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ORIGINAL RESEARCH

Optic nerve sheath diameter to diagnose psychogenic stroke

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Abstract

Background: Differential diagnosis between acute ischemic stroke and stroke mimics is important for initiating appropriate treatment. This study aimed to evaluate the diagnostic utility of optic nerve sheath diameter (ONSD) measured via cranial computed tomography (CT) for differentiating acute ischemic stroke from psychogenic stroke mimics in patients presenting to the emergency department (ED). **Methods**: This was a single-center, retrospective, case–control study. Patients were divided into two groups: patients with a definitive diagnosis of cerebrovascular accident (CVA) (Group 1, n = 612) and patients with psychogenic stroke-mimicking diseases (Group 2, n = 38). ONSD was measured using the initial brain CT scan performed upon admission to the ED. **Results**: The mean ONSD was 5.21 $\pm$ 0.71 mm in the overall population, and it was significantly higher in the acute CVA group (5.24 $\pm$ 0.73 mm) than in the psychogenic mimic group (4.32 $\pm$ 0.28 mm) ($p < 0.001$). In the receiver operating characteristics curve analysis conducted to assess the value of ONSD sufficient in distinguishing acute CVA from psychogenic stroke mimics, a cutoff of 4.87 mm yielded a sensitivity of 68.2% and a specificity of 95.5% (Area under the curve (AUC) = 0.885; 95% confidence interval (CI) = 0.838–0.932; $p < 0.001$). **Conclusions**: In patients presenting to the ED with suspected acute stroke, if ONSD is normal on brain CT, a more detailed history, neuropsychiatric examination and advanced neuroimaging should be performed before considering thrombolytic therapy to rule out the possibility of psychogenic stroke mimics.

Keywords

Ischemic stroke; Stroke mimics; Optic nerve sheath diameter

1. Introduction

Stroke is a significant cause of morbidity and mortality, affecting one in four people throughout their lifetime [1]. As an emergency condition, stroke requires rapid assessment of patients in the acute phase, including a thorough medical history, physical examination and noncontrast computed tomography (CT) [2]. The timely initiation of reperfusion therapy within 4.5 h of symptom onset is crucial for improving functional outcomes in patients with acute ischemic stroke [1–3]. However, some patients presenting with typical stroke symptoms actually experience stroke mimics, which can be functional (due to psychological or psychiatric disorders) or medical (due to conditions such as seizures, migraine, labyrinthitis and benign paroxysmal positional vertigo). Some of the patients diagnosed with ischemic stroke are patients with imitation stroke; this rate reaches up to approximately 17%, leading to unnecessary thrombolysis treatment to be administered to misdiagnosed patients [4–6]. Functional neurological disorders are among the most common conditions encountered by neurologists and neuropsychiatrists [5]. The symptoms of psychogenic stroke mimics are heterogeneous on onset and can be either acute or insidious. These symptoms can also resemble those of vascular stroke. Therefore, it is clinically challenging to differentiate patients with psychogenic stroke from those with vascular stroke [5, 7].

Optic nerve sheath (ONS) is a membrane that covers the optic nerve, which extends from the brain to the dura mater. The optic nerve and its sheath are collectively referred to as the optic nerve complex. The thickness of the complex is an important clinical indicator of certain disease conditions. The thickness of the retrobulbar region of the ONS is higher than that of the waist portion of the nerve. The thickness of the optic nerve complex may increase in patients with increased intracranial pressure (ICP). The bulbous portion of the nerve is located approximately 3 mm behind the eyeball. It is the most distensible part and most sensitive to the alterations in the ICP [8]. In the early 1960s, a British-Indian ophthalmologist, Hayreh, described the pathogenesis of optic disc edema and the mechanism by which intracranial hypertension is transmitted into the ONS [9].

Numerous studies have demonstrated that measuring the

optic nerve sheath diameter (ONSD) using CT or ultrasound has prognostic and diagnostic value in various neurological conditions, including subarachnoid hemorrhage, intracranial hypertension, stroke subtypes, seizures, complicated migraine and metabolic disorders [10–13]. Despite extensive studies, no study has investigated whether ONSD measurements can help differentiate psychogenic stroke mimics from true stroke cases and guide thrombolytic therapy decisions.

Therefore, this study aimed to explore whether ONSD can serve as a useful parameter for distinguishing true stroke from psychogenic stroke mimics.

2. Methods

2.1 Patient selection

In this study, data were collected by reviewing the hospital information management system database and patient files within the last 6 years (2019–2024). Patients aged ≥18 years who presented to the emergency department (ED) with symptoms of cerebrovascular accident (CVA), evaluated by emergency medicine specialists and neurologists, diagnosed with acute ischemic stroke by physical examination and cranial tomography, and who received thrombolytic therapy were included in the study.

2.2 Data collection

The demographic data of the patients, including age and sex, were recorded. The mean ONSDs of the right and left eyes were evaluated by two emergency specialists trained in radiological evaluation. They were blinded to the patients' clinical assessments and used the initial brain CT images obtained during the ED visits. To ensure standardization for the physicians measuring ONSD of the patients, measurements of 10% of the patients (39 patients) were conducted by physicians blinded to each other's results, with a 5% margin of error accepted in the measurements. The inter-rater agreement was determined by calculating the kappa value, which was 0.92.

2.3 Study groups

Patients were divided into two groups: patients with a definitive diagnosis of cerebrovascular accident (Group 1, n = 612) and patients with psychogenic stroke-mimicking diseases (Group 2, n = 38).

2.4 Measurement of the ONSD

ONSD was measured using the initial brain CT scan performed upon admission to the ED. Head CT was performed using a CT scanner (Canon Aquilion Lightning 160 slice/80 detector row Ultra Helical CT) (1385 Shimoishigami, Otawara City, Tochigi 324-8550, Japan) following the standardized brain CT protocol using noncontrast 1-mm contiguous slices.

All measurements were performed using the same window (WW 40, WL 250), contrast, and brightness settings. The ONSD was measured 3 mm behind the globe, immediately below the sclera. To obtain the most reliable diameter, the coronal plane was used to measure the transverse diameter of the ONSD (Fig. 1) [14].

2.5 Statistical analysis

Data were analyzed using SPSS (version 26.0, https://en.wikipedia.org/wiki/SPSS_Inc., Chicago, IL, USA). Descriptive statistics are presented as numbers and percentages (n (%)), means and standard deviations (Mean ± SD), and as medians and ranges. The conformity of the data to normal distribution was evaluated using the Kolmogorov-Smirnov test. In the univariate analysis, continuous variables that did not show normal distribution were expressed as median (interquartile range). Pearson's chi-square test was used to compare categorical variables, and Fisher's exact test was used when the number of variables was less than five. Student's *t*-test was used to compare two independent continuous variables. The diagnostic accuracy and predictive performance were assessed using receiver operating characteristic (ROC) curve analysis. Appropriate cutoff values were determined, and for parameters with an area under the curve (AUC) >0.600, sensitivity and specificity values were calculated. A p-value < 0.05 was considered statistically significant in all analyses.

3. Results

Among the patients diagnosed with stroke mimics as a result of clinical follow-up, advanced examination and neuropsychiatric evaluations, 25 patients with medical mimickers (such as migraine, epilepsy, hyponatremia and multiple sclerosis), 10 patients diagnosed with transient ischemic attack, and 6 patients who could not be followed up due to death (arrest at presentation and subsequent death despite intervention) were excluded from this study. Finally, 650 of 691 patients were included in the study, of whom 612 had a true stroke and 38 had psychogenic stroke mimics.

Of the 650 patients included in the study, 60% (n = 390) were male, and the mean age was 67.96 ± 13.10 years. The proportion of women in the psychogenic mimic group was significantly higher than that in the acute CVA group ($p = 0.004$). The mean age of the acute CVA group was 68.18 ± 13.07 years and that of the psychogenic mimic group was 64.32 ± 13.46 years, with no significant difference between the groups. All patients received thrombolytic therapy as indicated for acute stroke. Hemorrhagic complications were observed in 14.8% of the patients who received thrombolytic therapy. Moreover, hemorrhagic complications and mortality were observed only in the acute CVA group, and the difference was statistically significant ($p = 0.021$ and $p = 0.049$, respectively) (Table 1).

ONSD was assessed in all patients. The mean ONSD was 5.21 ± 0.71 mm. Comparison of ONSD values between the groups showed that the mean ONSD was significantly higher in the acute CVA group (5.24 ± 0.73 mm) than in the psychogenic mimic group (4.32 ± 0.28 mm) ($p < 0.001$) (Table 1; Fig. 2).

In the ROC curve analysis conducted to assess the value of ONSD sufficient in distinguishing between the acute CVA and psychogenic mimic groups, a cutoff of 4.87 mm yielded a sensitivity of 68.2% and a specificity of 95.5% (AUC = 0.885; 95% CI = 0.838–0.932; $p < 0.001$) (Fig. 3).

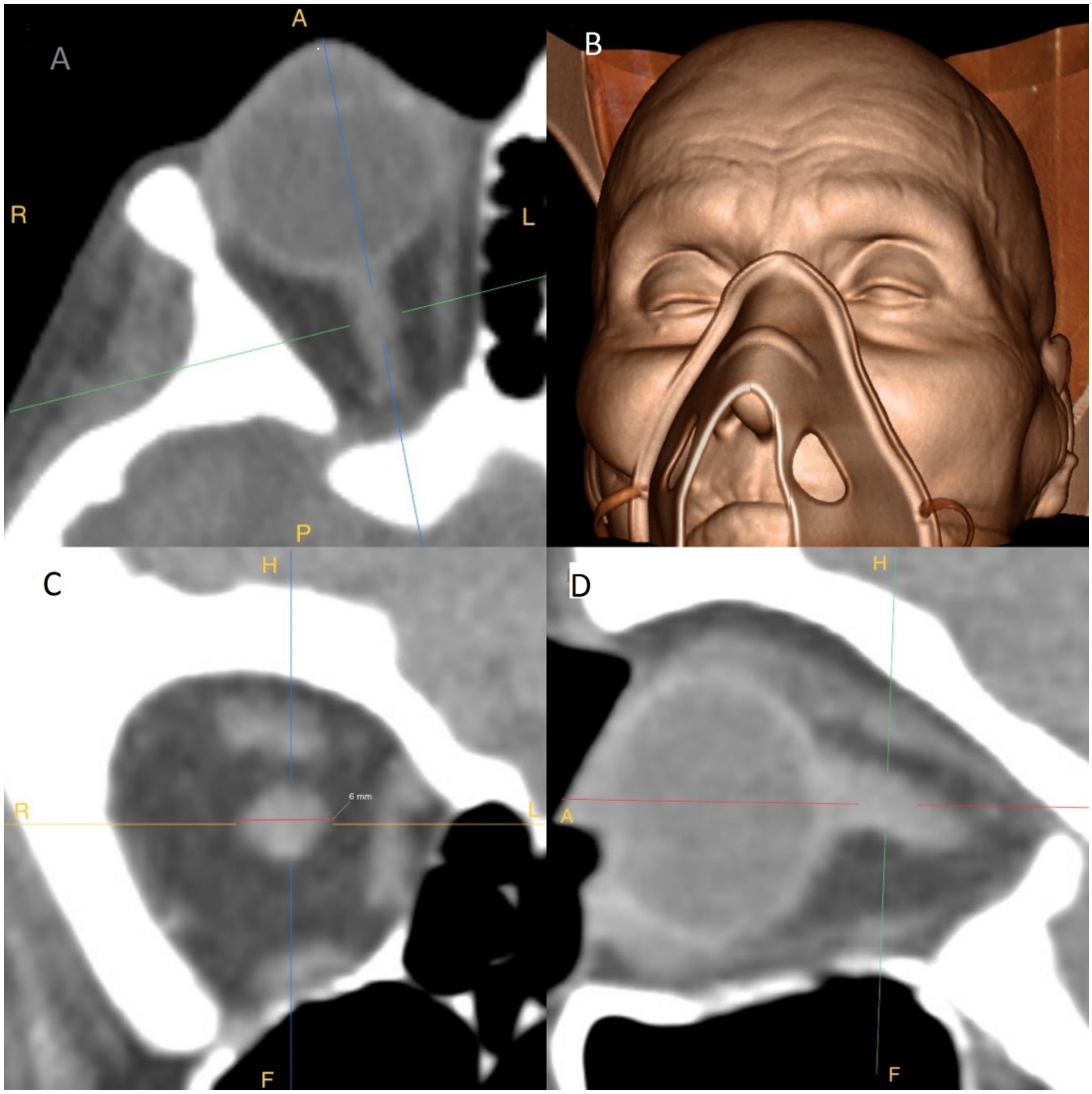

FIGURE 1. Measurement of optic nerve sheath (ONS) diameter. Perpendicular to both transverse (A) 3D Tomograpy Image (B) Coronal plane (C) and sagittal planes (D) of the optic nerve. The transverse diameter of the ONS was measured on the coronal plane.

TABLE 1. Comparison of demographic and clinical data between the groups.

Parameters	n (%)/Mean ± SD	Acute CVA group (n = 612) n (%)/Mean ± SD	Psychogenic mimic group (n = 38) n (%)/Mean ± SD	*p*
Age (yr)	67.96 ± 13.10	68.18 ± 13.07	64.32 ± 13.46	0.180[t]
Sex				
Men	390 (60.0)	385 (62.9)	12 (31.6)	0.004[p]
Woman	260 (40.0)	227 (37.1)	26 (68.4)	
ONSD (mm)	5.21 ± 0.71	5.24 ± 0.73	4.32 ± 0.28	<0.001[t]
Hemorrhagic Complication				
No	554 (85.2)	554 (85.2)	38 (100.0)	0.021[t]
Yes	96 (14.8)	96 (14.8)	0 (0.0)	
Mortality (30 d)				
No	575 (88.5)	575 (88.5)	38 (100.0)	0.049[t]
Yes	75 (11.5)	75 (11.5)	0 (0.0)	

[t]: Independent samples t-test; [p]: Pearson χ^2 Test; SD: Standard deviation; mm: millimeter; ONSD: optic nerve sheath diameter; CVA: cerebrovascular accident.

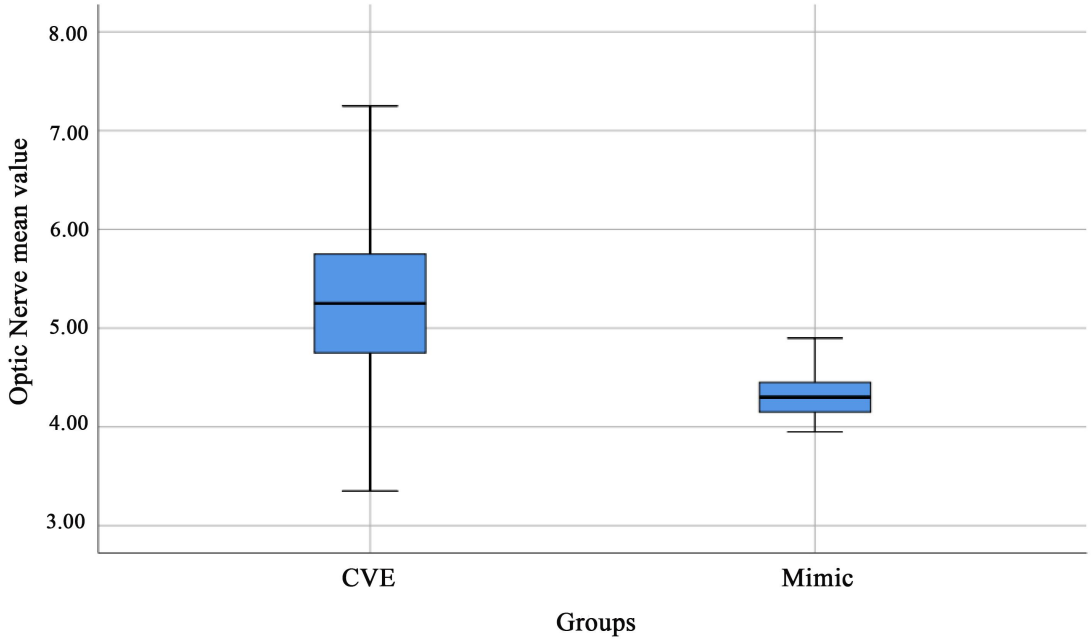

FIGURE 2. Mean optic nerve values. CVE: Cerebrovascular Event.

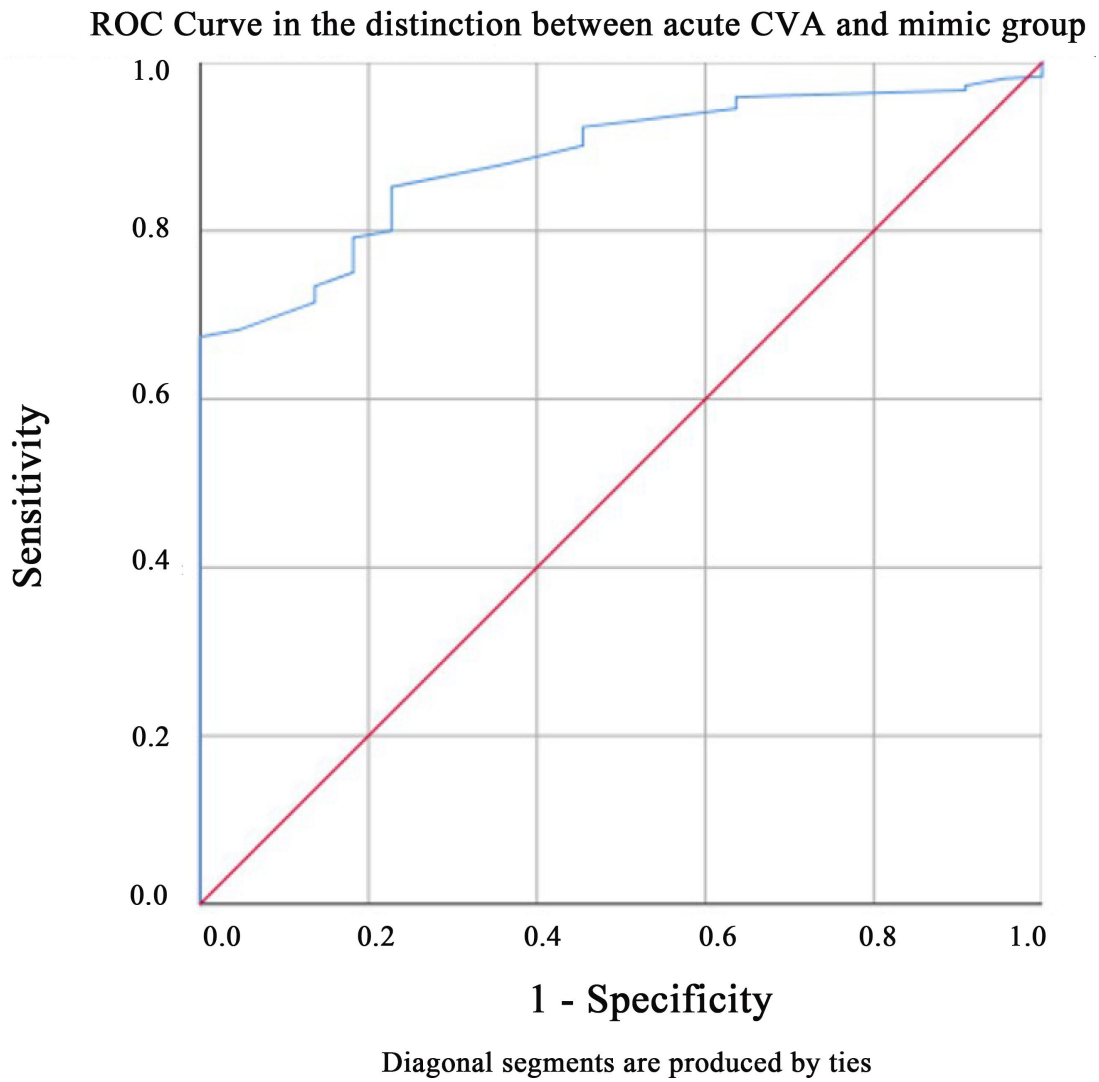

FIGURE 3. ROC curve analysis for the value of optic nerve sheath diameter sufficient in distinguishing between acute CVA and psychogenic mimic cases. ROC: receiver operating characteristic; CVA: cerebrovascular accident.

4. Discussion

This is the first clinical study to investigate the diagnostic value of ONSD for the differential diagnosis of ischemic and functional stroke mimics.

Patients with acute stroke are initiated on thrombolytic therapy after undergoing clinical assessment and cranial CT imaging in EDs. Therefore, patients with a clinically suspicious picture may need further investigations using magnetic resonance imaging (MRI). Although diffusion-weighted MRI is the most sensitive diagnostic tool for ischemic stroke, it is often not available in the ED and is unsuitable for meeting the door-to-needle time requirements for thrombolytic therapy [15].

Although stroke in patients presenting with clinical symptoms of acute stroke is detected based on medical history and physical examination after exclusion of hemorrhage and presence of mass using tomography, previous studies have reported that 2.8% to 24.8% of mimics, as reported in a large-scale review, receive a diagnosis of ischemic stroke and undergo thrombolytic therapy [15–17]. In a study conducted by Gargalas *et al*. [3], out of 1165 patients with stroke, 163 (14%) were identified as medical mimics and 98 (8.4%) as functional mimics [2]. In another study conducted in the Netherlands, 32 (5%) of the 669 patients admitted to the stroke unit after physical examination, CT and MRI were classified as having stroke mimics, 13 of whom were found to have conversion disorder [3]. In the present study, the rate of stroke mimics was 9.3%, with 5.62% being functional mimics. The stroke mimic rates in our study were consistent with those previously reported. We attribute the partial variations in rates between clinics to differences in expert evaluations, whether diffusion MRI was performed during the acute phase, and whether the clinics were stroke centers.

Multiple previous studies have reported that psychogenic stroke mimics are more common among women and tend to occur in younger age groups than in patients with true stroke [3, 5, 18]. Consistent with previous studies, the present study also found that the proportion of women was significantly higher in the psychogenic mimic group than in the CVA group ($p = 0.004$). However, no significant statistical difference was found between the groups in terms of mean age.

Numerous clinical studies have shown the diagnostic value of increased ONSD diameter secondary to intracranial hypertension in ischemic stroke cases [10, 13]. Batur *et al*. [19] reported that ONSD measurements are useful for both the diagnosis of ischemic stroke and the prediction of mortality in patients. Çömez *et al*. [20] reported that ONSD measurements can help diagnose stroke. Patel *et al*. [21] showed that ONSD was associated with mortality and poor functional activity in patients at 6 months. Similarly, in our study, we found that ONSD was higher in patients diagnosed with ischemic stroke. We attribute this increase in ONSD to increased ICP.

Although the mechanisms behind ICP elevation during seizures have not been elucidated previously, key factors include the Monro–Kellie hypothesis, molecular mechanisms of ICP and ictal activity, and increased blood flow during seizures because of depletion of energy reserves, all contributing to ICP elevation [22]. Another study conducted in patients with epilepsy found that ONSD increased in the first few hours in the postictal period after generalized tonic-clonic seizures and decreased over time [23]. Yılmaz *et al*. [24] found that bedside ONSD measurements, using ocular ultrasound effectively differentiated provoked seizures from unprovoked seizures. Demir *et al*. [25] reported a negative correlation between post-treatment ONSD and sodium levels at admission. In explaining this phenomenon, they argued that decreased sodium levels lead to increased cerebral edema and, thus, increased ICP [26]. A study of patients with migraine attacks showed that ONSD increased in patients with complicated migraine [27]. Based on the aforementioned evidence, patients with medical mimic stroke were excluded from the present study because they had increased ICP, which increases ONSD.

Furthermore, although no hemorrhagic complications were observed in patients with psychogenic mimics in our study, they received unnecessary thrombolytic therapy due to misdiagnosis.

In the present study, we found a significant difference in ONSD measurements between patients with acute CVA and psychogenic mimic stroke using brain CT. The absence of an increase in ONSD in patients with psychogenic stroke mimics was attributed to the lack of a pathology that would cause increased ICP due to psychogenic factors. Although psychogenic stroke mimics have distinct demographic and clinical characteristics, they may present with varying symptoms. Thus, patients with vascular stroke may present with similar symptoms, leading to missed diagnoses even after expert evaluations. The rate of stroke mimics is higher in stroke units [5].

The limitations of the present study include its retrospective design, single-center nature, and relatively small number of patients, which could introuduce bias and limit the generalizability of study findings. Multicenter prospective studies with larger cohorts are necessary to validate and generalize our results.

5. Conclusions

In patients presenting to the ED with suspected acute stroke, if ONSD is normal on brain CT, a more detailed history, neuropsychiatric examination and advanced neuroimaging should be performed before considering thrombolytic therapy because of the possibility of psychogenic stroke mimics. This issue requires further investigation to determine whether ONSD has a greater potential in detecting psychogenic stroke mimics.

ABBREVIATIONS

AUC, area under the curve; CT, computed tomography; CVA, cerebrovascular accident; ED, emergency department; MRI, magnetic resonance imaging; ONS, optic nerve sheath; ONSD, optic nerve sheath diameter; ROC, receiver operating characteristic; ICP, increased intracranial pressure; CI, confidence interval; CVE: Cerebrovascular Event.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this paper are available upon reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

İB—Conceptualization, Data curation, Investigation, Methodology, Supervision, Writing–original draft, Writing–review & editing. MD—Conceptualization, Data curation, Writing–review & editing. TÇ—Conceptualization, writing–original draft, Writing–review & editing. GA—Conceptualization, Writing–review & editing. KŞ—Conceptualization, Methodology, writing–original draft, Writing–review & editing. CK—Data curation, Formal analysis, Writing–original draft. FY—Conceptualization, Data curation, Writing–original draft. AB—Conceptualization, Data curation. MAE—Methodology, writing–original draft, Writing–review & editing.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Antalya Training and Research Hospital Clinical Research Ethics Committee (10/10/2024, Decision no: 15/4). The requirement for informed consent was waived by Antalya Training and Research Hospital Clinical Research Ethics Committee.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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