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Rethinking Non-arteritic anterior ischemic optic neuropathy: A pressure-mediated neurovascular failure rather than a primary ischemic event?

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Abstract
Background:
Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common form of acute optic neuropathy in the elderly, as well as a common cause of permanent vision loss. Despite longstanding categorization as an ischemic disease of the optic nerve head, the pathophysiology remains poorly understood and treatment remains lacking.

Purpose:
To critically re-evaluate the classical vascular paradigm of NAION and propose an updated model integrating structural, mechanical, and neurovascular mechanisms.

Methods:
A narrative, hypothesis-driven review of literature (landmark and recent 2015–2025 studies) was performed, focusing on optic nerve head anatomy, OCT/OCTA findings, and emerging concepts in neurovascular coupling and biomechanics.

Results:
The classic vascular model does not account for several key aspects of NAION: the key role of optic disc structure; the variability of presentation and outcome; and poor response to vascular therapy. A growing body of evidence supports a compartment-like mechanism for NAION in which structural crowding predisposes to pressure-induced microvascular compromise. Early axonal edema on imaging suggests that axonal compromise happens before irreversible damage, refuting the idea of primary infarction.

Conclusion:
NAION should be reconceptualized as a structurally predisposed, pressure-mediated neurovascular disorder in which ischemia represents a downstream consequence rather than the initiating event. This paradigm shift has important implications for diagnosis, risk stratification, and therapeutic development.

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I. Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is a form of anterior ischemic optic neuropathy that historically has been associated with and considered a model for ischemic conditions of the optic nerve head secondary to interrupted perfusion via the short posterior ciliary arteries [1-3]. From a clinical perspective, the cardinal feature of NAION is sudden onset of painless vision loss, potentially associated with edematous acute changes of the optic nerve head and variable patterns of visual field loss [4]. Yet despite large medical research, a coherent physiological schema about this condition has remained elusive. Important incongruities exist between fact and this traditional view: lack of demonstrable arterial occlusion in patients afflicted by NAION, the fixed association with a structurally crowded disc and the failure of treatment models developed on a pure vascular model [5-7]. Taken together, these lines of evidence point towards the likelihood that ischemia is not the primary initiating insult. Recent studies employing state-of-the-art structural and functional visualisation techniques have led to new understanding of the chronology and interdependency of the tissue alterations during steady exacerbations of NAION damage [8-10]. Also, this fresh evidence further favours a model based on mechanical, structural effects amplifying the injury in NAION: conceiving the disorder as neither an ischemic nor a non-ischemic form of optic neuropathy but rather as pressure mediated neuro-vascular disease of the optic nerve head.

Re-examining the vascular paradigm: strength without completeness

The current vascular model of NAION is well grounded in established anatomy and population-level epidemiology. The anterior optic nerve head receives blood supply from paraoptic branches of the short posterior ciliary arteries, together composing the circle of Zinn-Haller[11, 12]. Vascular systemic risk factors for NAION (hypertension, diabetes mellitus, hyperlipidemia) are consistently identified[13-16].

The limitations of this approach include the absence of direct histopathological and/or radiographic confirmation of the specific vascular phenomenon hypothesized. Unlike embolic or thrombotic vascular occlusions elsewhere, none of the various theories of NAION involving primary vascular dysfunction has been supported by demonstration of unambiguous vascular occlusion in the involved territory in any large number of patients [17]. Also, though OCTA studies have shown decreased peripapillary perfusion[18,19], the chronological relationship between vascular perfusion changes and structural changes remains unclear. In several studies, observed decreases in perfusion appear to follow observed increases in axonal swelling, suggesting that decrease in perfusion could be secondary to the increased pressure exerted by swollen axons[20, 21]. Most compellingly, multiple antiplatelet, anticoagulant, vasodilatory and corticosteroid therapies have been tested for clinical effectiveness; none has yielded large clinical response in a manner that has been reliably repeated[22]. If ischemia were responsible for damage, some of these would be expected to alter its clinical course, yet no therapy appears reproducibly efficacious.

The central role of structure: the "disc at risk" revisited

Perhaps the most reproducible finding in non-arteritic anterior ischemic optic neuropathy (NAION) is the small crowded "disc at risk"[23], where minimal or absent physiological cupping juxtaposes against increased axonal packing in a tight scleral canal[24]. What's more, this appearance is non-associative with the contralateral eye in impacted patients appearing similarly on many occasions. Affected eyes in turn appear at higher risk for future involvement[25]. Such bilateral symmetry argues strongly for structural predisposition to the pathophysiologic processes underlying disease. Recent imaging studies using high-resolution OCT to quantify and characterize optic nerve head morphology have begun to support the view that structural tissue volumes and densities are indeed lower in susceptible people [26,27]. Biomechanical models further suggest that such crowded appearances could well predispose to localized increases in intraocular pressure even with minimal degrees of edema[28]. Once and done, it may be argued then that our "disc at risk" is in fact an actively engaged - as opposed to passively associative - contributor to disease pathophysiology.

Is ischemia primary or secondary? A critical unresolved question

A key question in NAION pathogenesis is whether ischemia is the triggering mechanism or happens in conjunction with or following another process. The traditional model assumes primary vascular insufficiency directly causing axonal infarction but accumulating evidence challenges this model. OCT studies have shown that the earliest detectable structural finding in NAION is swelling of the retinal nerve fiber layer, showing axonal edema [29]. Thinning and associated visual field damage tend to follow. Also, OCTA findings, while showing reduced peripapillary vessel density in acute NAION, vary with regard to their onset time [32, 33]. An intriguing possibility is that these secondary changes lag behind mechanical effects from swelling in an enclosed space. As it pertains to primary infarction, some studies show onset of structural changes contemporaneous with or prior to ischemia. A primary model predicts immediate structural degeneration after infarction. Instead, series of OCTs consistently reveal a phase of swelling prior to atrophy[30], strongly showing a model where edema and then mechanical factors are primary. A plausible pathway to ischemia in the setting of swelling may involve compression of the very capillaries feeding this region. The observation of thinning following swollen stages suggests an impaction or kinking of vessels due to volumetric pressure. A reasonable conclusion: initial insult → axonal swelling →mechanical pressuring & impaction of vessels → ischemia.

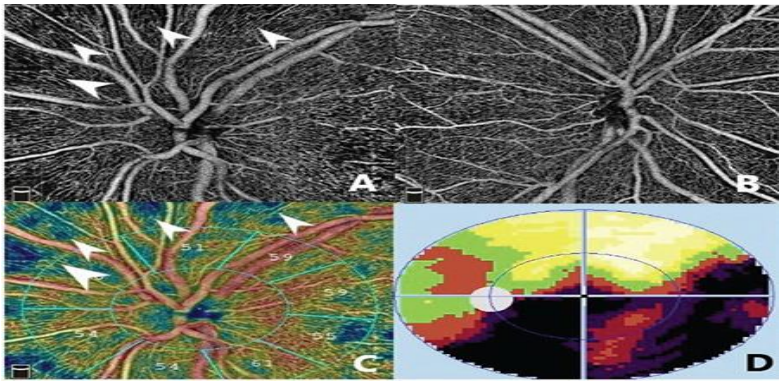

Figure 1: Optical coherence tomography angiography findings in acute NAION demonstrating sectoral hypoperfusion and structure – function correlation.(A) En face OCTA image of the radial peripapillary capillary (RPC) layer showing focal areas of hypoperfusion (white arrowheads).
(B) Corresponding cross-sectional OCTA image demonstrates preserved superficial vascular flow without evidence of large-vessel occlusion.
(C) Vascular density map highlighting reduced perfusion in the superior nasal peripapillary region.(D) Automated visual field showing an inferior altitudinal defect corresponding to the hypoperfused regions.These findings illustrate that microvascular attenuation may occur in the absence of demonstrable primary vascular obstruction, supporting a pressure-mediated secondary ischemic mechanism.Adapted from Hatice Kübra Sönmez et al [57]. Evaluation of Optic Disc Perfusion with Optical Coherence Tomography Angiography in Acute Non-arteritic Anterior Ischemic Optic Neuropathy. Turkish Journal of Ophthalmology. 2022;52(1):30–36. Licensed under Creative Commons Attribution (CC BY).

NAION as a compartment-like process: from hypothesis to framework The concept of NAION being a compartment syndrome of the optic nerve head provides a structure for understanding these observations. A compartment syndrome is characterized by elevated pressure within a limited anatomic space causing diminished perfusion and tissue damage. The ONH, surrounded by the scleral canal, meets several of these criteria [34]. Following some inciting event such as transient hypoperfusion or dysregulation of autoregulation, axonal swelling results. Because the disc is structurally crowded, axonal swelling results in elevated tissue
pressure and further capillary compression; so initiating a vicious cycle in which edema begets ischemia and ischemia begets edema [35]. Animal studies show early axonal swelling followed by irreversible neuronal loss [36] and clinical studies with OCT show a similar progression of events[37]. Also, this model would provide an explanation of variability, as small variations in structure could explain a given cycle stabilizing or escalating to irreversible damage. So, NAION exhibits characteristics of compartment syndromes elsewhere in the body.

Integrating vascular, structural, and neurovascular mechanisms
Nor fully-vacular nor purely-mechanical, I propose a general structure that combines both (see above picture). Systemic vascular and local perfusion instability act as initiators, structure-induced pressure-driven mechanical aggravation can be seen at the level of the optic nerve head and neurovascular coupling can control the metabolic-vascular dynamics [38]. The same principle might be applied to several neurodegenerative conditions, for which tissue susceptibility to degeneration is also a complex interplay of vascular, structure-related and metabolic factors. So, NAION would be but yet another example of neurovascular failure under a specific structural scenario.

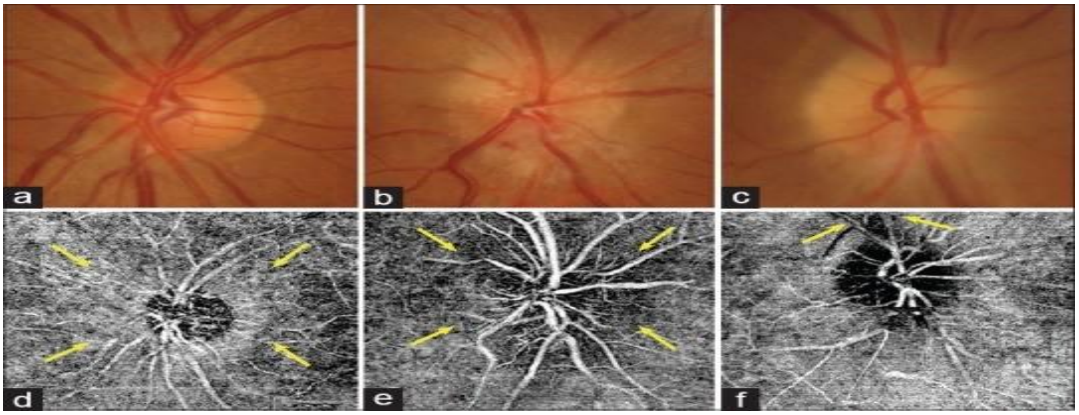

Figure 2: Comparison of optic disc appearance and peripapillary microvasculature in normal and NAION eyes using OCT angiography.

(a, d) Normal eye demonstrating intact peripapillary capillary network.(b, e) Eye with non-arteritic anterior ischemic optic neuropathy showing diffuse loss of the peripapillary microvascular cuff (yellow arrows).(c, f) Eye with NAION demonstrating focal sectoral loss of peripapillary vasculature (yellow arrows).

These findings illustrate the heterogeneous pattern of microvascular involvement in NAION, supporting the concept that perfusion abnormalities may be regionally variable rather than resulting from a single primary vascular occlusion.

Source: Reproduced from Gandhi U, Chhablani J, Badakere A, Kekunnaya R, Rasheed MA, Goud A, et al[57]. Optical coherence tomography angiography in acute unilateral nonarteritic anterior ischemic optic neuropathy: A comparison with the fellow eye and with eyes with papilledema. Indian J Ophthalmol. 2018;66(8):1144–1148. doi:10.4103/ijo.IJO_179_18. Licensed under Creative Commons Attribution-NonCommercial-ShareAlike 4.0 (CC BY-NC-SA).

Why treatments fail: a mismatch between mechanism and therapy

It is clear how the absence of an effective treatment for NAION, up to now, can be explained by this integrated model. The majority of current treatments act through vascular pathways, either enhancing perfusion or preventing thrombosis. But if mechanical forces and tissue pressures play major roles, then these approaches will likely never address the principal sources of injury[39]. The inability of optic nerve decompression surgery to provide benefit in clinical trials may have been wrongly construed as strong evidence that a compartment mechanism does not exist. Indeed, surgery on patients with NAION has occurred only well after irreparable damage has set in and so timed and technique may not actually be aimed at successfully mitigating edema in its earliest phases. Therapeutic intervention in the future may need to be geared towards early modulation of axonal swelling and microvascular patency, as well as neuroprotection from secondary injury.

Future directions: toward a testable model

The future of NAION investigation lies in solving the problem of whether ischemia is the first hit or secondary to structural/biomechanical failure in the optic nerve head (ONH). While advanced imaging modalities have failed to delineate the timing between the extent of axonal injury and microvascular compromise, it is important to redefine the pathogenesis. The OCTA finding -what we see is either primary or secondary;We can see reduced peripapillary vessel density on OCTA in NAION but still we do not know what is the cause and effect. The presence of microvascular attenuation in NAION is consistently documented on optical coherence tomography angiography (OCTA) but the interpretation thereof is limited due to its inability to set up cause and effect. Recent papers suggest microvascular attenuation could potentially happen as a secondary phenomenon to structural and axonal swelling, not primary [20, 21, 45]. Diverse findings regarding the diagnostic and prognostic value of OCTA underscore the issue [3, 4, 5]. Increasing data showing a potential increased severity of microvascular rarefaction in crowded discs or associated with optic disc drusen further shows the link between structural and microvascular crowding[3]. Future investigations should focus on hyperacute longitudinal investigations to illustrate the sequence of events leading from retinal nerve fiber layer (RNFL) swelling, to vessel dropout, to dysfunction, so clarifying whether it is the microvascular or structural changes that serve as the inciting factor in the pathogenesis of NAION.

The ONH is characterized by tissue deformation, lamina cribrosa translation and changing scleral canal dimensions that determine both axonal vitality as well as ocular perfusion0. Individual components,

like the lamina cribrosa can be subject to unique stress/ remodelling that may determine microvascular patency and axoplasmic flow. Recent advances using OCT derived structural maps in modelling allows quantification of ONH deformation and computation of effort fields showing localised distribution of biomechanical stress [46] by doing this potentially identifying susceptible neuronal populations. An individual with a structurally crowded disc would so appear susceptible to a pressure induced ischemia despite the presence of relatively slight alterations in perfusion. Future works should therefore delineate the patient specific mechanistic signature for susceptibility, axonal swelling vs effort and also investigate the predictive biomarker for biomechanical stress that portend clinical NAION-demonstrating, that NAION may transform from a stochastic event to a biomechanically stratified disease.

Artificial intelligence (AI) is revolutionizing ophthalmic care as deep learning models learn to fuse structural (OCT) and vascular (OCTA) data to detect occult retinal nerve fiber layer thinning, microvascular alterations and optic disc phenotypes which may not be immediately obvious to the eye of an expert clinician [52]. More critically, newer emerging AI architectures aim to transcend the limits of classification to embark on predictive modeling which identifies patients at risk for NAION based on ONH structure [10], predicts future axon loss and second eye involvement and incorporates systemic influences with ocular imaging to usher in the era of precision neuro-ophthalmology where disease risk can be quantified before irrecoverable injury. The relatively unexplored yet seminal determinant of NAION disease expression is neuro-vascular coupling -the homeostasis governing local blood supply by matching axon metabolic demand met with perfusion. Derailed coupling places axons susceptible to injury despite no primary blood vessel compromise. Emerging ONH perfusion being interlinked with structure and metabolism suggests potential autoregulatory failure contributing to disease emergence [20, 21] with added concomitant systemic disorders such as nocturnal hypotension or sleep apnea further upsetting perfusion metabolist matching[4]. NAION may in fact reflect focalled neuro-vascular uncoupling.

Perhaps the most critical future direction is the transition of the clinically descriptive diagnosis of NAION into a mechanistically testable model of disease. The pressure mediated neurovascular structure presented in this review yields multiple testable hypotheses -namely that: Structural Crowding precedes vascular compromise. Axonal Edema precedes Capillary Dropout. ONH biomechanical effort predicts Lesion Localization. Early Modulation of Edema Alters Disease -that can be evaluated in prospective multimodal studies that combine OCT, OCTA, functional metrics and computational modeling. A important aspect of successful study will be a focus on the early stage of disease, during which intervention remains potentially biological relevant. By defining Non-Arteritic Ischemic Optic Neuropathy (NAION) as a pressure mediated neurovascular disorder, several important advances can be envisioned in terms of clinical management. Current treatment modalities, mainly targeting vascular pathophysiology, fail to treat the associated mechanical/structural aspects of disease [3, 7, 9] Future strategies might include an emphasis on early modulation of axonal edema and tissue pressure; application of optic nerve head (ONH) biomechanical/structural modulation therapy; and/or deployment of neuroprotective drugs aimed at secondary injury cascades. Also, with application of artificial intelligence (AI) powered risk prediction and ONH biomechanics the possibility of detecting preclinical risk people for progression, by doing this transforming non-arteritic ischemic optic neuropathy (NAION) management from a clinically reactive to a preclinical proactive approach in ophthalmology and neuro-ophthalmology.

II. Summary

In this article I challenge the classic ischemic model for non-arteritic anterior ischemic optic neuropathy (NAION) and present a revised pathophysiologic model incorporating structural, mechanical and neurovascular mechanisms. Despite long being conceptualized as a disorder characterized by primary vascular insufficiency of the optic nerve head, there are important inconsistencies with this model, including lack of confirmatory vascular occlusive lesions, an overwhelming association with the "disc at risk" phenotype and little-to-no efficacy of treatments targeting the vasculature. Utilizing recent advances in optical coherence tomography (OCT) and OCT angiography (OCTA), I emphasize that early-onset axonal edema (retinal nerve fiber layer edema) is consistently seen before irreversible tissue damage, whereas reduction in microvascular perfusion happens only later, so challenging the concept of infarction-as-initiator and supporting ischemia-as-byproduct of increased tissue pressure in a structurally-crowded optic disc. I propose a compartment-like syndrome where axonal edema within a crowded optic disc leads to increased intra-tissue pressure, capillary compression and initiation of a vicious edema-ischemia cycle accounting for the variability in onset and progression of NAION. A structure is synthesized with systemic vascular risk factors serving as triggers; structural predisposition/biomechanics dictating progression, with neurovascular dysregulation amplifying disease progression, by doing this explaining the

consistent failure of historic therapies attempting to improve perfusion or prevent thrombosis. I conclude that NAION should now be reclassified as a pressure-mediated disorder, rather than mostly an ischemic optic neuropathy with ischemia acting as a down-stream effect. This model shift has immediate implications for clinical practice in terms of the need for early detection, structural risk-stratification and treatment strategies targeting axonal edema, tissue pressure and neuroprotection and encourages future studies examining the utility of longitudinal multimodal imaging and personalized mechanism-based therapy.

III. Conclusion

NAION has traditionally been viewed as a manifestation of vascular insufficiency, a model that does not fully explain its clinical characteristics. A growing body of evidence suggests that a different model - in which structural crowding and pressure-mediated effects play a more prominent role - may be more accurate. Instead of being considered an ischemic optic neuropathy, NAION can be viewed as a structurally predisposed, pressure-mediated neurovascular event, in which ischemia is a late effect rather than an initial cause. This view resolves long-standing paradoxes in NAION and suggests new directions for research and therapy that could in the end change the way we understand and treat this disease. The time is now right for a model shift in our thinking on this difficult problem. Model shifts have immediate implications for clinical practice: risk stratification needs to shift away from systemic vascular determinants and focus instead on the structure of the optic nerve head. Similarly, therapies will need to shift from enhancing perfusion to the early treatment of axonal edema and tissue pressure, if ongoing failure of therapeutic trials is to be avoided. I believe that NAION is fundamentally misclassified; a structurally triggered, pressure mediated neurovascular failure with ischemia as a downstream effect.

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Conflicts of interest
There are no conflicts of interest.

Ethical approval
Not applicable. This study is a narrative review and does not involve human participants or animals.

Informed consent
Not applicable.

Author contributions
Akshay Thakur conceptualized the study, performed the literature review and is the Corresponding author. Dr. Seema Yengkhom drafted the manuscript, and approved the final version.

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Data availability
No new data were generated or analyzed in this study.