NEU PRESS

SELCUK MEDICAL JOURNAL
SELÇUK TIP DERGİSİ

DOI:http:/dx.doi.org/10.30733/std.2026.01992
Selcuk Med J 2026;42(3): 295-300

OPEN

RESEARCH ARTICLE

Persistent Papilledema After Cerebral Venous Sinus Thrombosis: A Prospective Observational Cohort Study

Serebral Venöz Sinüs Trombozu Sonrası Persistan Papilödem: Prospektif Gözlemsel Kohort Çalışması

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ABSTRACT

Objective: Papilledema is a clinically significant manifestation of cerebral venous sinus thrombosis (CVST) and reflects intracranial hypertension resulting from impaired venous outflow. However, its severity and longitudinal course have not been adequately characterized. This study aimed to evaluate the longitudinal course of papilledema in patients with CVST and to identify clinical and radiological factors associated with persistent papilledema.

Materials and Methods: This prospective observational study included 37 adult patients with radiologically confirmed CVST. Papilledema severity was assessed at baseline and at six months using the Frisén grading scale. Persistent papilledema was defined as the presence of papilledema of any grade (≥1) at follow-up. Clinical and radiological variables, including venous sinus involvement and recanalization status, were analyzed.

Results: Papilledema was present in 14 patients (37.8%) at baseline. At six months, papilledema had resolved in 5 patients but persisted in 9 patients (24.3% of the cohort). The frequency of baseline Frisén grade 3–4 papilledema was significantly higher among patients with persistent papilledema than among those without persistent papilledema (66.7% vs. 7.1%; $p < 0.001$). Magnetic resonance imaging (MRI) data were available for 25 patients, and complete recanalization was observed more frequently in patients without persistent papilledema ($p = 0.007$). No significant associations were identified between persistent papilledema and demographic, clinical, or other radiological variables.

Conclusion: Papilledema may persist in a substantial proportion of patients with CVST. Higher baseline papilledema grades were more frequently observed among patients with persistent papilledema, whereas complete recanalization was more common among those without persistent papilledema. These findings support the importance of structured neuro-ophthalmological follow-up in patients with CVST.

Keywords: Cerebral venous sinus thrombosis, Intracranial hypertension, Papilledema, Prognosis

ÖZET

Amaç: Papilödem, serebral venöz sinüs trombozunun (SVST) klinik açıdan önemli bir bulgusudur ve bozulmuş venöz dönüşe bağlı intrakraniyal hipertansiyonu yansıtır. Ancak şiddeti ve zaman içindeki seyri yeterince tanımlanmamıştır. Bu çalışmada, SVST hastalarında papilödemin uzunlamasına seyrinin değerlendirilmesi ve persistan papilödem ile ilişkili klinik ve radyolojik faktörlerin belirlenmesi amaçlandı.

Gereç ve Yöntemler: Bu prospektif gözlemsel çalışmaya, radyolojik olarak doğrulanmış SVST tanılı 37 erişkin hasta dahil edildi. Papilödem şiddeti başlangıçta ve altıncı ayda Frisén derecelendirme ölçeği kullanılarak değerlendirildi. Persistan papilödem, takipte herhangi bir derecede papilödem varlığı (≥1) olarak tanımlandı. Venöz sinüs tutulumu ve rekanalizasyon durumu dahil olmak üzere klinik ve radyolojik değişkenler analiz edildi.

Bulgular: Başlangıçta 14 hastada (%37,8) papilödem mevcuttu. Altıncı ayda 5 hastada düzeldi, 9 hastada ise devam etti (kohortun %24,3'ü). Başlangıçta Frisén evre 3–4 papilödem görülme sıklığı, persistan papilödemi olan hastalarda olmayan hastalara göre anlamlı derecede daha yüksekti (%66,7'ye karşı %7,1; $p<0,001$). Manyetik rezonans görüntüleme (MRG) verileri 25 hastada mevcuttu ve tam rekanalizasyon, persistan papilödem olmayan hastalarda daha sık görüldü ($p = 0,007$). Demografik, klinik veya diğer radyolojik değişkenlerle anlamlı ilişki saptanmadı.

Sonuç: Papilödem, SVST'li hastaların önemli bir kısmında persiste edebilir. Daha yüksek başlangıç papilödem dereceleri, persistan papilödemi olan hastalarda daha sık gözlenirken; tam rekanalizasyon, persistan papilödemi olmayan hastalarda daha sık görüldü. Bu bulgular, SVST'de yapılandırılmış nöro-oftalmolojik takibin önemini desteklemektedir.

Anahtar Kelimeler: İntrakraniyal hipertansiyon, Papilödem, Prognoz, İntrakraniyal sinüs trombozu

Received: 28 April 2026 **Accepted:** 8 August 2026 **Published Online:** 14 September 2026

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Cite this article as: Sonmez MT, Arslan Mehdiyev D, Duman İlki C, Cam N, Mehdiyev Z, Uzun SU, Cillier AE. Persistent Papilledema After Cerebral Venous Sinus Thrombosis: A Prospective Observational Cohort Study. Selcuk Med J 2026;42(3): 295-300

Disclosure: *Author has not a financial interest in any of the products, devices, or drugs mentioned in this article. The research was not sponsored by an outside organization. Author has agreed to allow full access to the primary data and to allow the journal to review the data if requested.*

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is an uncommon cerebrovascular disorder, accounting for approximately 0.5–3% of all stroke cases and occurring more frequently among young adults and women (1, 2). The clinical presentation of CVST is highly heterogeneous and primarily reflects impaired cerebral venous drainage (3-5). Headache, seizures, and focal neurological deficits represent the most common presenting manifestations, whereas symptoms associated with intracranial hypertension constitute another important component of the disease spectrum (1, 4, 6). Among these manifestations, papilledema is a clinically significant finding resulting from elevated intracranial pressure secondary to venous outflow obstruction (7).

Papilledema in CVST develops predominantly because increased venous pressure impairs cerebrospinal fluid absorption at the arachnoid granulations (3, 4, 8). Although this pathophysiological mechanism differs from direct parenchymal injury, persistent intracranial hypertension may nevertheless lead to progressive optic nerve damage and visual impairment if not adequately recognized and monitored (2, 7). Despite its clinical relevance, papilledema in CVST has frequently been reported only as a dichotomous finding, with limited attention paid to its severity or temporal evolution (9). Recent studies have suggested that signs of intracranial hypertension may persist in a subset of patients despite apparent clinical stabilization (10). This observation indicates that papilledema may not always represent a transient manifestation confined to the acute phase of the disease and may instead necessitate ongoing follow-up (7, 10). However, longitudinal studies assessing papilledema severity using standardized grading systems remain scarce.

Furthermore, the relationship between specific patterns of venous sinus involvement and the clinical course of papilledema remains inadequately characterized. Thrombosis involving major venous outflow pathways may alter intracranial pressure dynamics and contribute to the persistence of papilledema in certain patients (4, 10). Therefore, a more comprehensive understanding of the longitudinal course of papilledema in CVST is of considerable clinical importance, as sustained intracranial hypertension may place patients at risk of progressive and potentially preventable visual impairment. The present study aimed to evaluate the longitudinal course of papilledema in patients with CVST using the Frisén papilledema grading scale at baseline and after six months of follow-up. Additionally, we sought to determine the frequency of persistent papilledema and to investigate its potential associations with clinical and radiological characteristics.

MATERIALS AND METHODS

Study Design and Participants

This prospective observational study included consecutive adult patients diagnosed with CVST who were admitted to Ankara Etlik City Hospital between July 2023 and December 2024. The diagnosis of CVST was established on the basis of the clinical presentation and was confirmed by neuroimaging findings, including magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV) or computed tomography venography (CTV) (1). Eligible participants were adults (≥18 years of age) with radiologically confirmed CVST who underwent ophthalmological and/or neurological assessment for papilledema at the time of diagnosis and were available for follow-up evaluation at six months. Patients were excluded if they had pre-existing optic nerve disorders, ocular diseases that could interfere with the assessment of papilledema, or incomplete clinical or neuroimaging data.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Ankara Etlik City Hospital Ethics Committee (approval number: Ankara Etlik City Hospital-EK1-2023-243). Written informed consent was obtained from all participants prior to study enrollment.

Clinical Assessment and Follow-up

Baseline demographic and clinical characteristics were recorded at the time of presentation. These data included age, sex, presenting symptoms, and neurological examination findings. Particular emphasis was placed on manifestations associated with intracranial hypertension, including headache and visual symptoms. For analytical purposes, comorbidities and risk factors were categorized into predefined groups. Metabolic and endocrine comorbidities included conditions such as diabetes mellitus, hypertension, hyperlipidemia, and thyroid disorders. Potential predisposing factors comprised smoking, migraine, polycystic ovary syndrome (PCOS), high-dose vitamin A exposure, and obesity. Transient provoking risk factors included oral contraceptive use, pregnancy, the postpartum period, and active sinusitis. Persistent provoking risk factors included malignancy, inherited or acquired thrombophilia (including antiphospholipid syndrome), and Behçet's disease.

All patients were followed for a period of six months following the initial diagnosis. Clinical evaluations were performed both at baseline and at the six-month follow-up visit.

Papilledema Assessment

The severity of papilledema was evaluated using the Frisén papilledema grading scale, a standardized clinical classification system ranging from grade 0 (normal optic disc) to grade 5 (severe papilledema). Fundoscopic examinations were performed at baseline and repeated at the six-month follow-up visit. All papilledema assessments were conducted by a single ophthalmologist with expertise in neuro-ophthalmology. Both eyes were evaluated separately, and Frisén grades were recorded individually for each eye at baseline and at the six-month follow-up assessment. For patient-level analyses, the highest papilledema grade observed in either eye was used as the representative measure of papilledema severity. Persistent papilledema was defined as the presence of any degree of papilledema (Frisén grade ≥1) at the six-month follow-up visit and was considered the primary outcome of the study.

Neuroimaging Evaluation

All patients underwent neuroimaging at the time of diagnosis. The diagnosis of CVST was confirmed by magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV) or by computed tomography venography (CTV).

All neuroimaging studies were reviewed by an interventional neurologist with expertise in cerebrovascular diseases. The anatomical location of venous sinus thrombosis was recorded for each patient. Venous sinus involvement was categorized according to the affected sinus segments, including the superior sagittal sinus, transverse sinus, sigmoid sinus, straight sinus, and other venous structures. The presence of single-sinus and multiple-sinus involvement was also documented.

Treatment

All patients received treatment in accordance with current clinical guidelines for cerebral venous sinus thrombosis (1). Anticoagulation therapy was initiated unless contraindicated, and additional therapeutic interventions were administered when clinically indicated. Treatment decisions were made by the treating physicians as part of routine clinical practice and were not influenced by the study protocol. Treatment-related variables were not included in the statistical analyses of the present study.

Statistical Analysis

Statistical analyses were performed using jamovi version 2.6.45 (The jamovi Project, Sydney, Australia). The normality of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation (SD) and compared using Student's t-test. Non-normally distributed variables were presented as median (interquartile range [IQR]) and compared using the Mann–Whitney U test.

Categorical variables were expressed as frequencies and percentages and were compared using the chi-square test or Fisher's exact test, as appropriate. A two-sided p value < 0.05 was considered statistically significant. Multivariable logistic regression analysis was not performed because of the limited sample size and the low number of outcome events.

RESULTS

Baseline Characteristics of the Study Population

A total of 37 patients with CVST were included in the study. The median age was 31 years (IQR 24–45; range, 18–71 years), and 67.6% (n = 25) were female. The median number of emergency department or outpatient clinic visits related to CVST prior to diagnosis was 1 (IQR 0–3). Headache and other nonspecific symptoms, including dizziness and nausea, constituted the most common initial presentation (73.0%), followed by focal neurological deficits (13.5%), encephalopathy (10.8%), and visual symptom–predominant presentation (2.7%). Seizures occurred in 21.6% of patients, while status epilepticus was observed in 5.4%. Papilledema was present at baseline in 14 patients. Metabolic and endocrine comorbidities were identified in 18.9% of the cohort. Potential predisposing factors were present in 51.4% of patients, whereas transient and persistent precipitating factors were observed in 51.4% and 62.2% of patients, respectively. Radiologically, multiple-sinus thrombosis was the most common finding, occurring in 86.5% of patients. Among individual venous sinus involvements, transverse sinus thrombosis was the most frequent (83.8%), followed by superior sagittal sinus thrombosis (37.8%). Venous infarction was detected in 32.4% of patients, and hemorrhagic components were present in 8.1%. The baseline demographic, clinical, and radiological characteristics of the study population are summarized in Table 1.

Persistent Papilledema

Among the 14 patients with papilledema at baseline, papilledema resolved during follow-up in 5 patients, whereas it persisted in 9 patients. Overall, persistent papilledema was observed in 9 of 37 patients (24.3%) at the six-month follow-up assessment. No significant associations were identified between persistent papilledema and age, sex, seizure occurrence, clinical presentation at symptom onset, venous infarction, or multiple-sinus thrombosis (all $p > 0.05$). However, the prevalence of high-grade papilledema at baseline (Frisén grades 3–4) was significantly greater among patients with persistent papilledema than among those without persistent papilledema without persistent papilledema (66.7% vs. 7.1%, $p < 0.001$).

Six-month MRI data were available for 25 of 37 patients (8/9 patients with persistent papilledema and 17/28 patients without persistent papilledema). Among these patients,

Table 1. Baseline demographic, clinical, and radiological characteristics of the study population

Characteristics	n (%) / median (IQR)
Demographics	
Age, years (median IQR)	31 (24–45)
Female sex, n (%)	25 (67.6)
Presenting symptoms	
Headache/nonspecific symptoms	27 (73.0)
Focal neurological deficit	5 (13.5)
Encephalopathy	4 (10.8)
Visual-symptom–predominant onset	1 (2.7)
Neurologic features	
Seizure, n (%)	8 (21.6)
Status epilepticus, n (%)	2 (5.4)
Papilledema at presentation, n (%)	14 (37.8)
Risk factors	
Metabolic/endocrine comorbidities, n (%)	7 (18.9)
Potential predisposing factors, n (%)	19 (51.4)
Transient precipitating factors, n (%)	19 (51.4)
Persistent precipitating factors, n (%)	23 (62.2)
Radiological findings	
Transverse sinus thrombosis, n (%)	31 (83.8)
Superior sagittal sinus thrombosis, n (%)	14 (37.8)
Multi-sinus thrombosis, n (%)	32 (86.5)
Venous infarction, n (%)	12 (32.4)
Hemorrhagic component, n (%)	3 (8.1)

Note: IQR: interquartile range. Visual-symptom–predominant onset refers to patients presenting primarily with visual complaints.

Table 2. Comparison of clinical and radiological features according to persistent papilledema status

Variable	Without Persistent Papilledema (n=28)	Persistent Papilledema (n=9)	p value
Age, years (median IQR)	32 (24.8-44.3)	26 (20-51)	0.357
Female, n (%)	20 (71.4%)	5 (55.6%)	0.432
Seizure, n (%)	7 (25.0%)	1 (11.1%)	0.649
Venous infarction, n (%)	11 (39.3%)	1 (11.1%)	0.220
Multi-sinus thrombosis, n (%)	24 (85.7%)	8 (88.9%)	1.000
Baseline papilledema Frisén grade 3-4, n (%)	2 (7.1%)	6 (66.7%)	<0.001
Complete recanalization, n (%)	13 (76.5%)	1 (12.5%)	0.007*
Incomplete recanalization, n (%)	4 (23.5%)	7 (87.5%)	

Note: Recanalization data were available for 25 patients and were evaluated based on six-month MRI findings. Incomplete recanalization comprised both partial recanalization and absence of recanalization. The p value was calculated using Fisher's exact test.
*Complete and incomplete recanalization represent complementary categories of the same variable; therefore, a single p value is reported.

Table 3. Comparison of patients with and without baseline papilledema

Variable	Without Papilledema (n=23)	Papilledema (n=14)	p value
Age, years (median IQR)	33 (25.5-43.5)	27 (21.5-45.0)	0.357
Female, n (%)	18 (78.3%)	7 (50.0%)	0.146
Seizure, n (%)	4 (17.4%)	4 (28.6%)	0.445
Superior sagittal sinus thrombosis, n (%)	6 (26.1%)	8 (57.1%)	0.085
Multi-sinus thrombosis, n (%)	20 (86.9%)	12 (85.7%)	1.000
Venous infarction, n (%)	8 (34.8%)	4 (28.6%)	1.000

Note: IQR: interquartile range. Age is presented as median (IQR); other variables are presented as n (%).

complete recanalization was significantly more common in those without persistent papilledema than in those with persistent papilledema (76.5% vs. 12.5%; Fisher's exact test, p = 0.007). Comparisons between patients with and without persistent papilledema are presented in Table 2.

Comparison Between Patients With and Without Baseline Papilledema

Of the 37 patients included in the study, 14 presented with papilledema at admission, whereas 23 had no evidence of papilledema. No significant differences were observed between the two groups regarding age, sex, seizure occurrence, venous infarction, multiple-sinus thrombosis, clinical presentation, or the distribution of risk factors (all p > 0.05). Superior sagittal sinus thrombosis was numerically more frequent among patients with papilledema than among those without papilledema (57.1% vs. 26.1%); however, this difference did not reach statistical significance (p = 0.085). Comparisons according to baseline papilledema status are summarized in Table 3.

DISCUSSION

In this prospective cohort of patients with CVST, papilledema was present at the time of diagnosis in more than one-third of patients. At the six-month follow-up, papilledema had resolved in most cases; however, it persisted in approximately one-quarter of the cohort. The frequency of baseline high-grade papilledema (Frisén grades 3–4) was significantly higher among patients with persistent papilledema than among those without persistent papilledema (66.7% vs. 7.1%, p < 0.001). In addition, differences in recanalization status were observed between patients with and without persistent papilledema. Complete recanalization at six months was more frequently observed in patients without persistent papilledema. No significant associations were identified between persistent papilledema and demographic characteristics, clinical presentation, seizure occurrence, or the radiological parameters evaluated, including venous infarction and multiple-sinus involvement. Taken together, these findings support careful follow-up of patients presenting with high-grade papilledema. The prevalence of papilledema in our cohort was consistent with the broad range reported in previous studies (4, 11-13). Papilledema was observed in 37.8% of patients at presentation. In the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT), papilledema was reported in 28.3% of 624 patients (14). Similarly, the large multicenter VENOST study reported papilledema in approximately 28% of 1,144 patients (9). The slightly higher prevalence observed in our cohort may be attributable to the systematic neuro-ophthalmological assessment performed at baseline. Both eyes were evaluated, and papilledema severity was graded using the standardized Frisén scale. This approach may have increased the detection of mild or early-stage optic disc edema in routine clinical practice.

Our findings indicate that papilledema may persist beyond the acute phase in a subset of patients with CVST. Nearly one-quarter of patients exhibited persistent papilledema at the six-month follow-up assessment. Although persistent papilledema does not necessarily indicate ongoing intracranial hypertension, previous studies have suggested that intracranial hypertension may persist in a subset of

patients following CVST and that the mean time required for papilledema resolution is approximately six months (3). Schuchardt et al. demonstrated that neuroimaging markers of intracranial hypertension, including optic nerve sheath enlargement and partial empty sella, may persist for several months despite clinical stabilization (10). Likewise, Wei et al. reported that severe intracranial hypertension at presentation, including papilledema and visual symptoms, was associated with poorer visual outcomes during follow-up (4). Collectively, these observations suggest that papilledema should not be regarded solely as a transient acute manifestation and support continued neuro-ophthalmological surveillance during follow-up (7). The most notable finding of our study was that baseline high-grade papilledema (Frisén grades 3–4) was significantly more frequent among patients with persistent papilledema at six months than among those without persistent papilledema. One possible explanation is that patients presenting with more severe papilledema may experience slower resolution of optic disc edema during follow-up. This observation suggests that the initial severity of papilledema may be associated with its subsequent clinical course. However, the mechanisms underlying this association could not be directly evaluated in the present study. Previous investigations have demonstrated that structural and hemodynamic changes within the optic nerve head may persist even after clinical improvement (7, 10, 15).

Recanalization status differed significantly between patients with and without persistent papilledema. Complete recanalization at six months was more frequently observed in patients without persistent papilledema than in those with persistent papilledema (76.5% vs. 12.5%, $p = 0.007$). Previous studies have suggested that persistent impairment of venous outflow may contribute to prolonged venous hypertension and delayed resolution of papilledema (2–4, 7). In this context, our findings suggest a potential relationship between recanalization status and the persistence of papilledema. However, intracranial pressure was not directly measured in our cohort, and the mechanisms underlying this association remain speculative. We did not identify significant associations between persistent papilledema and several clinical or radiological factors. Variables such as seizure occurrence, venous infarction, and multiple-sinus thrombosis were not associated with papilledema persistence. Although superior sagittal sinus thrombosis was numerically more frequent among patients with papilledema at presentation, this difference did not reach statistical significance. The superior sagittal sinus is a major site of cerebrospinal fluid (CSF) absorption through the arachnoid granulations, and its occlusion has been associated with more pronounced intracranial hypertension (4). Recent systematic reviews have likewise emphasized impaired CSF drainage at this location as an important mechanism contributing to optic disc swelling (2, 3). Nevertheless, the lack of statistical significance should be interpreted cautiously given the limited sample size of our cohort.

We found no clear association between papilledema and seizure occurrence. This finding supports the concept that CVST may manifest through distinct pathophysiological pathways. Recent clinical classifications distinguish between isolated intracranial hypertension syndromes and focal parenchymal syndromes in CVST (6). Seizures and focal neurological deficits are commonly associated with parenchymal injury, hemorrhagic infarction, or cortical vein involvement (16). In contrast, papilledema primarily reflects generalized intracranial pressure elevation resulting from impaired venous drainage. Recent studies have similarly reported that papilledema is not strongly associated with seizure occurrence in patients with CVST (17, 18). These findings have important clinical implications. Papilledema in CVST should not be considered merely a transient manifestation of the acute disease phase. Persistent papilledema may occur in a substantial proportion of patients and may threaten vision if not adequately monitored. Patients presenting with high-grade papilledema may benefit from closer neuro-ophthalmological follow-up, including regular funduscopic examinations and visual field assessments (4, 7). Accordingly, these findings may contribute to risk stratification and guide the intensity of follow-up in routine clinical practice.

Study Limitations

This study has several strengths. It was designed as a prospective cohort study with systematic follow-up at a predefined six-month interval. Papilledema severity was evaluated using the standardized Frisén grading scale rather than a simple binary classification. Furthermore, funduscopic examinations were performed by an ophthalmologist with expertise in neuro-ophthalmology, and both eyes were assessed separately. This approach enabled a more detailed evaluation of papilledema severity and its longitudinal course.

Several limitations should also be acknowledged. First, this was a single-center study with a relatively small sample size, which may have limited the statistical power to detect moderate associations in subgroup analyses. The limited sample size also precluded multivariable analysis; therefore, the observed associations should be interpreted as exploratory rather than as evidence of independent effects. Second, the proportion of hemorrhagic lesions in our cohort was lower than that reported in larger multicenter cohorts such as the ISCVT (14). This difference may partly reflect selection bias, as severely affected patients with extensive hemorrhagic lesions may not have been able to undergo detailed ophthalmological evaluation at baseline. In addition, visual field outcomes were not systematically available and therefore could not be incorporated into the analysis, despite their clinical relevance in patients with papilledema. Information regarding intracranial pressure–lowering therapies during follow-up was also not systematically collected. Consequently, the potential influence of treatment-related factors on papilledema persistence could not be reliably evaluated. Although some treatment-related differences were observed between groups, treatment allocation was not standardized and the sample size was insufficient to determine independent treatment effects. Furthermore, follow-up MRI data were not available for all patients, which may have introduced selection bias into the

analysis of recanalization and its association with papilledema persistence. Finally, intracranial pressure was not directly measured during follow-up; therefore, papilledema was used as a noninvasive clinical marker of intracranial hypertension.

CONCLUSION

In conclusion, papilledema is a common manifestation of CVST and may persist in a substantial proportion of patients during follow-up. The prevalence of baseline high-grade papilledema (Frisén grades 3–4) was higher among patients with persistent papilledema. Furthermore, complete recanalization at six months was observed more frequently in patients without persistent papilledema. In contrast, parenchymal lesions and focal neurological manifestations were not significantly associated with the persistence of papilledema. These findings underscore the importance of comprehensive neuro-ophthalmological evaluation at the time of diagnosis and continued follow-up thereafter, particularly in patients presenting with high-grade papilledema.

DECLARATIONS

Conflict of Interest: *The authors declared that there is no conflict of interest.*

Financial Disclosure: *The authors declare no financial conflicts of interest.*

Acknowledgements: *The authors declare that there are no individuals who contributed to the preparation of this manuscript but did not meet authorship criteria.*

Funding: *No financial support was received for this study.*

Author Contributions: *Concept: M.T.S. Design: M.T.S., D.A.M., C.D.İ. Data Collection or Processing: M.T.S., D.A.M., N.Ç., Z.M., S.Ü.U., A.E.Ç. Analysis or Interpretation: M.T.S., C.D.İ. Literature Search: M.T.S., D.A.M., C.D.İ. Writing: M.T.S., D.A.M., C.D.İ.*

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