Int Ophthalmol (2019) 39:597–604
https://doi.org/10.1007/s10792-018-0846-x

ORIGINAL PAPER

Retrobulbar and intraocular blood flow in anterior ischaemic optic neuropathy are linked to the functional impairment

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Received: 14 July 2017 / Accepted: 1 February 2018 / Published online: 13 February 2018

Abstract
Purpose Evaluation of ocular haemodynamics in patients with acute non-arteritic anterior ischaemic optic neuropathy (NAION) by colour Doppler imaging and fluorescein angiography and correlation of blood flow parameters to visual field loss and visual acuity.
Methods Blood flow velocities (peak systolic velocity (PSV), end-diastolic velocity (EDV)) of the ophthalmic artery (OA), central retinal artery (CRA) and nasal and temporal posterior ciliary arteries (PCAs) were measured via colour Doppler imaging. Resistive index (RI) of all vessels was calculated (PSV-EDV/PSV). Retinal arteriovenous passage times (AVP) were evaluated using fluorescein angiography (scanning laser ophthalmoscope) and digital image analysis. The visual field global index mean deviation (MD, 30-2 programme, Humphrey Field Analyzer) and visual acuity (logMar) was used for analysis of functional impairment after NAION.
Results Twenty patients (age: 64.62 ± 11.63 years) with acute NAION were included. Mean duration of symptoms was 7.6 ± 6.9 days. Mean defect was 15.4 ± 8.9 dB, AVP was determined with 1.66 ± 0.37 s. EDV of the CRA was significantly correlated to visual field MD ($r = 0.52$, $p = 0.017$) and AVP ($r = -0.49$, $p = 0.025$). The RI of the OA was significantly correlated to visual acuity ($r = 0.493$, $p < 0.037$). No significant correlations were recorded for the PCAs. A significant correlation was found between AVP and the EDV of the CRA ($r = -0.49$, $p = 0.025$).
Conclusion Decreased EDV in the CRA and increased RI in the OA seem to be linked to the functional damage in NAION. An improvement of the retrobulbar circulation might be beneficial in the treatment of NAION.

Keywords NAION · CDI · Fluorescein angiography · Retrobulbar haemodynamics · Ocular blood flow

Introduction

Non-arteritic anterior ischaemic optic neuropathy (NAION) is the second most common optic neuropathy and the most common cause for acute optic neuropathy in patients over 50 years [1]. The incidence in the US is estimated at 2.3–10.2 per 100,000 per year [2]. The vision loss was reported to frequently arise immediately upon awakening [3] with a high variability in further progression. In most patients, the initial course is static, whereas progressive courses of the disease have been reported [4]. Visual field defects are most commonly found altitudinally, usually in the

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inferior hemisphere [4]. The pathophysiology of NAION is not completely understood to date. The aetiology appears to be multifactorial and several risk factors have been identified. The ischaemic damage is most commonly believed to result from acute hypoperfusion, probably only temporarily, of the short posterior arteries (PCAs) [5–7]. Evidence of disturbed circulation in AION was previously reported. Via fluorescein angiography an increased retinal arteriovenous passage time (AVP) was described by Bertram et al. [8], and Arnold and Hepler [9] found markedly delayed filling of the optic nerve head capillaries in patients with acute AION. Furthermore, disturbed retrobulbar haemodynamics were detected by means of colour Doppler imaging (CDI) measurement [7, 10–14]. In most cases, impaired blood flow velocities in the central retinal artery (CRA) were measured [12–14]. It is most commonly believed that disturbed blood flow in the CRA is secondary to optic disc oedema and subsequent vessel compression with slower blood flow and lower velocities. Significant results for the posterior ciliary arteries (PCA) which play a critical role in the pathogenesis of NAION are rare, which may be caused by a high variability of CDI measurements of these vessels [15].

CDI allows the non-invasive measurement of the retrobulbar vessels including the PCAs. In this study, we investigated whether either retrobulbar haemodynamics or fluorescein angiographic findings are related to the clinical impairment in acute NAION.

Materials and methods

Patients

Twenty patients (13 males, 7 females, age: 64.62 ± 11.63 years) suffering from acute NAION were included in this study. The time from onset of symptoms until first presentation in our clinic was 7.6 ± 6.9 d (range 1–28 days). All examinations were performed in accordance with the Declaration of Helsinki for research involving human subjects. Informed consent was acquired from each participant, and the study was approved by the local ethics committee. A detailed ophthalmological examination was performed including visual field testing using the achromatic 30-2 standard (Humphrey Visual Field Analyzer II, Zeiss, Germany), Goldmann applanation tonometry and CDI measurement as well as arterial blood pressure measurement.

Fluorescein angiography with a scanning laser ophthalmoscope (Rodenstock, Ottobrunn, Germany) was performed to evaluate the AVP and for digital image analysis. We previously described the technique in greater detail [16]. A 40-degree observation centred on the optic nerve head was used. At the beginning of the angiography, 10% sodium fluorescein dye (2.5 cc Alcon, Freiburg, Germany) was injected into the antecubital vein. Image acquisition was performed with constant parameters until the maximum intensity level in the retinal veins had passed to avoid artefacts. The dynamic sequences were acquired with 25 images per second. The angiograms were analysed by digital image analysis (Matrox Inspector, Matrox Inc., Quebec, Canada). The retinal AVP was calculated using dye dilution curve analysis. The AVP represents the shortest passage of the fluorescein dye from the retinal arterioles to the venules representing retinal microcirculation. The intensity level for each image was calculated at a predetermined region of interest (ROI) consisting of the arterioles and venules, the extend corresponded to vessel diameter. All measurements were performed in the temporal superior and inferior arterioles and venules. The mean AVP was used for further analysis.

Retrobulbar blood flow velocities were measured by means of colour Doppler imaging using a 7.5 MHz linear phase-arrayed transducer (Siemens, Sonoline, Sienna). All measurements were taken by experienced investigators (NP, MK). The method was previously described in greater detail [11, 17]. After application of coupling gel, the transducer was gently placed onto the closed upper eyelid. All measurements were taken in supine position. CDI allows the measurement of the ophthalmic artery (OA), the CRA, and both nasal and temporal PCA. The peak systolic velocity (PSV) and end-diastolic velocity (EDV) were obtained from the measurements. The resistive indices were calculated as follows: resistive indices (RI): (PSV − EDV)/PSV.

Systemic and diastolic blood pressure and heart rate were recorded in supine position after a resting time of 5 min before any CDI measurements.

Mean arterial pressure as well as ocular perfusion pressure were calculated from the acquired data.

$$\text{MAP} = \text{diastolic blood pressure} + 1/3\,(\text{systolic} - \text{diastolic blood pressure})$$

$$\text{OPP} = 2/3\ \text{MAP} - \text{IOP}$$

Statistics and graph creation

Statistical analysis and graph creation was performed using Graph Pad Prism Software for Windows (GraphPad Software Inc., USA) as well as Matlab for Windows (Mathworks Inc.USA). Correlations were calculated with the Fisher's Transformation. A p value of ≤ 0.05 was regarded as statistically significant.

Results

Clinical parameters of the patients are displayed in Table 1

All recorded CDI parameters and the AVP are provided in Table 2.

Figure 1 shows the box-plot for the recorded AVP times.

The MD was significantly correlated to the EDV of the CRA ($r = 0.52$, $p = 0.017$, Fig. 2). Figure 2 shows the significant correlation between the end-diastolic velocity (EDV) in the central retinal artery (CRA) and visual field medial deviation (MD).

No significant correlation was found for either MD or AVP ($r = 0.09$, $p = 0.70$). In addition, no significant correlations were found between MD and CDI parameters for the OA and both TPCA and NPCA.

A significant correlation was found between the AVP and the EDV of the CRA ($r = -0.49$, $p = 0.025$, Fig. 3). The correlation between AVP and RI of the NPCA did not reach statistical significance ($r = -0.43$, $p = 0.062$). Visual acuity (LogMar) was not significantly correlated to AVP. The correlation between visual acuity and the EDV of the OA slightly did not reach statistical significance ($r = -0.463$, $p = 0.053$). A significant correlation

Table 2 Recorded CDI parameters for all patients

	Mean	SD
OA-PSV	34.53	9.28
OA-EDV	7.57	3.72
OA-RI	0.79	0.07
CRA-PSV	7.14	1.56
CRA-EDV	2.0	0.59
CRA-RI	0.72	0.07
NPCA-PSV	7.77	1.94
NPCA-EDV	2.68	0.91
NPCA-RI	0.66	0.07
TPCA-PSV	7.19	2.01
TPCA-EDV	2.64	0.79
TPCA-RI	0.59	0.29
AVP (in s)	1.66	0.37

OA ophthalmic artery, *CRA* central retinal artery, *NPCA* nasal posterior ciliary artery, *TPCA* temporal posterior ciliary artery, *PSV* peak systolic velocity, *EDV* end-diastolic velocity, *RI* resistive index, *AVP* arteriovenous passage time

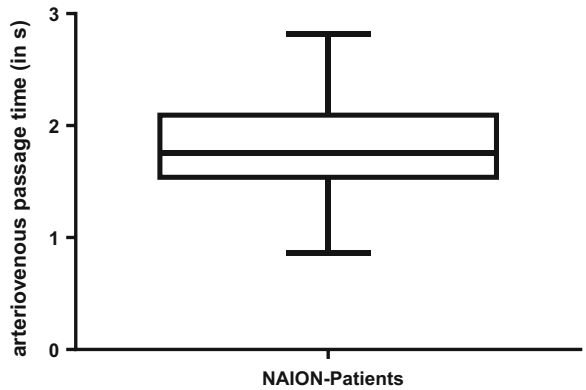

Fig. 1 Boxplot of the recorded AVP time in seconds

Table 1 Basic clinical parameters of all patients

Age (in years)		Visual field MD (in dB)		Visual acuity (LOG Mar)		IOP (in mmHg)		Systolic blood pressure (in mmHg)		Diastolic blood pressure (in mmHg)		MAP (in mmHg)		PP	
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
64.62	11.63	− 15.36	8.86	0.35	0.44	15.55	3.28	147.05	24.14	78.2	12.55	100.92	14.2	51.06	9.67

MD medial deviation, *IOP* intraocular pressure, *AVP* arteriovenous passage time, *MAP* mean arterial pressure, PP = perfusion pressure

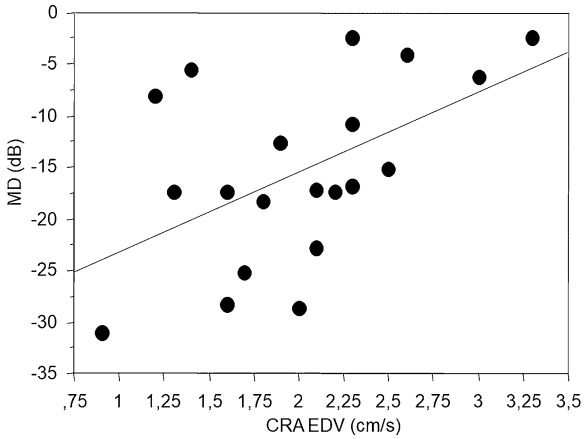

Fig. 2 Significant correlation between the end-diastolic velocity (EDV) in the central retinal artery (CRA) and visual field medial deviation (MD)

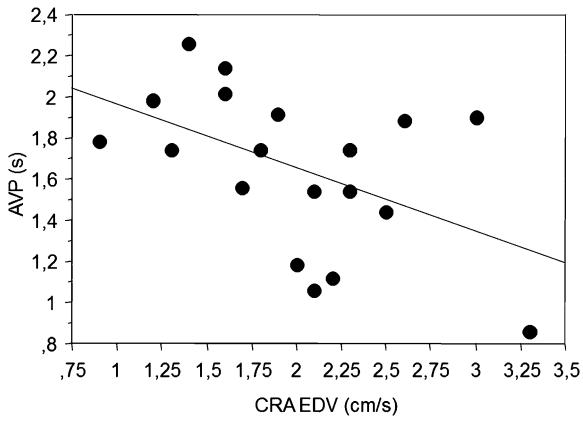

Fig. 3 Significant correlation between the end-diastolic velocity (EDV) in the central retinal artery (CRA) and arteriovenous passage time (AVP)

was recorded only for the RI in the OA and visual acuity ($r = 0.493$, $p < 0.037$, Fig. 4).

The respective correlation coefficients are provided in Table 3, 4 and 5.

Discussion

In this study, the retrobulbar haemodynamics were examined by CDI and the intraocular hemodynamics by fluorescein angiography in patients with acute NAION. To our knowledge, this is the first study using both diagnostic tools in the same patients, trying to find a correlation to the functional impairment.

The clinical impact of the CRA is somehow limited in NAION, as only the capillaries in the optic disc head nerve fibre layer and rarely a minor branch supplying the retrolaminar region are supplied by the CRA [18]. Nevertheless, disturbed blood flow in the CRA is the most consisting finding in all studies investigating AION by means of CDI (12–14). Furthermore, Wang et al. reported disturbed blood flow in these capillaries on the surface of the optic nerve head supplied by the CRA by scanning laser Doppler flowmetry [19]. Similar results were found by delayed filling of these capillaries in the fluorescein angiography in patients with AION [9]. We found similar results in delayed filling in our patients. To date, it is not completely understood why blood flow in the CRA is disturbed in NAION. We, among other authors, believe that it might be a secondary finding, due to the profound optic disc oedema in NAION patients. This assumption is supported by the findings of Jacquemin et al. [20], who reported reduced blood flow in the CRA

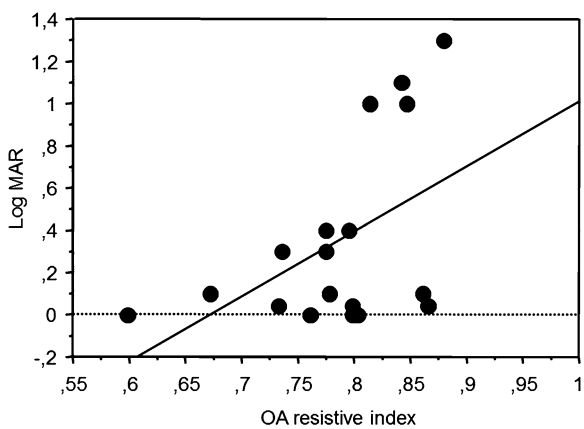

Fig. 4 Significant correlation between the resistive index (RI) in the ophthalmic artery (OA) and visual acuity (LogMar)

Table 3 Correlation coefficients and *p* values for the CDI parameters as well as AVP with visual field defect MD

Parameters	Correlation coefficient	*p* value
MD and AVP	0.092	0.703
MD and PSV of the OA	0.227	0.34
MD and EDV of the OA	0.168	0.485
MD and RI of the OA	− 0.122	0.613
MD and PSV of the CRA	0.396	0.085
MD and EDV of the CRA	**0.522**	**0.017**
MD and RI of the CRA	− 0.280	0.236
MD and PSV of the TPCA	0.390	0.089
MD and EDV of the TPCA	0.413	0.07
MD and RI of the TPCA	− 0.007	0.978
MD and PSV of the NPCA	0.144	0.549
MD and EDV of the NPCA	0.219	0.359
MD and RI of the NPCA	− 0.181	0.45

Statistically significant correlations are printed in bold

MD medial deviation (of the visual field testing), *AVP* arteriovenous passage time, *PSV* peak systolic velocity, *EDV* end-diastolic velocity, *RI* resistive index, *OA* ophthalmic artery, *CRA* central retinal artery, *TPCA* temporal posterior ciliary artery, *NPCA* nasal posterior ciliary artery

Table 5 Correlation coefficients and *p* values for the CDI parameters, AVP and visual acuity (VA)

Parameters	Correlation coefficient	*p* value
VA and AVP	− 0.350	0.157
VA and PSV of the OA	− 0.301	0.229
VA and EDV of the OA	− 0.463	0.053
VA and RI of the OA	**0.493**	**0.039**
VA and PSV of the CRA	0.264	0.296
VA and EDV of the CRA	0.102	0.692
VA and RI of the CRA	0.061	0.813
VA and PSV of the TPCA	0.245	0.333
VA and EDV of the TPCA	− 0.061	0.812
VA and RI of the TPCA	0.224	0.378
VA and PSV of the NPCA	0.339	0.172
VA and EDV of the NPCA	− 0.004	0.988
VA and RI of the NPCA	0.385	0.116

Statistically significant correlations are printed in bold

VA visual acuity (logMar), *AVP* arteriovenous passage time, *PSV* peak systolic velocity, *EDV* end-diastolic velocity, *RI* resistive index, *OA* ophthalmic artery, *CRA* central retinal artery, *TPCA* temporal posterior ciliary artery, *NPCA* nasal posterior ciliary artery

assessed via CDI in patients with optic nerve melanomas and subsequent oedema. Furthermore, Elvin et al. [21] found significantly increased blood flow resistances in the CRA in patients with optic neuritis and swollen optic discs in comparison with the unaffected eye.

Nevertheless, it is also possible that disturbed blood flow in the CRA represents a primary pathogenetic factor in AION.

In our study, we found a significant correlation between the EDV in the CRA and AVP, highlighting the disturbed blood flow in the CRA. Huber et al. [16] found the same association in patients suffering from NTG. The finding was interpreted as a sign of chronic ischaemia. There appears to be a similarity between the chronic ischaemia found in NTG patients and the acute ischaemia in NAION patients.

Table 4 Correlation coefficients and *p* values for the CDI parameters and AVP

Parameters	Correlation coefficient	*p* value
AVP and PSV of the OA	0.103	0.67
AVP and EDV of the OA	0.079	0.743
AVP and RI of the OA	− 0.041	0.867
AVP and PSV of the CRA	− 0.255	0.283
AVP and EDV of the CRA	**− 0.495**	**0.025**
AVP and RI of the CRA	0.375	0.105
AVP and PSV of the TPCA	− 0.216	0.365
AVP and EDV of the TPCA	− 0.297	0.207
AVP and RI of the TPCA	− 0.003	0.991
AVP and PSV of the NPCA	− 0.425	0.062
AVP and EDV of the NPCA	− 0.347	0.135
AVP and RI of the NPCA	0.006	0.98

Statistically significant correlations are printed in bold

AVP arteriovenous passage time, *PSV* peak systolic velocity, *EDV* end-diastolic velocity, *RI* resistive index, *OA* ophthalmic artery, *CRA* central retinal artery, *TPCA* temporal posterior ciliary artery, *NPCA* nasal posterior ciliary artery

The AVP time is the shortest circulation time between a peripapillary artery and its corresponding vein. It characterises the microcirculation of the retina in the examined sector. A prolongation of the AVP can be caused by vascular diameter decrease or increased resistance at the level of the lamina cribrosa. Furthermore, vascular dropout in ischaemic retinal diseases has previously been reported to result in a prolongation of the AVP [8, 22].

The correlations found in our work show that ocular blood flow in the CRA is related to the retinal microcirculation presented by the AVP. Therefore, one can assume that optic disc oedema is associated with either primary or secondary reduced blood flow in the optic disc and disturbed microcirculation in the complete retina. These findings imply that the ischaemic damage to the optic disc in NAION and the consequently disturbed microcirculation of the retina synergistically cause profound functional defects in AION patients. It would be interesting to see whether particularly disturbed blood flow velocities can be found in patients with progressive functional deterioration. Furthermore, it would be interesting to see whether the patients with improvement of the visual field over time after NAION show improved ocular blood flow as well and might even have an overall better ocular blood flow at the time of the primary NAION incident. Larger studies have to be conducted to answer these questions.

Furthermore, the visual field damage was significantly correlated to the EDV in the CRA as well. None of the other vessels' CDI parameters as well as AVP were significantly correlated to MD. Furthermore, no statistically significant correlations between the visual acuity and the recorded parameters apart from the RI in the OA were found.

The extent of the functional damage in our patients is correlated to the blood flow in the optic disc in NAION. Whether blood flow velocities are relevant for long-term outcomes in NAION patients have yet to be answered in future long-term studies.

Due to the lack of a direct healthy control group, we will compare the important factors for ocular blood flow, i.e., IOP, Systolic and Diastolic blood pressure (SBP and DBP) as well as ocular perfusion pressure (OPP) to values provided by the large-scale study conducted by Zheng et al. [23]. The authors found an average IOP in 3130 patients without glaucoma (aged 58.7 ± 11) of 15.3 ± 3.5 which is comparable to recorded IOP of 15.55 ± 3.28 found in our patients. The systolic and diastolic blood pressure values recorded in our patients appear to be significantly higher in comparison with the published recordings (SBP: 147.05 ± 24.14 compared to 134.5 ± 24.6 mmHg, DBP: 78.2 ± 12.55 compared to 61.4 ± 11.5 mmHg). However, the ocular perfusion pressure appears to be impaired compared to the previously recorded values in a healthy population (OPP: 51.06 ± 9.67 compared to 52.8 ± 9.3 mmHg) and seems to be similarly reduced as values recorded in patients suffering from open angle glaucoma provided by the same authors (51.6 ± 10.2 mmHg). In our study-group, we were unable to record any significant correlations between OPP and any of the CDI parameters.

A previously published morphological study suggested a form of compartment syndrome in the pathogenesis of AION causing ischaemia [24]. Interestingly, the loss of nerve fibres was at its greatest extent surrounding the CRA.

NAION therapeutic options are limited and somewhat unsatisfying. Optic nerve sheath fenestration (ONSF) was previously reported to be able to improve blood flow in the CRA and PCAs [10]; however, the results are questionable as the data were compared to the partner eye, which was normal in only 60% of the patients. Recently, the treatment method was investigated in the large-scale IONDT-trial, which proved that the clinical improvement in visual acuity is insufficient and accompanied by a greater risk of a visual acuity deterioration [25]. Therefore, the technique should only be considered in patients with progressive NAION, after all other treatment options provided insufficient results.

Most commonly corticosteroids are administered in NAION patients [4]. The exact mechanism is not yet understood. Some authors speculated that the faster dissolution of the optic disc oedema might lead to improved ocular perfusion. Furthermore, recently published work suggests an inflammatory component to play a major role in the cell damage in animal models of NAION [26, 27]. A relatively small randomised study by Rebolleda et al. [28] showed no improvement in visual outcome between treated and untreated subjects. Nevertheless, the most distinguished experts agree that corticosteroids appear to be beneficial in the treatment of NAION [4].

Confounding factors and limitations in our study are the rather small sample size of 20 patients and the vast user dependency of CDI measurements. One should always consider that CDI measurements are highly user dependent especially for the PCAs [15], and only the trunk of the PCAs with potential residual perfusion can be measured. Therefore, the measurements of blood flow in these vessels, which are critically involved in the pathogenesis of NAION, remains a challenge. Furthermore, we were unable to recruit an age-matched otherwise healthy control group for direct comparison of our CDI and AVP findings.

Conclusion

In this study, we were able to show that disturbed blood flow in the CRA is correlated to the visual defect and AVP and increased resistance in the OA can be correlated to the visual acuity in patients with acute NAION. It remains unclear to date whether these findings in the CRA are primary or secondary in NAION. Nevertheless, the critical involvement of ocular blood flow in this sight-threatening disease is eminent. Although previously used surgical methods to improve ocular blood flow in NAION patients have nowadays been abandoned, further studies on other possibilities to improve ocular blood flow should be considered as the critical involvement of retrobulbar and ocular blood flow is apparent.

Compliance with ethical standards

Conflict of interest The authors report no conflicts of interest and have no proprietary interest in any of the materials mentioned in this article.

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