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The predictive value of optic nerve sheath diameter measurement via ultrasound for intracerebral hemorrhage complicated by cerebral-cardiac syndrome

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Abstract

Objective This study aims to evaluate the clinical significance of ultrasound-based measurement of optic nerve sheath diameter (ONSD) in predicting intracerebral hemorrhage (ICH) complicated by cerebral-cardiac syndrome (CCS).

Methods Patients with ICH and who were treated in the intensive care unit (ICU) at Shijiazhuang People's Hospital between October 2021 and November 2022 were included in this study. Participants were divided into two groups: those with CCS and those without. Various clinical parameters, including sex, age, electrocardiogram (ECG) findings, myocardial markers, B-type natriuretic peptide (BNP) levels, Glasgow Coma Scale (GCS) score, ONSD, hematoma volume, and midline shift, were assessed. A binary logistic regression model and receiver operating characteristic (ROC) curve analysis were employed to determine the predictive value of each risk factor for ICH complicated by CCS.

Results ONSD measurements differed significantly between males and females, with males exhibiting larger ONSD values. Additionally, significant differences were observed in ONSD, hematoma volume, midline shift, and GCS scores between the CCS and non-CCS groups. A direct correlation was identified between ONSD and both hematoma volume and midline shift. Multiple regression analysis demonstrated that ONSD, hematoma volume, and GCS score are independent risk factors for predicting ICH complicated by CCS. ROC curve analysis for ONSD in predicting ICH with CCS revealed an area under the curve (AUC) of 0.80, with an optimal cutoff value of 5.88 cm, yielding a sensitivity of 83% and a specificity of 79%. When ONSD, hematoma volume, and GCS score were combined, the predictive accuracy improved, with an AUC of 0.880.

Conclusion Males tend to have larger ONSD measurements compared to females. Ultrasound is a valuable tool for measuring ONSD, comparable to computed tomography, and is useful in detecting intracranial hypertension and mass effect. ONSD, hematoma volume, and GCS score are independent predictors of ICH complicated by CCS, and their combined use enhances predictive accuracy.

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Introduction

Intracerebral hemorrhage (ICH) is characterized by the rupture of the intracranial blood vessel due to non-traumatic causes, leading to the direct extravasation of blood into the brain parenchyma and the formation of a localized hematoma. In some cases, the hemorrhage may extend into the ventricles and subarachnoid space, resulting in neurological deficits and cognitive impairments [1–3]. ICH is associated with high mortality and disability rates, with mortality ranging from 35 to 52% within 30 days of onset. Furthermore, both the incidence and mortality of ICH increase with advancing age [4].

Research conducted in China and other countries indicates that cerebral conditions such as ICH, cerebral infarction, acute craniocerebral trauma, and subarachnoid hemorrhage (SAH) may lead to secondary cardiovascular dysfunction or cardiac injury. These secondary effects may present as subendocardial hemorrhage, arrhythmias, myocardial ischemia, or heart failure, even in the absence of preexisting cardiac diseases. This phenomenon is referred to as cerebral-cardiac syndrome (CCS) [5]. CCS is common among individuals with cerebrovascular diseases [5–7] and often complicates their clinical course, leading to a poorer prognosis [8].

The diagnosis of CCS primarily relies on cardiac markers such as electrocardiogram (ECG) abnormalities, elevated troponin levels, cardiac enzymes, and color echocardiography. However, given the overlap between the risk factors for ICH and those for cardiovascular diseases, individuals with ICH and a history of cardiac disease may present with symptoms and risk factors related to both cerebral and cardiac conditions. As a result, cardiac impairments secondary to craniocerebral pathologies may be overlooked. Therefore, in addition to traditional cardiovascular indicators, it is crucial to identify alternative markers that can enable the timely diagnosis and treatment of CCS.

Some researchers in China have suggested that the occurrence of CCS may be linked to a significant increase in intracranial pressure (ICP) and excessive stimulation of the cardiac sympathetic nervous system [9]. They propose a correlation between elevated ICP and the onset of CCS, prompting interest in non-invasive methods for monitoring ICP. Among these methods, ultrasound measurement of optic nerve sheath diameter (ONSD) has gained attention, demonstrating a sensitivity of 95% and a specificity of 92% in assessing ICP, highlighting its potential utility in clinical practice [10]. While computed tomography (CT) and magnetic resonance imaging (MRI) are also capable of evaluating ONSD and ICP [10, 11], ultrasound offers several distinct advantages, including ease of use, repeatability, bedside application, safety, affordability, and the absence of radiation exposure.

Currently, limited literature exists on the use of ultrasound-based ONSD measurements to predict CCS. This study aims to address this gap by analyzing ONSD in patients with ICH, comparing those with CCS (CCS group) to those without CCS (non-CCS group). The objective is to assess the significance of ONSD as a predictive marker for ICH complicated by CCS.

Materials and methods

Material

Study participants

A total of 100 patients with ICH and admitted to Shijiazhuang People's Hospital between October 2021 and November 2022 were initially identified as potential candidates for this study. Following a screening process, 82 patients with confirmed ICH based on CT scans were included.

Inclusion and exclusion criteria of patients with ICH

Inclusion criteria

The diagnosis of ICH was based on the Chinese Guidelines for the Diagnosis and Treatment of Intracerebral Hemorrhage (2019) [12]. The inclusion criteria were as follows:

(1) Acute onset of symptoms.
(2) Presence of localized neurological deficits (occasionally comprehensive neurological deficits), often accompanied by headache, vomiting, elevated blood pressure, and varying degrees of impaired consciousness.
(3) Evidence of hemorrhage on head CT or MRI.
(4) Exclusion of non-vascular causes of cerebral hemorrhage.

Patients were included if the onset of disease occurred within 72 h or less prior to enrollment. Informed consent was obtained from all participants or their families. The study protocol was approved by the hospital's ethics committee.

Exclusion criteria

Exclusion criteria were as follows:

(1) ICH attributed to cerebral organic pathologies such as tumors, SAH, or trauma.
(2) History of cardiac diseases, severe liver or kidney dysfunction, thyroid disorders, electrolyte imbalances, or infectious diseases prior to hospitalization.
(3) History of eye trauma, surgery, tumors, or other ophthalmic diseases. Patients with significant differences in ONSD between the eyes were also excluded.
(4) Poor visualization of the optic nerve sheath or artifacts during ultrasonography.
(5) Inability to cooperate with the examination due to restlessness or other factors.
(6) Refusal to provide informed consent.

Criteria for diagnosing CCS

The diagnosis of CCS was based on the following criteria [13]:

(1) Absence of a prior history of heart disease.
(2) Confirmed diagnosis of ICH.
(3) Presence of secondary cardiac damage, manifesting as one or more of the following: (i) Electrocardiographic changes, including ST segment elevation or depression, T wave abnormalities, or a prolonged QT interval [14]. (ii) Echocardiographic evidence of impaired left ventricular diastolic function, abnormal ventricular wall motion, reduced left ventricular ejection fraction, or other cardiac functional abnormalities. (iii) Elevated peripheral levels of cardiac markers, such as troponin [15], creatine kinase-MB (a myocardial enzyme), and B-type natriuretic peptide (BNP) [16].

Criteria for case dropout or termination

Patients were withdrawn from the study if they:

(1) Were unresponsive to follow-up or had incomplete data.
(2) Chose to withdraw from the study, as expressed by themselves or their families.

Study methods

General data collection

Data collection for all participants was conducted on the day of admission and included the following parameters:

(1) General Data: Information regarding the sex and age of the patients was recorded.
(2) Medical History: The presence of hypertension and diabetes mellitus was documented.
(3) Clinical Characteristics: The Glasgow Coma Scale (GCS) score was assessed upon admission. The location of the cerebral hemorrhage was identified, and the hematoma volume was calculated. For patients with regular hematomas, the volume was determined using the formula: length × width × depth / 2. For patients with irregular hematomas, the formula length × width × depth / 3 was applied [17]. The midline shift was evaluated by measuring the displacement of the transparent interphase relative to the ideal midline on CT scans.
(4) Cardiac Indicators: Cardiac data collected included ECG results at admission, levels of myocardial enzymes, serum troponin, BNP, and echocardiographic findings.
(5) Inflammatory Indicators: Inflammatory markers, including interleukin-6 (IL-6), serum amyloid A (SAA), C-reactive protein (CRP), and procalcitonin (PCT), were measured upon admission.
(6) Other Data: ONSD measurements were recorded.

Measuring ONSD using ultrasound

Before undergoing ONSD measurements, all patients refrained from taking diuretics or receiving surgical intervention. The healthcare professionals conducting the measurements had been trained in bedside ultrasound techniques for ONSD assessment. The Philips CX50 portable color Doppler ultrasound machine was used for the measurements.

The procedure for measuring ONSD was as follows:

- The patient was positioned supine with the head aligned centrally.
- Transparent eye patches were applied to protect the eyes.
- An appropriate amount of coupling gel was applied to the ultrasound probe to avoid exerting excess pressure on the eyeball.
- The probe was placed gently on the eyelid and adjusted both vertically and horizontally to obtain the optimal imaging plane, ensuring clear visualization of the eyeball structures, optic nerve, and blood vessels.
- The ONSD was measured 3 mm posterior to the eyeball, perpendicular to the optic nerve's axis, using an electronic caliper.
- Measurements were taken twice in both the cross-sectional and sagittal planes of each eye, with the process repeated eight times.
- The average ONSD was then calculated based on these eight measurements.

Statistical methods

Data analysis was performed using SPSS version 25.0. All continuous variables were initially tested for normality. Data that followed a normal distribution are presented as mean ± standard deviation (SD), and comparisons between groups were made using the t-test. For non-normally distributed variables, data are expressed as median and interquartile range (M [IQR]), with group comparisons analyzed using the Mann–Whitney U test. Categorical variables are represented as counts and percentages (n [%]), and comparisons between groups were conducted using the chi-squared test.

Variables that showed statistically significant differences were included in a binary logistic regression model to identify independent risk factors for the development of CCS following ICH. The predictive accuracy of each variable for ICH complicated by CCS was assessed using receiver operating characteristic (ROC) curve analysis. A significance level of $\alpha = 0.05$ was established, with P-values less than 0.05 considered statistically significant. Additionally, the correlation between ONSD and both hematoma volume and midline shift was evaluated using Spearman's rank correlation coefficient.

Results

1. General characteristics of study participants

Initially, 100 patients were considered for the study, and after applying the predefined exclusion criteria, 82 patients were enrolled. Of these, 55 were males (67.1%) and 27 were females (32.9%), with an average age of 59.8 ± 14.7 years (Table 1).

2. Comparison of clinical indicators between the CCS and non-CCS groups

Among the 82 patients with ICH, 54 developed CCS, resulting in an incidence rate of 65.9% (54/82). No statistically significant differences were found between the CCS and non-CCS groups in terms of age, sex, location of hemorrhage, frequency of hydrocephalus, presence of diabetes mellitus or hypertension, or levels of CRP, PCT, SAA, and IL-6 ($P > 0.05$). However, ONSD, hematoma volume, midline shift, and GCS scores differed significantly between the groups ($P < 0.05$) (Table 2).

3. Comparison of ONSD by sex and age

A significant difference in ONSD was observed between males and females, with males exhibiting larger ONSD values ($P < 0.05$). No statistically significant differences in ONSD were observed across different age groups within either the CCS or non-CCS group ($P > 0.05$) (Table 3).

Table 1 General data of the study participants

Age (Years)	**59.77 ± 14.66**
Male/Female (n)	55/27
High blood pressure (n [%)]	56.1
Diabetes mellitus (n [%)]	15.9
Site of intracerebral hemorrhage (ICH) (n [%)]	100
Cerebral lobe	32.9
Cerebellum	2.4
Brainstem	4.9
Thalamus	12.2
Basal ganglia	47.6
CRP [mg/L, (M(Q)]	4.73 (49.71)
PCT [ng/ml, (M(Q)]	0.1 (0.24)
IL-6 [ng/L, (M(Q)]	54.07 (108.75)
SAA [mg/L, (M(Q)]	5 (63.02)
Glasgow Coma Scale (GCS) score (M [Q])	6 (3)
ONSD [mm, (M(Q)]	5.84 ± 0.64
Midline shift (cm, M [Q])	0.4 (0.6)
Hematoma volume (cm, M [Q])	44.5 (43.5)

4. Correlation between ONSD and hematoma volume and midline shift

NSD measured via ultrasound showed a positive linear correlation with hematoma volume ($r = 0.541$, $P < 0.000$) and midline shift ($r = 0.428$, $P < 0.000$) as measured by CT (Figs. 1 and 2).

5. Independent risk factors for ICH complicated by CCS

The variables that showed statistically significant differences—ONSD, hematoma volume, midline shift, and GCS score—were included in a binary logistic regression analysis. Collinearity analysis revealed no significant interactions between the variables. The regression analysis identified ONSD, hematoma volume, and GCS score as independent risk factors for the development of CCS in patients with ICH ($P < 0.05$) (Table 4).

6. Accuracy of ONSD in predicting ICH complicated by CCS

The predictive value of ONSD in identifying CCS among patients with ICH was assessed using the ROC curve. The area under the curve (AUC) was 0.802 (95% confidence interval [CI]: 0.708–0.895, $P < 0.000$), with a sensitivity of 83% and specificity of 79%. The optimal

Table 2 Comparison of clinical data between the two groups of patients with ICH

Indicators	Patients with ICH		χ^2/Z/t value	*P* value
	CCS group	Non-CCS group		
Age (Years)	59.61 ± 15.79	61.14 ± 12.34	0.609	0.544
Male/Female (n)	39/15	16/12	1.898	0.168
High blood pressure [n (%)]	33 (61.1)	13 (46.4)	0.432	0.511
Diabetes mellitus [n (%)]	12 (22.2)	6 (27.3)	0.412	0.748
Site of intracerebral hemorrhage (ICH) [n (%)]	54 (100)	28 (100)	1.002	0.980
Cerebral lobe	18 (33.3)	9 (32.1)		
Cerebellum	1 (1.9)	1 (3.6)		
Brainstem	3 (5.6)	1 (3.6)		
Thalamus	6 (11.1)	4 (14.3)		
Basal ganglia	26 (48.1)	13 (46.4)		
Hydrocephalus	2 (3.2)	1 (3.6)	0.0094	0.923
CRP (mg/L)	1.95 (24.71)	1.65 (22.98)	−0.998	0.318
PCT (ng/ml)	0.07 (0.24)	0.05 (0.11)	−0.841	0.4
IL-6 (ng/L)	25.60 (118.94)	25.15 (35.47)	−1.828	0.072
SAA (mg/L)	5 (60.21)	5 (135.72)	−0.963	0.336
Glasgow Coma Scale (GCS) score	6 (4)	8 (4)	−3.219	0.001*
ONSD (mm)	6.07 ± 0.56	5.39 ± 0.53	−5.292	0.000*
Midline shift (cm)	0.45(0.9)	0.4(0.5)	−2.931	0.003*
Hematoma volume (cm^3)	61.07 ± 34.33	28.39 ± 16.87	−5.778	0.000*
Surgical intervention *[n (%)]*	39 (72.2)	21 (75)	0.072	0.788

*Indicates statistical significance ($P < 0.05$)

Table 3 Comparison of ONSD across different sexes and age groups

		ONSD	t	*P*
Sex	Male	5.96 ± 0.65	−0.314	0.754
	Female	5.58 ± 0.54		
Age (Years)	< 65	5.82 ± 0.64	−2.638	0.010*
	≥ 65 years	5.87 ± 0.65		

*Indicates statistical significance ($P < 0.05$)

cutoff value for ONSD in predicting CCS was determined to be 5.88 cm (Fig. 3).

7. Combined predictive efficiency of ONSD, hematoma volume, and GCS score

Logistic regression analysis identified ONSD, hematoma volume, and GCS score as independent predictors for CCS. The predictive performance of each variable individually was compared to the combined predictive power of all three variables. Individually, ONSD, hematoma volume, and GCS score had AUC values of 0.802 (95% CI: 0.708–0.895, $P < 0.001$), 0.808 (95% CI: 0.714–0.901, $P < 0.001$), and 0.716 (95% CI: 0.600–0.832, $P < 0.001$), respectively. When these indicators were combined, the AUC increased to 0.880 (95% CI: 0.807–0.953, $P < 0.001$), indicating that the combination of ONSD, hematoma volume, and GCS score provided the most accurate prediction for the development of CCS in patients with ICH (Table 5; Fig. 4).

Discussion

Traditionally, mortality following strokes has predominantly been attributed to neurological deficits. However, emerging research suggests that cardiovascular complications play a significant role in stroke-related mortality [6, 18]. Craniocerebral conditions can directly or indirectly impair cardiac function, resulting in arrhythmias, cardiac dysfunction, heart failure, and other complications. Cardiac impairments following ICH are associated with early mortality and poor outcomes [19–22]. Previous studies have reported that the incidence and mortality rates in patients with CCS are eight and four times higher, respectively, compared to those without CCS [23].

Patients who develop CCS often present with various clinical manifestations, including ECG changes, abnormal myocardial enzyme levels, elevated BNP, and other cardiac anomalies. Among these, ECG changes are considered the most common and sensitive markers. Reports indicate that 32–75% of patients with stroke without pre-existing cardiac conditions exhibit ECG change

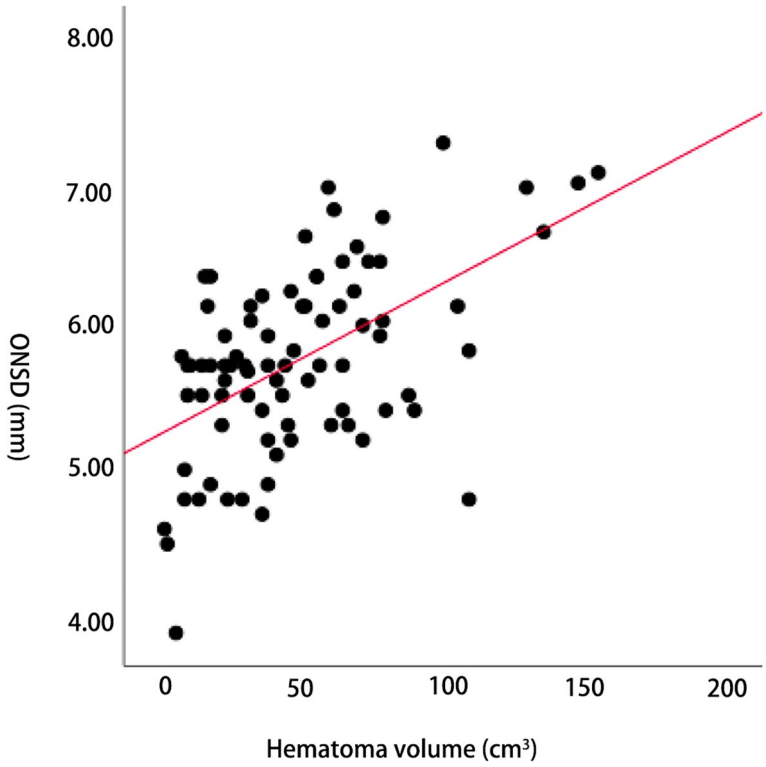

Fig. 1 ONSD and hematoma volume

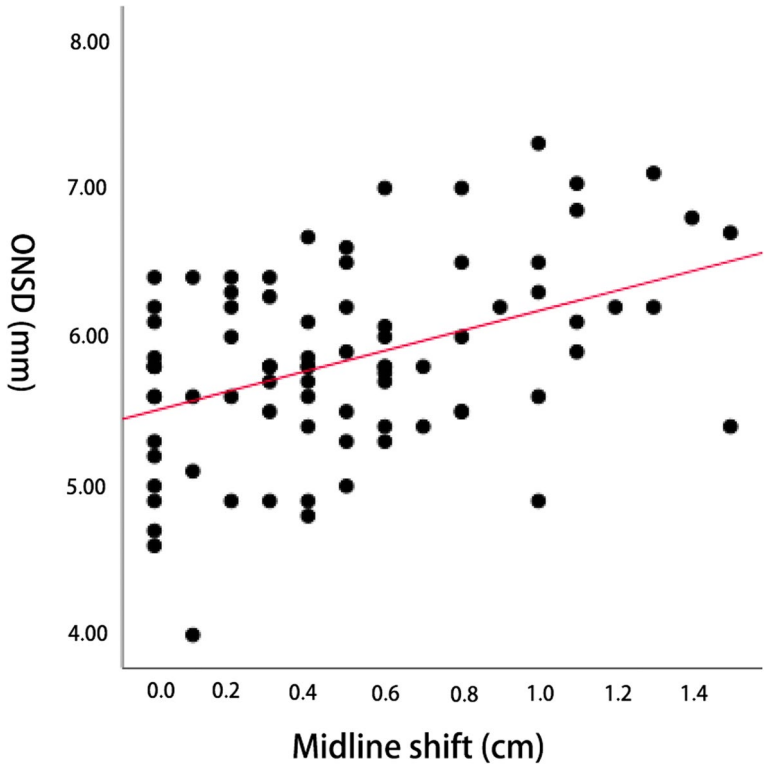

Fig. 2 ONSD and midline shift

Table 4 Analysis of independent risk factors for the occurrence of CCS in patients with ICH

	OR	95%CI	*P* value
ONSD	5.865	1.287–26.729	0.022*
Midline shift (cm)	0.837	0.083–8.407	0.880
Hematoma volume (cm^3)	1.055	1.015–1.096	0.007*
Glasgow Coma Scale (GCS) score	0.724	0.534–0.981	0.037*

*Indicates statistical significance ($P < 0.05$)

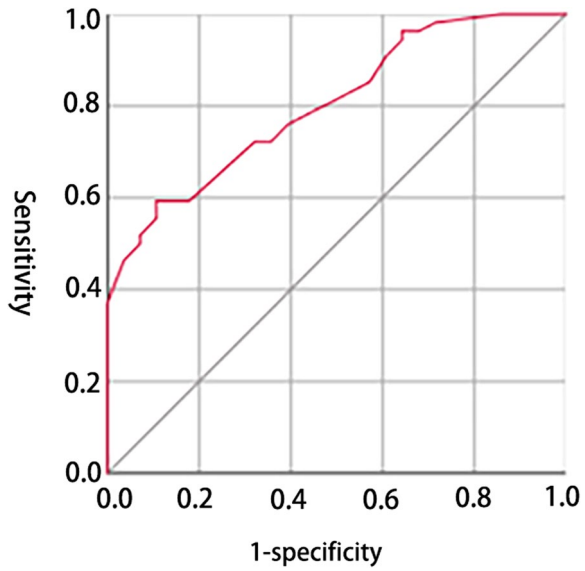

Fig. 3 ROC curve of ONSD in predicting CCS

[24]. Left ventricular dysfunction is frequently observed in patients with acute ischemic stroke (AIS) [25], ICH [25], or SAH [26]. Common findings in patients with stroke include reduced left ventricular ejection fraction and diastolic dysfunction. Approximately 20–40% of patients with ICH or SAH have elevated troponin levels [27, 28]. Both patients with elevated troponin levels and those without may experience left ventricular dysfunction. Furthermore, elevated troponin levels are associated with reduced ejection fraction and abnormalities in ventricular wall motion [28].

Myocardial enzyme abnormalities typically manifest within three days following craniocerebral trauma, with elevated creatine kinase-MB levels being the most frequently observed [9]. Previous studies have reported an incidence rate of CCS in acute stroke patients ranging from 62 to 90% [29]. In the present study, 82 patients with ICH were included after excluding those with pre-existing cardiac conditions. Of these, 54 patients developed CCS, resulting in an incidence rate of 65.9% (54/82). Among the CCS group, 46 patients exhibited ECG changes, 33 had abnormal myocardial enzyme levels, 18 had elevated troponin levels, 22 showed increased BNP levels, and 19 demonstrated echocardiographic abnormalities.

Existing literature suggests that ECG changes may be associated with the type of acute cerebrovascular disease, the location of the lesion, and the severity of the condition [30]. However, in this study, no statistically significant differences were observed in the location of ICH between the CCS and non-CCS groups. This indicates that the site of hemorrhage may not significantly influence the development of secondary CCS in this cohort. Nevertheless, this study had several limitations, including a relatively small sample size and varying severity of cerebral trauma within the cohort. Larger studies are needed to further investigate the relationship between lesion location and the development of secondary CCS.

The mechanism underlying the concurrent occurrence of ICH and CCS remains incompletely understood. It is thought to be primarily associated with micro-ribonucleic acid (microRNA) dysregulation and conditions triggered by cerebral trauma, such as systemic inflammatory response syndrome [31], splenic immune regulation [32], hypothalamic-pituitary-adrenal axis activation [33], increased catecholamines, autonomic nervous system dysfunction, dysbiosis of the gut microbiota, and cell-derived microparticles [6]. The immune- inflammatory response and neurohumoral mechanisms are currently recognized as predominant contributors to this phenomenon [34].

Table 5 Comparison of predictive efficiency of ROC curves for each factor curve

	AUC	*P* value	95% CI	
			Upper limit	Lower limit
ONSD	0.802	0.000*	0.708	0.895
Hematoma volume (cm^3)	0.808	0.000*	0.714	0.901
Glasgow Coma Scale (GCS) score	0.716	0.001*	0.600	0.832
Prediction using a combination of indicators	0.880	0.000*	0.807	0.953

*Indicates statistical significance ($P < 0.05$)

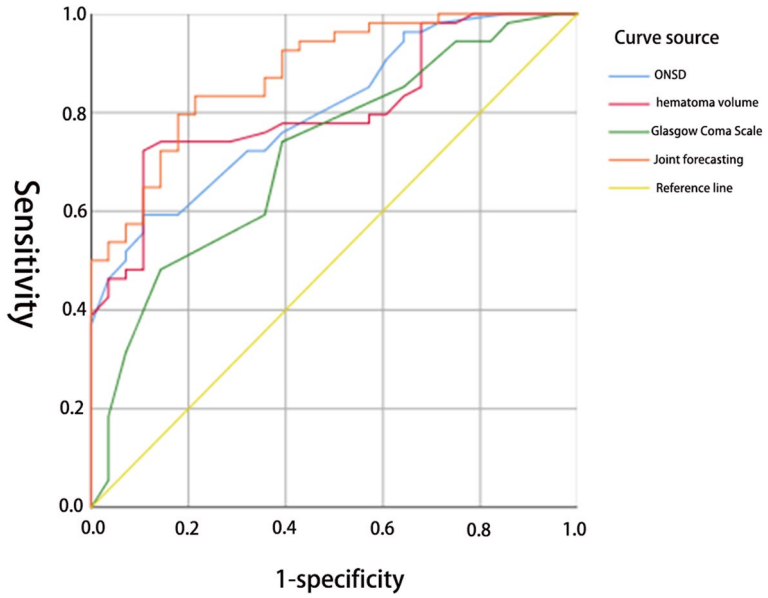

Fig. 4 Comparison of ROC curves of the three predictors and their combination

In this study, inflammatory indicators including IL-6, SAA, CRP, and PCT were analyzed in both the CCS and non-CCS groups. However, no statistically significant differences were observed in these inflammatory indicators between the two groups. The lack of strict control over factors relevant to complications may explain this finding. Further investigation is warranted to determine whether these inflammatory indicators serve as risk factors for concurrent occurrence of ICH with CCS.

Patients with ICH often experience intracranial hypertension due to the mass effect of hematoma formation cerebral edema, hematoma enlargement, and disturbances in cerebrospinal fluid circulation [3]. Elevated ICP can lead to cerebrovascular disorders, cerebral tissue displacement, and consequent cerebral tissue damage, resulting in autonomic nervous system dysfunction that affects heart regulation. The utility of ONSD as an accurate indicator for monitoring ICP has been well-established. This study further explored the accuracy of ONSD in predicting the development of CCS.

The optic nerve originates as an extension of the central nervous system's white matter tracts and is enclosed by the optic nerve sheath, which consists of three layers of meninges that terminate posterior to the eyeball. The space between the soft and hard layers of the optic nerve sheath, referred to as the sheath space, is connected to the cranial subarachnoid space. When ICP increases, the elastic optic nerve sheath is compressed, displacing cerebrospinal fluid into the subarachnoid space of the optic nerve sheath and causing the sheath to expand. This leads to an enlargement of the ONSD, forming the theoretical basis for using ONSD to assess elevated ICP. Research has shown that ONSD begins to expand within minutes following an increase in ICP, well before the development of papilledema [35].

In recent years, there has been increasing interest in non-invasive methods for monitoring ICP. Ultrasound measurement of ONSD has emerged as a reliable and accurate predictor of elevated ICP, gaining significant attention in scientific research [10].

Some studies suggest that ONSD may be influenced by factors such as sex [36], age, height, body weight, body mass index (BMI), and head circumference. However, conflicting results have been reported. Maude [37] and Chen [38] argue that ONSD is not correlated with these variables, while other researchers support a correlation. The inconsistency across studies highlights the need for further investigation. In this study, comparison of ONSD between different sexes and age groups revealed no statistically significant difference in ONSD between patients aged 65 years or older and those younger than 65. However, a significant difference in ONSD was observed between males and females, with males exhibiting higher ONSD values than females. Interestingly, the presence of ICH, whether or not complicated by CCS, did not show any correlation with sex or age, but was associated with ONSD.

Under normal circumstances, slight differences in ONSD between males and females have been documented, with males generally exhibiting slightly larger ONSD values [39, 40]. This difference is believed to be related to anatomical variations between sexes. However,

these differences are typically minor and may not be clinically significant. Therefore, while differences in sex should be considered when using ONSD as a predictive marker, they are unlikely to be a primary influencing factor. Further research is necessary to better understand the impact of sex on ONSD and its clinical significance in different contexts.

Additionally, ONSD was found to be a predictive factor for CCS secondary to ICH, with a cutoff value of 5.88 mm identified for predicting the occurrence of CCS. Thotakura et al. [41] reported significant correlations between ONSD and GCS score, between GCS score and radiological indicators such as midline shift, and between ONSD and radiological markers. Similarly, in this study, ONSD demonstrated a linear correlation with both midline shift and hematoma volume, suggesting that ONSD has predictive capability comparable to that of CT in detecting increases in ICP and mass effect.

Moreover, ONSD, hematoma volume, midline shift, and GCS score are well-established factors associated with poor prognosis and increased mortality in patients with ICH [42–44]. Midline shift is frequently associated with brainstem deformation and critical elevations in ICP [45]. A study by Sun [44], which included 149 patients with ICH, found that 65 patients showed an increased rate of midline shift on follow-up CT scans, with this increase identified as an independent factor for mortality. Additionally, large hematoma volume is recognized as a significant risk factor for early mortality in patients with ICH [46].

The pathophysiological mechanisms leading to cerebral tissue impairments in the ultra-acute phase of ICH involve direct damage to cerebral tissue due to hematoma compression, as well as the subsequent formation of cerebral edema around the hematoma within several days. This edema exacerbates the compressive effect of the hematoma on surrounding cerebral tissue, contributing to more severe and sustained secondary cerebral injury.

The GCS is widely used as a clinical indicator for assessing the level of consciousness and the severity of illness in patients with ICH. It is a well-established predictor of both mortality and disability [47], with lower initial GCS scores typically associated with larger hemorrhage volumes and more severe cerebral injuries [48, 49]. In the present study, ONSD, hematoma volume, and GCS score were all identified as independent predictors of CCS in patients with ICH. Specifically, higher ONSD measurements, larger hematoma volumes, and lower GCS scores were associated with an increased likelihood of ICH concurrent with CCS.

Although this study did not directly assess patient prognosis or mortality rates, the findings suggest a potentially poor outcome in cases where ICH is complicated by secondary CCS. Thus, these indicators may reflect an adverse prognosis to some degree.

Stroke and coronary artery disease often share common risk factors, and post-stroke cardiac abnormalities may be overlooked in clinical settings. This study identified ultrasound measurements of ONSD, hematoma volume, and GCS score as significant predictors for the development of CCS secondary to ICH. Additionally, monitoring ICP is critical in the diagnosis and management of ICH. ONSD, as a non-invasive method for monitoring ICP, not only provides an indication of ICP levels but also predicts the likelihood of CCS, making it a valuable diagnostic tool. The findings of this study may offer new insights and guidance for the diagnosis and treatment of CCS secondary to ICH, providing useful information for clinical practitioners.

However, several limitations of this study must be acknowledged. As a single-center study with a relatively small sample size, the accuracy of the findings and conclusions requires further validation through multicenter studies involving larger populations. Moreover, to specifically assess the effects associated with ICH, individuals with pre-existing conditions such as hyperthyroidism and cardiac diseases were excluded to minimize the likelihood of CCS-related complications. Further research is necessary to investigate the role of neurohumoral factors, including catecholamines and other relevant mechanisms, in the development of CCS in patients with ICH.

Conclusion

An individual's sex significantly influences ONSD, with males typically exhibiting higher ONSD measurements compared to females. Ultrasound-based ONSD measurements exhibit a positive correlation with midline shift and hematoma volume as observed on CT scans. Similar to CT, ultrasound is a reliable tool for measuring ONSD and facilitates the detection of intracranial hypertension and mass effect. Compared to the non-CCS group, individuals in the CCS group exhibited larger ONSD values, greater midline shift, larger hematoma volume, and lower GCS scores. ONSD was identified as an independent risk factor for the development of ICH complicated by CCS, with a cutoff value of 5.88 cm. Ultrasound measurements of ONSD, along with hematoma volume and GCS score, are independent predictors of the likelihood of ICH concurrent with CCS. Moreover, the combined use of these three indicators enhances predictive accuracy, surpassing the predictive capability of each indicator when used individually. The integration of ONSD, hematoma volume, and GCS score offers greater predictive value in assessing the risk of CCS in patients with ICH.

Abbreviations

ONSD	Optic nerve sheath diameter
ICH	Intracerebral hemorrhage
CCS	Cerebral-cardiac syndrome
ICP	Intracranial pressure
GCS	Glasgow Coma Scale
ROC	Receiver operating characteristic
AUC	Area under the curve
MRI:	Magnetic resonance imaging
CT	Computed tomography
EVD	External ventricular drainage
LP	Lumbar puncture
SAH	Subarachnoid hemorrhage
AIS	Acute ischemic stroke
CPP	Cerebral perfusion pressure
MAP	Mean arterial blood pressure

Authors' contributions

Cheng H, Shen XH conceived the idea and conceptualised the study. Jiang JR, Zang HL, Yang WJ, Jing LX, Wang H collected the data. Wang H, Jing LX, Yang WJ, Zang HL analysed and interpreted the data. Shen XH, Fan WZ statistically analyzed the data. Cheng H obtained financing. Fan WZ, Jiang JR drafted the manuscript. Cheng H made critical revisions to the intellectual content of the manuscript. All authors read and approved the final draft.

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Data availability

The datasets used or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was conducted with approval from the Ethics Committee of Shijiazhuang People's Hospital (No.2024-008). This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for publication

All participants signed a document of informed consent.

Competing interests

The authors declare no competing interests.

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