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Emergency assessment of vertigo using CT optic nerve sheath diameter: A novel approach to etiology differentiation and prognosis

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Abstract

Background: Differentiating central from peripheral vertigo in the emergency department (ED) remains a major diagnostic challenge, as early clinical findings frequently overlap. Computed tomography (CT) is widely used for initial imaging but has limited sensitivity for posterior fossa lesions. Measurement of the optic nerve sheath diameter (ONSD) on CT has emerged as a promising surrogate marker of intracranial pressure (ICP), yet its utility in vertigo patients has not been fully elucidated. *Methods:* We retrospectively analyzed 294 adult patients who presented to a tertiary ED with acute vertigo between January 2022 and December 2023. Patients were classified as central (n=119) or peripheral (n=175) vertigo based on clinical and radiological findings. ONSD was measured bilaterally at 3 mm behind the globe on axial CT images. Comparisons between groups and subgroups were performed using independent samples t-test, one-way ANOVA with Bonferroni post-hoc test, and Chi-square analysis. Associations with hospitalization and mortality were also assessed. *Results:* Mean ONSD values were significantly higher in central vertigo (5.13 ± 0.68 mm) than in peripheral vertigo (4.81 ± 0.41 mm, $p<0.001$). Among central subgroups, patients with infarction (5.18 ± 0.63 mm) and hemorrhage (5.22 ± 0.82 mm) exhibited increased diameters, whereas those with cerebellar masses demonstrated lower ONSD (4.62 ± 0.65 mm), closer to peripheral values. Right-sided lesions produced the largest diameters (5.27 ± 0.64 mm), followed by left-sided (5.00 ± 0.72 mm) and no lesion (4.81 ± 0.42 mm) groups ($p<0.001$). Hospitalized patients had higher ONSD (5.12 ± 0.67 mm) compared to discharged patients (4.80 ± 0.41 mm, $p<0.001$). Mortality cases (n=5) displayed markedly elevated values (6.24 ± 1.04 mm) versus survivors (4.92 ± 0.52 mm, $p<0.001$). *Conclusions:* CT-derived ONSD is a valuable adjunct for differentiating central from peripheral vertigo and may predict hospitalization and mortality. Importantly, cerebellar masses exhibited distinct ONSD behavior compared to acute vascular lesions, underscoring the need to consider pathophysiological heterogeneity in interpretation. Integration of ONSD into ED protocols may enhance risk stratification and clinical decision-making.

Keywords: Vertigo, emergency department, optic nerve sheath diameter (ONSD), computed tomography

INTRODUCTION

Vertigo is one of the most frequent neurological complaints in the emergency department (ED), accounting for approximately 2–3% of all ED visits worldwide.[1,2] While the majority of cases are benign and peripheral in origin, a considerable proportion of patients harbor central causes, including cerebellar infarction, hemorrhage, and mass lesions, which are associated with significant morbidity and mortality if not promptly recognized.[3,4] Differentiating central from peripheral vertigo in the acute setting is often challenging, as initial clinical presentations may overlap, and misdiagnosis can lead to delayed treatment and adverse outcomes.[5]

Neuroimaging is crucial in the diagnostic evaluation of vertigo. Computed tomography (CT) frequently serves as the primary imaging technique in the emergency department owing to its accessibility and rapidity; nevertheless, its sensitivity in detecting early ischemic changes or minor posterior fossa lesions is constrained.[6] In recent years, the measurement of optic nerve sheath diameter (ONSD) has emerged as a

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promising non-invasive indicator of intracranial pressure (ICP), due to the direct communication between the subarachnoid area surrounding the optic nerve and the cerebral compartment.[7] ONSD enlargement has been correlated with acute rises in ICP in various conditions, including traumatic brain injury, stroke, intracerebral hemorrhage, and hydrocephalus.[8-10]

Initial studies established the diagnostic and prognostic significance of ONSD in several clinical settings. For instance, ultrasonographic or CT-based ONSD measurements have been shown to correlate with invasive ICP monitoring and to predict outcomes in patients with large hemispheric infarction or subarachnoid hemorrhage.[8,11,12] However, research focusing on patients presenting with vertigo, specifically comparing ONSD values in central versus peripheral etiologies, remains limited. Furthermore, little is known about whether different central vertigo subtypes—such as cerebellar infarction, hemorrhage, or mass lesions—exhibit distinct ONSD profiles, despite their varied pathophysiological mechanisms.

Given the need for rapid and reliable differentiation of vertigo etiologies in the ED, investigating the role of CT-derived ONSD measurements may provide a valuable diagnostic adjunct. Therefore, the aim of the present study was to evaluate the utility of ONSD measured on cranial CT in distinguishing central from peripheral vertigo, to explore differences across central subtypes, and to assess the association of ONSD values with hospitalization and mortality outcomes.

METHOD

Study design and ethical considerations

This study was structured as a retrospective, observational, and cross-sectional inquiry at the Emergency Medicine Clinic at University of Health Sciences Bağcılar Training and Research Hospital, a prominent tertiary emergency department in Istanbul, Turkey. All procedures were conducted in compliance with the ethical norms established by the institutional and national research committees, as well as the 1964 Helsinki Declaration and its subsequent amendments. The local institutional ethics committee examined and approved the study protocol. To ensure confidentiality, patient identifiers were removed and only anonymized data were used. As no direct interventions were performed, the requirement for informed consent was waived due to the retrospective design of the study.

Study population

The study group comprised adult patients who presented to the emergency department with vertigo from January 1, 2022, to December 31, 2023. A total of 9,724 patients were assessed for dizziness-related issues during this period. *A priori* power analysis was conducted utilizing G*Power version 3.1.9.7 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany) to ascertain the minimum necessary sample size. Based on prior literature evaluating ONSD differences between central and peripheral vertigo populations, we assumed a moderate effect size (Cohen's d = 0.5; equivalent to $\eta^2 \approx 0.06$ for ANOVA). With a two-tailed alpha level of 0.05 and 95% confidence (power = 0.95), the analysis indicated that at least 119 patients with central vertigo and 175 with peripheral vertigo were required. The final study sample of 294 patients met these requirements, ensuring adequate statistical power to detect clinically meaningful differences in ONSD values between groups. The high patient volume and the multidisciplinary structure of our hospital, which routinely receives referrals for dizziness from neurology, otorhinolaryngology, and internal medicine departments, ensured a heterogeneous and representative study sample.

Inclusion and exclusion criteria

Patients were eligible if they were 18 years of age or older, had undergone cranial computed tomography (CT) during their ED visit, and had complete clinical, laboratory, and imaging data available in the hospital information system. Cases were excluded if they were younger than 18 years, had a history of trauma, did not undergo CT examination, or had incomplete medical records. This rigorous selection process was designed to maintain the homogeneity of the study cohort and minimize bias.

Data collection

Demographic and clinical parameters, including age, sex, diagnosis, mean arterial pressure, hospitalization status, and mortality outcomes, were collected. The primary variable of interest was the ONSD, measured bilaterally on CT scans. The mean ONSD value for each patient was calculated as the average of both eyes. These values were compared between central and peripheral vertigo groups and further analyzed across subgroups such as lesion laterality (right/left), hospitalization, and survival status.

ONSD measurement technique

Cranial CT images were retrieved and reviewed through the Picture Archiving and Communication System (PACS). Measurements were performed in the axial plane, at a distance of 3 mm posterior to the globe, perpendicular to the axis of the optic nerve. This location has been established as the most reliable point for detecting intracranial pressure (ICP) changes, as it reflects distension of the optic nerve sheath in direct communication with the subarachnoid space.[8,9,13] Both right and left optic nerves were measured, and the mean value was calculated. Measurements were independently performed by at least two observers experienced in neuroradiology, and discrepancies were resolved by consensus. Previous studies have validated CT-derived ONSD measurements against invasive ICP monitoring and demonstrated strong correlations, thereby supporting the reliability of this method.[8,10]

All cranial CT examinations were performed using a multidetector CT scanner (e.g., Siemens Somatom Definition, Siemens Healthineers, Erlangen, Germany) available in our emergency department. Images were acquired with 5-mm slice thickness for routine brain evaluation, and thin-slice reconstructions (1.0–1.5 mm) were generated in the orbital plane for accurate ONSD measurements. The typical acquisition parameters included a tube voltage of 120 kVp and an effective tube current of 200–250 mAs, with standard brain kernel reconstruction. Images were reviewed in a soft tissue window (window level: 40 HU; window width: 100 HU), which provided optimal visualization of the retrobulbar segment of the optic nerve. ONSD was measured bilaterally as recommended in previous studies.[8,9]

Statistical analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). For categorical variables, the frequencies and percentages were used to convey the data, whereas mean ± standard deviation (SD) was used to express the continuous variables. When comparing two groups, the independent samples t-test was utilized; when comparing several groups, the one-way analysis of variance (ANOVA) was utilized. In order to ascertain differences between groups, Bonferroni post-hoc testing was conducted when statistically significant differences were distinguished. Statistical significance was defined as having a p-value of less than 0.05.

RESULTS

A total of 294 patients were included in the study, with a mean age of 61.35 ± 16.24 years (range: 18–94). Of these, 143 (48.6%) were female and 151 (51.4%) were male. The most common diagnosis was peripheral vertigo (n=175, 59.5%), followed by cerebellar infarction (n=93, 31.6%), cerebellar hemorrhage (n=14, 4.8%), and cerebellar mass (n=12, 4.1%). Brain CT revealed no lesion in 175 (59.5%) cases, right-sided lesions in 55 (18.7%), and left-sided lesions in 64 (21.8%). Hospital admission was required in 126 patients (42.9%), and overall mortality was 1.7% (n=5) (Table 1). There was no significant difference in optic nerve sheath diameter (ONSD) between male and female patients for right, left, or mean values (all $p>0.05$). Similarly, age stratification did not reveal a significant difference (Table 1).

In contrast, a clear distinction was observed between peripheral and central vertigo. Patients with central vertigo had significantly larger ONSD values compared to those with peripheral vertigo (right: 5.12 ± 0.69 mm vs. 4.81 ± 0.42 mm, $p<0.001$; left: 5.14 ± 0.68 mm vs. 4.80 ± 0.43 mm, $p<0.001$). Mean ONSD was also higher in central vertigo (5.13 ± 0.68 mm vs. 4.81 ± 0.41 mm, $p<0.001$). The proportion of patients with ONSD ≥5 mm was significantly greater in the central vertigo group (Table 2). When subgroups of central vertigo were analyzed, cerebellar infarction (5.18 ± 0.63 mm) and cerebellar hemorrhage (5.22 ± 0.82 mm) exhibited significantly higher mean ONSD values compared to peripheral vertigo (4.81 ± 0.41 mm) (all $p<0.001$). In contrast, patients with cerebellar mass demonstrated lower ONSD measurements (4.62 ± 0.65 mm), similar to peripheral vertigo cases (Table 2).

The presence of a lesion on cranial CT was associated with higher ONSD values across all measurement sites. Patients with right-sided lesions had the largest diameters, with right ONSD measuring 5.27 ± 0.64 mm, compared to 5.00 ± 0.72 mm in patients with left-sided lesions and 4.81 ± 0.42 mm in those without lesions ($p<0.001$). Left ONSD measurements were likewise elevated in patients with right-sided lesions (5.12 ± 0.61 mm) and left-sided lesions (5.16 ± 0.73 mm) compared with lesion-negative cases (4.80 ± 0.43 mm, $p<0.001$). Analysis of mean ONSD values demonstrated a similar pattern, with averages of 5.19 ± 0.62 mm for right-sided lesions and 5.08 ± 0.72 mm for left-sided lesions, compared with 4.81 ± 0.41 mm for

Table 1: Baseline characteristics of the study population (n=294) and comparison of ONSD measurements by gender

			mean ± SD		
Age (year)			61.35 ± 16.24		
			n	**%**	
Gender	Female		143	48.6	
	Male		151	51.4	
Diagnose	Peripheral vertigo		175	59.5	
	Cerebellar infarction		93	31.6	
	Cerebellar hemorrhage		14	4.8	
	Cerebellar mass		12	4.1	
Lesion	No lesion		175	59.5	
	Lesion on the right		55	18.7	
	Lesion on the left		64	21.8	
Hospitalization	No		168	57.1	
	Yes		126	42.9	
Mortality	No		289	98.3	
	Yes		5	1.7	
Total			294	100	
		n(%)	**mean ± SD**	**t**	**p**
Right ONSD (mm)	Female	143(48.6)	4.94±0.59	0.182	0.856
	Male	151(51.4)	4.93±0.54		
Left ONSD (mm)	Female	143(48.6)	4.93±0.61	-0.360	0.719
	Male	151(51.4)	4.95±0.53		
ONSD (mm)	Female	143(48.6)	4.93±0.59	-0.091	0.928
	Male	151(51.4)	4.94±0.52		

Values are presented as mean ± SD or number (%). Comparisons of continuous variables between two groups were performed using the independent samples t-test.

patients without radiological findings (p<0.001) (Table 3).

Clinical outcomes showed a significant relationship with ONSD. Among patients who required hospitalization, mean ONSD was 5.12 ± 0.67 mm, which was significantly higher than the 4.80 ± 0.41 mm recorded in patients discharged directly from the emergency department (p<0.001). Outcome analysis by survival demonstrated that patients who died (n=5) had substantially higher ONSD values across all measurements. Mean ONSD in the mortality group was 6.24 ± 1.04 mm compared to 4.92 ± 0.52 mm in survivors (p<0.001). This difference was consistently observed in both right and left ONSD measurements, with right ONSD averaging 6.09 ± 0.95 mm in the mortality group versus 4.92 ± 0.54 mm in survivors, and left ONSD averaging 6.39 ± 1.14 mm in non-survivors compared with 4.91 ± 0.52 mm in survivors (p<0.001) (Table 4).

DISCUSSION

The present study is one of the few to evaluate the role of cranial CT–derived ONSD in differentiating central from peripheral vertigo in the emergency department. Our findings indicated that the mean ONSD was markedly elevated in central vertigo (5.13 ± 0.68 mm) in comparison to peripheral vertigo (4.81 ± 0.41 mm). Importantly, subgroup analyses revealed striking differences among central causes: cerebellar infarction (5.18 ± 0.63 mm) and cerebellar hemorrhage (5.22 ± 0.82 mm) were associated with elevated ONSD, whereas cerebellar mass lesions (4.62 ± 0.65 mm) showed values close to those of the peripheral group. Moreover, outcome-related analyses

Table 2: Comparison of ONSD measurements between peripheral and central vertigo groups and subgroup analyses according to vertigo etiology

		n(%)	mean ± SD	t	p
Right ONSD (mm)	Peripheral Vertigo	175(59.5)	4.81±0.42	-4.774	<0.001
	Central Vertigo	119(40.5)	5.12±0.69		
Left ONSD (mm)	Peripheral Vertigo	175(59.5)	4.80±0.43	-5.1274	<0.001
	Central Vertigo	119(40.5)	5.14±0.68		
ONSD (mm)	Peripheral Vertigo	175(59.5)	4.81±0.41	-5.105	<0.001
	Central Vertigo	119(40.5)	5.13±0.68		
		Peripheral Vertigo n(%)	**Central Vertigo n(%)**	**X2**	**p**
Right ONSD	<5 mm	101(57.7)	49(41.2)	7.752	0.005
	>5 mm	74(42.3)	70(58.8)		
Left ONSD	<5 mm	118(67.4)	52(43.7)	16.357	<0.001
	>5 mm	57(32.6)	67(56.3)		
ONSD	<5 mm	101(57.7)	53(44.5)	4.930	0.026
	>5 mm	74(42.3)	66(55.5)		
		n(%)	**mean ± SD**	**F**	**p**
Right ONSD (mm)	Peripheral vertigo	175(59.5)	4.81±0.42a	13.236	<0.001
	Cerebellar infarction	93(31.6)	5.18±0.65b		
	Cerebellar hemorrhage	14(4.8)	5.24±0.81b		
	Cerebellar mass	12(4.1)	4.55±0.66a		
Left ONSD (mm)	Peripheral vertigo	175(59.5)	4.80±0.43a	12.545	<0.001
	Cerebellar infarction	93(31.6)	5.19±0.63b		
	Cerebellar hemorrhage	14(4.8)	5.20±0.86b		
	Cerebellar mass	12(4.1)	4.70±0.68a		
ONSD (mm)	Peripheral vertigo	175(59.5)	4.81±0.41a	13.212	<0.001
	Cerebellar infarction	93(31.6)	5.18±0.63b		
	Cerebellar hemorrhage	14(4.8)	5.22±0.82b		
	Cerebellar mass	12(4.1)	4.62±0.65a		

Values are presented as mean ± SD or number (%). Continuous variables were compared with the independent samples t-test (two groups) or one-way analysis of variance (ANOVA) with Bonferroni post-hoc test (multiple subgroups). Groups sharing the same superscript letter are not significantly different (Bonferroni post-hoc, $p < 0.05$) Categorical comparisons (<5 mm vs >5 mm thresholds) were evaluated using the Chi-square test.

indicated that patients requiring hospitalization had higher mean ONSD (5.12 ± 0.67 mm vs 4.80 ± 0.41 mm in non-admitted), and those who died had markedly elevated values (6.24 ± 1.04 mm vs 4.92 ± 0.52 mm in survivors). These findings provide both diagnostic and prognostic insights and highlight important nuances in interpreting ONSD in vertigo populations.

ONSD is a well-established non-invasive surrogate marker of ICP. The optic nerve sheath is continuous with the intracranial subarachnoid space; therefore, acute rises in ICP are transmitted directly to the sheath, producing measurable distension.[14,15] This mechanism explains the elevated ONSD observed in acute cerebellar infarction and hemorrhage in our cohort. Our results are consistent with recent prospective stroke studies demonstrating ONSD elevations in acute ischemic and hemorrhagic subtypes, with mean values exceeding 5.5 mm in patients with large infarctions or hemorrhages.[16,17] In particular, Lee *et al*. reported that thin-slice CT ONSD and ONSD/eyeball transverse diameter (ETD) ratios predicted poor functional outcomes in ischemic

Table 3: Comparison of ONSD measurements according to lesion laterality on cranial CT

	Lesion / ONSD	n	mean ± SD	F	p
Right ONSD (mm)	No Lesion	175	4.81±0.42a	15.485	<0.001
	Lesion Right	55	5.27±0.64b		
	Lesion Left	64	5.00±0.72b		
Left ONSD (mm)	No Lesion	175	4.80±0.43a	13.944	<0.001
	Lesion Right	55	5.12±0.61b		
	Lesion Left	64	5.16±0.73b		
ONSD (mm)	No Lesion	175	4.81±0.41a	13.759	<0.001
	Lesion Right	55	5.19±0.62b		
	Lesion Left	64	5.08±0.72b		

		No Lesion		Lesion Right		Lesion Left		X^2	p
		n	%	n	%	n	%		
Right ONSD	<5 mm	101	57.7	17	30.9	32	32	12.066	0.002
	>5 mm	74	42.3	38	69.1	32	32		
Left ONSD	<5 mm	118	67.4	23	41.8	29	29	16.505	0.001
	>5 mm	57	32.6	32	58.2	35	35		
ONSD	<5 mm	101	57.7	23	41.8	30	30	5.234	0.073
	>5 mm	74	42.3	32	58.2	34v	34		

Values are presented as mean ± SD or number (%). Continuous data were compared using one-way ANOVA with Bonferroni post-hoc test. Groups sharing the same superscript letter are not significantly different (Bonferroni post-hoc, $p < 0.05$). Categorical group comparisons were performed with the Chi-square test.

stroke, underscoring the clinical utility of accurate ONSD measurements.[11] Similarly, Zhu *et al*. demonstrated that an ONSD >5.7 mm or ONSD/ETD >0.25 was associated with higher in-hospital mortality in comatose stroke patients.[18] These results are consistent with our mortality subgroup findings, where mean ONSD was 6.24 mm.

The most intriguing finding of our study was the cerebellar mass subgroup, where mean ONSD (4.62 ± 0.65 mm) was significantly lower than in infarction or hemorrhage, and nearly indistinguishable from peripheral vertigo. This discrepancy reflects fundamental differences in pathophysiology. Cerebellar tumors usually progress over weeks to months, allowing compensatory mechanisms—CSF redistribution, enhanced absorption, venous compliance, and cranial elastance—to delay increases in ICP (19). Consequently, ONSD may remain within the normal range until decompensation occurs. In line with this, Kalim *et al*. reported only modest ONSD elevations in posterior fossa tumors preoperatively, with significant increases seen only in patients with hydrocephalus or peritumoral edema, and postoperative regression of ONSD after resection.[20] Among posterior fossa tumors, Kanjanakangwankul *et al*. demonstrated that tumor volume alone is not sufficient to predict hydrocephalus; peritumoral vasogenic edema and indicators of ventricular obstruction were also significant predictors.[19] Thus, our findings reinforce that not all central lesions cause ONSD enlargement, and clinicians should avoid interpreting normal ONSD as excluding central pathology. This nuance has substantial clinical implications: a patient with cerebellar mass may present with normal ONSD yet still harbor a life-threatening lesion.

From a clinical standpoint, our results support incorporating ONSD into emergency diagnostic pathways for vertigo. Elevated ONSD values (>5.0 mm on CT) should raise suspicion for acute central causes such as infarction or hemorrhage. Moreover, our finding that hospitalized patients and especially those with fatal outcomes had higher ONSD values (up to 6.24 mm) suggests prognostic potential, consistent with prior reports linking ONSD to poor outcomes in stroke and traumatic brain injury.[18,21] However, our data also caution that normal ONSD does not exclude central lesions, particularly slowly progressive tumors. In these cases, ONSD should be interpreted in conjunction with clinical presentation, neurologic examination,

Table 4: Comparison of ONSD measurements according to hospitalization and mortality outcomes

	Hospitalization	n(%)		mean ± SD		t	p
Right ONSD (mm)	No	168(57.1)		4.81±0.41		-4.701	<0.001
	Yes	126(42.9)		5.11±0.68			
Left ONSD (mm)	No	168(57.1)		4.79±0.43		-5.291	<0.001
	Yes	126(42.9)		5.13±0.67			
ONSD (mm)	No	168(57.1)		4.80±0.41		-5.076	<0.001
	Yes	126(42.9)		5.12±0.67			
		No		**Yes**		**X2**	**p**
		n	**%**	**n**	**%**		
Right ONSD	<5 mm	98	58.3	52	41.3	8.389	0.004
	>5 mm	70	41.7	74	58.7		
Left ONSD	<5 mm	115	68.5	55	43.7	18.160	<0.001
	>5 mm	53	31.5	71	56.3		
ONSD	<5 mm	98	58.3	56	44.4	5.568	0.018
	>5 mm	70	41.7	70	55.6		
	Mortality	**n(%)**		**mean ± SD**		**t**	**p**
Right ONSD (mm)	No	289(98.3)		4.92±0.54		-4.779	<0.001
	Yes	5(1.7)		6.09±0.95			
Left ONSD (mm)	No	289(98.3)		4.91±0.52		-6.119	<0.001
	Yes	5(1.7)		6.39±1.14			
ONSD (mm)	No	289(98.3)		4.92±0.52		-5.529	<0.001
	Yes	5(1.7)		6.24±1.04			
		No		**Yes**		**X2**	**p**
		n	**%**	**n**	**%**		
Right ONSD	<5 mm	149	51.6	1	20.0	1.959	0.162
	>5 mm	140	48.4	4	80.0		
Left ONSD	<5 mm	169	58.5	1	20.0	2.984	0.084
	>5 mm	120	41.5	4	80.0		
ONSD	<5 mm	153	52.9	1	20.0	2.138	0.144
	>5 mm	136	47.1	4	80.0		

Values are presented as mean ± SD or number (%). Continuous variables were compared with the independent samples t-test. Categorical group comparisons were analyzed using the Chi-square test.

and neuroimaging findings such as edema or hydrocephalus.

Several methodological issues merit discussion. CT slice thickness influences measurement accuracy; thin-slice (0.6–0.75 mm) reconstructions yield better discrimination than routine 4–5 mm slices.[11] The use of the ONSD/ETD ratio has been recommended to adjust for interindividual variation in globe size.[11,22] In our study, only absolute ONSD was available, but future work should incorporate ratio metrics. Inter-observer variability is another concern; prior studies report intraclass correlation coefficients of 0.75–0.9 with standardized training, but lower without it.[23] Furthermore, different modalities (CT, ultrasound, MRI) may yield systematically different values; while CT is widely available in EDs, ultrasound permits bedside monitoring and may detect dynamic changes in ICP.[10] Harmonization across modalities remains an unmet need.

Our study also adds to the literature on prognostic applications of ONSD. Elevated values in our hospitalized and deceased subgroups parallel findings in large hemispheric infarction and malignant MCA infarction, where ONSD predicts herniation risk and mortality.[17,18] Notably, our mortality subgroup's mean ONSD of 6.24 mm lies well above established thresholds for intracranial hypertension, reinforcing the strong association between severe ICP elevation and fatal outcome. Thus, ONSD measurement in vertigo patients may offer not only diagnostic but prognostic guidance in acute care.

This study has several limitations. First, it is retrospective and thus subject to selection bias. Second, subgroup sizes, particularly for cerebellar masses, were small, limiting statistical power. Third, ONSD was measured only on CT, not ultrasound or MRI, and only at a single time point. Fourth, we did not correlate ONSD directly with invasive ICP monitoring, the gold standard. Finally, CT slice thickness and inter-observer variability may have influenced absolute values. Furthermore, although ONSD measurements were performed by consensus, observers were not blinded to clinical or outcome data, which may have introduced bias; future studies should include blinded readers and report inter-observer reliability using ICC.

Future research should focus on prospective studies including larger numbers of cerebellar masses, with serial ONSD measurements before and after interventions, to clarify temporal dynamics. The integration of ONSD/ETD ratio, multimodal imaging, and clinical variables into risk stratification models may enhance diagnostic and prognostic accuracy. Furthermore, standardized measurement protocols are essential to reduce variability and facilitate broader clinical implementation.

In addition, although inter-observer consensus was used for ONSD measurements in this study, future investigations should report inter-rater reliability using intraclass correlation coefficients (ICC) to further validate the reproducibility of CT-based ONSD assessment. Another promising avenue is the application of receiver operating characteristic (ROC) curve analyses to derive etiology-specific cut-off values for differentiating central subtypes, such as infarction, hemorrhage, and cerebellar mass lesions. Establishing reliable thresholds may enhance the diagnostic accuracy and clinical integration of ONSD into emergency protocols.

In conclusion, our study demonstrates that CT-measured ONSD can differentiate central from peripheral vertigo and has prognostic relevance in terms of hospitalization and mortality. Elevated ONSD values in cerebellar infarction and hemorrhage reflect acute ICP elevation, whereas near-normal values in cerebellar masses highlight the limitations of ONSD in slowly progressive lesions. Clinicians should therefore interpret ONSD in the context of clinical presentation and imaging findings rather than as a stand-alone marker. These insights expand the role of ONSD as a valuable adjunct in emergency evaluation of vertigo while underscoring the need for nuanced, etiology-aware interpretation.

DISCLOSURE

Ethical approval: The study was approved by the institutional review board (Date:26/07/2024, Decision No:2024/07/08/061).

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