

# EVOLUTION AND PROGNOSIS IN ANTERIOR OPTIC NEUROPATHY (NOIA) - IMPACT ON QUALITY OF LIFE OF PATIENTS

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**Abstract:** *The aim of the paper was to answer the two following aspects of anterior ischemic optic neuropathy (NOIA): Issues related to the onset of NOIA and diagnosis strategy: role and place of laboratory investigations in NOIA; and the second one: the Evolution and prognosis of NOIA and the impact on the quality of life of patients.*

*The study included 109 patients selected from those who presented NOIA suggestive symptoms. The research was structured as a retrospective, longitudinal investigation of the lot. To be included in the survey was used the selection method based on inclusion and exclusion criteria of the study. The lot of the study included subjects of both sexes, aged between 51 and 78 years, from rural and urban areas. Ischemic optic neuropathy is a formidable disease which can rapidly lead to loss or damaged of the eye function, with major impact on quality of life of the patients.*

**Key words:** *ischemic optic neuropathy.*

## 1. Introduction

### 1.1. Aim and working hypothesis

The paper aim was to answer the following aspects of anterior ischemic neuropathy (NOIA):

1. NOIA debut issues and diagnosis strategy: role and place of laboratory investigations in NOIA
2. Evolution and prognosis NOIA and quality of life of patient.

## 2. Material and methods

The study included 109 patients selected from those who presented NOIA

suggestive symptoms. The research was structured as a retrospective, longitudinal investigation of the lot.

To be included in the survey was used selection method based on inclusion and exclusion criteria of the study. Group in the study included subjects of both sexes, aged between 51 and 78 years from rural and urban areas.

### 2.1. Inclusion and exclusion criteria of study

The criteria for inclusion in the study: decreased visual acuity, visual field deficits and suggestive ophthalmoscopic changes. Exclusion criteria show certain

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ocular conditions responsible for visual acuity changes and highlight some neurological deficits-substrate of visual field changes in absence of specific ophthalmological changes.

## 2.2. NOIA treatment methodology

Its object is to reduce ischemic aggressive factor and improve local tissue metabolism.

Drug therapy included corticosteroid treatment administered at the beginning as general and local therapy in order to reduce papilloedema and reinstate the tissue permeability, vasodilator therapy being used to increase local blood flow, and to treat underlying diseases: cardiovascular, diabetes and dyslipidemia. Also the drug therapy in NOIA may include neuroprotective treatment.

Neuroprotection may be:

- Indirect, when acting on local risk factors: local hemodynamic alterations, increase in intraocular pressure by changing the pressure of local infusion
- Direct, is a set of measures seeking to increase the threshold for survival of neurons.

## 3. Survey Results

### 3.1. NOIA Epidemiology

NOIA study included a group of 109 patients (52 men and 57 women) aged between 51 and 78 years.

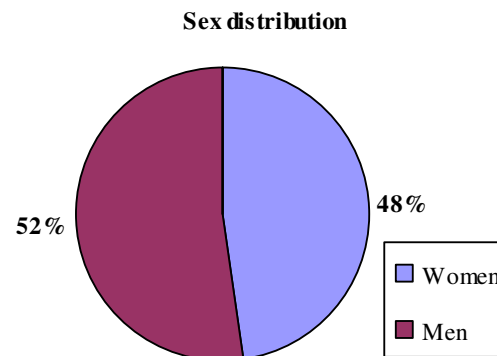
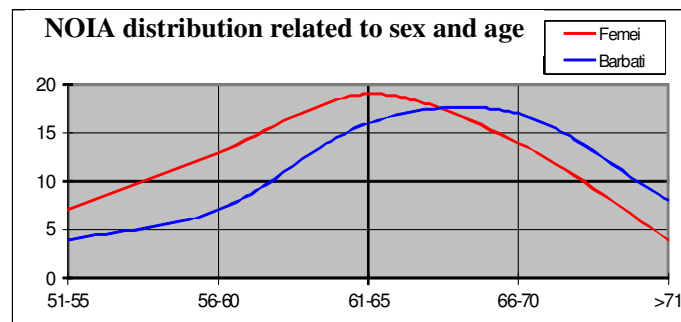


Fig. 1. *Sex distribution*

NOIA is a disease with incidence increased with the age, mean age of 63.6 years (females with manifestation of early disease), with a slight preponderance in urban population and more frequently in women than in men (sex ratio F / B = 1.1 / 1).



Sex	Age				
	51-55	56-60	61-65	66-70	>71
W	7	13	19	14	4
M	4	7	16	17	8

### 3.2. NOIA Etiology

#### NOIA diagnostic algorithm

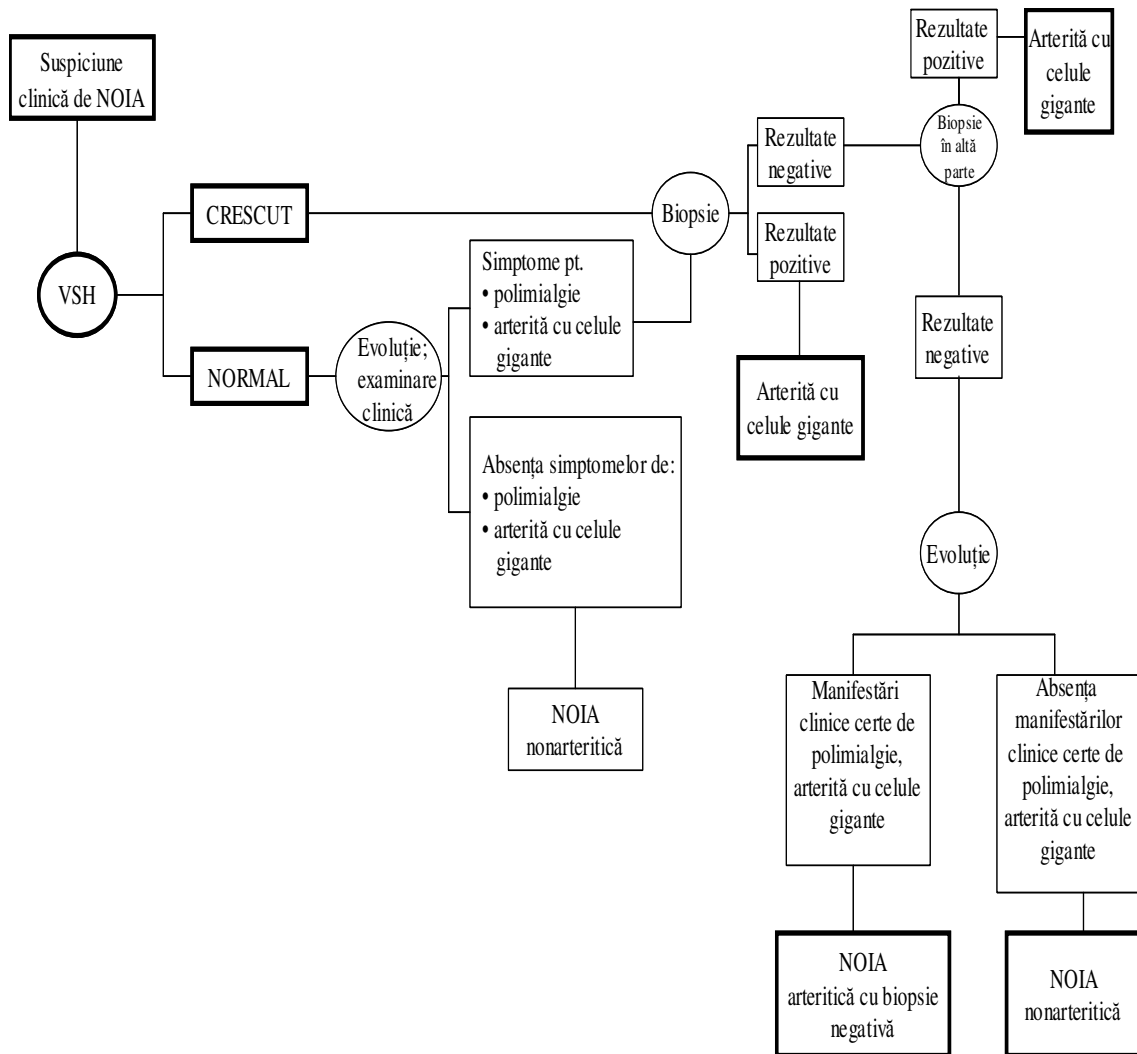
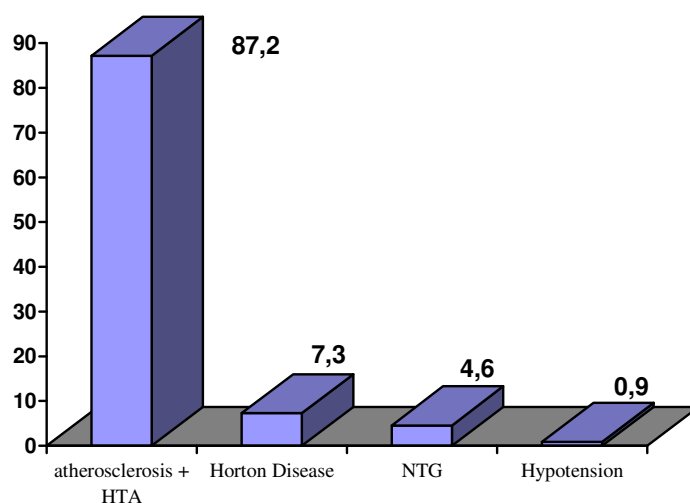


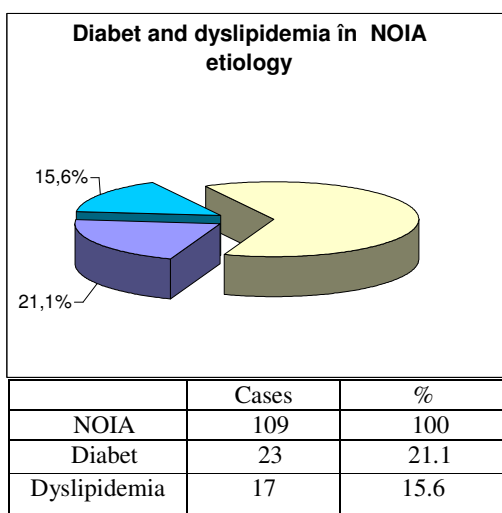
Fig. 3. NOIA diagnostic algorithm

Study results showed the following etiological aspects:

- atherosclerosis + HTA in 87.2% of cases (97 patients);
- Giant cell arteritis in 7.3% of cases (8 patients);
- Normal static tension glaucoma in 4.6% of cases (5 patients);
- Hypotension brutal, prolonged, in 0.9%.

**NOIA etiology**Fig. 4. *NOIA etiology*

The study mentioned the following diseases associated with NOIA diabetes U.S. (21.1%) and dyslipidemia (15.6%).

Fig. 5. *Diabet and dyslipidemia in NOIA etiology***3.3. NOIA at beginning**

The onset of disease for the cases studied was: suddenly in 101 cases (92.6%), three new cases with slowly progressive arthritic NOIA (2.8%) and slow, seemingly insignificant, in five cases (4.6%).

Table 1

NOIA at beginning	No. of cases	Percentage
Suddenly	101	92,6%
Slowly progressive	3	2,8%
Seemingly insignificant	5	4,6%

In 92.6% of cases NOIA had a sudden onset, the dominant clinical symptom was the decreasing visual acuity installed in an apparently normal eye status.

**3.4. Examination AV**

Loss of vision in the study group was installed: sharply to 92.8% of cases (52)

patients; intensity deficit is variable and slowly progressive vision to 2.8% of cases (three patients). In five cases (4.6%) there was decreased AV.

AV	1-0,5 (1-1/2)	0,5-0,1 1/2-1/10	0,1-0,02 1/10-1/50	0,02-0,002 1/50-1/500	0,001 PMM	0,0005 PL
No. of cases	5	25	32	31	12	4
Percentage	4,6%	22,9%	29,4%	28,4%	11%	3,7%

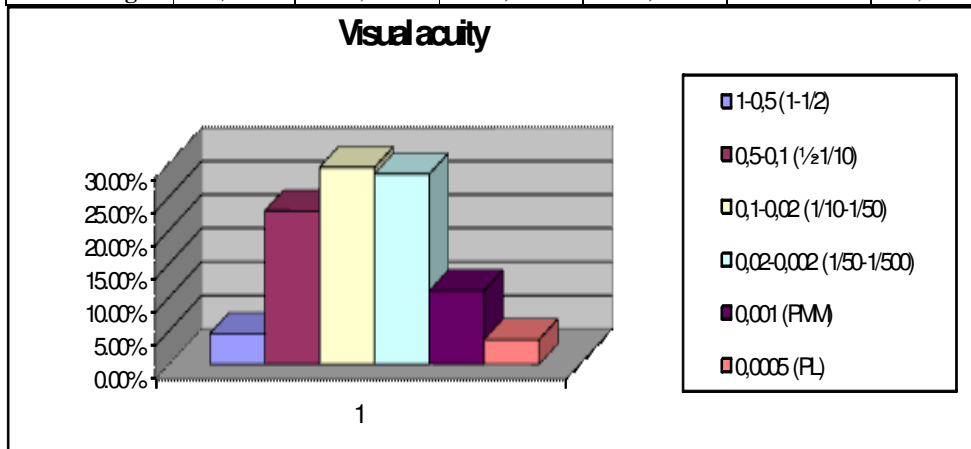


Fig. 6. Visual acuity at the first exam

AV was variable: from no effect to AV 1/2000.

Table 2

AV Decrease	No. of cases	Percentage
Suddenly	101	92,6
Slowly progressive	3	2,8
No AV decrease	5	4,6

Visual acuity evolution was: improvement to 31.2% cases (34 patients), decreased to 12.8% AV cases (14 patients), no changes to AV at 56% (61 patients).

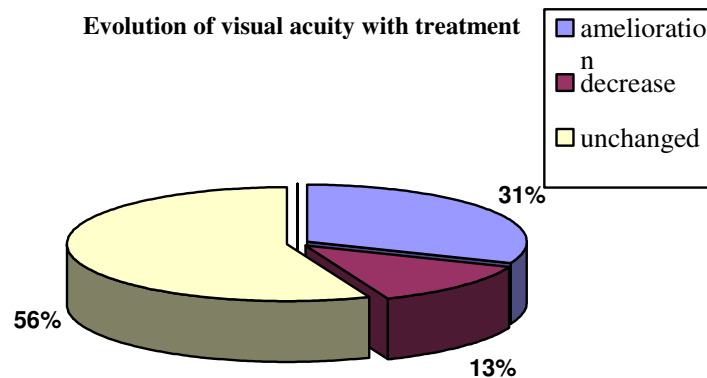


Fig. 7. Evolution of VA acuity treatment

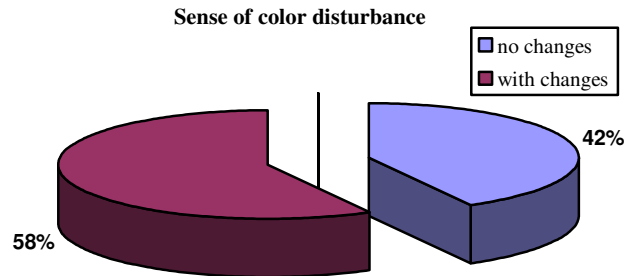


Fig. 8. *Sense of color disturbance*

### 3.5. Visual acuity after treatment

#### Color sense examination

It was done in 62 patients (56.8%) with VA better than 1/50.

The results were as follows:

- 36 patients (58% of cases) showed the axis red-green dyschromatopsia
- 26 patients (42%) showed no changes in color sense

#### Fundus examination eye

It was done to the whole group of 109 patients and was:

- Ischemic papillary edema was present in 94 cases (86.2%);
- Juxtapapilare bleeding was found in 45 cases (43.1%);
- Soft exudates Juxtapapilare were observed in 21 cases (19.2%).
- Trajectory and caliber vascular changes were found in all cases.

- Papillary excavation was found from the start in five cases (4.6%);
- Progressive optic atrophy, in 42.2% overall and sectoral 33% of cases.

Diagnosis is mainly clinical NOIA fundus examination with a significant weight.

Pathological manifestation of ischemic papillary edema is the most important and constantly found (86.2%).

### 3.6. Visual Field Examination (CV)

VF examination was done in 63 patients (57.8%) and were found:

- Central spot in 23.8% of cases (15 patients);
- Lower altitude deficit to 61.9% of cases (39 patients);
- Arciforme deficits in 14.3% of cases (9 patients).

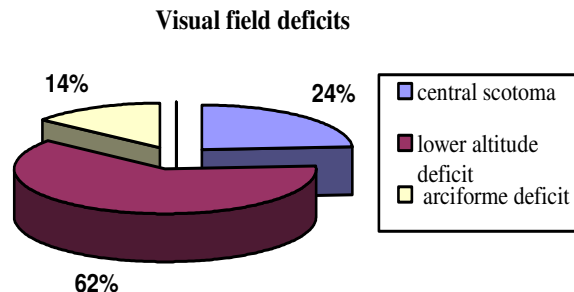


Fig. 9. *Visual field deficits*

CV was repeated at intervals of 7-14 days revealed the following:

- Improving CV from 34.9% of cases (22 patients);
- CD did not change significantly from 46% (29 patients);
- Worsening CV of 19.1% (12 patients).

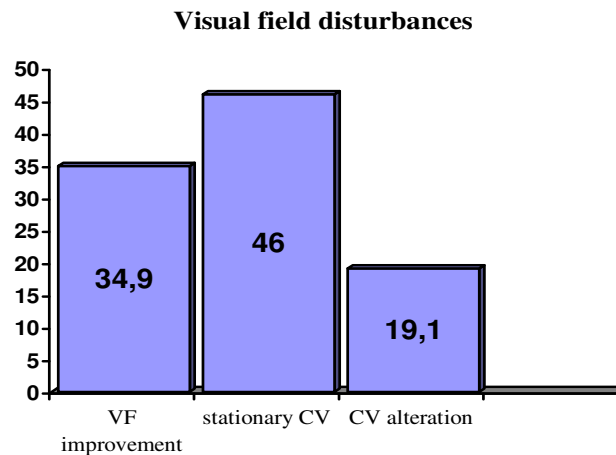


Fig. 10. Visual field disturbances

#### 4. Discussion and comments

##### 4.1. Epidemiological aspects of NOIA

NOIA is the attribute of age, averaged age who experienced disease was 63.6 years, with a maximum incidence in women between 51-66 years and in men after the age of 66 years.

##### 4.2. Etiopathogenic aspects

Decompensation of the hemodynamic balance from the head of optic nerve may be induced by degenerative changes, inflammatory and functional spastic changes reaching the vascular bed of the head of the optic nerve. Study results highlight the increasing number of atherosclerotic degenerative processes associated with hypertension in the etiology NOIA. In all cases where we face a unilateral NOIA arthritic on one eye, it is required to consider the possibility and major risk of disease to spread to the other eye.

##### 4.3. NOIA onset

Regarding the NOIA onset was revealed that in most cases was acute in nature, and the symptom which dominated the clinical picture was the decreased visual acuity. Sudden onset was seen mainly in patients with degenerative etiology NOIA, ischemic status manifested on a relatively good eye.

##### 4.4. The clinical examination and laboratory examinations in the diagnosis and monitoring NOIA

Visual acuity is the main reason for the request of the ophthalmological examination. Bilateral decreased vision was found in a small proportion (2.75% in the group had studied).

Low percentage of visual acuity improvement with treatment underscores the seriousness of the disease and limited effectiveness of treatment in these cases.

Disturbance of the sense of color affect information transmission paths in NOIA and give rise to a stable axis red-green dyschromatopsia.

Fundus examination is a routine that is of great importance in guiding the clinical diagnosis and NOIA. Reveal any papillary ischemia associated with sudden and steep decrease of vision suggests the diagnosis of acute ischemia of the optic nerve head.

Visual field examination is of great importance in clinical diagnosis and monitoring disease progression. In NOIA, we find dynamic visual field deficits, depending on disease progression. Improving sight from 34.9% of cases underscores the need for prompt treatment.

#### 4.5. Treatment of ischemic optic neuropathy

Treatment is neuroprotective trying to reduce the damage of ganglion cell axons.

Neuroprotective medication must achieve the following objectives:

- To reduce or inhibit excess amino acids, free radicals, intracellular calcium;
- To increase the supply of neurotrophic factors.

Neuroprotective medications most commonly used is represented by:

- Topical medication: Betaxolol and Brimodinine, Dorzolamide
- Systemic medication: as channell blockers meantime

In NOIA emergency treatment is aimed to reduce ischemic factor in the optic nerve head and to improve local tissue metabolism. Medications used included: corticosteroids, anti-ischemic, neuroprotective, disorders of pre-existing cardiovascular disease, diabetes, dyslipidemia.

#### 5. Conclusions

1. NOIA has a sudden onset in most cases (92.6% of cases).
2. NOIA etiology study highlights the variability and difficulty of disease etiology.
3. Visual acuity is the dominant symptom of the clinical picture.
4. Persistence of visual deficit in nearly 70% of cases emphasizes the seriousness of the disease and limited effectiveness of treatment.
5. Deficits campimetric lower altitude (cvafricanic orhemianopsia) were the most frequent changes (61.9%).
6. Ischemic optic neuropathy are formidable disease which can rapidly lead to loss or damaged eye function.
7. There is potential for improvement in reducing disability and that requires a great need for prompt diagnosis and rapid treatment in all cases.

#### References

1. Cernea, P., Munteanu, G.: *Opticopatia*. Bucuresti. Ed. Medicala, 1983. 17-364.
2. Hayreh, S.S.: *Anterior ischemic optic neuropathy*. In: *Arch. Neurol.*, 1981, 38, p. 675-688.
3. Orgul, S., Meyer, P., Cioffi, G.A.: *Physiology of Blood Flow Regulation and Mechanisms Involved in Optic Nerve Perfusion*. In: *J.Glaucoma*, 1995, 4, p. 427-443.
4. Collingnon-Brach, Jackueleine. *Treatment og Glaucomatous Optic Neuropathy*. The University of Liege, Departament of Ophthalmology, Report, 2000, p. 5-59.
5. Arnold, A.C.: *Ischemic Optic Neuropathie*. In: Yanoff M., Duker J.S., Ophthalmology, Mosby International Limited, St. Louis, CD-ROM Edition, 1998, section 11.7.
6. Chisalita, D.: *Tratamentul glaucomului pozitiv cu unghi deschis*. In: *Oftalmologia*, 2001, Supl., p. 26-58.
7. Glaser, J.S.: *Topical Diagnosis: Prechiasmal Visual Pathways*. In: *Duane's Ophthalmology*, Lippincott-Raven Publishers, Inc., 1998, CD-ROM Edition, vol.2, cap.5.