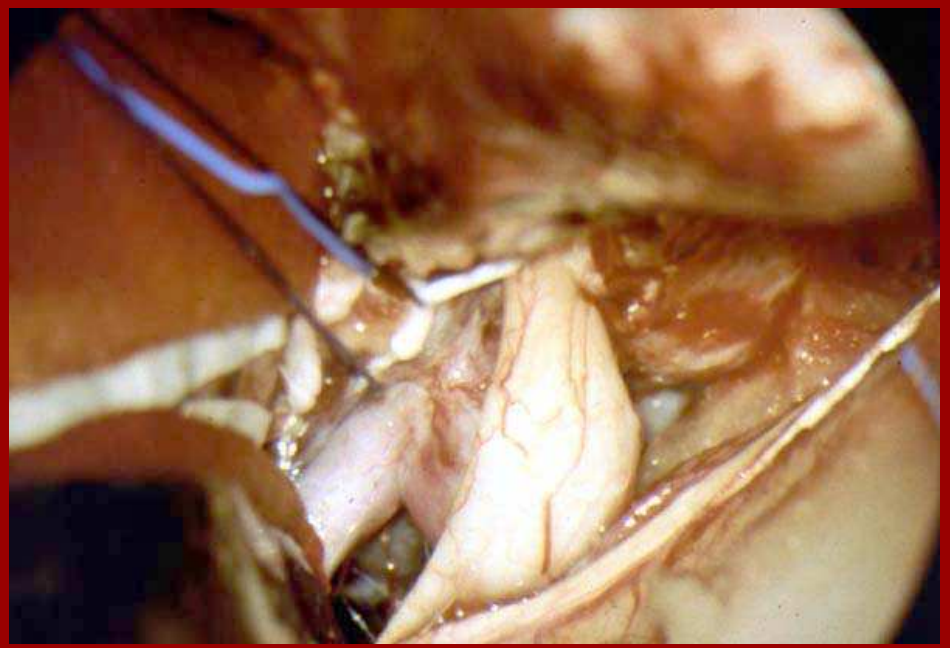
ACTITUD ANTE UNA PERDIDA DE VISION EN UN PACIENTE CON SOSPECHA DE ENFERMEDAD DE HORTON

VII REUNION NACIONAL DE ACTUALIZACION EN ENFERMEDADES AUTOINMUNES SISTEMICAS PARA RESIDENTES

ZARAGOZA, 19 OCTUBRE 2013



Dr Fernando Martínez Valle
Servicio Medicina Interna
Unidad de Enfermedades Sistémicas
Hospital Vall d'Hebró
Barcelona

Paciente de 75 años de edad que acude por pérdida visual súbita de ojo I

ANTECED

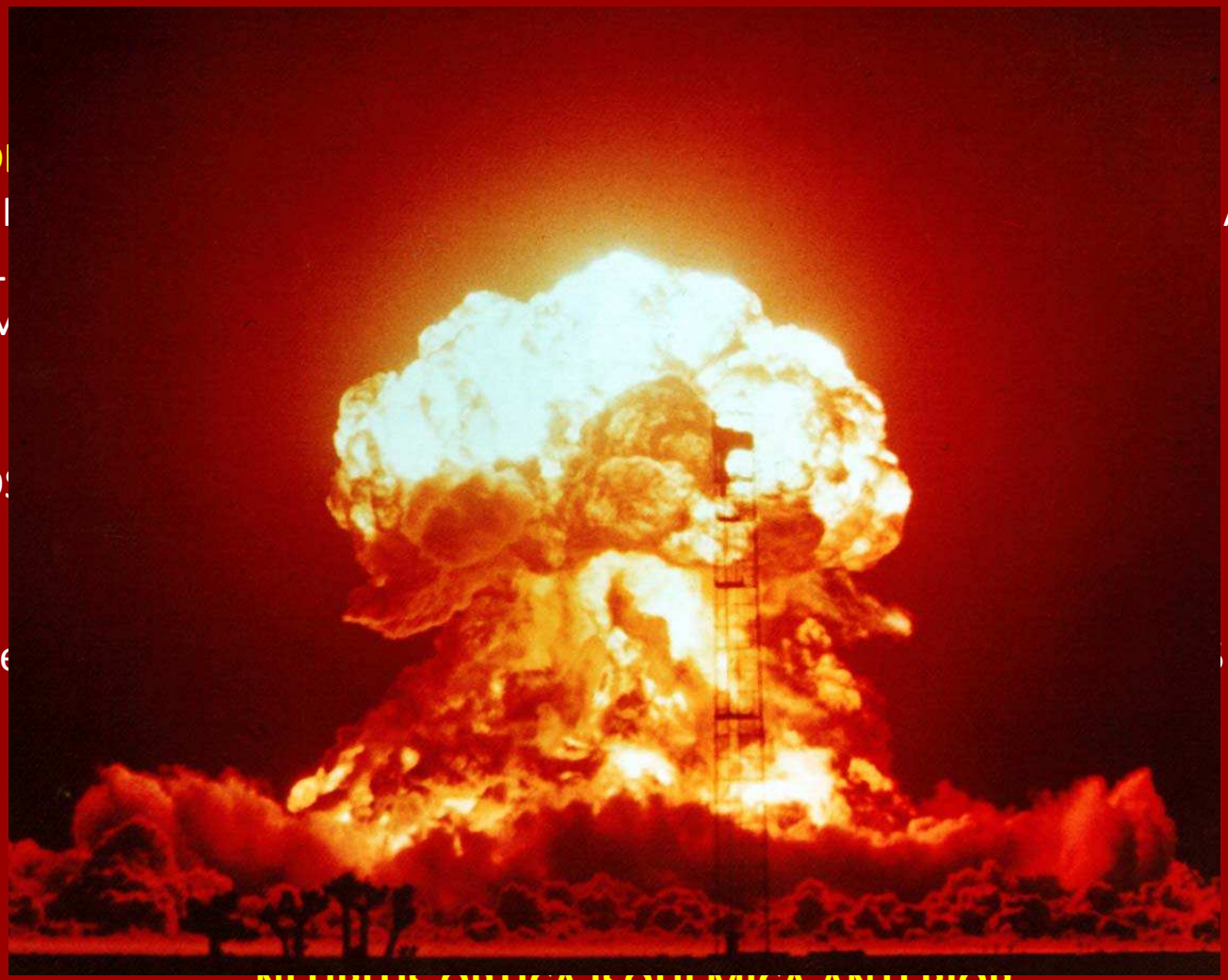
HIPERTE ACC, TZD)

HIPERGL

DISLIPEM

EPISODIO

Hb d

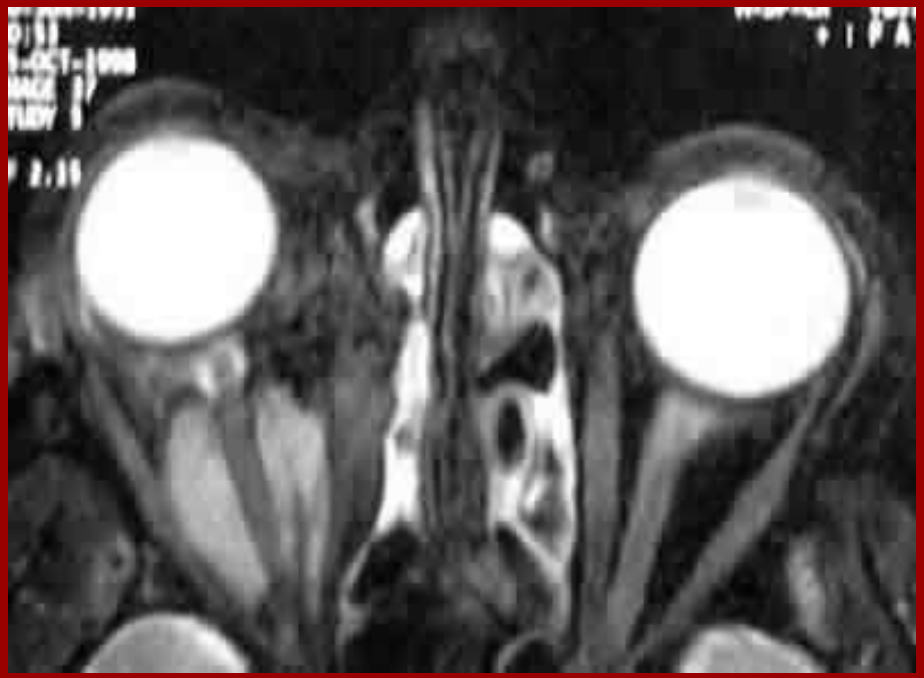


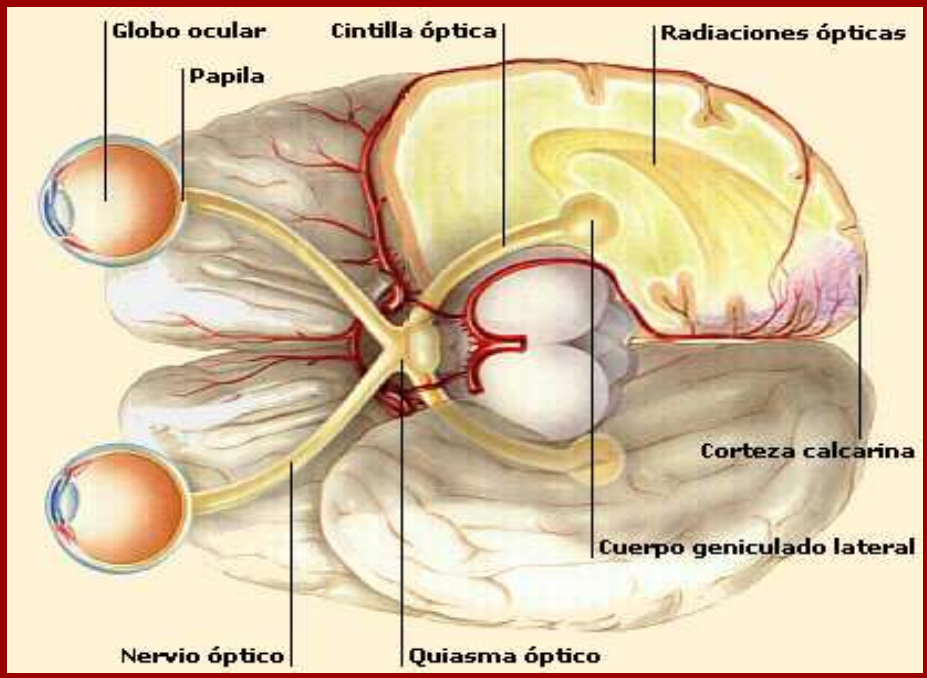
NEURITIS OPTICA ISQUEMICA ANTERIOR

NEUROPATIA OPTICA ISQUÉMICA

NEUROPATIA OPTICA ISQUÉMICA ANTERIOR (NOIA)

NEUROPATIA OPTICA ISQUÉMICA POSTERIOR (NOIP)





NEURITIS OPTICA ISQUEMICA ANTERIOR:

- **ETIOLOGIA ARTERITICA:** ENFERMEDAD DE HORTON
  PAN
  LES

- **NO ARTERITICA**

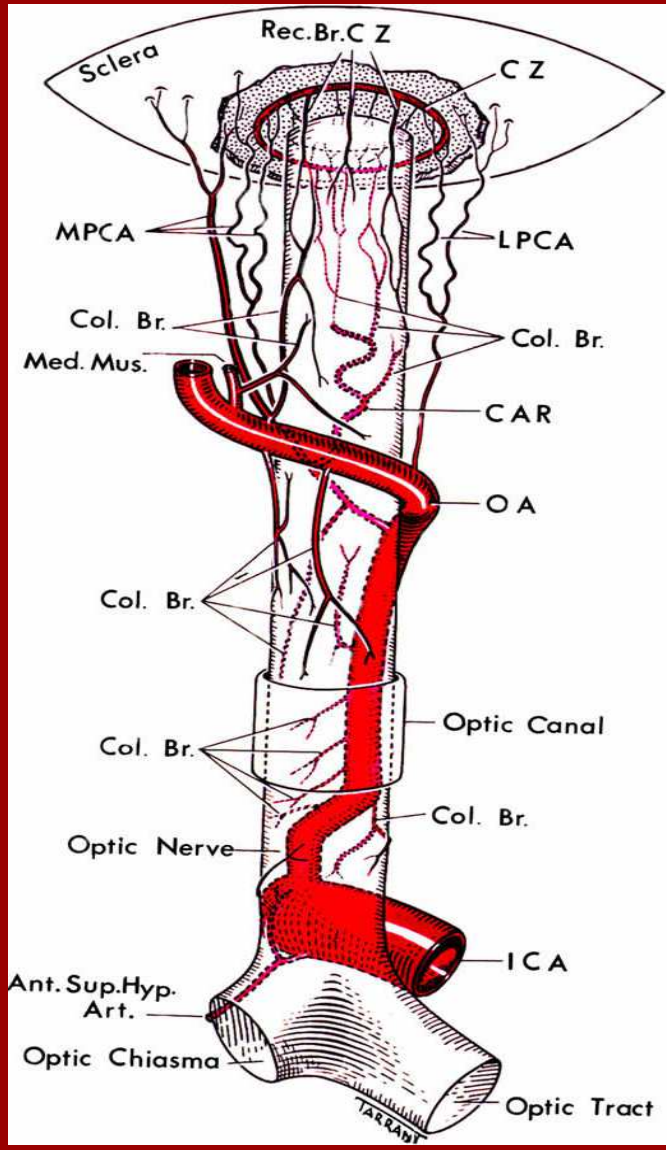
VASCULARIZACION DEL NERVIO OPTICO

-PARTE ANTERIOR DEL NERVIO OPTICO (cabeza, papila):

ARTERIAS CILIARES POSTERIORES

- PARTE POSTERIOR DEL NERVIO OPTICO:

DIFERENTES COLATERALES

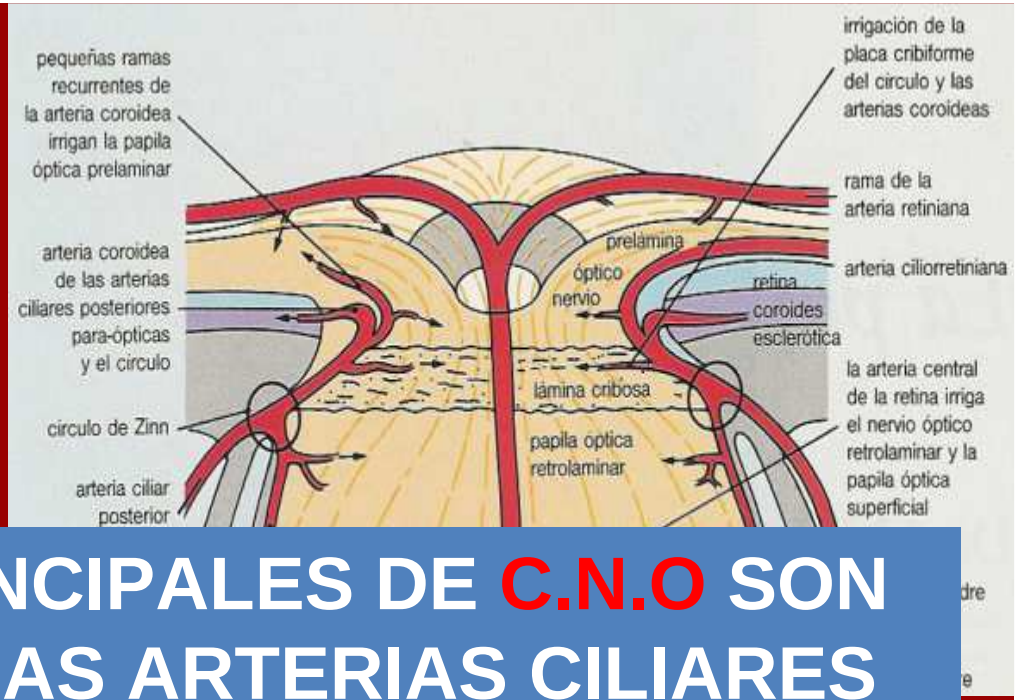


CABEZA DEL NERVIO OPTICO:

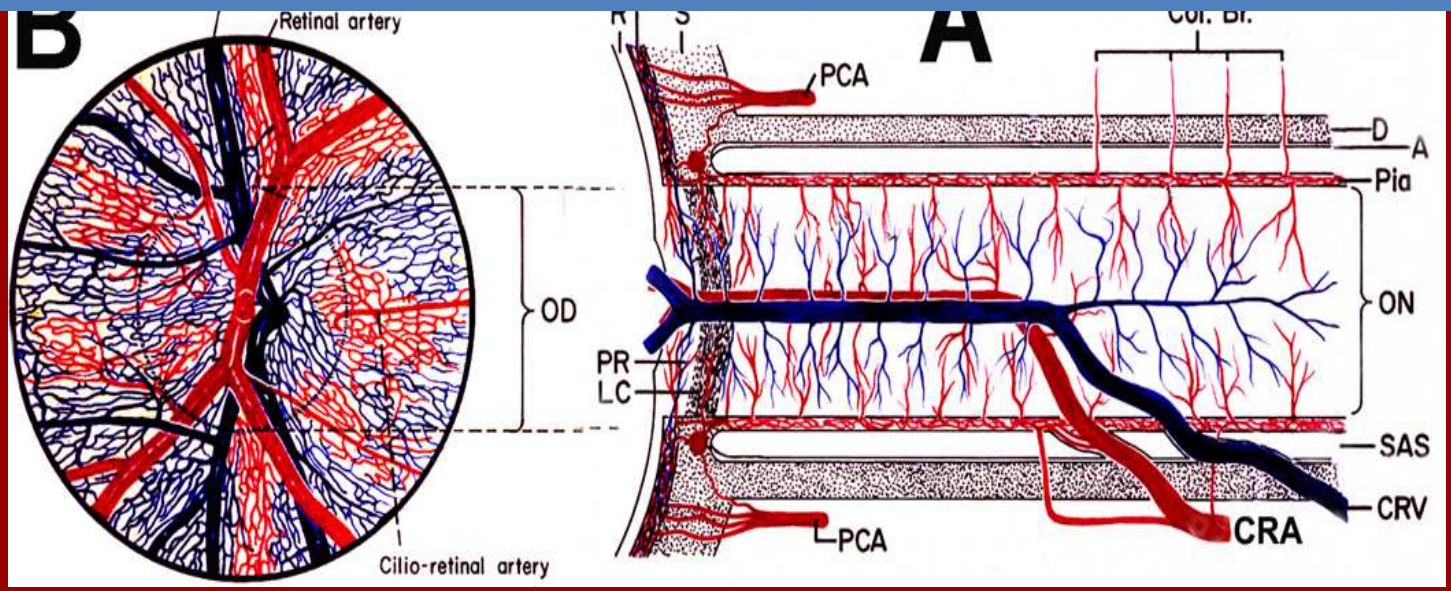
superficie de fibras nerviosas

región prelaminar

lámina cribosa

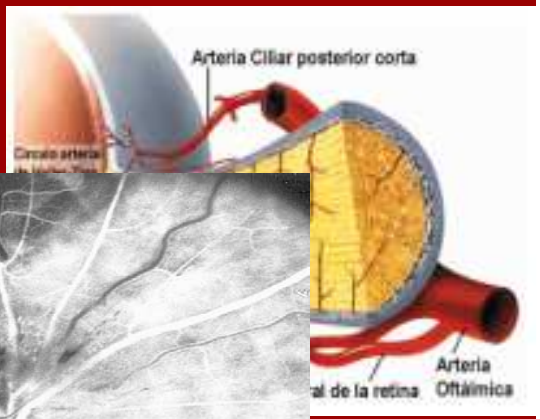


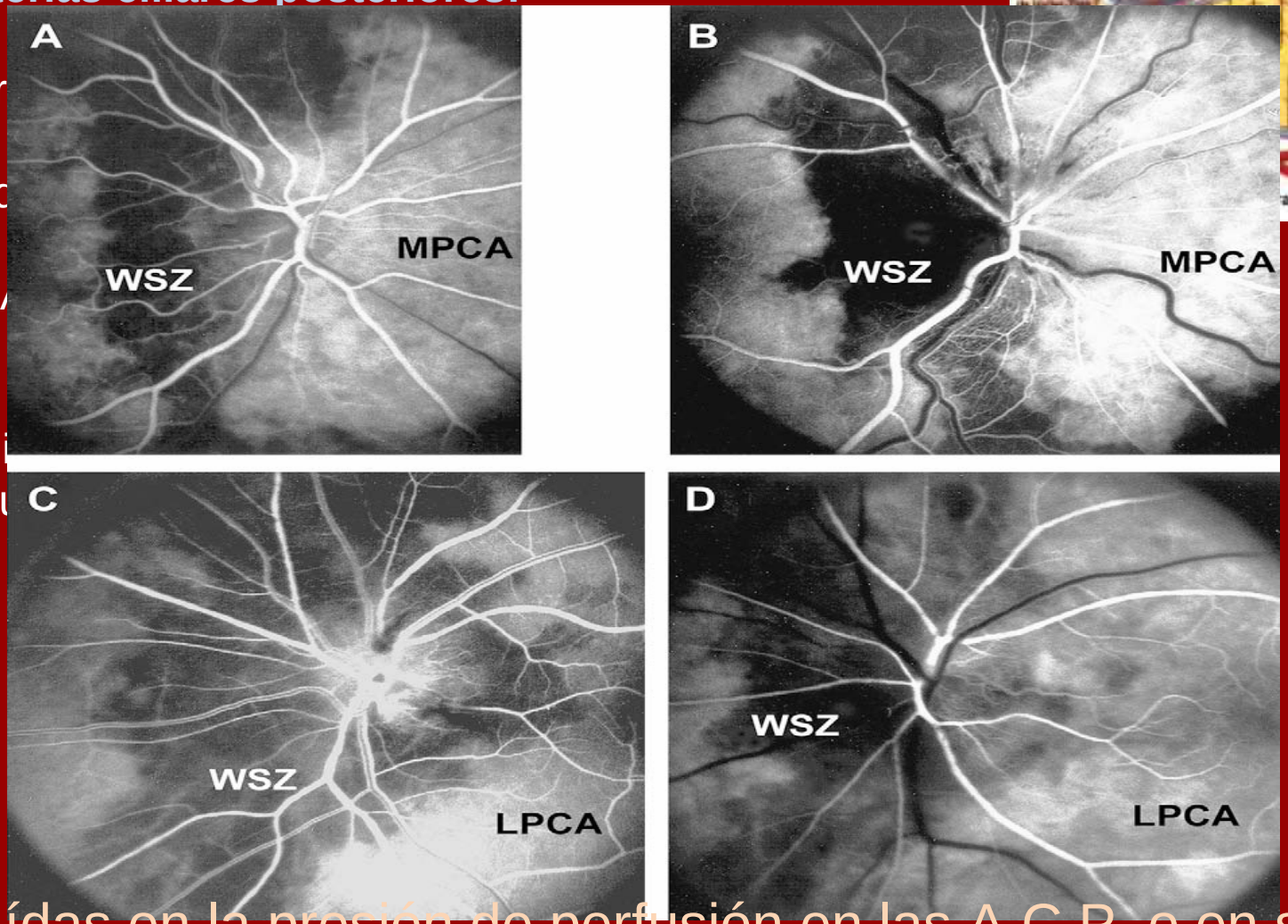
NUTRIENTES PRINCIPALES DE C.N.O SON LAS RAMAS DE LAS ARTERIAS CILIARES POSTERIORES



Las arterias ciliares posteriores:

- entr
- mec
- las A
C.N.O.
- la di
tejido su





Caídas en la presión de perfusión en las A.C.P. o en sus ramas, conlleva la isquemia de las zonas de penumbra

AUTORREGULACION DEL FLUJO DE LA C.N.O

AUTORREGULACION: mantener el flujo continuo a pesar de los cambios de presión

Alteraciones de la autorregulación se pueden producir en:

- HTA
- DIABETES MELLITUS
- ARTERIOESCLEROSIS
- HIPOTENSION
- EDAD

NOIA

Afectación del flujo dependiente de las A.C.P a nivel de la C.N.O.

ETIOLOGIA DE LA NEURITIS OPTICA ISQUEMICA ANTERIOR

1.- NOIA debido a lesiones trombóticas de ACP

1.1.- trombosis secundarias a vasculitis (ACG, SLE, PAN)

EN LAS CAUSAS TROMBÓTICAS Y EMBÓLICAS SUELE HABER UN DAÑO PERMANENTE Y MASIVO EN LA C.N.O CUYA EXTENSIÓN VA A DEPENDER DEL TAMAÑO DE LA ACP AFECTADA

- Inicio súbito
- Excavación profunda del nervio óptico
- Evidencia de oclusión de ACP en la angiografía fluoresceínica

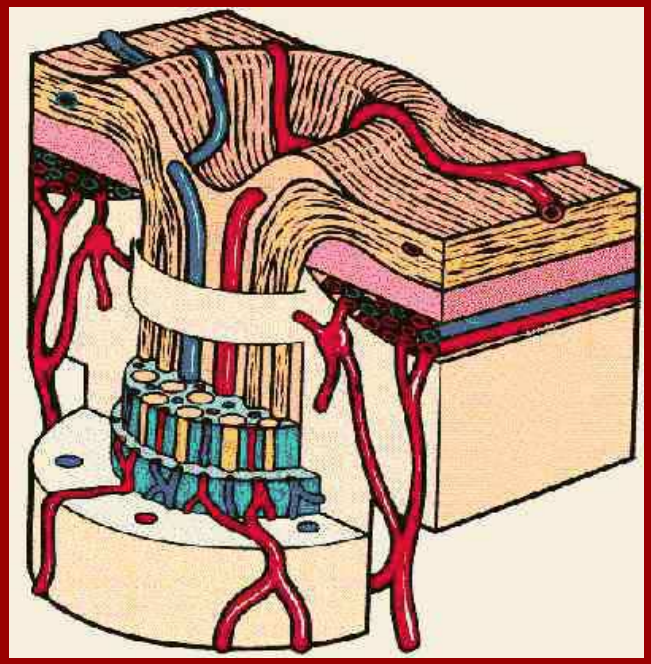
3.- NOIA secundaria a hipoperfusión transitoria de las arterias nutrientes de la C.N.O

- Caída transitoria de la presión de perfusión
- pacientes susceptibles: NOIA no arterítica
- no oclusión de las A.C.P
- causa más frecuente de NOIA

- CAÍDA DE TA (nocturno)
- ESTENOSIS DE ARTERIA CARÓTIDA INTERNA U OFTÁLMICA
- ALTERACIONES HEMATOLÓGICAS

FACTORES OCULARES ASOCIADOS A NA-NOIA

1.- Excavación pequeña o ausente en el nervio óptico



2.- Aumento de presión intraocular

3.- Edema del disco óptico

4.- Localización de las zonas de penumbra dependientes de las A.C.P en relación a la C.N.O.

5.- Alteraciones de la autoregulación en las arterias que irrigan la C.N.O.

AFECTACION BILATERAL EN LA NOIA

NA-NOIA: riesgo de afectación contralateral del 25% a los 3 años

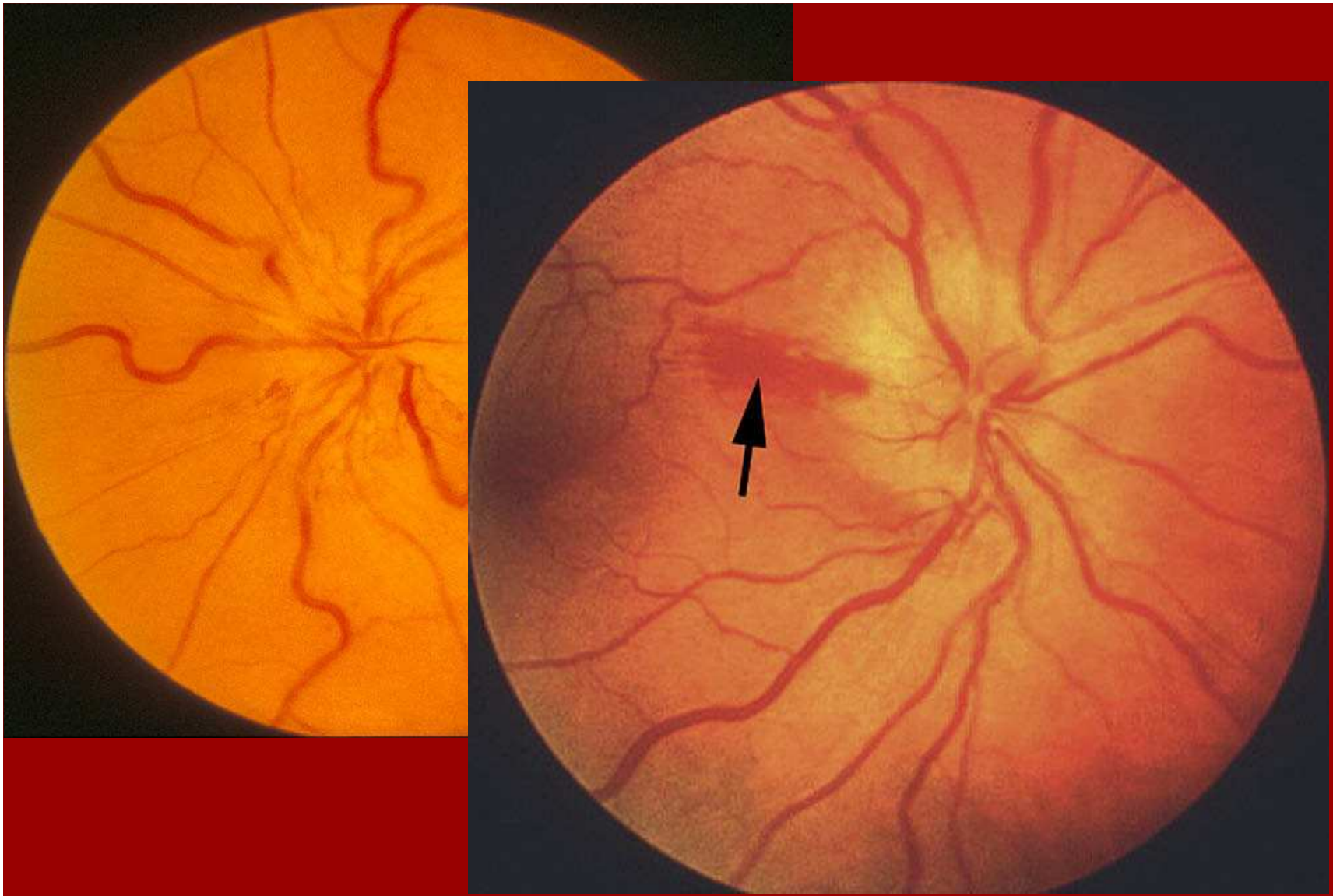
A-NOIA: en los casos de bilateralidad, el 95% estará presente en el momento de la consulta hospitalaria (paciente sin tratamiento)

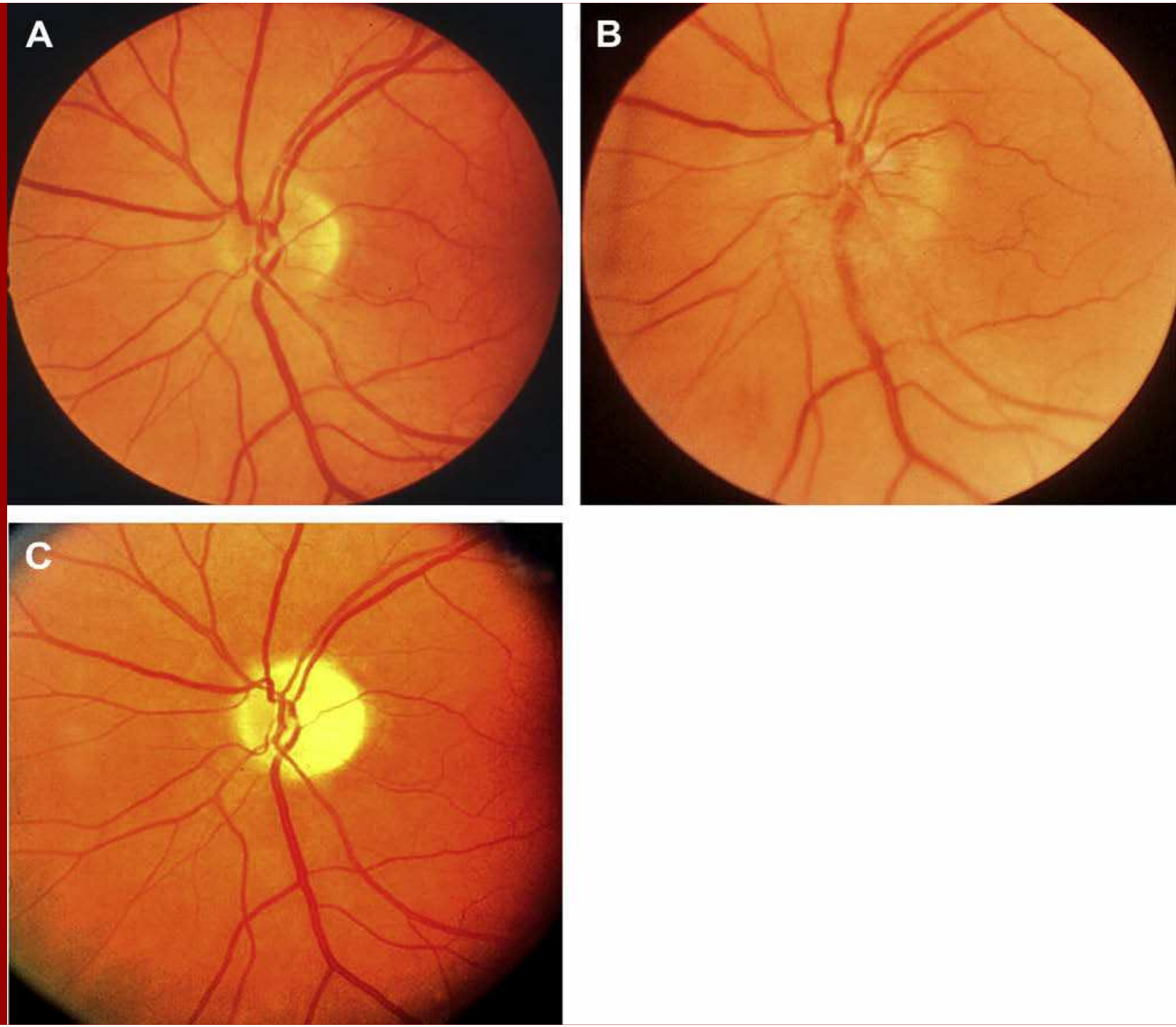
pero unos pocos casos la presentan al cabo de 3-4 días de haber consultado (bajo tto con corticoides).

ALTERACIONES OFTALMOSCÓPICAS

NA-NOIA

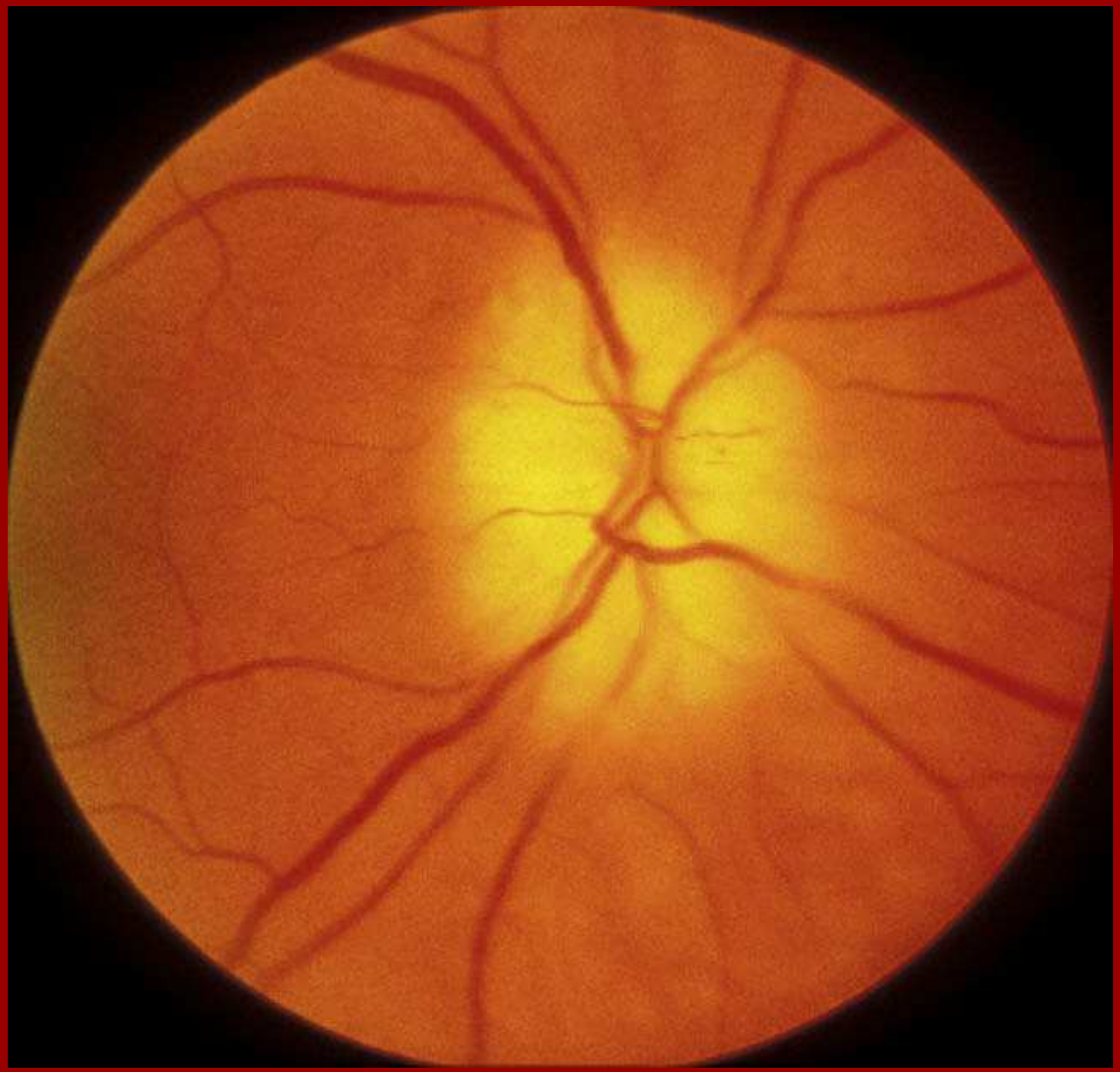
- Disco óptico edematoso (puede ser asimétrico)
- hemorragias en astilla frecuentes
- Gradualmente el disco óptico se vuelve pálido (se inicia a las dos semanas) y el edema desaparece
- A los 2-3 meses, desaparece edema y disco óptico pálido

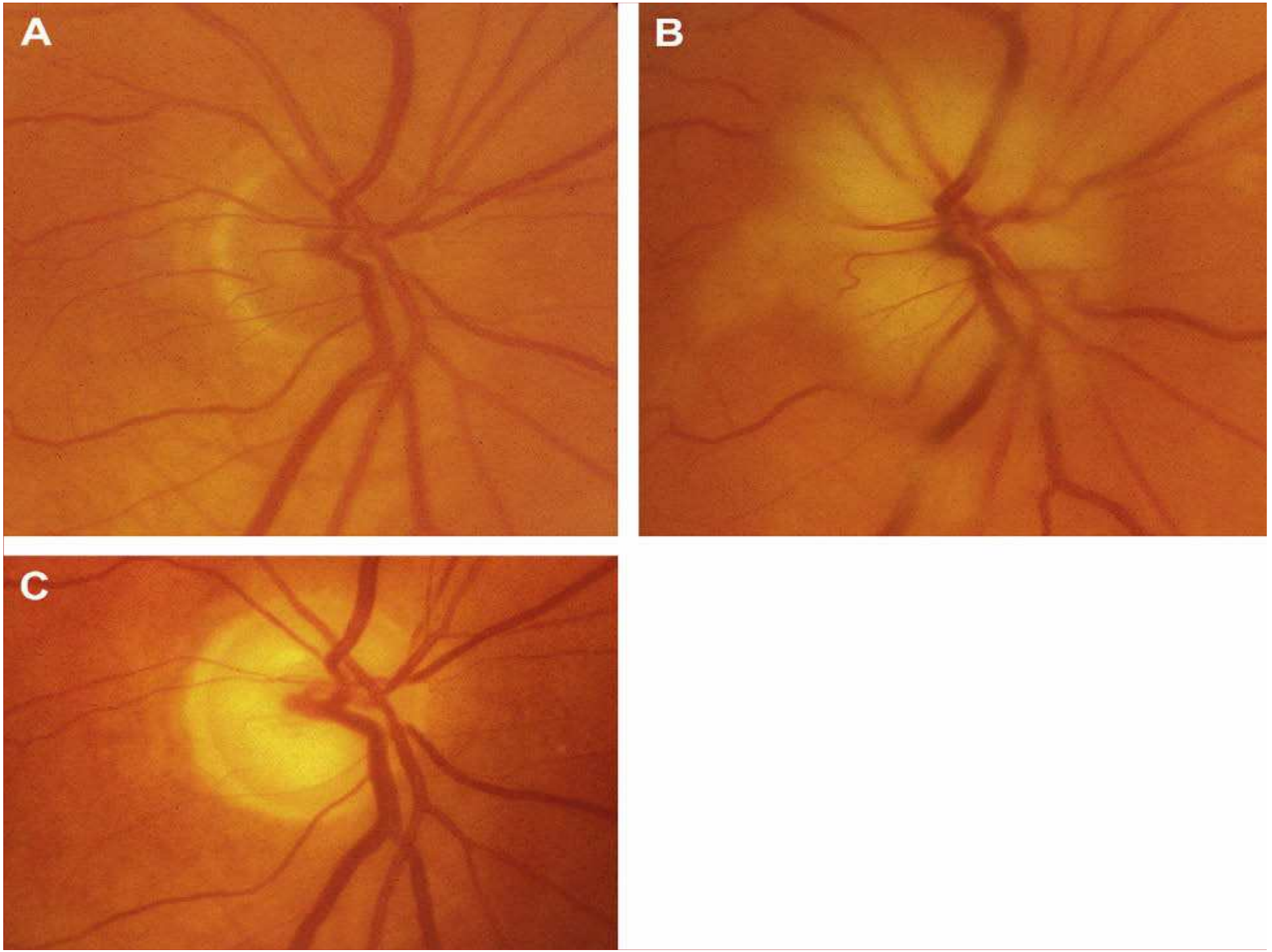



A
B
C

A-NOIA

- En el **69%** de los casos, aspecto “chalky” característico
- Patrón evolutivo de la papila similar al de la NA-NOIA
- En fases evolucionadas, la excavación es igual a la que aparece en el disco óptico glaucomatoso (pero más pálida)




A
B
C

MANIFESTACIONES CLÍNICAS

A-NOIA

- La presencia de amaurosis fugax o diplopia es muy sugestiva de GCA

- Pérdida visual masiva y precoz sugestivo de GCA:

  54% en GCA
  14% en NA-NOIA

NA-NOIA

- Defecto más frecuente:

  - sector inferior nasal (absoluto o relativo)
  - inferior altitudinal
  - escotoma central

- No clínica de patología sistémica ni síntomas cefálicos

Hasta un 20% de los pacientes con ACG diagnosticados mediante BAT, no presentan síntomas sistémicos ni cefálicos

¿SE DEBE INICIAR TRATAMIENTO CON CORTICOIDES?

BENEFICIO CLARO EN PACIENTES AFECTOS DE ACG

¿ESTA INDICADO SU USO EN NA-NOIA?

Brit J. Ophthal (1974) **58**, 981

Anterior ischaemic optic neuropathy

III. Treatment, prophylaxis, and differential diagnosis

SOHAN SINGH HAYREH

Department of Ophthalmology, University of Iowa

Group	*Systemic cortico-steroid therapy*	*No. of patients*	*Visual acuity (per cent.) during follow-up*		
			No change	*Improvement*	*Deterioration*
I	With therapy and temporal arteritis	11	64	9	27
II	With therapy and no temporal arteritis	8	12·5	75	12·5
III	No therapy and no temporal arteritis	6	83	17	Nil

Graefes Arch Clin Exp Ophthalmol (2008) 246:1029–1046
DOI 10.1007/s00417-008-0805-8

NEURO-OPHTHALMOLOGY

Non-arteritic anterior ischemic optic neuropathy: role of systemic corticosteroid therapy

Sohan Singh Hayreh · M. Bridget Zimmerman

Objective To investigate systematically the role of systemic corticosteroid therapy in non-arteritic anterior ischemic optic neuropathy (NA-AION).
Methods The study consists of a cohort of 613 consecutive patients (696 eyes), first seen in our clinic from 1973 to 2000. Of this cohort, 312 patients (364 eyes) voluntarily opted for systemic steroid therapy, and 301 (332 eyes) for no treatment. At first visit, all patients in both groups had a

Table 3 Visual acuity change from initial visit to optic disc edema (ODE) resolution

Visual acuity at initial visit	Steroid started ≤2 weeks from onset (n=261 eyes)			Steroid started >2 weeks from onset (n=101 eyes)		
	n	Number (%) of eyes		n	Number (%) of eyes	
		Improved	Worsened		Improved	Worsened
20/15–20/30	140	–	21 (15%)	47	–	3 (6%)
20/40	22	1 (5%)	3 (14%)	11	0 (0%)	1 (9%)
20/50–20/60	29	6 (21%)	0 (0%)	15	1 (7%)	0 (0%)
20/70–20/100	26	9 (35%)	1 (4%)	8	2 (25%)	0 (0%)
20/200–20/400	11	7 (64%)	0 (0%)	4	1 (25%)	0 (0%)
Counting fingers or worse	33	15 (45%)	0 (0%)	16	5 (31%)	0 (0%)
VA 20/70 or worse	70	31 (44%)	1 (1%)	28	8 (29%)	0 (0%)

Table 9 Comparison of percent improvement in visual acuity* among those with initial visual acuity of 20/70 of worse that were seen within 2 weeks on NA-AION onset who received steroid therapy within two weeks of onset and those that did not receive any steroid

Follow-up period	Percent with improved visual acuity* (95% Confidence Interval)		Odds ratio (95% CI) for VA improvement (steroid/no steroid)	*p* value
	Steroid	No steroid**		
At ODE resolution	44.2% (33.1%, 56.0%)	21.2% (13.2%, 32.2%)	2.95 (1.42, 6.17)	0.004
3 months from first visit	47.1% (35.8%, 58.7%)	16.7% (9.6%, 27.3%)	4.45 (2.03, 9.75)	0.0002
6 months from first visit	69.8% (57.3%, 79.9%)	40.5% (29.2%, 52.9%)	3.39 (1.62, 7.11)	0.001
1 year from first visit	72.2% (60.2%, 81.6%)	39.0% (27.6%, 51.7%)	4.06 (1.92, 8.57)	0.0002

*Estimates obtained from the repeated measures logistic regression model fitted using the generalized estimating equations (GEE) method
**From cohort of patients in our natural history study of NA-AION [28].
CI = Confidence interval; NA-AION = Non-arteritic anterior ischemic optic neuropathy; ODE = Optic disc edema; VA = Visual acuity

Table 10 Comparison of percent improvement in visual field defect* between those with initial moderate to severe visual field defect and were seen within 2 weeks of NA-AION onset who received steroid therapy within 2 weeks of onset and those that did not receive any steroid

Follow-up period	Percent with improved visual field* (95% confidence interval)		Odds ratio (95% CI) for VF improvement (steroid/no steroid)	*p* value
	Steroid	No steroid**		
At ODE resolution	36.6% (29.7%, 43.9%)	19.6% (13.8%, 27.0%)	2.36 (1.41, 3.96)	0.001
3 months from first visit	37.7% (30.8%, 45.0%)	19.7% (13.9%, 27.2%)	2.47 (1.48, 4.12)	0.0006
6 months from first visit	40.1% (33.1%, 47.5%)	24.5% (17.7%, 32.9%)	2.06 (1.24, 3.40)	0.005
1 year from first visit	40.0% (33.0%, 47.3%)	24.7% (17.8%, 33.1%)	2.03 (1.23, 3.36)	0.006

*Estimates obtained from the repeated measures logistic regression model fitted using the generalized estimating equations (GEE) method
**From cohort of patients in our natural history study of NA-AION [28]
CI = Confidence interval; NA-AION = Non-arteritic anterior ischemic optic neuropathy; ODE = Optic disc edema; VF = Visual field

Graefes Arch Clin Exp Ophthalmol (2013) 251:255–260
DOI 10.1007/s00417-012-1995-7

NEUROPHTHALMOLOGY

Visual and anatomical outcomes of non-arteritic anterior ischemic optic neuropathy with high-dose systemic corticosteroids

Gema Rebolleda · Marta Pérez-López · Pilar Casas-LLera · Inés Contreras · Francisco José Muñoz-Negrete

	Steroid therapy group (n=9)	Untreated group (n=27)	P value
Visual acuity onset (logMAR)	0.69 (±0.57) [0.34; 1.07]	0.4 (±0.82) [0.18;0.9]	0.173
Median change VA	−0.032 (±0.21) [−0.3; 0.04]	0.00 (±0.84) [−0.14; 0.52]	0.28
MD onset (dB)	−19.7(±6.3) [−23.7; −12.9]	−17.4 (±6.9) [−26.3; −15.5]	0.874
MD 6 months (dB)	−21.9 (±5.3) [−24.8; −15.2]	−18.4 (±4.6) [−26.1; −10.6]	1.00
Change MD (final–initial)	−0.56 (±5.03) [−3.6; 0.7]	1.27 (±6.9) [−2.14; 5.2]	0.213
PSD onset (dB)	10.3 (±2.7) [7.7; 12.9]	11.8 (±4.2) [7.7; 13.2]	0.548
PSD 6 months (dB)	11.7 (±2.7) [8.5; 12.8]	9.3 (±4.1) [7.1; 11.7]	0.213
Change PSD (initial–final)	−0.02 (±1.9) [−1.6; 0.6]	0.91 (±4.8) [−0.75; 2.9]	0.07

CF, counting fingers; HM, hand motion; MD, mean deviation; PSD, pattern standard deviation; * P values for Mann–Whitney test

Conclusion High-dose systemic steroid treatment did not show any beneficial effect in visual and anatomic outcomes when given during the acute phase of NAION. Furthermore, it caused serious complications in a third of the patients treated.

Visual improvement with corticosteroid therapy in giant cell arteritis. Report of a large study and review of literature

Sohan Singh Hayreh[1], Bridget Zimmerman[2] and Randy H. Kardon

[1]Department of Ophthalmology and Visual Sciences, College of Medicine, University of Iowa, Iowa, USA
[2]Department of Biostatistics, College of Public Health, University of Iowa, Iowa, USA

Acta Ophthalmol. Scand. 2002: 80: 353–367

Is intravenous steroid therapy more effective than oral therapy in causing visual improvement in GCA patients?

Table 3. Comparison of patients/eyes with intravenous (IV) steroid therapy versus oral steroid therapy.

Variable		Comparison of patients: IV therapy (n = 41 patients)		Comparison of patients: Oral therapy only (n = 43 patients)	
Initial dose (mg equivalent of Prednisone)		(*n* = 40)*		(*n* = 43)	
	55	-		1	(2%
	60	-		3	(7%
	80	-		24	(56%
	100–160†	-		15	(35%
	300–330	3	(8%)	-	
	500	3	(8%)	-	
	800–850	5	(12%)	-	
	1000	29	(72%)	-	
Involved eye	Right	11	(27%)	13	(30%)
	Left	12	(29%)	18	(42%)
	Both	18	(44%)	12	(28%)
		Fisher's exact test p = 0.310			
Gender	Male	13	(32%)	10	(23%)
	Female	28	(68%)	33	(77%)
		Fisher's exact test p = 0.466			
Age at first visit to our clinic	Mean ± SD	78.1 ± 6.5		75.1 ± 7.8	
	Median (IQR)	78.1 (74.6–82.1)		76.6 (70.8–78.9)	
	Range	63.1–93.4		57.1–92.5	
		Wilcoxon rank-sum test p = 0.068			
		(*n* = 41)		(*n* = 42)‡	
Initial ESR mm/hour Westergren	Mean ± SD	86.8 ± 30.0		71.0 ± 35.4	
	Median (IQR)	86 (64–112)		67 (40–102)	
	Range	32–134		5–140	
		Wilcoxon rank-sum test p = 0.037			

central visual field was found in seven eyes (in six patients). Visual improvement was seen in 7% of 41 patients treated initially with intravenous steroids versus 5% (p = 0.672) of 43 patients treated with oral steroids only. Comparison of

Visual improvement with corticosteroid therapy in giant cell arteritis. Report of a large study and review of literature

Sohan Singh Hayreh[1], Bridget Zimmerman[2] and Randy H. Kardon[1]

[1]Department of Ophthalmology and Visual Sciences, College of Medicine, University of Iowa, Iowa, USA
[2]Department of Biostatistics, College of Public Health, University of Iowa, Iowa, USA

Acta Ophthalmol. Scand. 2002: 80: 353–367

Methods: Clinical data were collected systematically on 84 consecutive patients (114 eyes) with visual loss, all of whom had GCA confirmed by temporal artery biopsy and treated by us with high-dose systemic corticosteroid therapy. The patients were treated between 1974 and 1999 and data were compiled retrospectively. All patients underwent a detailed visual and ophthalmic evaluation at the initial visit and at every follow-up. This included visual field testing (with a Goldmann perimeter). All were treated with systemic corticosteroid therapy (intravenous followed by oral in 41 patients and oral only in 43 patients).

Conclusions: In our study, only 4% of eyes with visual loss due to GCA improved, as judged by improvement in both VA and central visual field (by kinetic perimetry and Amsler grid). The data also suggest that there is a better ($p = 0.065$) chance of visual improvement with early diagnosis and immediate start of steroid therapy. Improvement in VA without associated improvement in the central visual field or Amsler grid may simply represent a learned ability to fixate eccentrically with more effective use of remaining vision: this factor could help explain a number of reported cases in the literature of improved VA after steroid treatment for GCA. To prevent further visual loss in either eye and for management of systemic manifestations of GCA, all patients must be treated on a long-term basis with adequate amounts of systemic corticosteroids.

Aspirin therapy in nonarteritic anterior ischemic optic neuropathy
Beck, Roy W;Sohan Singh Hayreh;Podhajsky, Patricia A;Eng-Suan Tan;Moke, Pamela S
American Journal of Ophthalmology; Feb 1997; 123, 2; ProQuest Health & Medical Complete
pg. 212

Aspirin Therapy in Nonarteritic Anterior Ischemic Optic Neuropathy

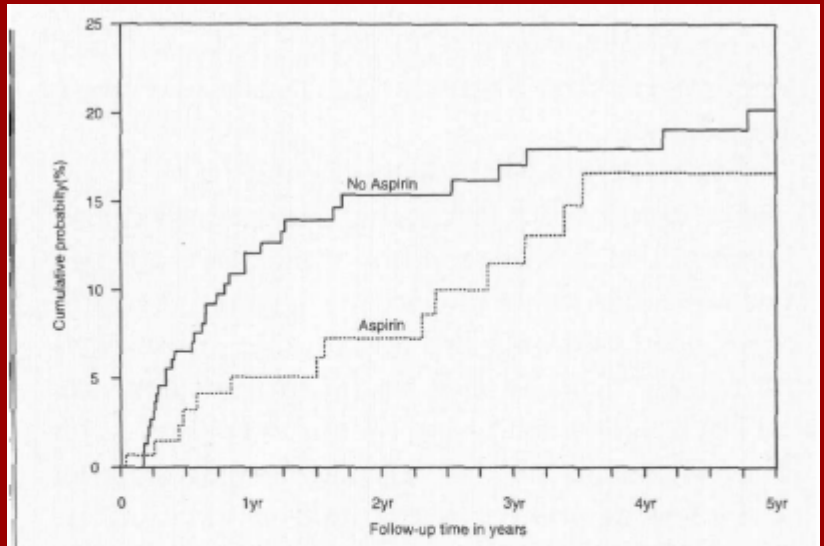


Figure. Kaplan-Meier curves showing cumulative probability of nonarteritic anterior ischemic optic neuropathy in the fellow eye in the aspirin-user and no-aspirin groups. See data in Table 2 regarding the censoring in each interval (by the Mantel log rank test, $\chi^2 = 1.54$, $P = .22$).

TABLE 2. Baseline Characteristics of Patients With no Baseline NAION in Fellow Eye by Incidence of New NAION in Fellow Eye

Variable	New NAION		No new NAION		*P* Value
	No.	(%)	No.	(%)	
Total number	48	(15)	278	(85)	
Age					
<65	22	(15)	124	(85)	0.75
≥65	26	(14)	154	(86)	
Sex					
Male	28	(14)	171	(86)	0.72
Female	20	(16)	107	(84)	
Study group					
Careful follow up	19	(18)	84	(82)	0.46
Surgery	16	(16)	82	(84)	
Nonrandomized	13	(10)	112	(90)	
Baseline visual acuity*					
Better than 20/64	13	(11)	110	(89)	0.10
20/64 to better than 20/200	10	(12)	72	(88)	
20/200 or worse†	24	(20)	95	(80)	
Smoking status*					
Current	8	(19)	35	(81)	0.55
Previous	19	(16)	100	(84)	
Never	14	(12)	104	(88)	
Vascular condition*					
No	13	(10)	114	(90)	0.06
Yes	35	(18)	163	(82)	
Diabetes*					
No	31	(12)	222	(88)	0.02
Yes	17	(24)	55	(76)	
Hypertension*					
No	20	(12)	149	(88)	0.12
Yes	28	(18)	128	(82)	
Myocardial infarction*					
No	40	(14)	245	(86)	0.27
Yes	8	(20)	32	(80)	
Cerebrovascular accident*					
No	46	(15)	267	(85)	0.96
Yes	2	(17)	10	(83)	
Transient ischemic attack*					
No/don't know	48	(15)	265	(85)	0.19
Yes	0		12	(100)	
Aspirin use (at baseline)‡					
No	31	(13)	206	(87)	0.25
Yes	17	(20)	70	(80)	
Aspirin use (after baseline)§					
No	35	(15)	205	(85)	0.65
Yes	13	(15)	73	(85)	

1.- Iniciar tratamiento con corticoides, al menos 1mgr/kg/día

2.- Si DM iniciar AAS (prevención secundaria), o si se sospecha etiología arterítica.

3.- Identificar posibles cofactores que hicieran posible causa de NA-NOIA.

4.- Revisión de tratamientos hipotensores

PRUEBAS COMPLEMENTARIAS: BAT, ECO doppler, ¿PET?

GRACIAS