# COLOR DOPPLER IMAGING OF ORBITAL VESSELS IN THE DIAGNOSIS OF ANTERIOR ISCHEMIC OPTIC NEUROPATHIES

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#### **1. INTRODUCTION**

Anterior ischemic optic neuropathies (AIONs), represented by a segmental infarction of the optic nerve head (ONH) supplied by the posterior ciliary arteries (PCAs), can be: non-arteritic (NA-AIONs), which are a multifactorial disease, and arteritic (A-AIONs), due to giant cell arteritis (GCA).

#### 2. PURPOSE

To investigate the clinical and ultrasound characteristics of the orbital vessels, and of the branches of external carotid arteries (ECAs), in patients with unilateral acute AION, which help differentiate newly diagnosed NA-AIONs from A-AIONs, which require immediate steroid treatment, in order to protect the fellow eye from going blind.

#### **3. PATIENTS AND METHODS**

In this prospective, comparative, observational study, 80 consecutive patients with clinical suspicion of unilateral acute AION were examined at admission, and in the first week of evolution, following a complex protocol including color Doppler imaging (CDI) of the orbital (retrobulbar) vessells.

#### 4. RESULTS

#### Table I. The comparison of major features of A-AIONs and NA-AIONs

FEATURE	A-AIONs (16 patients)	NA-AIONs (64 patients)	
Age (mean, years)	72.7+/-7.5	57.3+/-10.6	
Sex ratio	Female > male (10:6 cases)	Female=male (33:31 cases)	
Associated symptoms	New temporal headache, jaw claudication, abnormal TAs on palpation, scalp tenderness (75%-12 cases)	Pain occasionally noted (4.7%-3 cases)	
Visual acuity	81.3% (13 cases) < 20/200	65.6% (42 cases) > 20/200	
Optic disc	<ul> <li>Pale diffuse optic disc edema (figure 1C) (87.5%-14 cases) &gt; hyperemic partial optic disc edema (12.5%-2 cases)</li> <li>Cup normal (100%-16 cases)</li> </ul>	<ul> <li>Hyperemic optic disc edema (figure 1A) (90.6%-58 cases)</li> <li>&gt; pale diffuse optic disc edema (9.4%-6 cases)</li> <li>Cup small (85.9-55 cases) &gt;cup normal (14.1%-9 cases)</li> </ul>	
History of amaurosis fugax	25% (4 cases)	No	
ESR (mm/h) C-reactive protein (mg/l)	<ul> <li>&gt;50 (87.5%-14 cases)</li> <li>&gt; 5 (93.8%-15 cases)</li> </ul>	<ul> <li>&lt;50 (95.3%- 61 cases)</li> <li>&lt; 5 (96.9%-62 cases)</li> </ul>	
Sonographic features in temporal arteritis as part of GCA	<ul> <li>"Dark halo" sign-(75%-12 cases)</li> <li>Stenosis of branches of external carotid arteries-(31.2%-5 cases)</li> </ul>	No	
Temporal artery biopsy (TAB)	Granulomatous inflammation of the media layer - (100%-16 cases)	No TAB	
Color Doppler Imaging of the retrobulbar (orbital) vessels	Severe diminished blood flow velocities in the PCAs, especially on the affected side, and high RI in all retrobulbar vessels, in both orbits - (100%-16 cases)	Blood flow velocities and RI in PCAs were preserved (100% - 64 cases)	
Fluorescein fundus angiography	Disc and choroid filling delay ( <b>figure</b> <b>1D</b> ) (100%-16 cases)	Disc filling delay) ( <b>figure</b> <b>1B</b> ) (100%-64 cases)	
Treatment	Corticosteroids (100%-16 cases)	None proved	

#### CDI of the retrobulbar vessels features

#### 1). Spectral Doppler analysis of the orbital vessels in A-AION.

• In the first week of evolution, it revealed undetectable or severe diminished blood flow velocities in both PCAs (**figure2A-D**), especially on the affected side, with an increased resistance index (RI) in all retrobulbar vessels, in both orbits.



#### Figure 2. A-AION - (A, B) - CDI of temporal PCAs of both eyes, (C, D) - CDI of nasal PCAs of both eyes.

Less abnormalities were observed in the central retinal arteries (CRAs) (figure 3
 A, B), and in the ophthalmic arteries (OAs) (figure 3 C, D).



Figure 3. A-AION - (A, B) - CDI of CRAs of both eyes. (C, D) - CDI of OAs of both eyes. 2). Spectral Doppler analysis of the retrobulbar vessels in NA-AION.

- By contrast, in NA-AION, blood flow velocities and RI in PCAs were generally preserved.
- In the first week of evolution, this analysis revealed only a slight decrease of peak systolic velocities (PSV) in PCAs (nasal and temporal) in the affected eye, compared to the unaffected eye (**figure 4 A-D**), and a very slight decrease of PSV in CRA of the affected eye, due to papillary edema (**figure 4 E, F**). In OAs, PSV were variable: normal to decreased, according to ipsilateral internal carotid artery status.





NA-AION (left eye) Fig 1A. Fundus photography *with hyperemic, diffuse edema of the optic disc.* 

#### **5. CONCLUSIONS**



NA-AION (left eye) Fig 1B. Fluorescein angiography *with disc filling delay*.

#### × Vmax 11.2cm/s xVd 4.42cm/s RI 0.60 max 10.7cm/s xVd 3.78cm/s RI 0.647 x 5.21cm/s ≈ 3.4?cm/s FVmax 13.2cm/s xVd 5.28cm/s RI 0.600

Figure 4. NA-AION - (A, B) - CDI of temporal PCAs of both eyes, - (C, D) - CDI of nasal PCAs of both eyes, - (E,F) - CDI of CRAs of both eyes.

## Table II. The threshold values of RI in the orbital vessels and the corresponding values of Se, Sn, PPV, and NPV.

Arteries	CRA	PCA t	PCA n	OA
Cutt off point	0.68	0.7	0.67	0.8
Se	0.75	0.87	0.87	1
Sp	0.82	0.97	0.94	0.97
PPV	0.5	0.87	0.77	0.89
NPV	0.93	0.97	0.97	1



A-AION (left eye) Fig 1C. Fundus photography *with pale*, *diffuse edema of the optic disc*.

A-AION (left eye) Fig 1D. Fluorescein angiography *with disc and choroid filling delay*.

a). A combination of a history of amaurosis fugax before an abrupt, painless, and severe loss of vision of the involved eye, with concomitant diffuse pale optic disc edema was extremely suggestive of A-AION. However, none of these symptoms were found in NA-AION patients.

b). Because findings of temporal arteries ultrasound did not correlate with eye complications in A-AION patients, CDI of the orbital vessels is of critical importance. It allows the detection and monitoring of alterations in orbital blood flow, especially of the posterior ciliary arteries (PCAs), which correspond with the clinical features of A-AION.

c). CDI of the orbital vessels enables prompt differentiation between A-AIONs and NA-AIONs, supporting the evidence of involvement of the entire trunk of the PCAs in the A-AION. In contrast, in the NA-AION, the impaired flow to the optic nerve head is distal to the PCAs themselves, possibly at the level of the paraoptic branches.