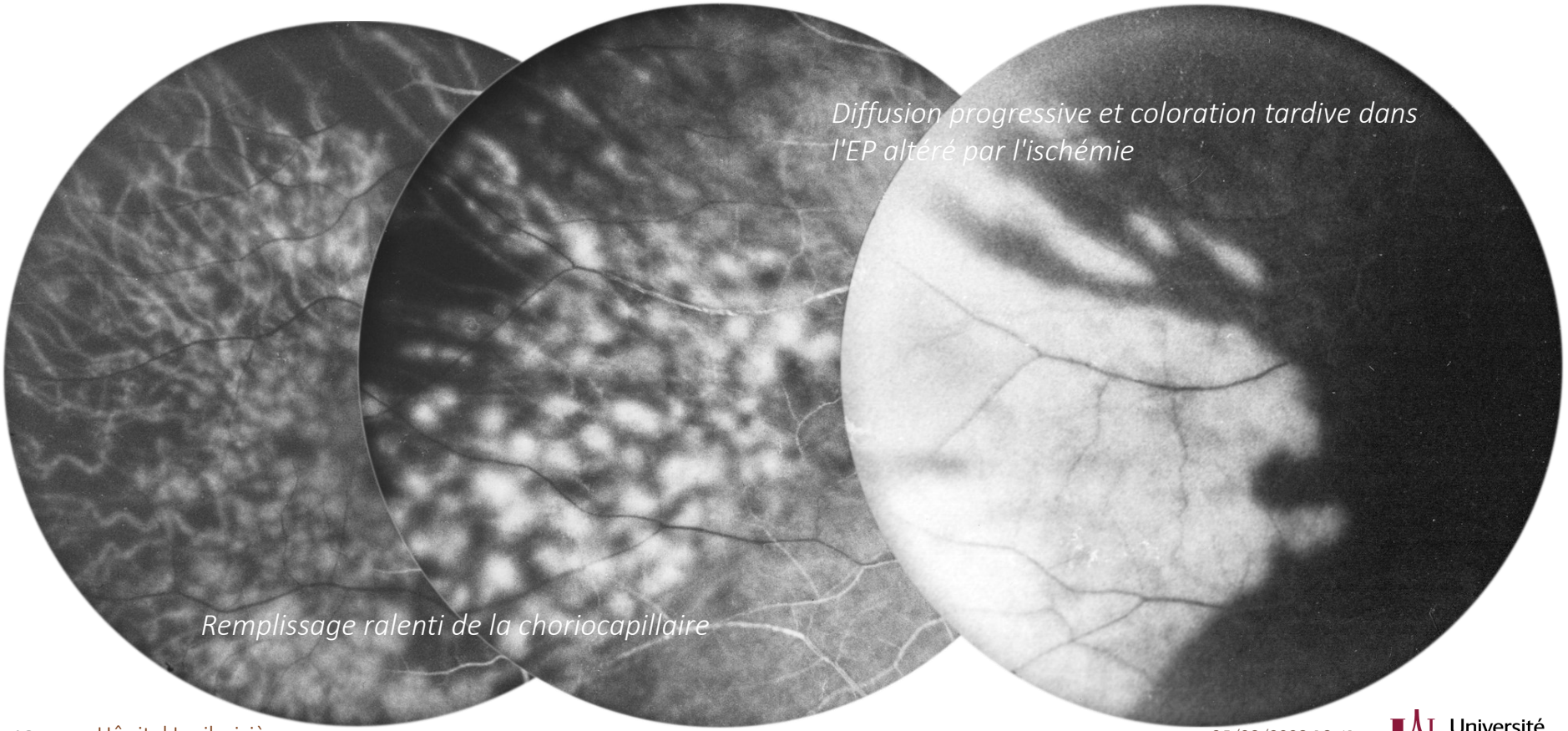


Ischémie choroïdienne aigüe en secteur

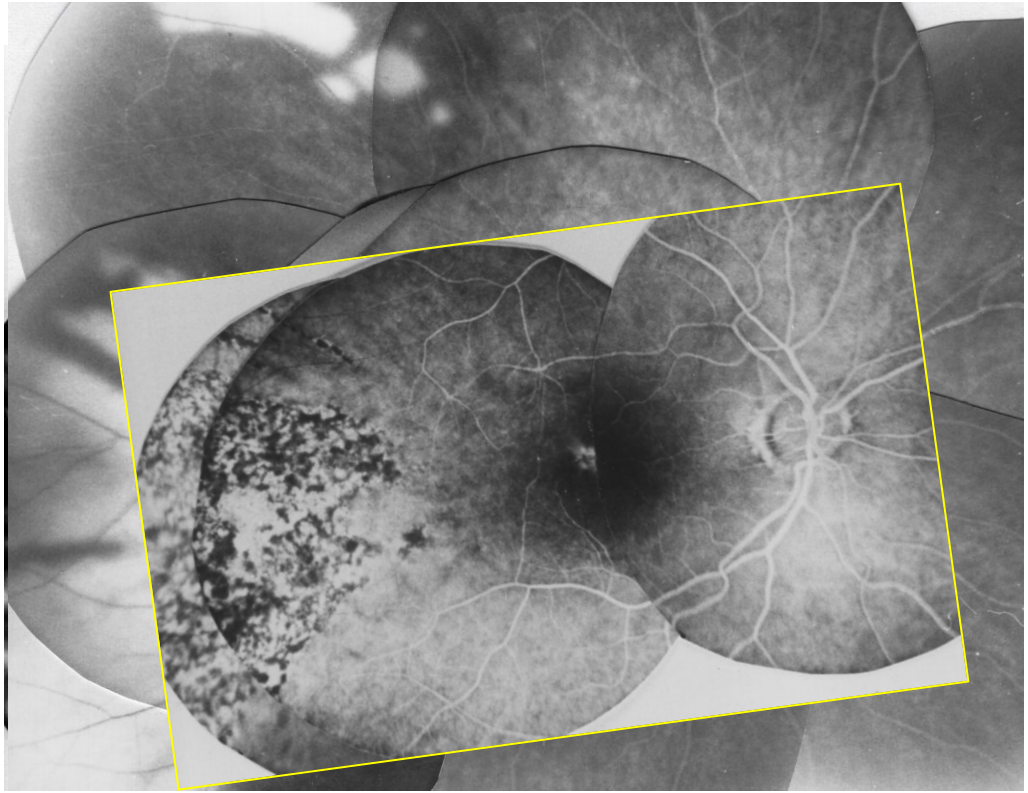


OACR avec épargne cilio-rétinienne

Ischémie choroïdienne aigüe en secteur

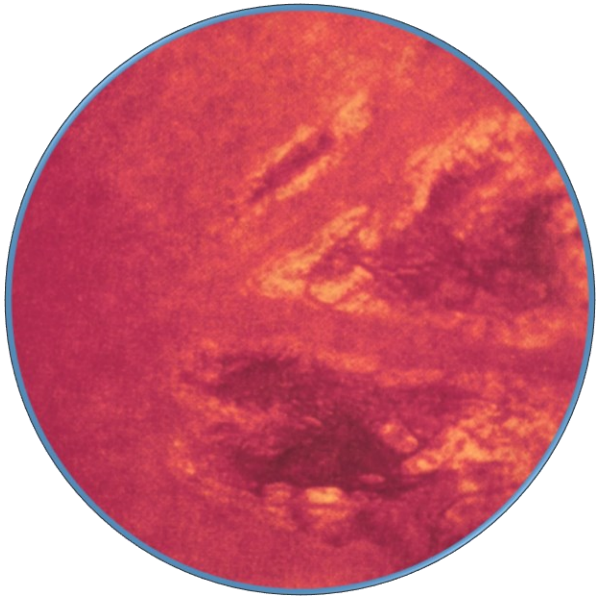


Ischémie choroïdienne en secteur



SYNDROME TRIANGULAIRE

P. Amalric Bull Soc Oph Fr , 1963



Maladie de Horton / Giant cell arteritis

- Hayreh SS : Eye 1990
  - "If angiography is performed during the first days after the onset of arteritic AION, it almost always shows massive choroidal non filling, corresponding to the occluded PCA"

Hayreh, S. S. (1990). "Anterior ischaemic optic neuropathy. Differentiation of arteritic from non-arteritic type and its management." Eye (Lond) 4 (Pt 1): 25-41.

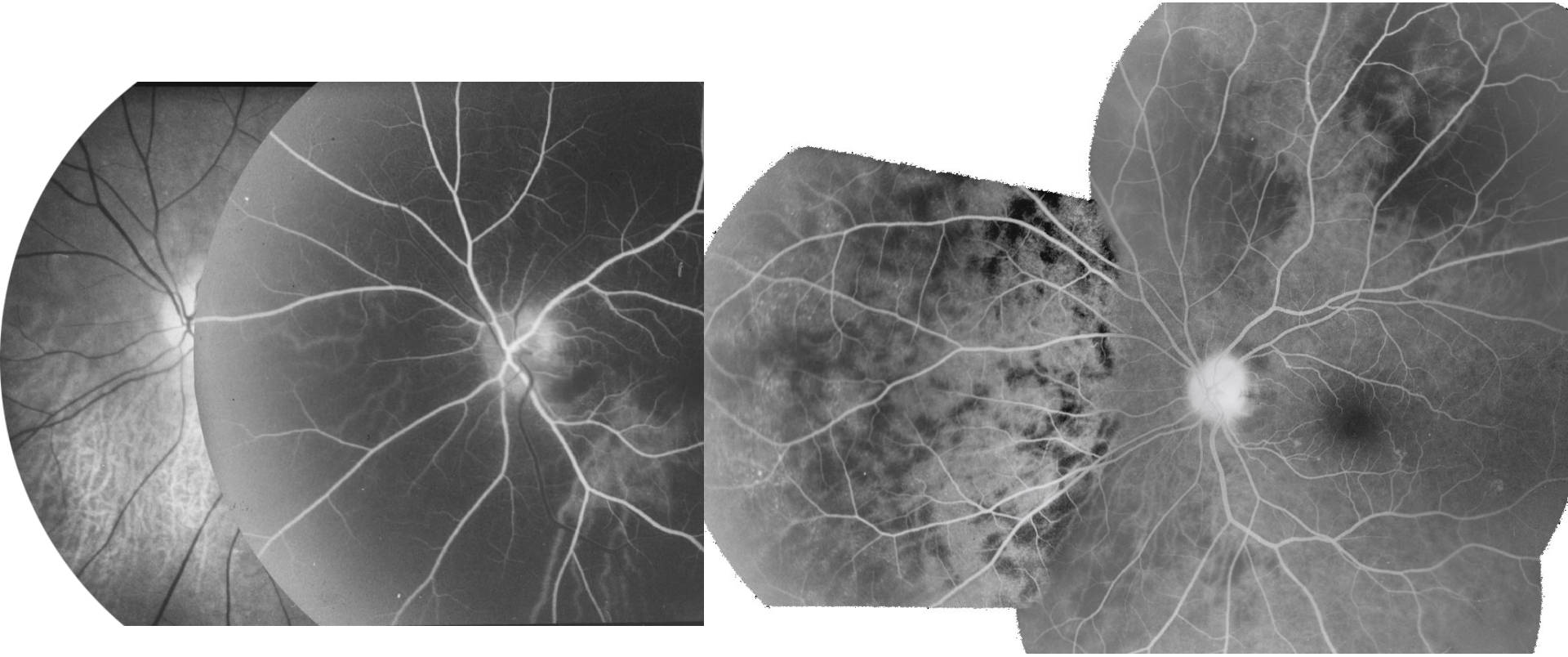
- Mack HG : J Cl Neuroopht 1991
  - 13 cas de maladie de Horton : 11 NOIA, 2 cécités transitoires
  - comparées à des NOIA non artéritiques
    - Retard de perfusion choroïdienne manifeste dans le Horton

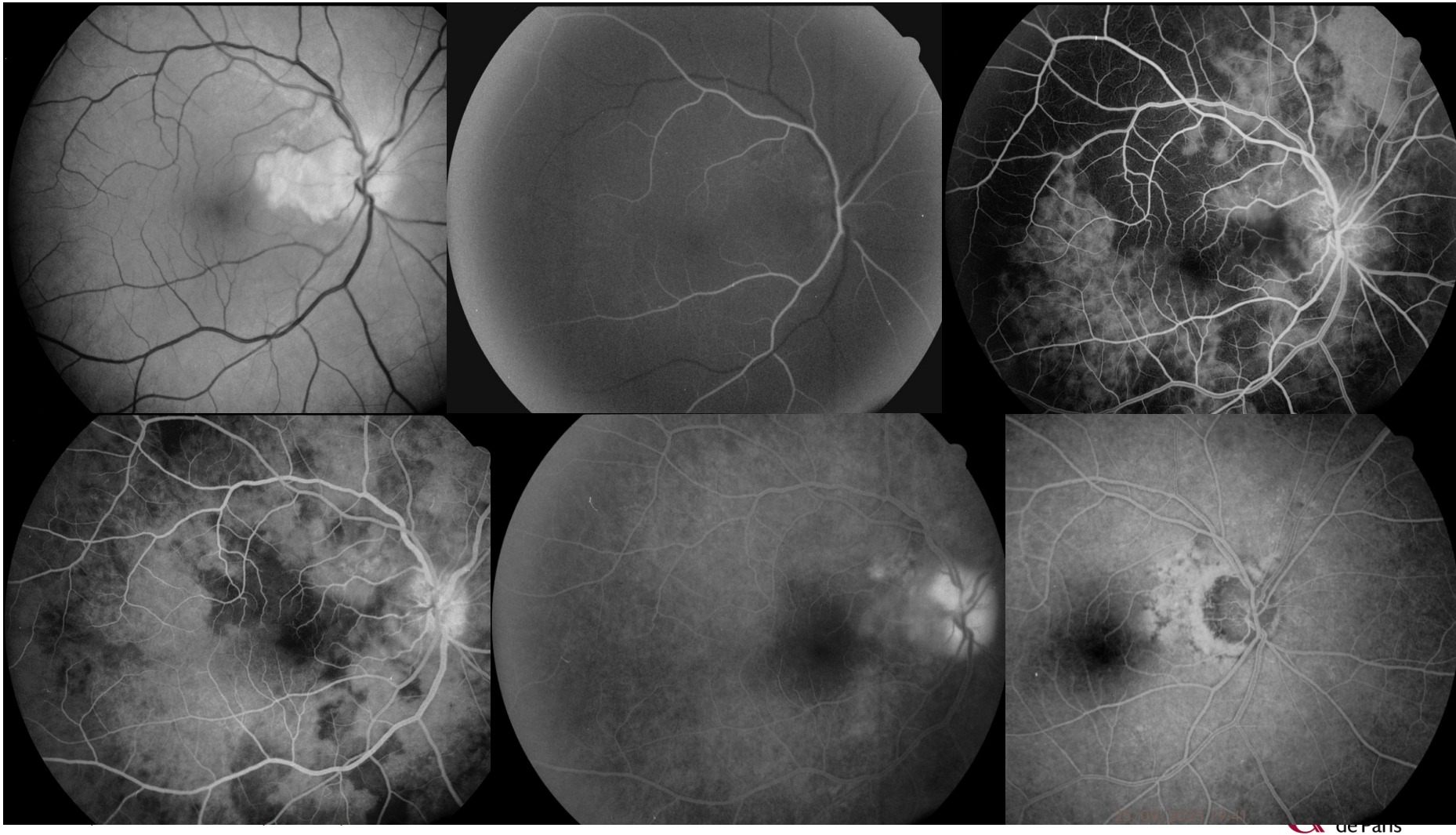
Mack HG, O'Day J, Currie JN. Delayed choroidal perfusion in giant cell arteritis. J Clin Neuro-ophthalmol. 1991 Dec;11(4):221-227.

- Siatkowski , Gass : Am J Oph 1993
  - 16 cas de NOIA au cours du Horton
  - comparés à 35 cas de NOIA non artéritique
    - Retard de perfusion choroïdienne manifeste dans le Horton

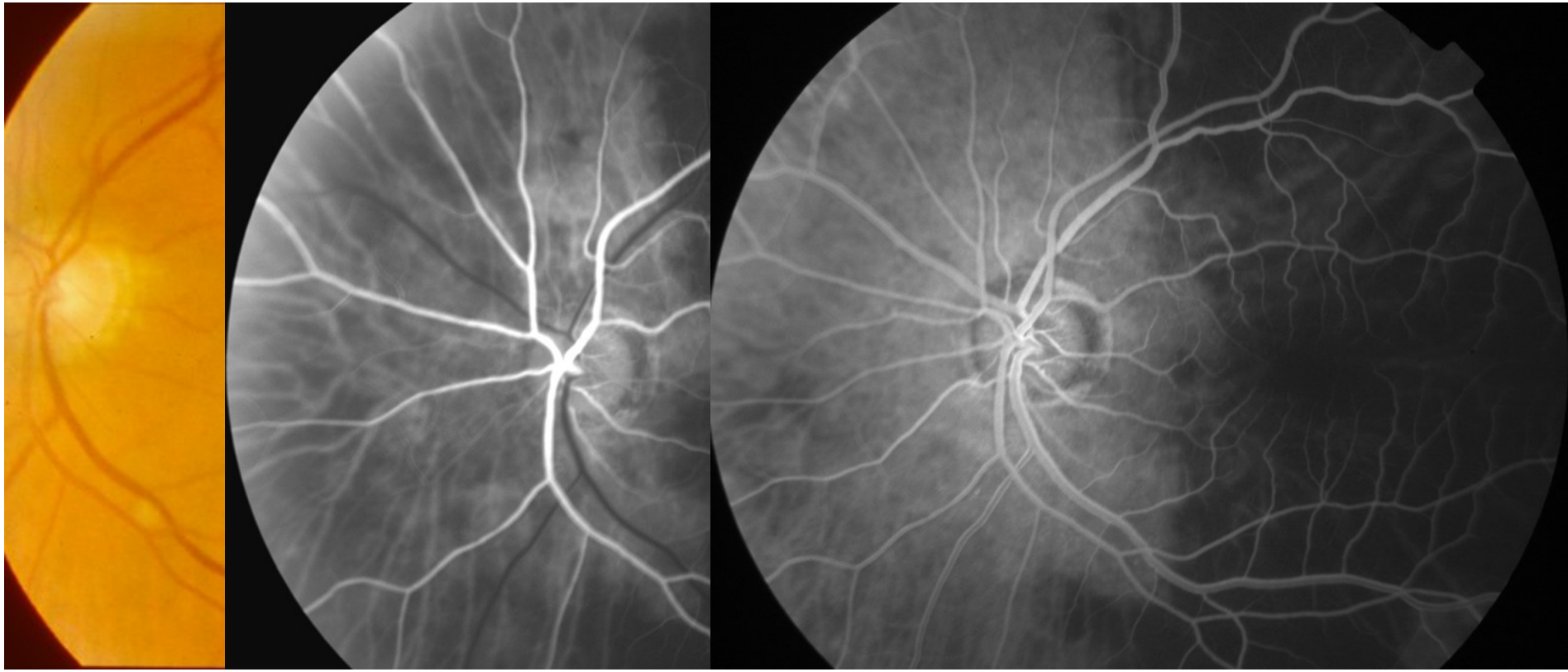
Siatkowski RM, Gass, JDM, Glaser JS. et al. Fluorescein Angiography in the Diagnosis of Giant Cell Arteritis. Am J Ophthalmol, 1993 115,1, 57-63

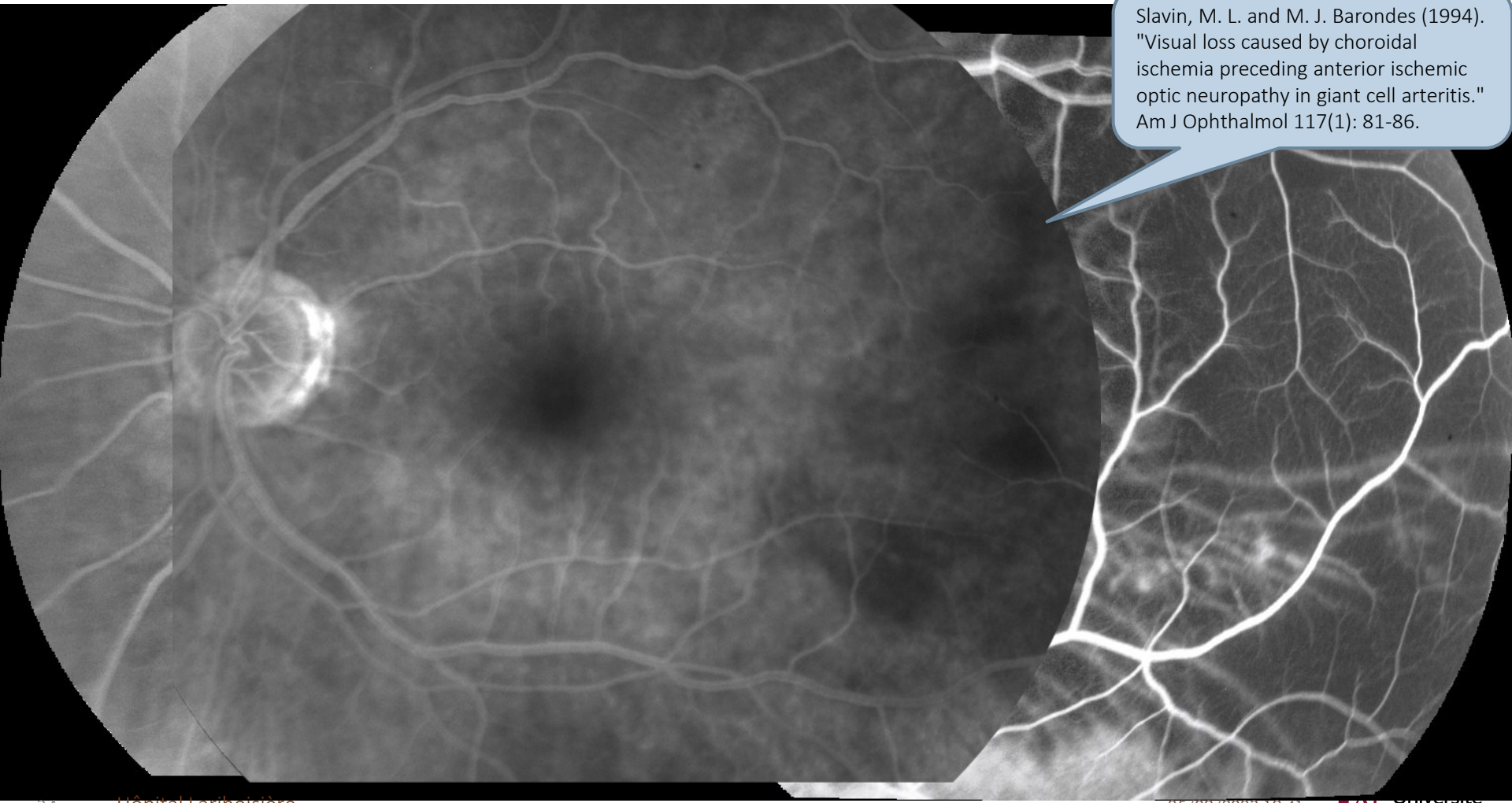
Maladie de Horton / Giant cell arteritis





Amaurose transitoire , Maladie de Horton

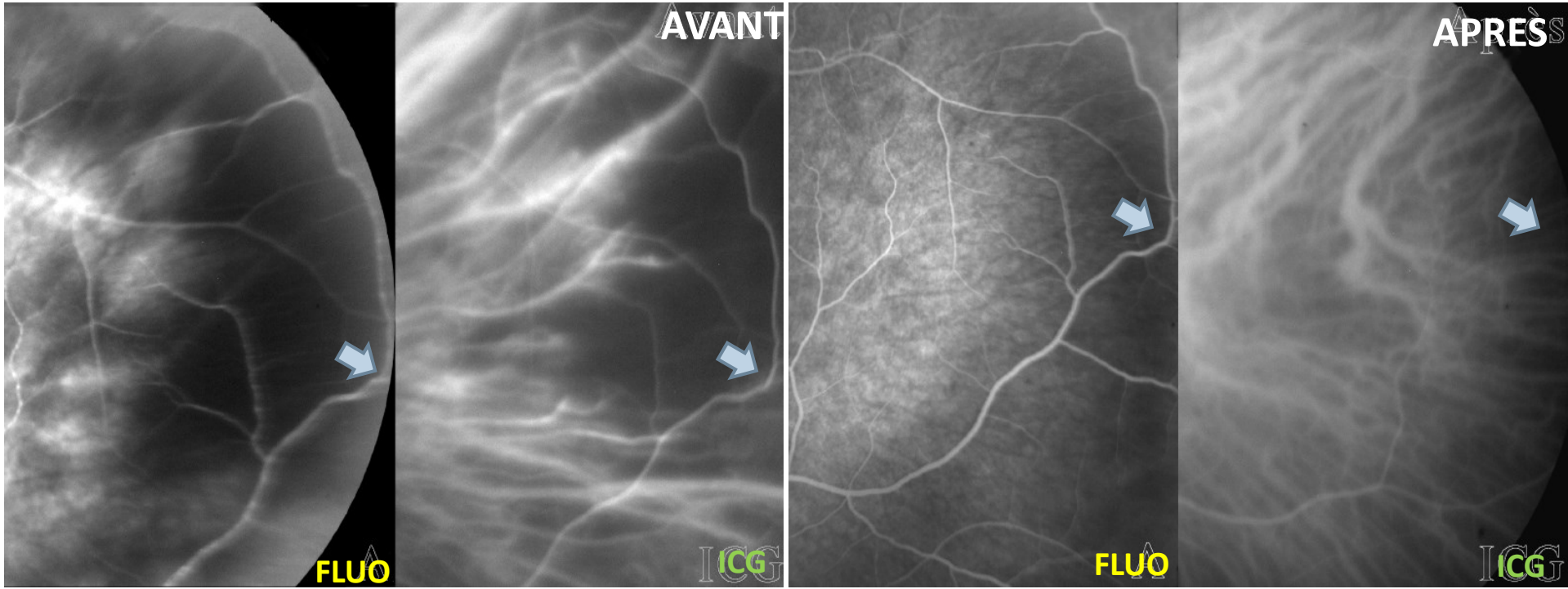




Slavin, M. L. and M. J. Barondes (1994). "Visual loss caused by choroidal ischemia preceding anterior ischemic optic neuropathy in giant cell arteritis." Am J Ophthalmol 117(1): 81-86.

Amaurose transitoire, Maladie de Horton

Évolution sous traitement



Ischémie choroïdienne Maladie de Horton

- Publications récentes

Choroidal ischemia as one cardinal sign in giant cell arteritis

Antonio M. B. Casella[1*], Ahmad M. Mansour[2], Souza EC[3], Rodrigo B. do Prado[1], Rodrigo Meirelles[4], Keye Wong[5], Salma Yassine[6] and Mário Luiz R. Monteiro[3]

Int J Retin Vitr. 2022;8(1):69.

Avec les lésions PAMM, les nodules cotonneux, la NOIAA et l'OACR, l'ischémie choroïdienne est un indicateur angiographique clé dans le diagnostic de la maladie de Horton.

Giant Cell Arteritis: The Experience of Two Collaborative Referral Centers and an Overview of Disease Pathogenesis and Therapeutic Advancements

Dammacco R et al. Clin Ophthalmol (Auckl, NZ). 2020;14:775-793.

Seulement 1 cas sur 20 d'ischémie choroidiennne , mais pas d'angiographie systématique

REVIEW ARTICLE OPEN

Check for updates

Giant cell arteritis: reviewing the advancing diagnostics and management

Edward J. Bilton[1,2] and Susan P. Mollan[1,2,3]

Eye. 2023;37(12):2365-2373.

Presentation	Ocular manifestations of Giant Cell Arteritis
Very common	Anterior ischaemic optic neuropathy
Common	Cotton wool spots
	Cilio-retinal Artery Occlusion
	Central Retinal Artery Occlusion
	Cranial nerve palsy
	Extra ocular muscle ischaemia
Rare	Posterior ischaemic optic neuropathy
	Choroidal ischaemia

Ischémie choroïdienne en secteur:
autres étiologies

TRIANGULAR SIGN OF AMALRIC IN INTRAVASCULAR LYMPHOMA

James Clay Bavinger, MD,* Ali G. Hamedani, MD,† Vivian Lee, MD,*
Alexander J. Brucker, MD,* Tomas S. Aleman, MD*

Retin Cases Brief Rep. 2022;16(1):20-24.

Amalric triangular sign in a case of central retinal artery occlusion combined with posterior ciliary artery occlusion – Case report

Soojin Lim[a], Cheng-Kuo Cheng[a,b,c,*], Yi-Hsuan Li[a]

Am J Ophthalmol Case Rep. 2018;11:149-152.

Sténose carotidienne

THE SPECTRUM OF AMALRIC TRIANGULAR CHOROIDAL INFARCTION

Julia Nemiroff, MD,* Nopasak Phasukkijwatana, MD, PhD,*† Veronika Vaclavik, MD,‡§¶
Aaron Nagiel, MD, PhD,* Eric R. Holz, MD,** David Sarraf, MD*††

Retin Cases Brief Rep 2017;11 Suppl 1:S113-S120.

Maladie de Horton
Dissection carotidienne
Lupus Erythémateux Disséminé

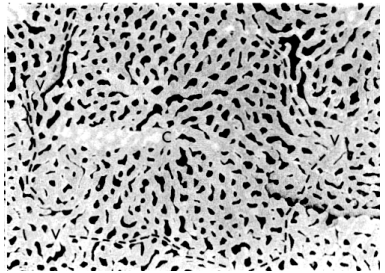
- On retiendra:
  - Devant une NOIAA
    - Une ischémie choroïdienne en secteur , signe le diagnostic de Maladie de Horton
  - Devant une amaurose fugace
    - Réaliser une angiographie peut permettre de suspecter maladie de Horton

- ISCHÉMIE CHOROÏDIENNE EN SECTEUR
  - Maladie de Horton
- ISCHÉMIE CHOROÏDIENNE MULTIFOCALE
  - **Toxémie gravidique**
  - Rétinopathie hypertensive maligne
  - Maladie de Harada
  - Expérimentation animale
  - Épithéliopathie en plaques
  - Choroidite serpigineuse

Ischémie choroïdienne multifocale

- D'abord décrite lors de la toxémie gravidique et HTA maligne
  - Gitter, K. A., et al. (1968). Toxemia of pregnancy. An angiographic interpretation of fundus changes." Arch Ophthalmol 80(4): 449-454.
  - Klien, B. A. (1968). Ischemic infarcts of the choroid (Elschinig spots). A cause of retinal separation in hypertensive disease with renal insufficiency. A clinical and histopathologic study." Am J Ophthalmol 66(6): 1069-1074.
  - Stern, W. H. and J. T. Ernest (1974). "Microsphere occlusion of the choriocapillaris in rhesus monkeys." Am J Ophthalmol 78(3): 438-448.

- Occlusion choroidienne multifocale aigüe
  - Taches jaunes de l'EP
  - DR séreux exsudatif
  - Remplissage choroidien en mosaïque
  - Diffusion et accumulation tardive de colorant
  - Guérison avec taches d' Elschnig



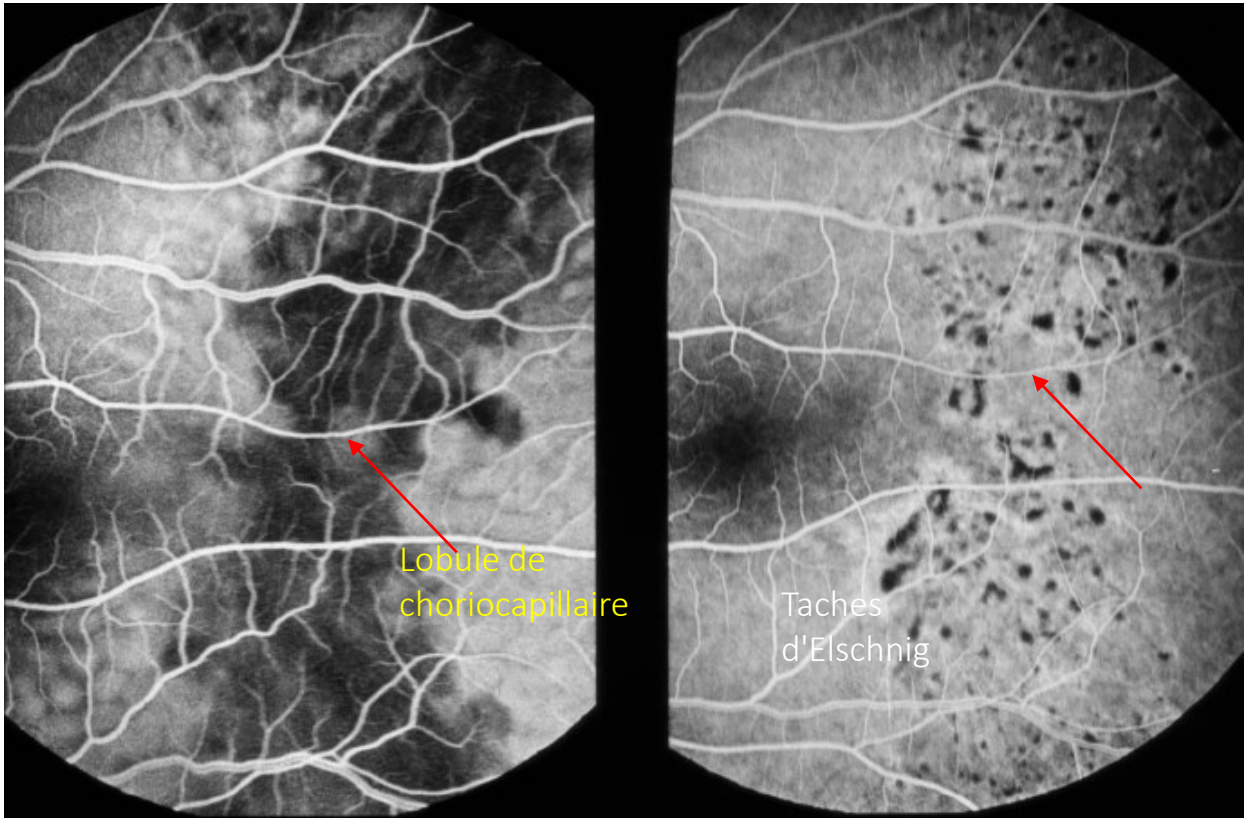
Toxémie gravidique

Femme de 24 ans , pré-éclampsie, baisse d'AV bilatérale après l'accouchement

- Décollement de rétine exsudatif

Toxémie gravidique

Phase aigüe



Guérison

Ischémie choroïdienne multifocale

- Paradoxalement les lésions ischémiques de l'EP sont plus exudatives lorsque co-existent :
  - unités de la choriocapillaire encore perfusées
  - et d'autres non-perfusées
  - ou que l'hypoperfusion est incomplète

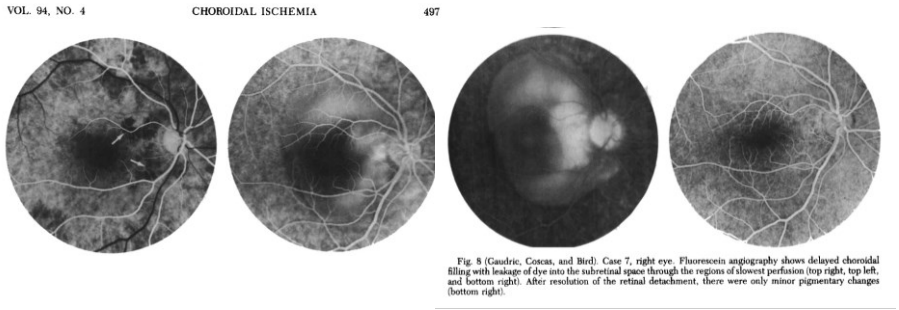


Fig. 8 (Gaudric, Coscas, and Bird). Case 7, right eye. Fluorescein angiography shows delayed choroidal filling with leakage of dye into the subretinal space through the regions of slowest perfusion (top right, top left, and bottom right). After resolution of the retinal detachment, there were only minor pigmentary changes (bottom right).

CHOROIDAL ISCHEMIA

A. GAUDRIC, M.D., AND G. COSCAS, M.D.
Paris, France
AND
A. C. BIRD, M.D.
London, England

Nine patients, seven women and two men, ranging in age from 21 to 86 years, had generalized vascular disease and significant deficits in choroidal perfusion but no retinal vascular changes. Because these perfusion abnormalities can be identified only by fluorescein angiography and the fundus appears to be normal, choroidal ischemia may be more common than has been realized. Infarction of the choroid, retinal pigment epithelium, and outer retina are usually unassociated with retinal detachment; even ischemia severe enough to induce retinal detachment seldom produces significant atrophy.

Choroidal ischemia has received less attention than retinal vascular disease. Published reports describe two well-defined and distinct manifestations of reduced choroidal blood flow in patients with generalized vascular disease. Large triangular infarcts may occur in the acute phase in the form of areas of opacification of the retinal pigment epithelium and outer sensory retina[1,2] or, later, in the form of well-defined regions of scarring.[3,4] These lesions have been ascribed to obstruction of short posterior ciliary arteries and have their counterparts in experimental arterial obstruction in animals.[5] The second form of disease typically occurs in accelerated hypertension in which there is multifocal opacification of the outer retina and serous detachment of the sensory retina. It has been suggested that there is widespread obstruction of choroidal arterioles in such cases.

During the last decade there has been progressively more support for the theory that choroidal vascular disease may be responsible for retinal pigment epithelial ischemia in the young in the absence of obvious vascular disease elsewhere,[6-8] appearing as multifocal opacification of the retinal pigment epithelium[9] or as retinal detachment,[10] although the evidence for ischemia in such cases is circumstantial.

We examined a series of nine patients with documented generalized vascular disorders and a wide variety of manifestations of presumed choroidal vascular disease.

Accepted for publication May 24, 1982.
From the Centre Hospitalier Intercommunal, Paris, France (Drs. Gaudric and Coscas), and the Institute of Ophthalmology, Moorfields Eye Hospital, London, England (Dr. Bird).
Reprint requests to A. C. Bird, M.D., Institute of Ophthalmology, Moorfields Eye Hospital, City Road, London EC1V 2PD, England.

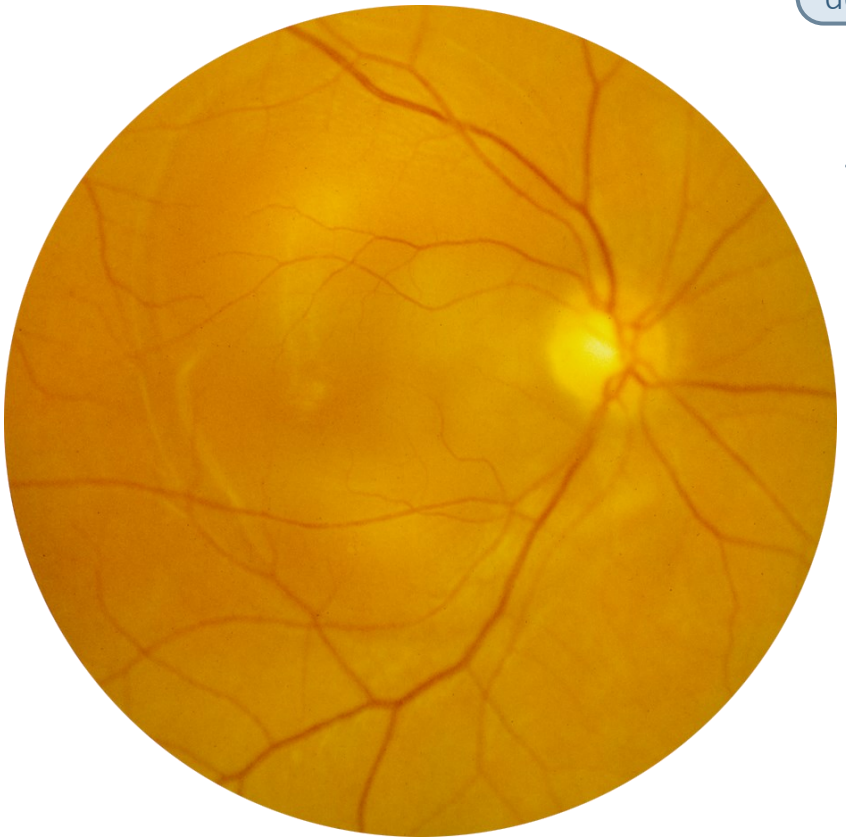
SUBJECTS AND METHODS

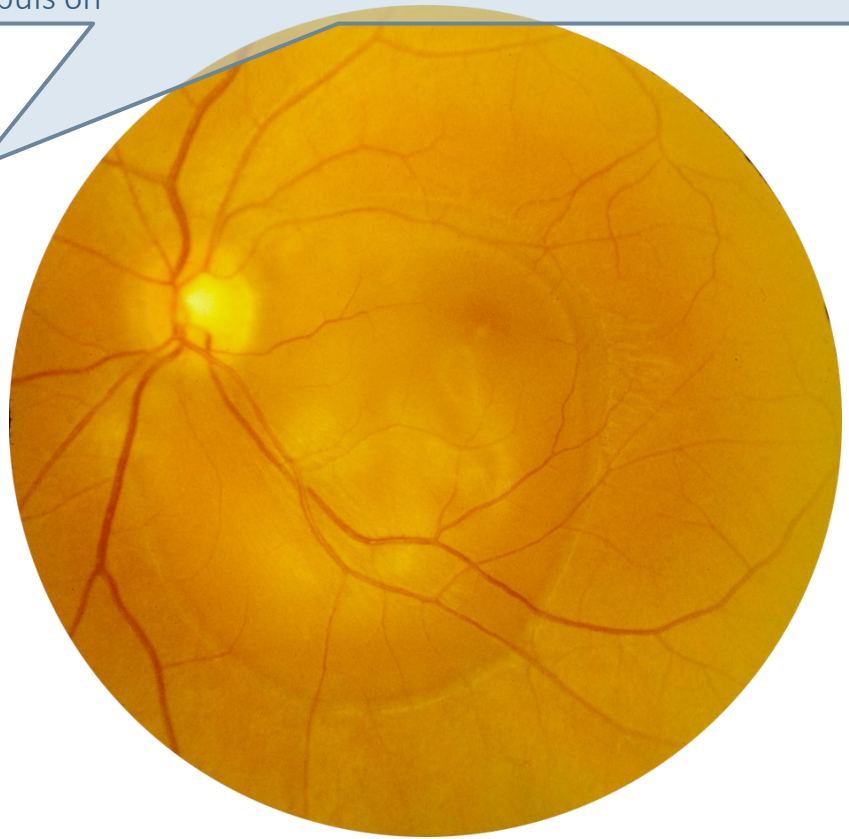
Nine patients with generalized vascular disease had well-documented disturbances of choroidal circulation in one eye (four patients) or both eyes (five patients) without significant retinal vascular dis-

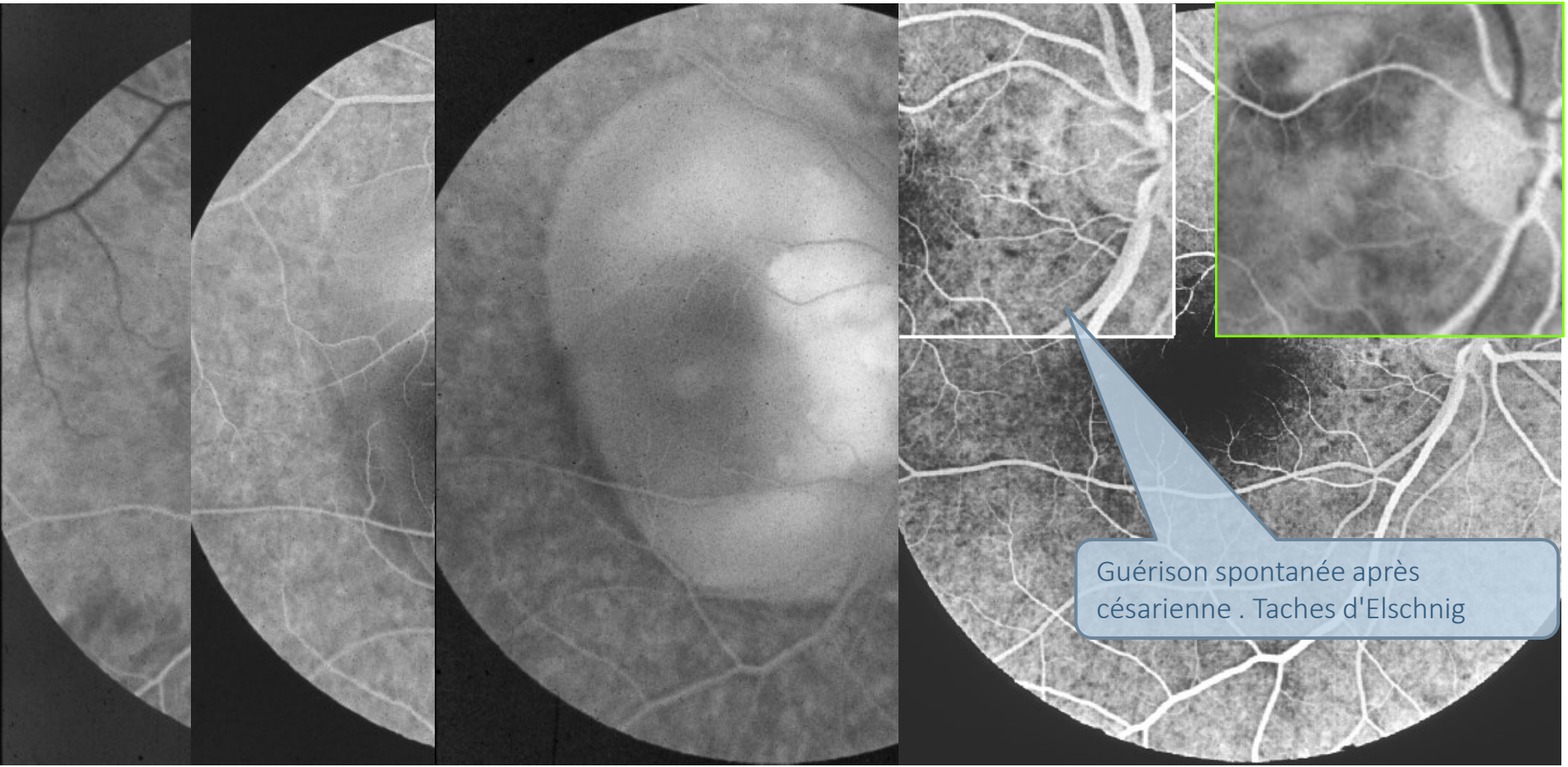
Am J Ophthalmol. 1982;94(4):489-498.

Toxémie gravidique

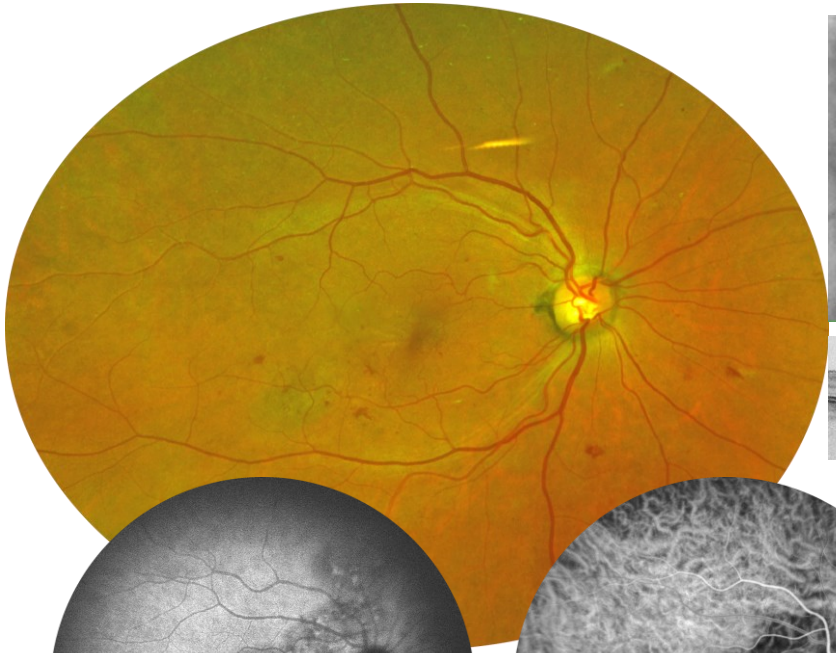
Femme de 32 ans, hospitalisée pour menace de toxémie gravidique , 33 ème semaine de grossesse , baisse d'AV depuis 6h

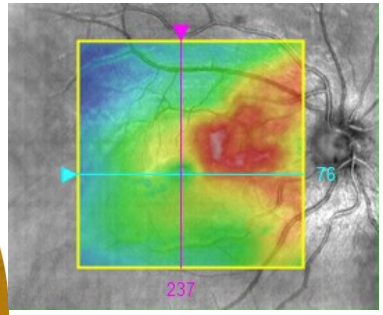


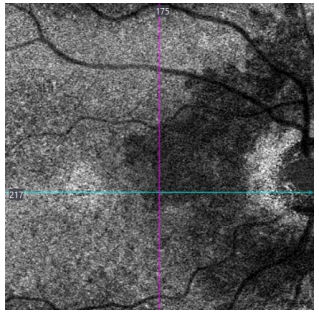


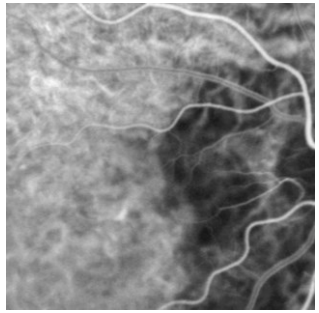

Guérison spontanée après césarienne . Taches d'Elschnig

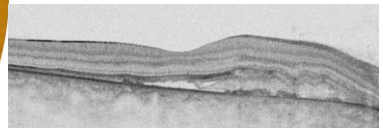
Toxémie gravidique

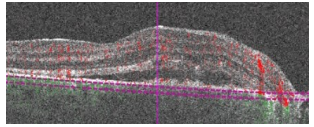


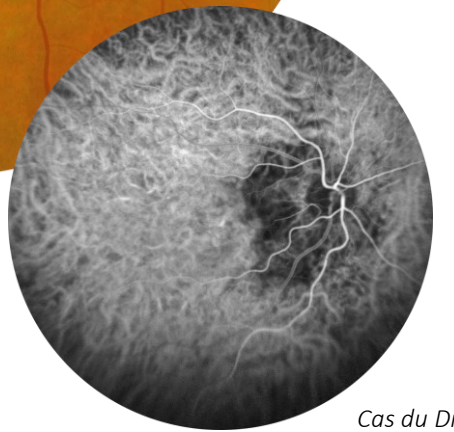


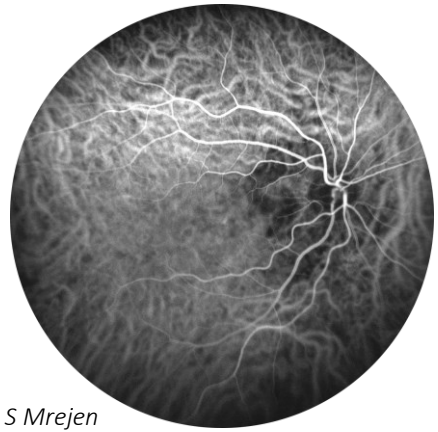












Toxémie gravidique, ischémie choroïdienne multifocale

Publications récentes

Serous retinal detachment in preeclampsia and malignant hypertension

Christopher Seungkyu Lee[1,2] · Eun Young Choi[1] · Minsub Lee[3] · Heesuk Kim[2] · Hyewon Chung[3]

Eye. 2019;33(11):1707-1714.

Les patientes atteintes de prééclampsie pourraient être considérées comme n'ayant pas atteint un seuil "malin" pour le développement de la rétinopathie.
Une placentation anormale provoque une ischémie placentaire chronique et un stress oxydatif, induisant la libération de substances dans la circulation : radicaux libres, lipides oxydés et de facteurs antiangiogéniques,, qui sont responsables du dysfonctionnement endothélial généralisé observé dans la prééclampsie.

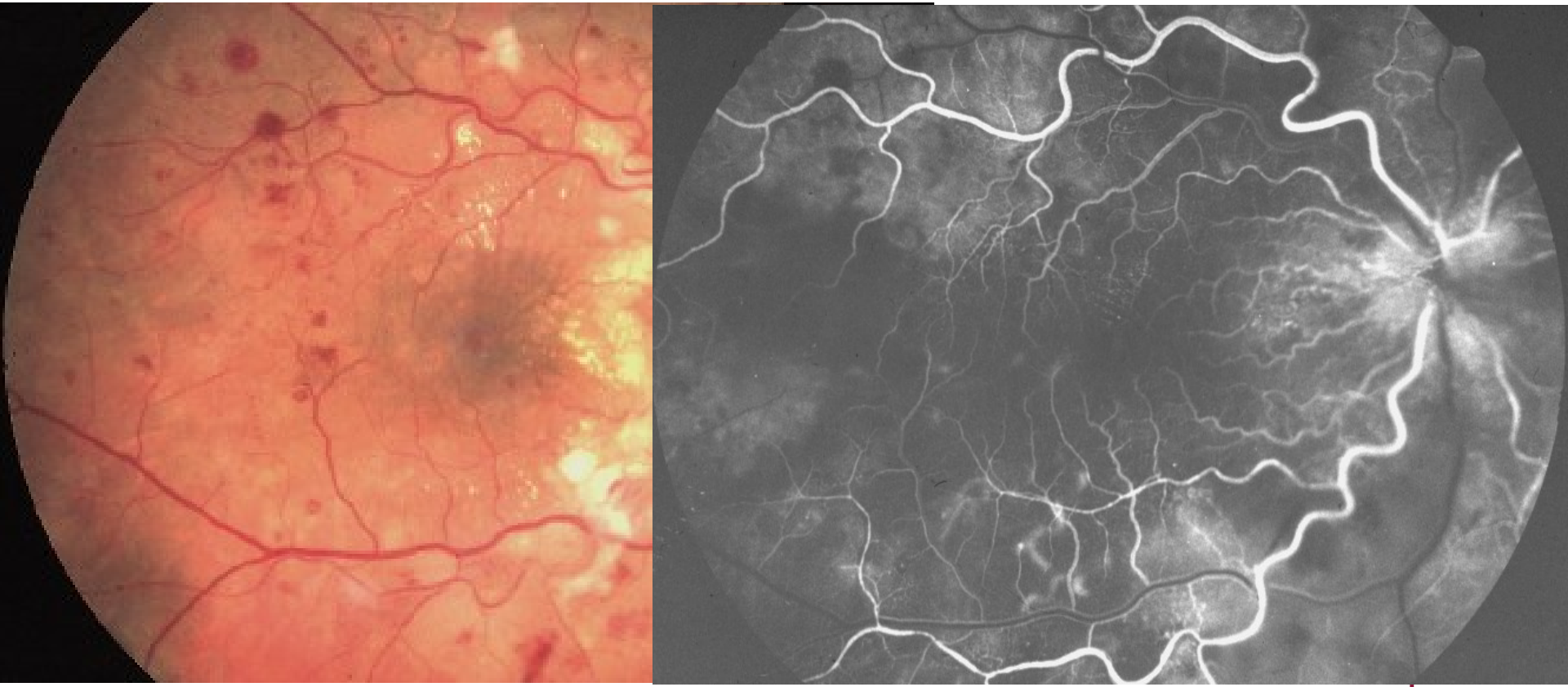
Diminished choroidal blood flow in hypertensive and preeclamptic third trimester pregnancies using optical coherence tomography angiography

Alaa E. Fayed[1,2]*, Mohamed M. Thabet[3], Marwa Metwally Salama[1], Malak El Shazly[1]

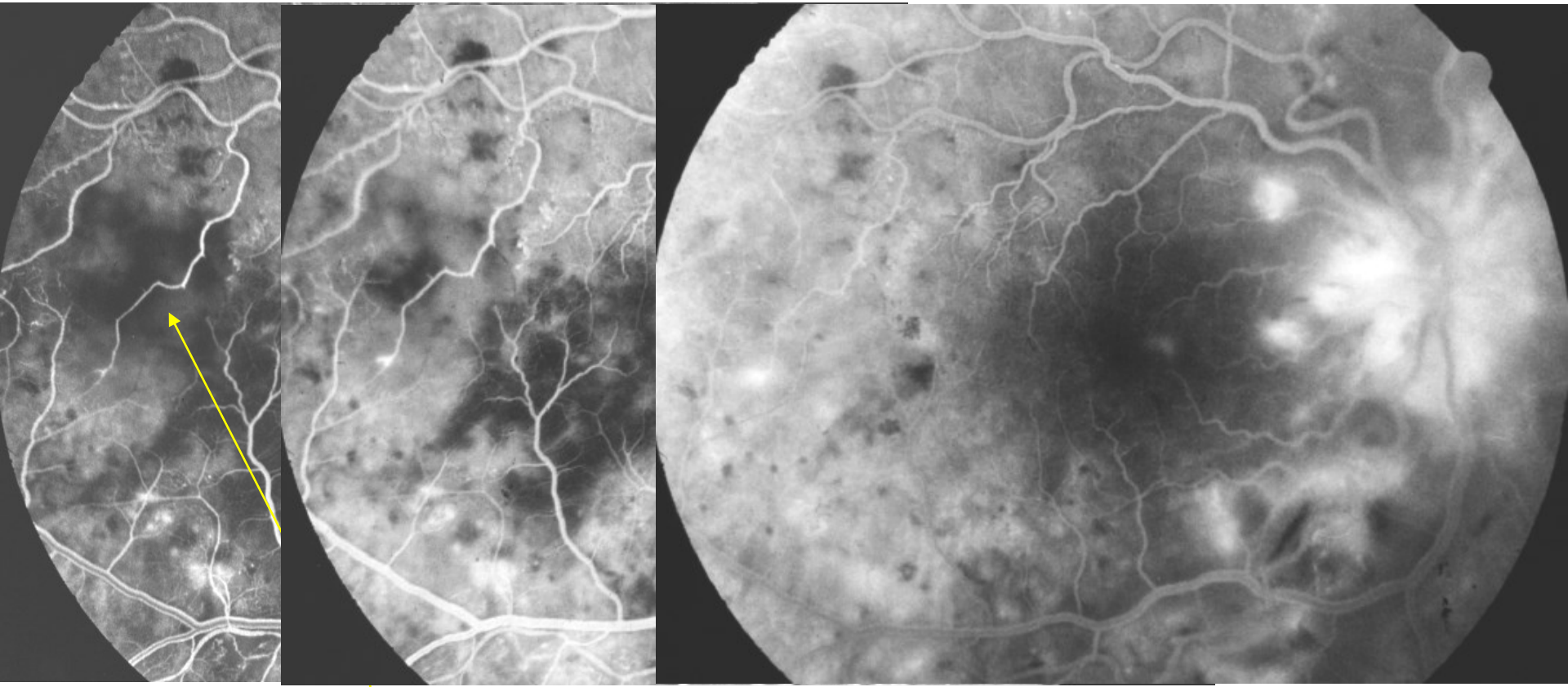
Etude en OCTA : Les grossesses compliquées de prééclampsie ont un flux sanguin choroïdien plus faible sur l'OCTA, que les grossesses avec hypertension systémique et les grossesses saines

- ISCHÉMIE CHOROÏDIENNE EN SECTEUR
  - Maladie de Horton
- ISCHÉMIE CHOROÏDIENNE MULTIFOCALE
  - Toxémie gravidique
  - **Rétinopathie hypertensive maligne**
  - Maladie de Harada
  - Expérimentation animale
  - Épithéliopathie en plaques
  - Choroidite serpigineuse

Rétino Choroïdopathie Hypertensive



Rétino Choroïdopathie Hypertensive



Rétino Choroïdopathie Hypertensive

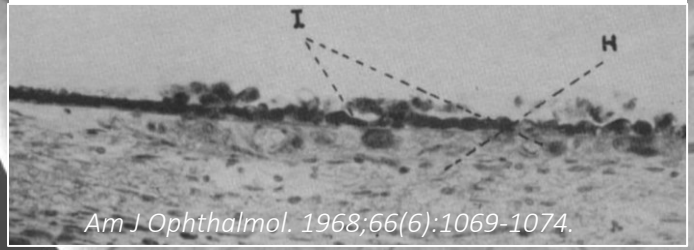
Après traitement de l'HTA

ISCHEMIC INFARCTS OF THE CHOROID (ELSCHNIG SPOTS)

A CAUSE OF RETINAL SEPARATION IN HYPERTENSIVE DISEASE WITH RENAL INSUFFICIENCY

A CLINICAL AND HISTOPATHOLOGIC STUDY

BERTHA A. KLIEN, M.D.
Chicago, Illinois



Am J Ophthalmol. 1968;66(6):1069-1074.

Taches d'Elschnig

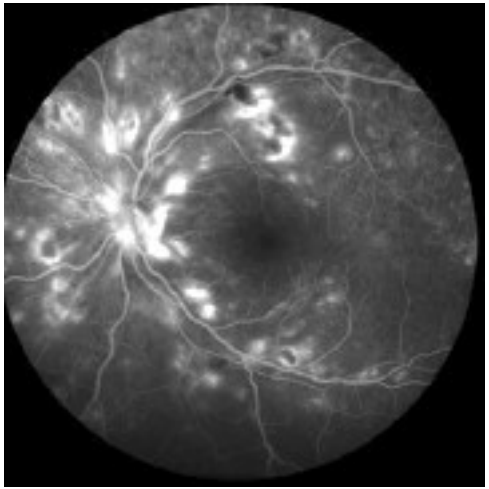
Rétinochoroidopathie hypertensive, OCTA

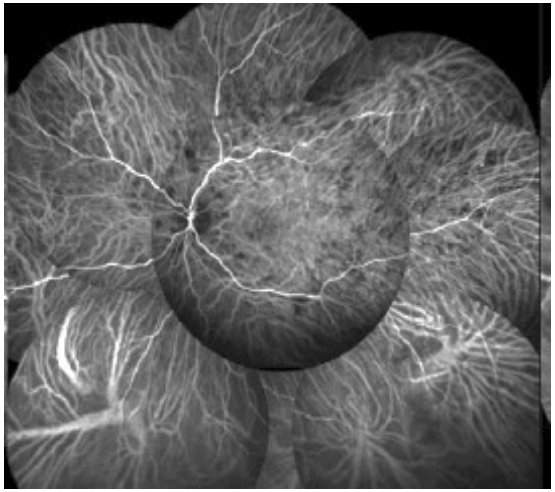
Case report

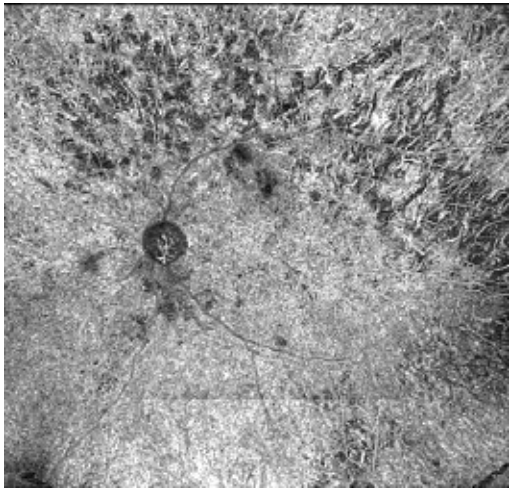
Hypertensive choroidopathy: Multimodal imaging and the contribution of wide-field swept-source oct-angiography

Am J Ophthalmol Case Reports. 2019;13:131-135.

Amina Rezkallah[a], Laurent Kodjikian[a,b], Amro Abukhashabah[a], Philippe Denis[a], Thibaud Mathis[a,b,*]



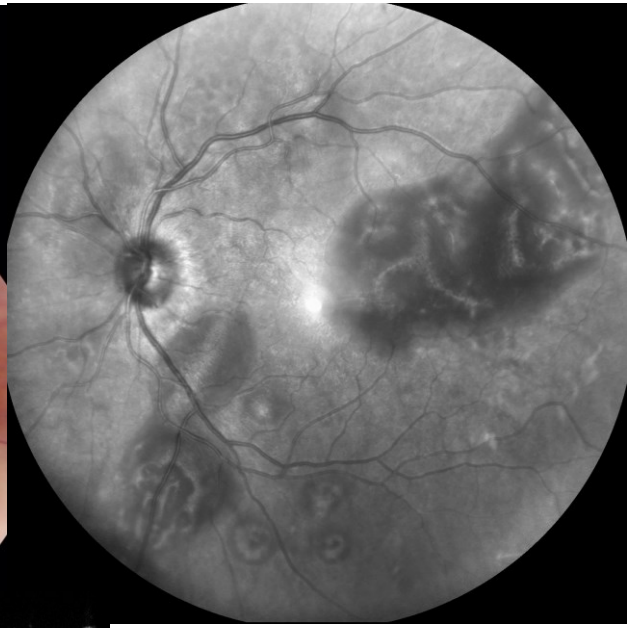


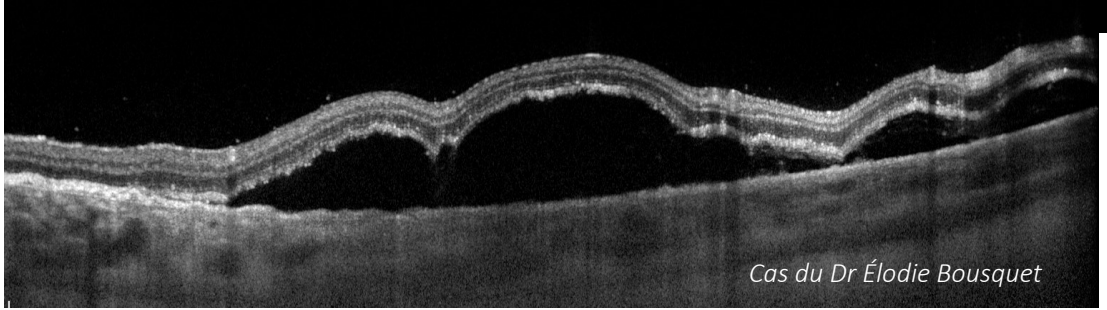


Autres causes d'ischémie choroïdienne multifocale

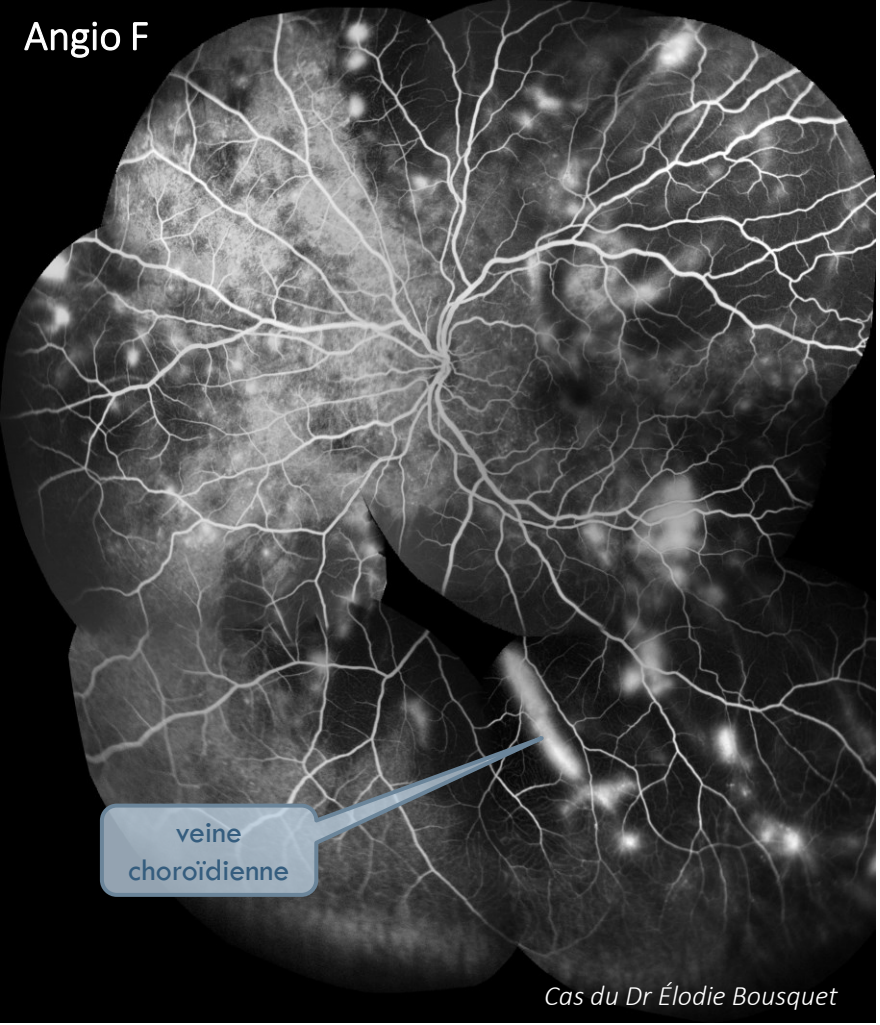
- Syndrome catastrophique des antiphospholipides
- Purpura thrombotique thrombocytopénique
- Cocaïne
- Périartérite noueuse
- ...

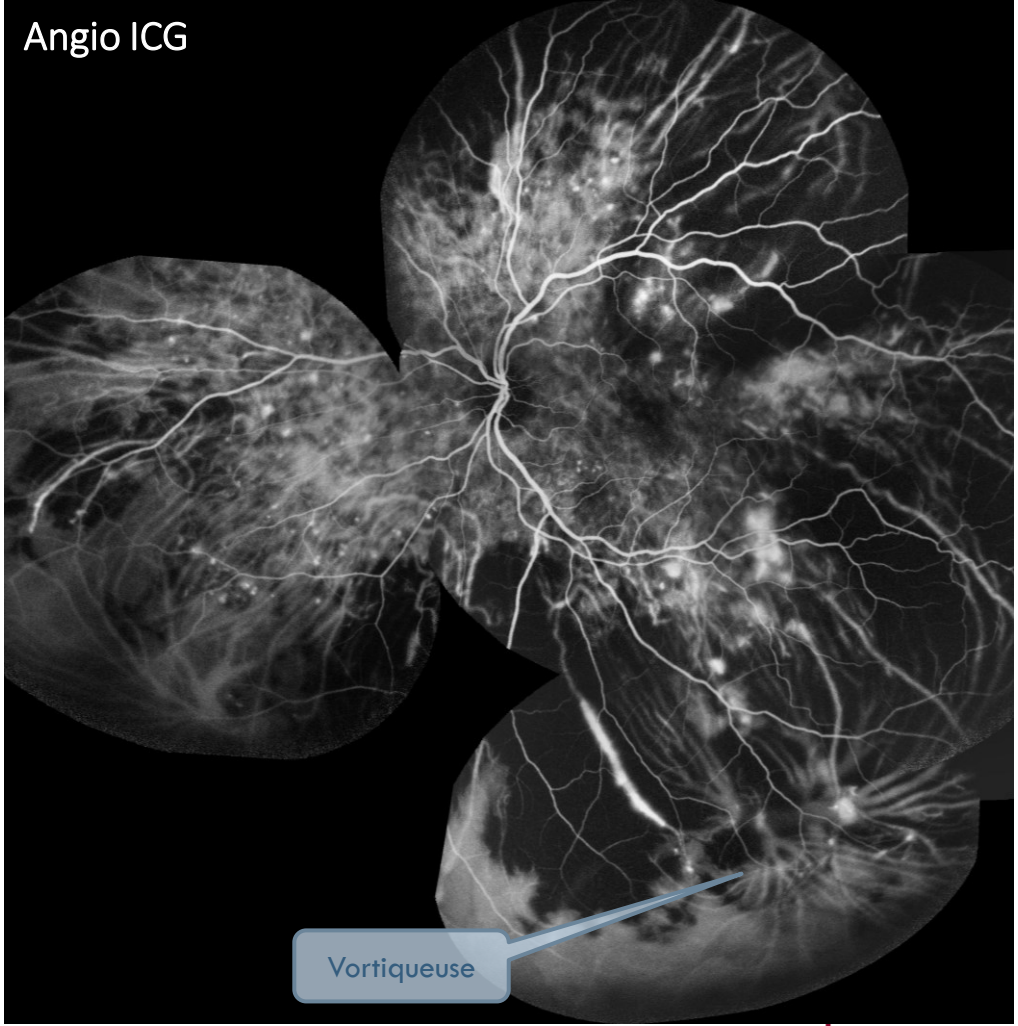
Syndrome catastrophique des anti-phospholipides



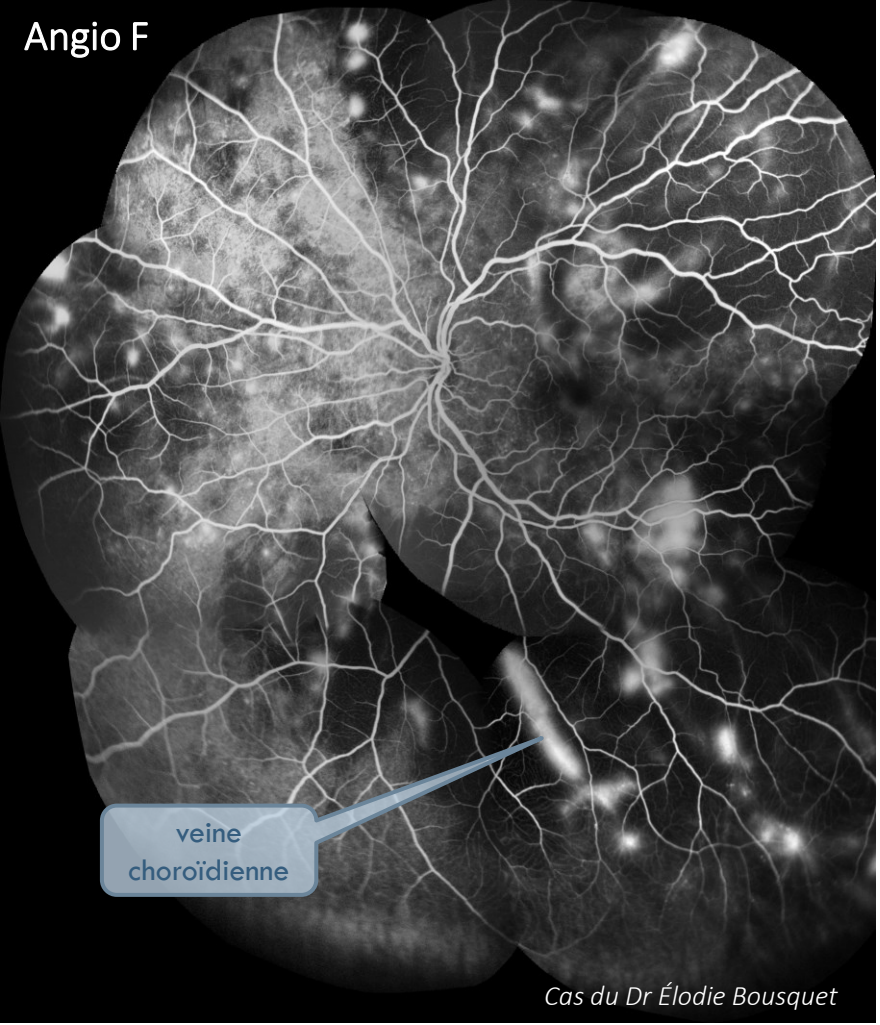


Femme de 30 ans
Antécédents de Syndrome des anti phospholipides et d'embolie pulmonaire
35 éme semaine de grossesse
Baisse soudaine d'AV bilatérale HELLP syndrome


Angio F
veine choroïdienne
Cas du Dr Élodie Bousquet

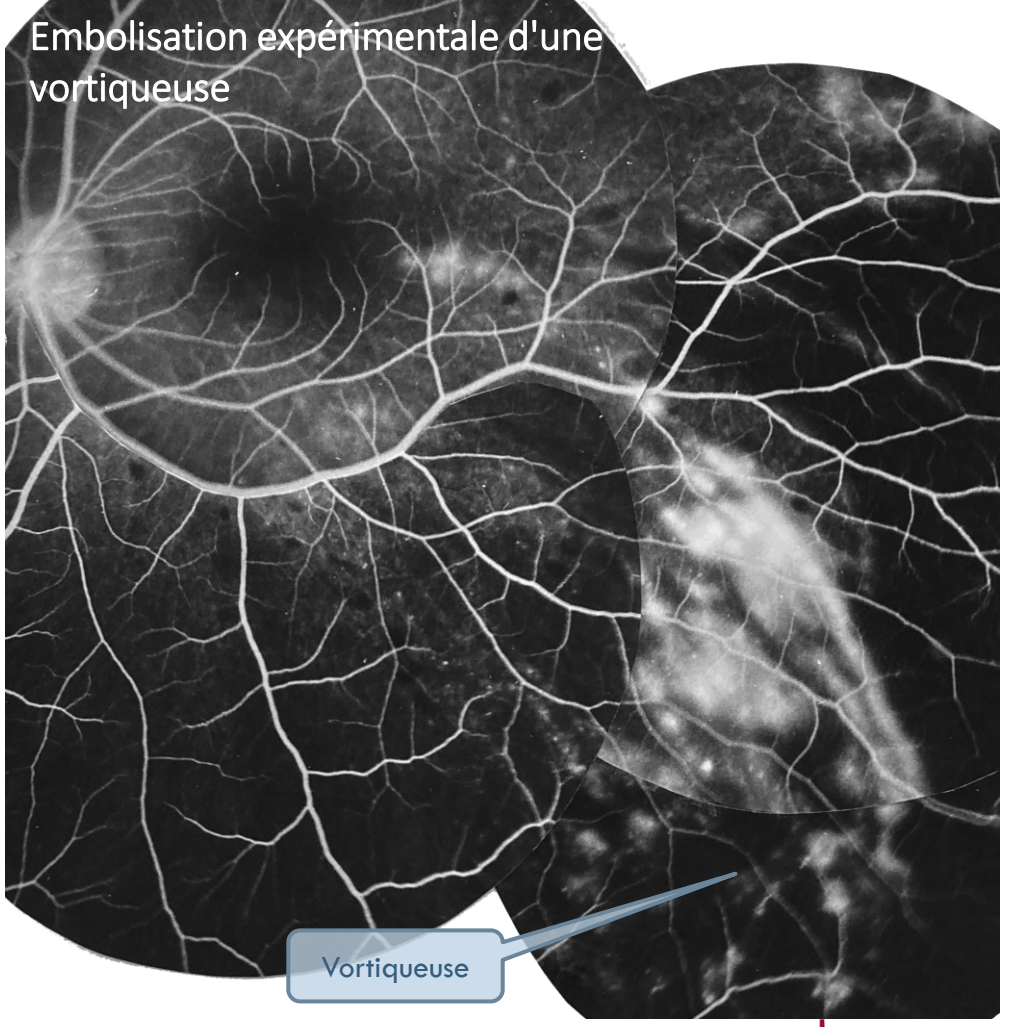

Angio ICG
Vortiqueuse

Angio F

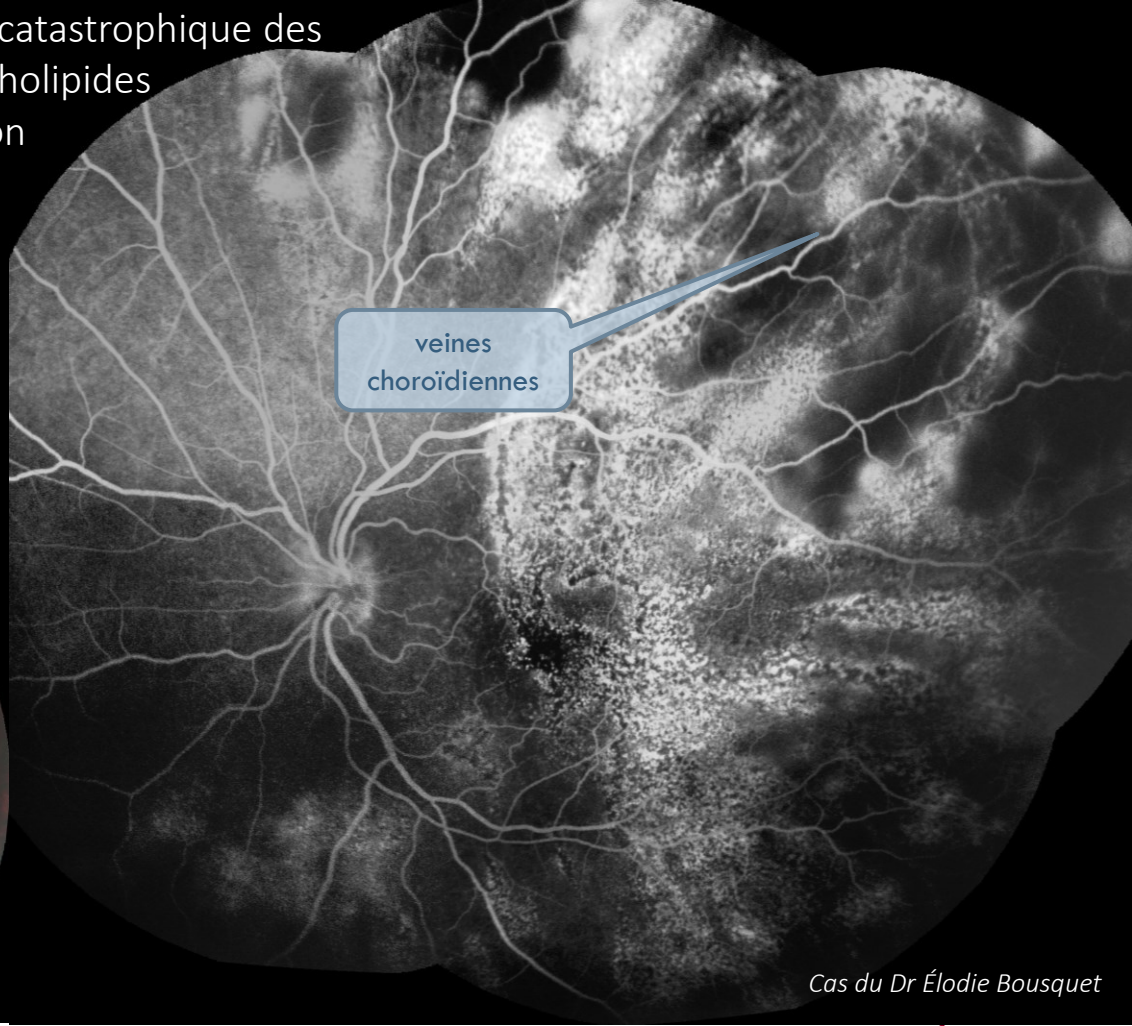


Cas du Dr Élodie Bousquet

Embolisation expérimentale d'une vortiqueuse



Syndrome catastrophique des anti-phospholipides
Guérison



Cas du Dr Élodie Bousquet

CAPS : autres publications

CATASTROPHIC ANTIPHOSPHOLIPID SYNDROME AND POSTERIOR OCULAR INVOLVEMENT

Case Series of 11 Patients and Literature Review

Morel N, Bonnet C, Mehawej H, et al. Retina. 2021;41(11):2332-2341.

11 cas : Occlusion choroïdienne 36% des cas
Revue littérature ,14 cas : occlusion choroïdienne 50% des cas

Les occlusions vasculaires rétiniennes étaient les plus fréquentes

The diagnosis and clinical management of the catastrophic antiphospholipid syndrome: A comprehensive review

Ricard Cervera[a,*], Ignasi Rodríguez-Pintó[b], Gerard Espinosa[a]

Cervera R, Rodríguez-Pintó I, Espinosa G.. Journal of autoimmunity. 2018;92:1-11.

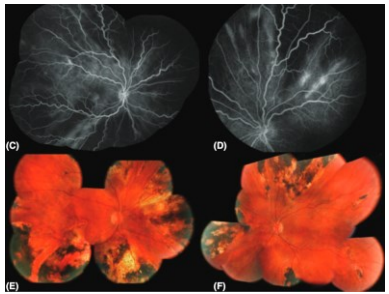
Microangioathie Thrombotique non-infalmmatoire au cours d'un syndrome des antiphospholipides

HELLP syndrome : CAPS durant la grossessse
Hemolysis, Elevated Liver enzyme, Low Platelet

Occlusion of choroidal vessels in a patient with catastrophic antiphospholipid syndrome

Matus Rehak,[1] Petra Meier,[1] Eva Bühner,[1] Sirak Petros[2] and Peter Wiedemann[1]

Acta ophthalmologica. 2010;89(6):595-596.



Purpura Thrombotique Thrombocytopénique

Maladie de Moschcowitz

Serous Retinal Detachments in Thrombotic Thrombocytopenic Purpura

Scott R. Lambert, MD; Katherine A. High, MD; Edward Cotlier, MD; Edward J. Benz, Jr, MD

Arch Ophthalmol. May 31, 2008:1-3.

Le pupura thrombocytopénique thrombotique (PTT) est une maladie d'étiologie inconnue, caractérisée par des microthrombi dans les artérioles et les veinules, qui entraînent à leur tour une thrombocytopénie, une anémie hémolytique, des symptômes neurologiques, une insuffisance , de la fièvre, et rarement des DSR par ischémie choroïdienne

Ocular findings in thrombotic thrombocytopenic purpura (Moschcowitz's disease)

S. P. B. PERCIVAL

Br J Ophthalmol. 1970;54(2):73-78.

Fibrin Clots in the Choriocapillaris and Serous Detachment of the Retina[1]

DAVID G. COGAN

Ophthalmologica. 1976;172:298-307.

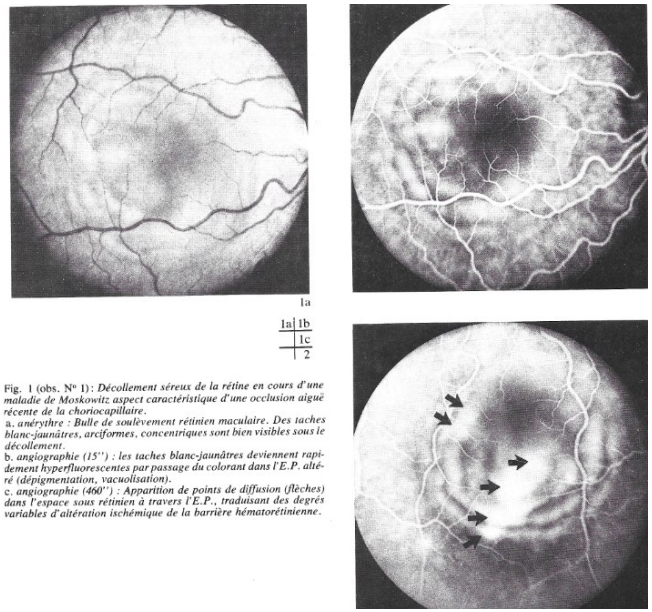
Obstruction choriocapillaire au cours de la maladie de Moschowitz

(à propos de 2 cas : analyse angiographique et anatomopathologique)

Coscas G, Gaudric A, Dhermy P, Vernant JP, Cordonnier C. J Fr Ophtalmolo. 1981;4(2):101-111..

Purpura Thrombotique Thrombocytopénique

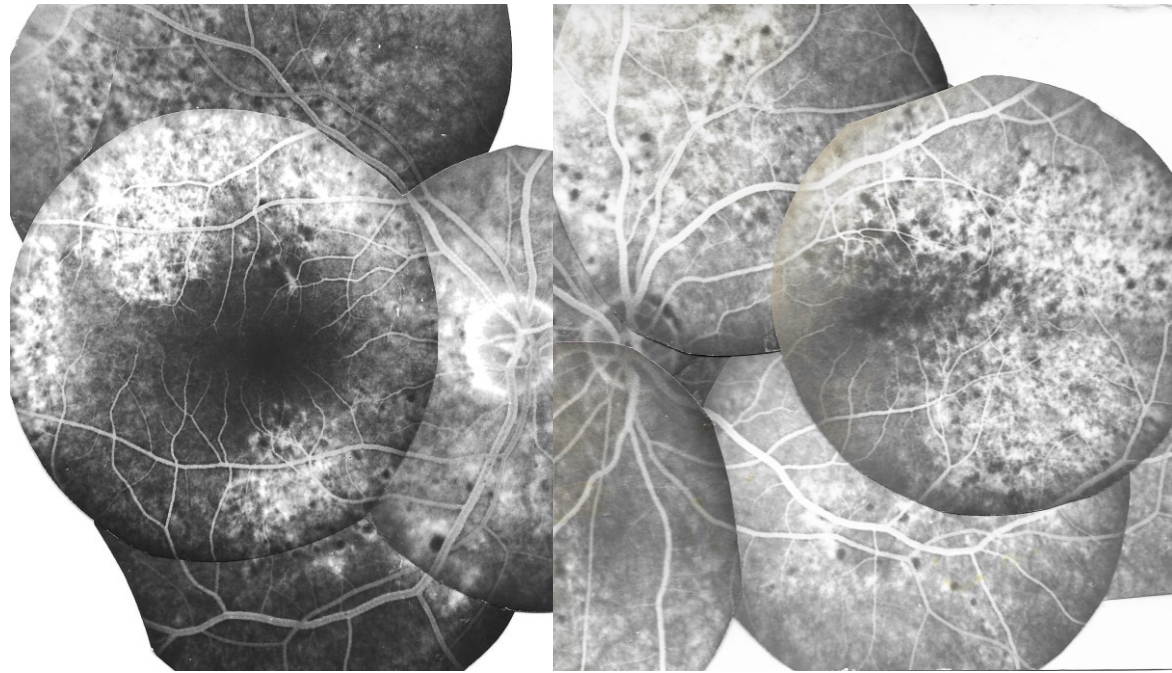
Maladie de Moschcowitz



1a

1a | 1b
1c
2

Fig. 1 (obs. N° 1) : *Décollement séreux de la rétine en cours d'une maladie de Moskowitz aspect caractéristique d'une occlusion aiguë récente de la choriocapillaire.*
a. *anérythre : Bulle de soulèvement rétinien maculaire. Des taches blanc-jaunâtres, arciformes, concentriques sont bien visibles sous le décollement.*
b. *angiographie (15'') : les taches blanc-jaunâtres deviennent rapidement hyperfluorescentes par passage du colorant dans l'E.P. altéré (dépigmentation, vacuolisation).*
c. *angiographie (460'') : Apparition de points de diffusion (flèches) dans l'espace sous rétinien à travers l'E.P., traduisant des degrés variables d'altération ischémique de la barrière hématorétinienne.*



Coscas G, Gaudric A, Dhermy P, Vernant JP, Cordonnier C. Occlusion choriocapillaire au cours de la maladie de Moschowitz. Journal Français Ophtalmologie. 1981;4(2):101-111.

Histologie et expérimentation

- Ischémie choroïdienne expérimentale

Stern WH, Ernest JT. Microsphere occlusion of the choriocapillaris in rhesus monkeys. Am J Ophthalmol. 1974; 78 : 438-448	*Injection de microsphères dans la choriocapillaire par voie artérielle*
Gaudric A, Sterkers M, Coscas G. Retinal Detachment After Choroidal Ischemia. Am J Ophthalmol. 1987;104:364-372.	*Injection de microsphères dans la choriocapillaire par voie vortiqueuse*

Embolisation de la choriocapillaire par voie artérielle

MICROSPHERE OCCLUSION OF THE CHORIOCAPILLARIS IN RHESUS MONKEYS

WALTER H. STERN, M.D., AND J. TERRY ERNEST, M.D.
Chicago, Illinois

A number of authors have used intravascular microspheres to study occlusive disease of the choriocapillaris.[1-4] In the majority of these studies, microspheres traveled into the ophthalmic artery resulting in spheres in both the retinal and choroidal circulations causing difficulty in certain cases in interpretation of the resulting pathology. To overcome this difficulty, Collier[4] injected spheres retrograde via the vortex veins of cats. This resulted, however, in extensive serous detachments of the retina.

To overcome these difficulties the present study was undertaken in which microspheres were delivered to the choriocapillaris via the short posterior ciliary arteries. This technique demonstrated the pattern of arterial blood flow to the macula. The microspheres could be limited to the temporal portion of each eye studied allowing comparison of changes between normal and abnormal areas of the same eye. It was possible to make clinical correlations with various human disease entities involving choriocapillaris occlusion.

MATERIALS AND METHODS

Twelve male and female rhesus monkeys weighing 5 to 6 kg were tranquilized with intramuscular phencyclidine (Sernylan), 2.0 mg/kg, and anesthesia was obtained with pentobarbital sodium administered intravenously. Through a midline neck incision the common carotid artery was isolated and followed to its bifurcation into internal and external carotid arteries. The external carotid artery was then tied off with 4-0 silk suture (Fig. 1). The short posterior ciliary arteries were exposed through a lateral orbital approach. The lateral wall of the orbit was removed, the lateral rectus muscle was cut 5 mm posterior to its insertion, and a clamp attached to the muscle to draw the whole eye forward. The orbital fat and fascia surrounding the optic nerve were then carefully removed until the lateral short posterior ciliary arteries could be clearly seen. A few drops of tolazoline (Priscoline) were usually placed over the arteries as they tended to go into spasm during the manipulations. A heavy silk suture was then placed around the optic nerve 1 to 2 mm posterior to the globe between the lateral short posterior ciliary artery branches and the optic nerve, and over the medial short posterior ciliary artery branches (Fig. 2).

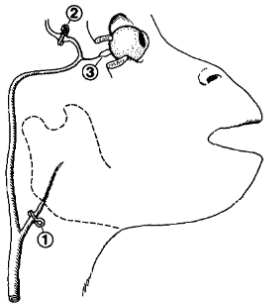
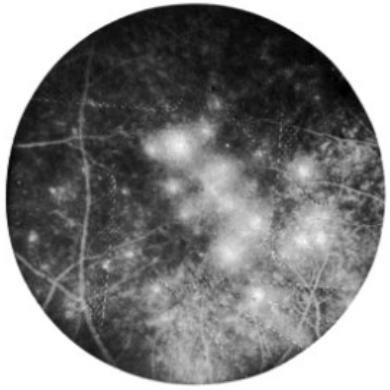


Fig. 1 (Stern and Ernest). The vascular supply to the eye in the rhesus monkey. Clamp 1 is on the external carotid artery. Clamp 2 is on the internal carotid artery distal to the ophthalmic artery. Ligature (3) is around the optic nerve.

From the Eye Research Laboratories, Department of Ophthalmology, University of Chicago, Chicago, Illinois. This study was supported in part by Public Health Service grants EY 00792 and EY 00004 from the National Eye Institute, National Institutes of Health.

Reprint requests to J. Terry Ernest, M.D., Eye Research Laboratories, 950 E. 59th St., Chicago, IL 60637.

B



D

Quelques diffusions de colorant en bordure du territoire ischémique

Décollement séreux rétinien seulement chez des singes rendus hypertendus

Embolisation de la choriocapillaire par voie vortiqueuse

Retinal Detachment After Choroidal Ischemia

Alain Gaudric, M.D., Margaret Sterkers, M.D., and Gabriel Coscas, M.D.

Injection of a 15-μm microsphere suspension through one or two vortex veins of nine monkey eyes caused various degrees of sectorial choroidal ischemia, which were documented by fluorescein angiography and electron microscopy. The severity of the lesions to the fundus depended on the volume of microspheres injected (0.4 to 1.6 ml of a suspension of 600,000 microspheres/ml). Three hours after embolization white patches appeared in the retinal pigment epithelium as well as a posterior pole serous retinal detachment in five eyes. Delayed choroidal filling was noted in the quadrant involved, but a few choriocapillaris units slowly perfused, leading to fluorescein leakage in the serous retinal detachments. Histologic examination showed various types of damage to the retinal pigment epithelium, including vacuolization and cell membrane rupture.

SEROUS RETINAL DETACHMENTS occurring in toxemia of pregnancy, disseminated intravascular coagulation, Harada's disease,[1] and other systemic disorders, are ascribed to multifocal choroidal ischemia,[2-8] but their pathogenesis is not clearly defined. Vascular hypertension in animals produced serous retinal detachment.[10] Embolization of the choriocapillaris by an arterial route did not result in retinal detachments in normotensive animals.[11] We induced serous retinal detachments in monkey eyes by injecting 15-μm microspheres into the choriocapillaris through a temporal vortex vein.[12,13] Angiographic and histologic studies demonstrated that choroidal venous stasis and ischemia of the retinal pigment epithelium were responsible for the serous retinal detachments in monkey eyes.

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Reprint requests to A. Gaudric, M.D., Clinique Ophtalmologique Universitaire, Hôpital Intercommunal —40, avenue de Verdun 94010 Créteil, France.

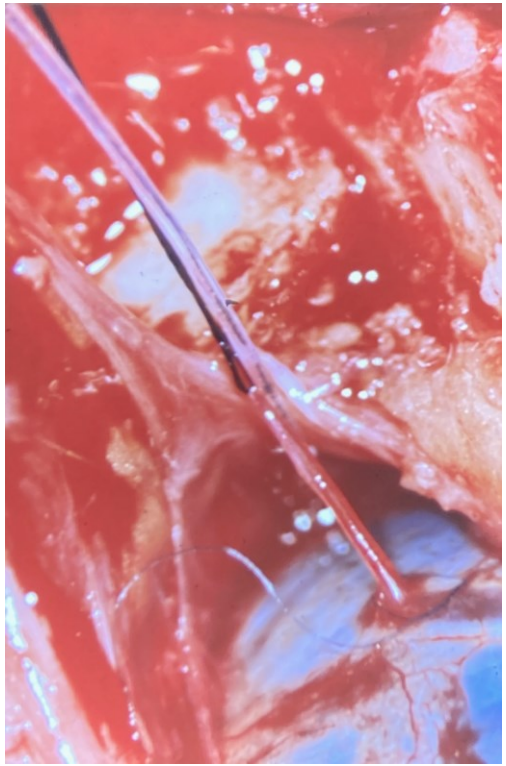
Material and Methods

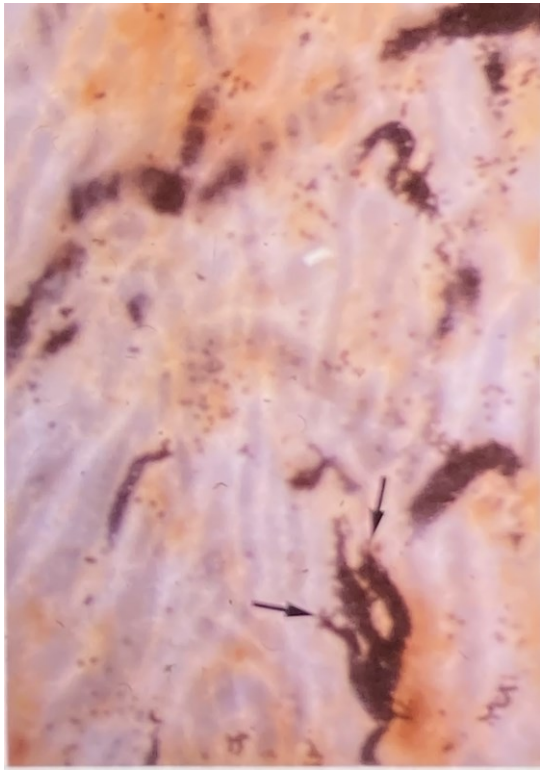
Five adult monkeys of the *Papio papio* and *Patas* species were tranquilized with intramuscular ketamine (10 mg/kg of body weight) and anesthetized with intravenous pentobarbital sodium. Systolic blood pressure did not rise above 140 mm Hg during anesthesia. After lateral orbitotomy, either the superotemporal or inferotemporal vortex vein was exposed and catheterized with a Teflon tube (outside diameter, 0.4 mm; inside diameter, 0.2 mm). The catheter had previously been filled with a saline suspension of polystyrene microspheres, 15 ± 5 μm in diameter, at a concentration of 600,000 microspheres/ml. The microsphere suspension was injected over a five- to ten-minute period either into one temporal vortex vein or successively into both veins. The total volume of suspension injected varied from 0.4 to 1.6 ml. The vortex veins were then ligated and cauterized. Fluorescein angiography was done in all ten eyes, one to three hours after injection. Six of the ten eyes were then enucleated. Three other eyes were observed for one to three weeks, and fluorescein angiography was performed before enucleation. In the single control eye, 1.2 ml of saline containing no microspheres was injected through a temporal vortex vein, which was then ligated (Table).

After enucleation, Eyes 3, 4, 5, and 10 were fixed in Bouin's solution and Eyes 1, 6, 7, 8, and 9 were fixed in glutaraldehyde. In Eye 2, a flat preparation of the choroid was done.

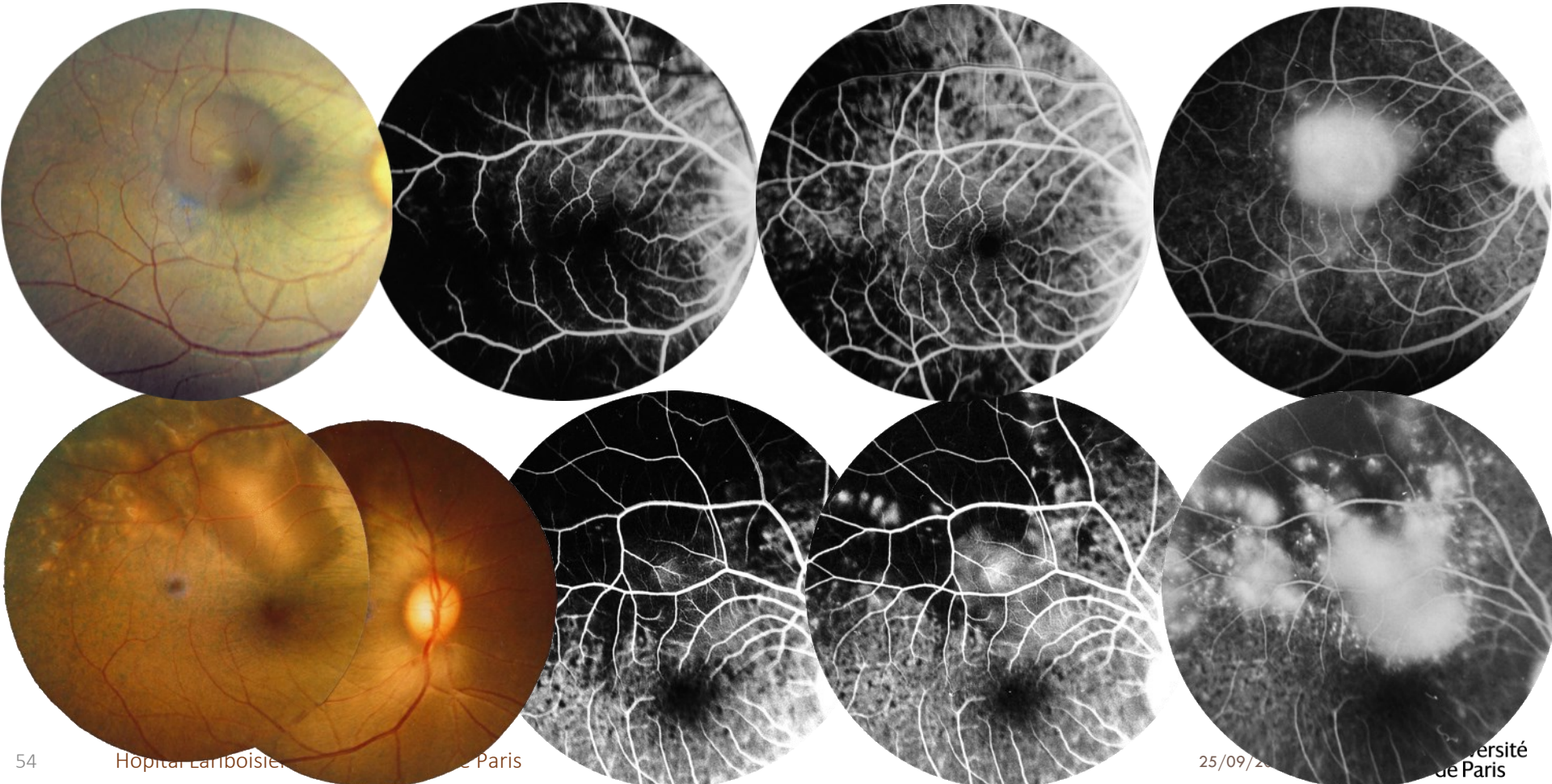
Results

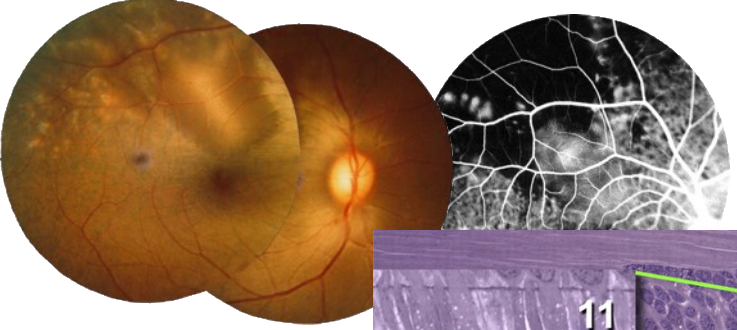
In the area drained by the embolized vein, aggregates of microspheres were slightly visi-

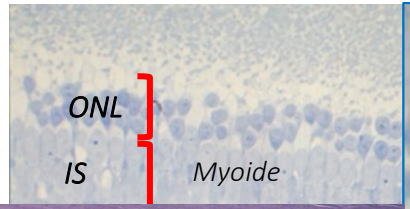


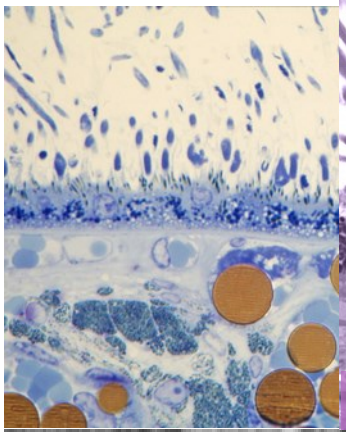


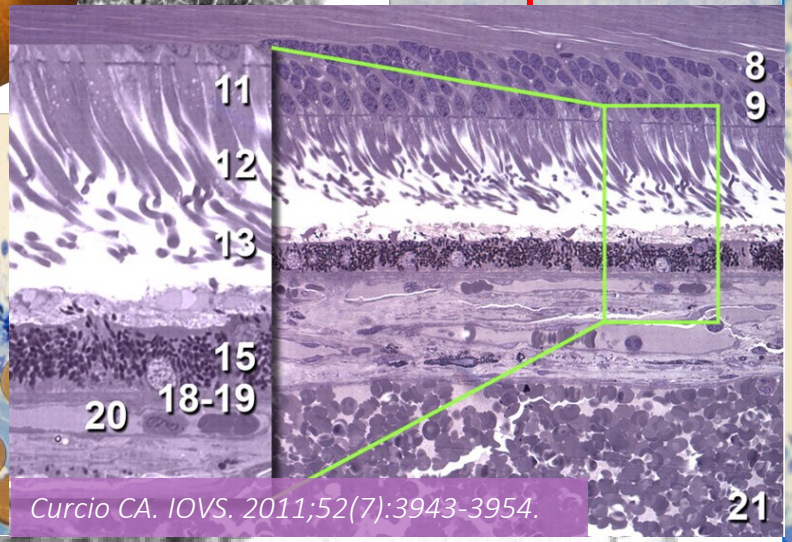
Microsphere embolization of the choroid in monkey eyes. AJO 1987











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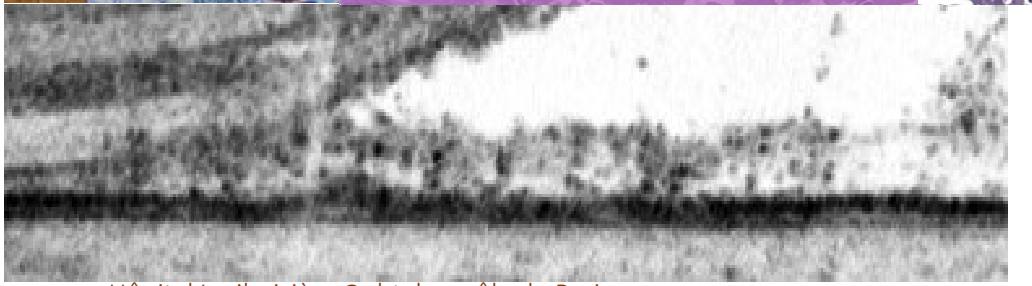
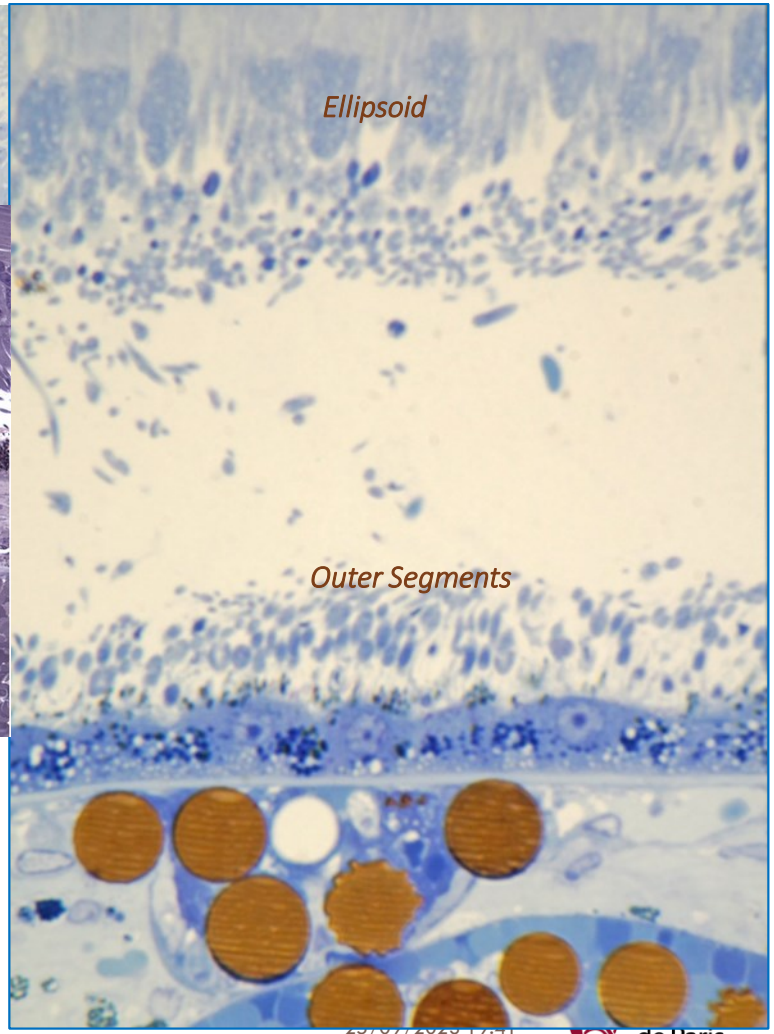
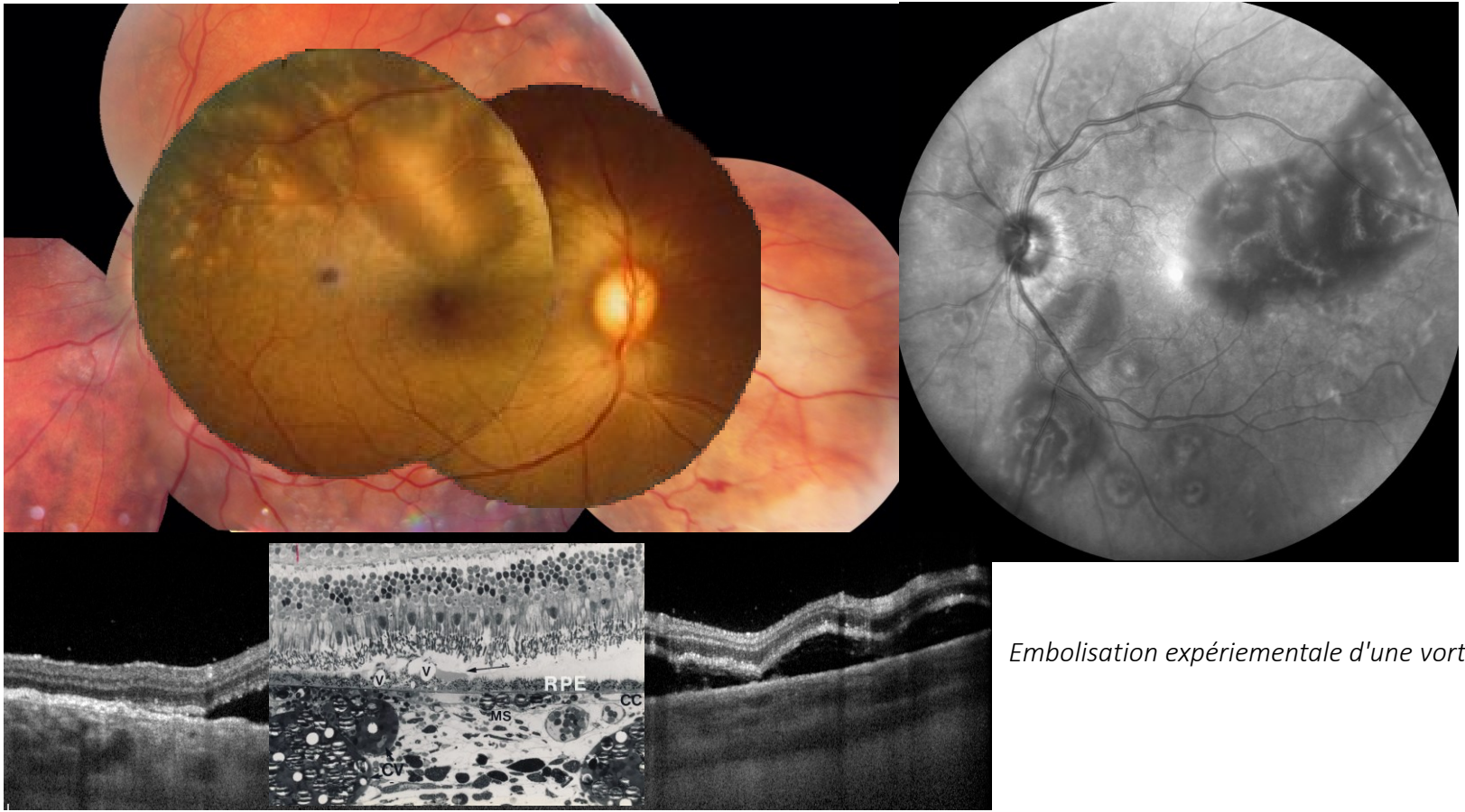
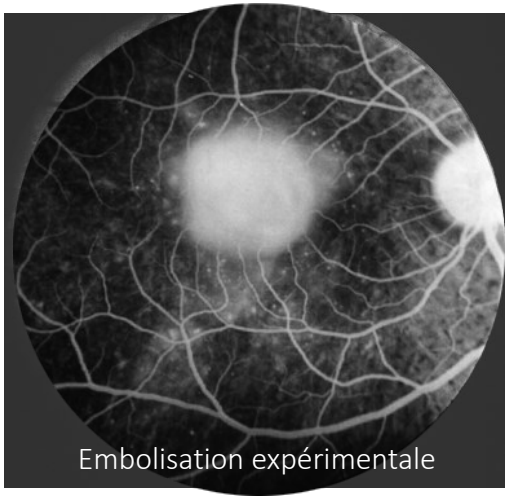


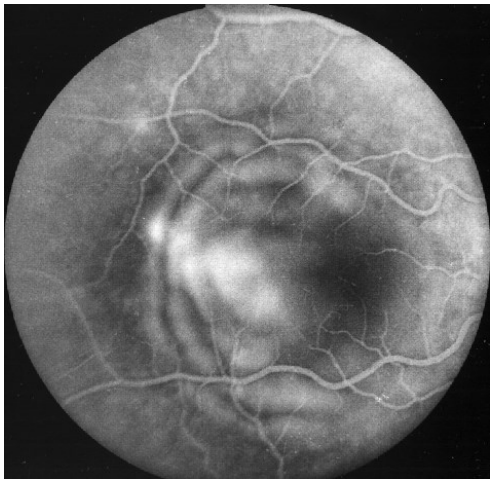


Syndrome catastrophique des anti-phospholipides



Embolisation expériementale d'une vortiqueuse

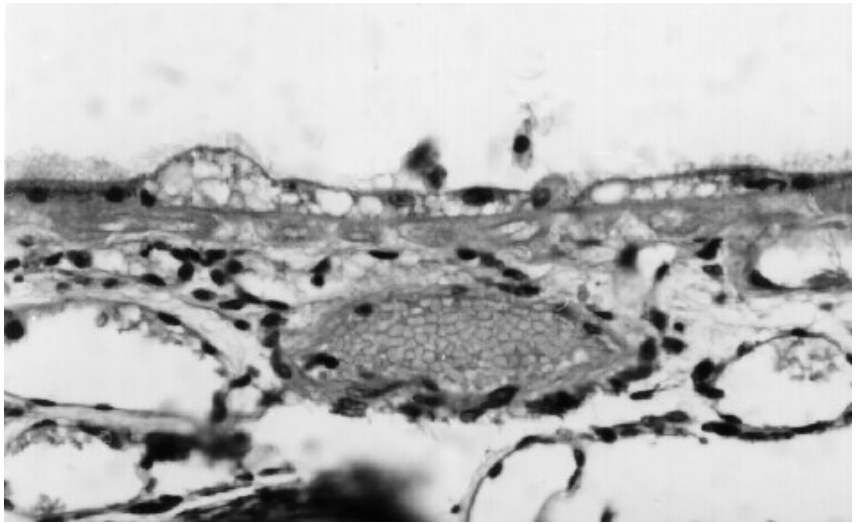

Embolisation expérimentale

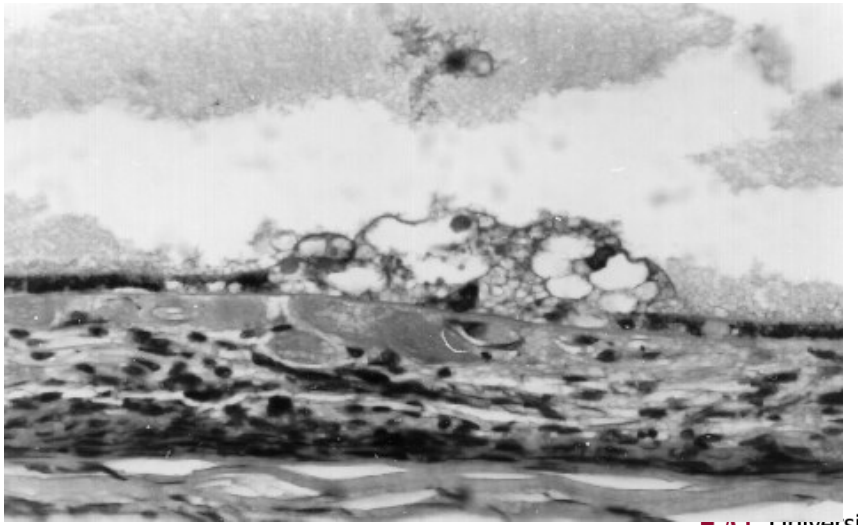


Maladie de Moschowitz

- capillary blood stasis
- vacuolization of RPE cells
- exudative retinal detachment

Obstruction de la choriocapillaire au cours de la maladie de Moschowitz. G Coscas, A Gaudric , P Dhermy et al . J Fr Ophtalmol 1981. 4: 101-111





Retinal Detachment After Choroidal Ischemia

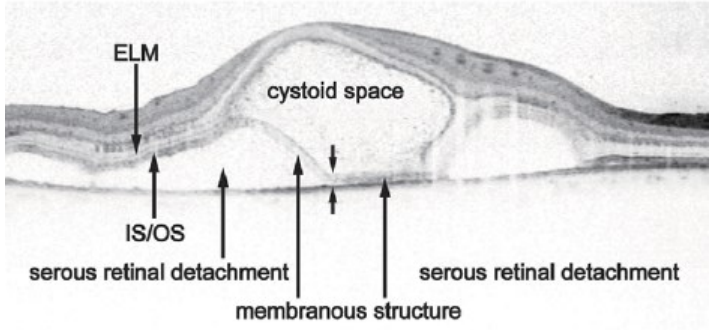
Alain Gaudric, M.D., Margaret Sterkers, M.D., and Gabriel Coscas, M.D.

Injection of a 15-μm microsphere suspension through one or two vortex veins of nine monkey eyes caused various degrees of sectorial choroidal ischemia, which were documented by fluorescein angiography and electron mi-

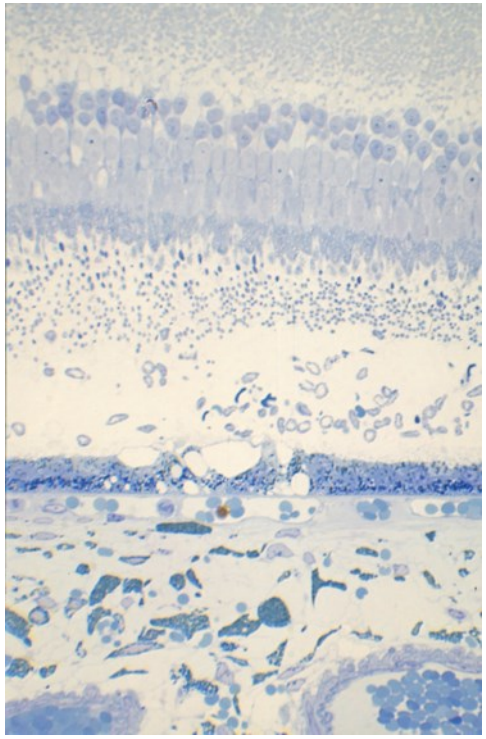
the retinal pigment epithelium were responsible for the serous retinal detachments in monkey eyes.

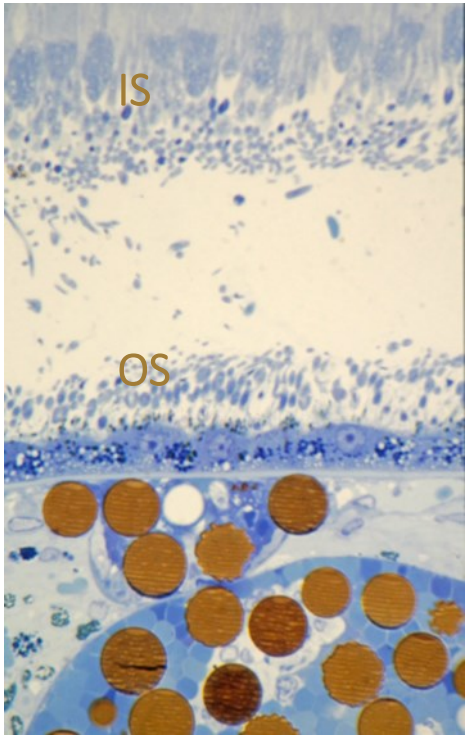
Acute Vogt–Koyanagi–Harada Disease in Enhanced Spectral-Domain Optical Coherence Tomography

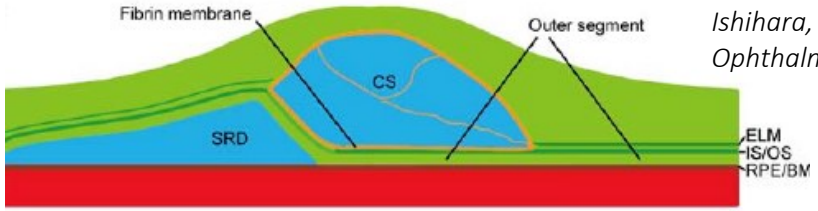
Kenji Ishihara, MD, Masanori Hangai, MD, Mihori Kita, MD, Nagahisa Yoshimura, MD



the membranous structure and cystoid spaces characteristic of this disease involve splitting off of outer segments of the photoreceptor layer from the inner segments and formation of a membranous structure







Ishihara, Yoshimura et al , Ophthalmology 2009

Maladie de Horton
Toxemie gravidique, eclampsie

Rétinochoroidopathie Hypertensive
Lupus érythémateux disséminé
Syndrome des anti-phospholipides
Coagulation intravasculaire disséminée,
Maladie de Moschowitz
Sclérodermie systémique
Syndrome de Goodpasture
Periarterite noueuse
Syndrome Hyperéosinophilique,
Syndrome de Churg and Strauss
Infiltration tumorale de la choroide
Maladie de Harada

Conclusion

- L'ischémie choroidienne comporte 2 variantes
  - l'ischémie en secteur (occlusion des artères ciliaires)
  - l'ischémie multifocale (occlusion choriocapillaire)
- Le diagnostic repose sur l'angiographie
  - qui donne des images caractéristiques à la phase aigüe
- L'ischémie choroidienne en secteur associée à une NOIA doit faire évoquer une Maladie de Horton

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Merci de votre attention,

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