



Evaluation of Peripapillary Vitreous Dynamics in Patients with Acute Nonarteritic Anterior Ischemic Optic Neuropathy

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Abstract

Objectives: To evaluate peripapillary and vitreomacular interface configurations using spectral-domain optical coherence tomography (SD-OCT) in patients with acute nonarteritic anterior ischemic optic neuropathy (NAION) and to compare findings with age-matched controls.

Materials and Methods: This single-center, retrospective, observational study included 60 patients with acute NAION and 60 age- and sex-matched healthy controls. Circumpapillary retinal nerve fiber layer scans and macular SD-OCT images were analyzed. Assessed peripapillary vitreoretinal interface parameters included peripapillary vitreoretinal adhesion, vitreoretinal traction, complete peripapillary vitreous detachment, peripapillary folds, superficial vascular protrusions, and hyperreflective vitreous dots. Vitreomacular interface characteristics were also evaluated.

Results: Peripapillary vitreoretinal adhesion was more frequently observed in NAION eyes compared with controls (50% vs. 25%, $p=0.04$). Hyperreflective vitreous dots were significantly more common in NAION eyes (68.3% vs. 8.3%; $p<0.001$), with a higher mean count (5.31 ± 6.05 vs. 0.50 ± 1.54 ; $p<0.001$). Peripapillary folds were also more prevalent in NAION (41.7% vs. 1.7%; $p<0.001$). Although peripapillary superficial vascular protrusions were common in both groups, mean counts differed significantly ($p<0.001$). Among 34 NAION eyes with

longitudinal follow-up, 29.4% demonstrated interval changes in vitreoretinal interface configuration.

Conclusion: Acute NAION is associated with distinct peripapillary vitreoretinal interface characteristics detectable by SD-OCT. These findings highlight structural differences in vitreoretinal interface configuration in acute NAION. However, their temporal relationship to ischemic injury remains uncertain.

Keywords: NAION, optical coherence tomography, vitreous detachment

Introduction

Nonarteritic anterior ischemic optic neuropathy (NAION) typically presents with a history of sudden painless unilateral visual loss, visual field defects, and optic disc edema in people over 50 years of age.¹ NAION is generally attributed to impaired perfusion within the short posterior ciliary artery circulation.^{2,3} Anatomically crowded optic discs appear particularly susceptible to perfusion imbalance, suggesting that structural and microenvironmental factors at the optic nerve head may influence disease expression.^{1,2} Optic disc edema in NAION is primarily thought to result from axoplasmic flow stasis secondary to impaired perfusion of the anterior optic nerve.⁴

Age-related changes at the vitreoretinal interface, including posterior vitreous detachment (PVD), are common in older adults and are known to contribute to a spectrum of vitreoretinal disorders.⁵ In certain configurations, persistent attachment of the posterior hyaloid at or near the optic nerve head has been described as vitreopapillary adhesion or traction.^{6,7,8} Recent optical coherence tomography (OCT)-based observations have identified vitreoretinal interface variations in some eyes with NAION, raising the question of whether consistent

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peripapillary structural patterns coexist in the acute phase of the disease.^{7,8,9} However, it remains unclear whether such findings represent incidental age-related changes, secondary alterations related to disc edema, or disease-associated interface characteristics.

The development of spectral-domain (SD) OCT has enabled high-resolution visualization of both macular and peripapillary vitreoretinal interface configurations.^{10,11} Although the vitreomacular interface has been extensively studied in macular disease, the peripapillary interface in NAION has received comparatively limited and heterogeneous investigation.

Accordingly, the present study aimed to evaluate peripapillary vitreoretinal and vitreomacular interface characteristics using SD-OCT in patients with acute NAION and to compare these findings with age- and sex-matched controls.

Materials and Methods

Study Design and Participants

This retrospective observational study included newly diagnosed patients with acute NAION evaluated at the Neuro-ophthalmology Unit of the University of Health Sciences Türkiye, Ankara Training and Research Hospital between June 2016 and December 2024. The study adhered to the tenets of the Declaration of Helsinki and was approved by the institutional review board of the University of Health Sciences Türkiye, Ankara Training and Research Hospital (protocol number: E-24-330, decision no: 330/2024, decision date: 18.12.2024). Informed consent was obtained from all participants.

Acute NAION was diagnosed based on sudden, painless unilateral vision loss in individuals older than 50 years, accompanied by diffuse or sectoral optic disc edema and corresponding visual field defects, with no alternative ocular, neurologic, or systemic cause identified. Only patients presenting within 15 days of symptom onset were included to ensure assessment during the acute phase. Patients with previous NAION sequelae, optic disc edema attributable to etiologies other than NAION (e.g., papilledema or inflammatory optic neuropathy), prior intraocular surgery (except uncomplicated cataract surgery), ocular trauma, uveitis, or other retinal or optic nerve diseases were excluded. A small optic disc was defined clinically based on the fundusoscopic appearance of a crowded optic nerve head with a small or absent physiologic cup. No predefined quantitative optic disc area cutoff was used.

Age- and sex-matched control subjects were randomly selected from individuals undergoing routine

ophthalmologic examination. Inclusion criteria were age greater than 50 years, spherical equivalent between +5 and -5 diopters, astigmatism less than 3 diopters, clear media, and the absence of retinal or optic nerve pathologies such as glaucoma, diabetic retinopathy, macular degeneration, and optic disc anomalies. Only one eye per control subject was included.

Collected data included age, sex, medical history, best-corrected visual acuity (BCVA), intraocular pressure measured by Goldmann applanation tonometry, slit-lamp biomicroscopy and dilated fundus examination findings, and SD-OCT imaging of the optic disc region and macula.

OCT Acquisition and Image Evaluation

All OCT images were obtained using the Heidelberg Spectralis system (Heidelberg Engineering, Heidelberg, Germany). Peripapillary retinal nerve fiber layer (RNFL) imaging was performed using a standardized 3.4-mm-diameter circular scan centered on the optic disc. Macular scans were acquired using the standard SD-OCT macular protocol.

All OCT scans were reviewed by a single experienced neuro-ophthalmologist (G.O.) masked to the study hypothesis. Image quality was assessed prior to review, and scans with insufficient quality to evaluate the vitreoretinal interface were excluded.

Evaluation of the Peripapillary Vitreoretinal Interface

Because circumpapillary RNFL scans do not directly image the optic nerve head surface, the study evaluated peripapillary vitreoretinal interface configurations rather than direct vitreopapillary attachment over the optic disc.

The following peripapillary parameters were assessed on circumpapillary RNFL scans: peripapillary vitreoretinal adhesion, peripapillary vitreoretinal traction, peripapillary vitreous detachment, peripapillary folds, peripapillary superficial vascular protrusions, and hyperreflective vitreous dots.

Peripapillary vitreoretinal adhesion was defined as visible contact between the posterior hyaloid and the inner retinal surface along the circumpapillary scan in at least one quadrant, without associated inner retinal deformation ([Figure 1a](#)). Peripapillary vitreous detachment was defined as the absence of visible posterior hyaloid attachment along the entire 360-degree circumpapillary scan ring. Peripapillary vitreoretinal traction was defined as visible peripapillary vitreoretinal adhesion accompanied by structural retinal deformation at the site of attachment, including focal elevation or tenting of the inner retinal surface ([Figure 1b](#)). In the absence of retinal deformation,

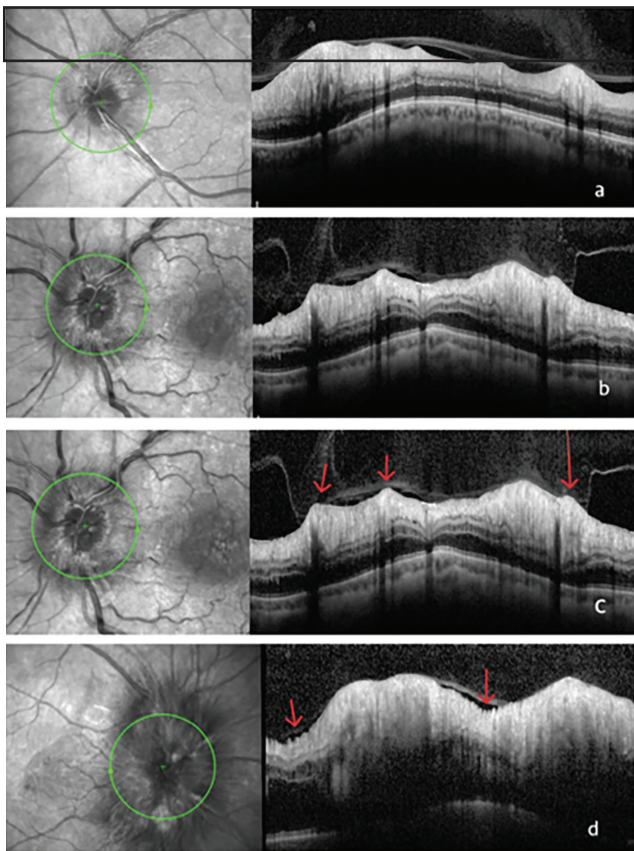


Figure 1. Spectral-domain optical coherence tomography circular circumpapillary retinal nerve fiber layer B-scans centered on the optic disc. a) Peripapillary vitreoretinal adhesion in the left eye of a patient with nonarteritic anterior ischemic optic neuropathy (NAION). b) Peripapillary vitreoretinal traction in the left eye of a patient with NAION. c) Red arrows indicate peripapillary superficial vessel protrusions in the left eye of a patient with NAION. d) Red arrows indicate peripapillary folds in the right eye of a patient with NAION

the finding was classified as adhesion rather than traction. Peripapillary superficial vascular protrusion was defined as focal anterior displacement of superficial retinal vessels above the RNFL contour (Figure 1c). Peripapillary folds were defined as concentric or spiral undulations of the inner retinal surface within the RNFL layer (Figure 1d). Hyperreflective vitreous dots were defined as discrete punctate hyperreflective foci within the peripapillary vitreous exceeding background speckle reflectivity. Dots were manually counted on the circumpapillary RNFL scan and recorded per eye.

Evaluation of the Vitreomacular Interface

Macular SD-OCT scans were used to evaluate vitreomacular interface features. Vitreomacular adhesion

was defined as perifoveal vitreous detachment with persistent foveal attachment and the absence of retinal distortion. Vitreomacular traction required persistent vitreomacular attachment accompanied by structural retinal changes, including inner retinal deformation, pseudocyst formation, macular schisis, cystoid macular edema, or subretinal fluid. Epiretinal membrane (ERM) was defined as a hyperreflective membrane on the inner retinal surface. Lamellar macular hole was defined as a partial-thickness foveal defect with preservation of the outer retinal layers.

Statistical Analysis

Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to assess the normality of continuous variables. Continuous variables were expressed as mean±standard deviation, and comparisons between the NAION and control groups were performed using the Mann-Whitney U test. Categorical variables were expressed as frequencies and percentages and were compared using the chi-square test. All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Results

Demographics and Ophthalmologic Examination

A total of 60 eyes from 60 patients with acute NAION and 60 eyes from 60 age- and sex-matched healthy controls were included. The mean age was 60.9 ± 8.4 years in the NAION group and 61.3 ± 9.7 years in the control group ($p=0.912$). Each group consisted of 27 females and 33 males. Mean BCVA was significantly lower in the NAION group (0.46 ± 0.36) compared with controls (0.86 ± 0.16 ; $p=0.017$). Thirty-four NAION patients (56.7%) had follow-up longer than six months and were included in the longitudinal analysis. Baseline demographic and clinical findings are summarized in Table 1.

Peripapillary Vitreoretinal Interface Findings

On circumpapillary RNFL scans at baseline, peripapillary vitreoretinal adhesion was identified in 30 NAION eyes (50%). Peripapillary vitreoretinal traction was observed in 12 eyes (20%). Complete peripapillary vitreous detachment was present in 2 eyes (3.3%). Compared with controls, NAION eyes showed a significantly higher frequency of peripapillary vitreoretinal adhesion ($p=0.04$). The distribution of peripapillary vitreoretinal interface configurations is summarized in Table 2.

Longitudinal Peripapillary Changes

Among the 34 NAION eyes with follow-up imaging, 10 (29.4%) demonstrated interval changes in peripapillary

Table 1. Demographic and clinical characteristics of patients with acute NAION and age-matched controls

Variable	NAION (n=60)	Controls (n=60)	p value
Age, years	60.9±8.4	61.3±9.7	0.912
Female sex, n (%)	27 (45)	27 (45)	1.000
BCVA, Snellen decimal	0.46±0.36	0.86±0.16	0.017
IOP, mmHg	12.95±3.55	13.15±4.74	0.554
Mean RNFL thickness, µm	203.64±48.27	100.93±8.49	<0.001

NAION: Nonarteritic anterior ischemic optic neuropathy, BCVA: Best-corrected visual acuity, IOP: Intraocular pressure, RNFL: Retinal nerve fiber layer

Table 2. Peripapillary vitreoretinal interface configuration and peripapillary structural features in acute NAION and controls

Parameter	NAION (n=60)	Controls (n=60)	p value
Normal peripapillary vitreoretinal interface, n (%)	16 (26.7)	31 (51.7)	0.04
Peripapillary vitreoretinal adhesion, n (%)	30 (50.0)	15 (25.0)	0.04
Peripapillary vitreoretinal traction, n (%)	12 (20.0)	9 (15.0)	0.473
Peripapillary vitreous detachment, n (%)	2 (3.3)	5 (8.3)	0.245
Hyperreflective vitreous dots present, n (%)	41 (68.3)	5 (8.3)	<0.001
Hyperreflective dot count, mean ± SD	5.31±6.05	0.50±1.54	<0.001
Peripapillary superficial vascular protrusions present, n (%)	53 (88.3)	58 (96.7)	0.084
Number of vascular protrusions, mean ± SD	2.51±1.84	3.45±1.47	<0.001
Peripapillary folds present, n (%)	25 (41.7)	1 (1.7)	<0.001
Small optic disc, n (%)	56 (93.3)	10 (16.7)	<0.001

NAION: Nonarteritic anterior ischemic optic neuropathy, SD: Standard deviation

vitreoretinal interface configuration. Eight eyes (23.5%) progressed from adhesion to traction, and two eyes (5.9%) developed complete peripapillary vitreous detachment.

Additional Peripapillary Structural Findings

Hyperreflective vitreous dots were detected significantly more frequently in NAION eyes (41 eyes, 68.3%) compared with controls (5 eyes, 8.3%) ($p<0.001$). The mean number of hyperreflective dots per eye was also higher in the NAION group (5.31 ± 6.05 vs. 0.50 ± 1.54 ; $p<0.001$). Peripapillary superficial vascular protrusions were common in both groups (88.3% in NAION vs. 96.7% in controls; $p=0.084$). However, the mean number of protrusions per eye differed significantly (2.51 ± 1.84 vs. 3.45 ± 1.47 ; $p<0.001$). Peripapillary folds were identified in 25 NAION eyes (41.7%) compared with 1 control eye (1.7%; $p<0.001$). These findings are summarized in [Table 2](#).

Vitreomacular Interface Findings

At baseline, vitreomacular adhesion was present in 15 NAION eyes (25.0%), vitreomacular traction in 3 eyes (5.0%), ERM in 4 eyes (6.7%), complete PVD in 5 eyes (8.3%), and lamellar macular hole in 1 eye (1.7%). The

overall prevalence of vitreomacular interface abnormalities was significantly higher in the NAION group compared with controls ($p<0.001$). A summary of vitreomacular interface findings is provided in [Table 3](#).

Among NAION patients with follow-up imaging, 10 eyes (29.4%) developed ERM not present at baseline. Interval macular interface changes were observed in 12 eyes (35.3%), including progression to PVD (26.5%), vitreomacular traction (5.9%), and vitreomacular adhesion (2.9%). A summary of longitudinal vitreomacular interface changes is provided in [Table 4](#).

Discussion

In the present study, we evaluated peripapillary and macular vitreoretinal interface configurations in patients with acute NAION using SD-OCT. Compared with age-matched controls, NAION eyes demonstrated a higher prevalence of detectable peripapillary vitreoretinal adhesion, hyperreflective vitreous dots, and peripapillary folds. Nearly one-third of patients with longitudinal follow-up exhibited interval interface changes, further indicating that vitreoretinal interface alterations are frequently observed in acute NAION.

Table 3. Vitreomacular interface findings

Parameter	NAION (n=60), n (%)	Controls (n=60), n (%)	p value
Normal vitreomacular interface	32 (53.3)	50 (83.3)	<0.001
Abnormal vitreomacular interface	28 (46.7)	10 (16.7)	<0.001
Epiretinal membrane	4 (6.7)	1 (1.7)	0.172
Lamellar macular hole	1 (1.7)	0 (0)	0.317
Vitreomacular traction	3 (5.0)	0 (0)	0.081
Vitreomacular adhesion	15 (25.0)	9 (15.0)	0.173
Posterior vitreous detachment	5 (8.3)	5 (8.3)	1.00

NAION: Nonarteritic anterior ischemic optic neuropathy

Table 4. Longitudinal changes in the vitreomacular interface (NAION eyes, n=34)

Parameter	n (%)
Any interval macular interface change	12 (35.3)
Development of epiretinal membrane	10 (29.4)
Progression to posterior vitreous detachment	9 (26.5)
Progression to vitreomacular traction	2 (5.9)
Progression to vitreomacular adhesion	1 (2.9)

NAION: Nonarteritic anterior ischemic optic neuropathy

Age-related vitreous liquefaction leads to progressive syneresis and a reduction in gel volume, generating tractional stress at residual sites of vitreoretinal adhesion. Under physiological conditions, these forces are generally well tolerated. However, when PVD occurs anomalously, persistent peripapillary attachment may produce vitreopapillary adhesion or traction.¹² Clinical observations indicate that persistent vitreous adhesion can transmit mechanical forces to the optic nerve head.^{13,14} Hayreh et al.^{1,2} demonstrated that even minimal reductions in optic disc perfusion may precipitate ischemia in anatomically crowded discs, underscoring the intrinsic vulnerability of these eyes to circulatory imbalance. More recent work has suggested that vitreopapillary traction represents part of a broader spectrum of vitreopapillary interface abnormalities that can influence optic nerve head biomechanics, and in certain contexts may also affect local vascular dynamics.^{14,15} In this setting, persistent vitreous adhesion may influence local peripapillary biomechanics. In our cohort, peripapillary interface differences were more frequently observed in acute NAION than in controls. These observations suggest that variations in peripapillary vitreoretinal interface configuration may coexist with acute NAION. However, whether such interface characteristics represent pre-existing structural features, secondary responses to disc edema, or epiphenomena of ischemic injury remains uncertain.

OCT-based studies have reported shallow adhesions, microfolds, and localized vitreous configuration changes as common accompanying features.⁸ Li et al.⁷ further demonstrated that incomplete papillary vitreous detachment is highly prevalent in NAION and can produce dynamic, intermittent traction on the optic disc, manifesting as peripapillary wrinkles, superficial vessel protrusions, and localized surface distortion-features that may mirror the interface abnormalities identified in our cohort. Hondur et al.¹⁴ indicated that persistent or residual vitreous adhesion may influence prelaminar and peripapillary tissue biomechanics, contributing to localized vessel wall deformation and altered microvascular behavior in susceptible discs. The peripapillary folds observed in our cohort may reflect structural changes associated with acute disc edema rather than definite tractional deformation. Overall, our results indicate that peripapillary interface characteristics differ in NAION, although it is not possible to determine whether these findings precede ischemia or develop as a consequence of disc swelling.

The detection of hyperreflective dots within the peripapillary vitreous was a notable observation in our NAION cohort. To our knowledge, vitreous-based hyperreflective dots have not been systematically described in patients with NAION. Previous reports, including the recent case series by Santos et al.¹⁶, described hyperreflective foci within the retinal layers, where they were interpreted as activated microglia, inflammatory cells, or proteinaceous debris following ischemic injury. In contrast, our observations demonstrated hyperreflective foci located within the vitreous compartment, suggesting that they may represent a different phenomenon from previously described intraretinal hyperreflective foci. Comparable vitreous hyperreflective dots have been reported in other inflammatory or ischemic retinal diseases, particularly uveitis and central retinal artery occlusion, where they are considered OCT correlates of inflammatory cell aggregates or reactive vitreous debris.^{17,18} These observations raise the possibility that vitreous hyperreflective dots in NAION may

be related to local inflammatory or ischemia-associated changes; however, their origin remains uncertain. They may correspond to inflammatory cells, erythrocytes, or microhemorrhagic material. Alternatively, some may represent vitreous collagen condensations, age-related vitreous opacities, or imaging artifacts, and their exact nature cannot be determined based on OCT findings alone. Correlation with disc hemorrhage was not systematically evaluated in our cohort, which should be considered a limitation of this study.

We observed interval changes in vitreomacular interface configuration, including ERM formation and progression toward partial PVD or vitreomacular traction. These findings may reflect dynamic remodeling of the vitreoretinal interface in the setting of acute NAION; however, the underlying mechanisms cannot be determined from imaging data alone. Such alterations could represent structural remodeling occurring during the course of the disease rather than a direct effect of ischemia itself. Histopathologic and prospective imaging studies would be required to clarify the biological mechanisms involved. Similar post-ischemic remodeling phenomena have been reported in diabetic macular edema and retinal vein occlusion.^{19,20} NAION is likely multifactorial, involving structural crowding, disc edema, and microvascular dysregulation. Peripapillary interface abnormalities should therefore be interpreted as accompanying structural findings within a multifactorial process rather than primary etiologic factors.

Study Limitations

This study has several limitations. Its retrospective design and modest sample size preclude causal inference. Not all patients underwent longitudinal OCT follow-up, limiting our ability to characterize the temporal evolution of vitreoretinal interface changes. In addition, the control group did not undergo longitudinal imaging and was not matched for systemic microvascular comorbidities. Conditions such as diabetes and hypertension may independently influence vitreoretinal interface characteristics and could partially account for some of the observed differences. The absence of a comorbidity-matched disease-control cohort should therefore be considered when interpreting these findings. Because the control group consisted of healthy eyes without optic disc edema, the present study cannot determine whether peripapillary folds, superficial vascular protrusions, or retinal contour alterations are specific to NAION or represent nonspecific structural consequences of optic disc swelling. Comparative studies including other causes of optic disc edema, such as papilledema or

inflammatory optic neuropathy, are required to clarify the specificity of these findings.

Furthermore, the study did not directly assess vitreopapillary adhesion over the optic nerve head but rather evaluated peripapillary retinal surface configurations using circumpapillary scans. Additionally, the OCT acquisition protocol was not specifically designed for comprehensive staging of PVD. Although both peripapillary and macular vitreoretinal interface configurations were evaluated, complete PVD staging could not be reliably performed retrospectively. Therefore, peripapillary vitreous adhesion should be interpreted within the context of age-related vitreous attachment rather than as an inherently pathologic finding.

Finally, all OCT images were evaluated by a single observer, which may have introduced observer bias, particularly in the assessment of qualitative vitreoretinal interface features. Interobserver agreement was therefore not assessed, and future studies using masked independent graders would improve the reproducibility of these findings. Despite these limitations, the structural differences observed between NAION and control eyes suggest that vitreoretinal interface configuration warrants further investigation in this setting.

Conclusion

Acute NAION is associated with distinct peripapillary and vitreomacular interface characteristics detectable by SD-OCT. Hyperreflective vitreous dots and surface contour irregularities were observed more frequently in NAION eyes than in controls, and NAION eyes demonstrated changes in vitreoretinal configuration over follow-up. These findings highlight structural differences at the vitreoretinal interface in the setting of acute NAION, although their temporal sequence and clinical significance remain uncertain. Prospective studies incorporating standardized imaging protocols and appropriate disease-control cohorts are needed to further clarify the relevance of these interface characteristics in NAION.

Ethics

Ethics Committee Approval: The study adhered to the tenets of the Declaration of Helsinki and was approved by the institutional review board of the University of Health Sciences Türkiye, Ankara Training and Research Hospital (protocol number: E-24-330, decision no: 330/2024, decision date: 18.12.2024).

Informed Consent: Informed consent was obtained from all participants.

Declarations

Authorship Contributions

Surgical and Medical Practices: G.O., K.K., Ö.C., G.S., Concept: G.O., Ö.C., Design: G.O., Ö.C., G.S., Data Collection or Processing: G.O., Analysis or Interpretation: G.O., N.Ü., A.B., Literature Search: G.O., K.K., Writing: G.O.

Conflict of Interest: No conflict of interest was declared by the authors.

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