

The Vienna Intracranial Hypertension Score for Predicting Elevated Cerebrospinal Fluid Opening Pressure in Patients Evaluated for Idiopathic Intracranial Hypertension

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Abstract

Background.

Increasing recognition of radiological and ophthalmological findings suggestive of intracranial hypertension has created diagnostic uncertainty in patients evaluated for suspected idiopathic intracranial hypertension (IIH). We aimed to identify independent predictors of elevated cerebrospinal fluid opening pressure (CSF-OP) and derive a clinical prediction model for pre-lumbar puncture risk stratification.

Methods.

This retrospective study included adults from the Vienna Intracranial Hypertension (VIH) database who underwent diagnostic lumbar puncture for suspected IIH with documented CSF-OP, brain MRI, and comprehensive ophthalmological assessment. Univariable and multivariable logistic regression models were used to identify predictors of elevated CSF-OP ($\geq 25\text{cmH}_2\text{O}$) and to derive the Vienna Intracranial Hypertension Score (VIHS). The diagnostic performance of the VIHS was evaluated using receiver operating characteristic (ROC) analysis. In addition, the VIHS was compared to the Pre-Lumbar Puncture Idiopathic Intracranial Hypertension Scale (PLIHS).

Results.

A total of 148 patients were included (median age 31.5 years [range 18–81], 87.2% female), of whom 86 (58.1%) had elevated CSF-OP. In multivariable analyses, age ≤ 40 years (OR 3.37, 95% CI 1.21–10.0; 1 point), body mass index $\geq 30 \text{ kg/m}^2$ (OR 3.44, 95% CI 1.27–9.98; 1 point), papilledema severity (Frisén grade ≥ 2 ; OR 2.78, 95% CI 1.03–7.85; 1 point), and transverse sinus stenosis (OR 4.61, 95% CI 1.67–13.9; 2 points) were independently associated with elevated CSF-OP and incorporated into the VIHS. The VIHS demonstrated superior discriminative performance compared with the externally validated PLIHS (AUC 0.79, 95% CI 0.71–0.87 vs. 0.67, 95% CI 0.57–0.77), with increasing VIHS categories associated with a shift toward higher CSF-OP and a probability of elevated CSF-OP of 87.8% among patients with VIHS ≥ 4 .

Conclusions.

The VIHS provides a pragmatic tool for pre-lumbar puncture risk stratification in suspected IIH, with high scores identifying patients with a high probability of elevated CSF-OP. Low scores, however, do not reliably exclude elevated CSF-OP.

1. Background

Idiopathic intracranial hypertension (IIH) is a disorder characterized by elevated intracranial pressure (ICP) in the absence of an identifiable structural, vascular, infectious, or neoplastic cause.¹ The condition predominantly affects women of reproductive age with obesity and can lead to disabling headaches, pulsatile tinnitus, visual disturbances, and irreversible visual loss if left untreated.² Despite advances in neuroimaging and ophthalmological assessment, the diagnosis is challenging and lumbar puncture (LP) with measurement of cerebrospinal fluid (CSF) opening pressure (OP) remains a cornerstone of the diagnostic workup and is required for confirmation of the diagnosis according to current diagnostic criteria.^{3,4}

At the same time, clinicians, particularly at specialized centres, are increasingly confronted with patients referred because of findings suggestive of IIH. These include MRI abnormalities classically associated with IIH, such as empty sella (ES), optic nerve sheath distension (ONSD), optic nerve tortuosity (ONT), posterior globe flattening (PGF), or transverse sinus stenosis (TSS), which, although characteristic of IIH if observed in combination, are not specific for the disorder.^{5,6} In parallel, ophthalmological assessment frequently identifies conditions that may mimic papilledema, including crowded optic discs, optic disc drusen, or equivocal optic disc swelling. Consequently, distinguishing incidental radiological findings or pseudopapilledema from true manifestations of raised ICP has become an increasingly common challenge in neurological and ophthalmological practice.

To facilitate pre-LP risk stratification, Raggi et al. proposed the Pre-LP Idiopathic Intracranial Hypertension Scale (PLIHS), combining clinical, ophthalmological, and radiological features associated with elevated CSF-OP.⁷ Although the score demonstrated promising diagnostic performance in its derivation cohort, some components, such as tinnitus, are common and diagnostically non-specific, potentially limiting discrimination in broader patient populations. Moreover, the model did not incorporate quantitative ophthalmological biomarkers, including optical coherence tomography (OCT) and transbulbar sonography, which may provide additional diagnostic value. These limitations highlight the need for a refined, pragmatic prediction model incorporating objective markers of elevated CSF-OP, while independent validation of the PLIHS provides an appropriate benchmark against which such a model can be evaluated.

The aims of this study were therefore to identify independent clinical, ophthalmological, and radiological predictors of elevated CSF-OP, and to derive a pragmatic clinical prediction model, the Vienna Intracranial Hypertension Score (VIHS) for estimating the probability of elevated CSF-OP prior to LP. As a secondary objective, we externally validated the diagnostic performance of the PLIHS in a well-characterised cohort of patients undergoing evaluation for suspected IIH.

2. Methods

Study cohort and study design

This retrospective observational study was conducted using data from the Vienna Idiopathic Intracranial Hypertension (IIH) database, a prospective multidisciplinary registry jointly maintained by the Department of Neurology and the Department of Ophthalmology at the Medical University of Vienna.⁸ The registry comprises standardized case report forms capturing demographic data, body weight, disease-specific parameters, as well as documentation of diagnostic and therapeutic procedures. All examinations are performed by specialized neurologists and neuro-ophthalmologists, and patients are treated in accordance with best practice based on the Austrian consensus recommendations.⁹

For the present analysis (database closed at 26 March 2026), consecutive adults undergoing diagnostic evaluation for suspected IIH were eligible if they fulfilled the following criteria: i) age ≥ 18 years, ii) documented CSF-OP obtained during diagnostic LP, iii) brain MRI performed within 4 weeks before LP, and iv) comprehensive ophthalmological assessment performed within 7 days before LP. Patients with IIH without papilledema (IIH-WOP) were excluded from the analysis.

Lumbar puncture

A standardized diagnostic LP was performed using a Quincke type point, 20GA 3.50 IN, 0.9x88 mm (Braun Melsungen AG, Germany) spinal needle with a spinal manometer (Rocket Medical®, UK). LP was performed in the left lateral decubitus position with the legs repositioned after dural puncture to reduce abdominal compression. A stabilized CSF-OP reading was recorded as fluctuations in the manometer settled. After measuring CSF-OP, CSF was drained until a target of 10–15 cm CSF was reached (maximum of 50 ml CSF drained). CSF closing pressure was recorded prior to removal of the spinal needle.

Ophthalmological assessment

Fundoscopy was executed using the Frisén staging scale to rate and categorize papilledema severity from stage 0 (no papilledema) to stage 5 (severe papilledema).¹⁰

Best-corrected visual acuity was assessed using Sloan charts at distance following subjective refraction. Results were given in logarithm of the minimum angle of resolution (logMAR).¹¹

Automated visual field testing (Humphrey Field Analyzer, Carl Zeiss Meditec, Jena, Germany) was performed using 30 – 2 Swedish Interactive Threshold Algorithm (SITA)-Fast protocols, quantifying the mean deviation (MD) in decibels (dB) of all test locations compared to age-matched controls.^{8,10}

OCT imaging was performed without pupil dilation in both eyes of each person using spectral-domain OCT (Spectralis OCT, Heidelberg Engineering, Heidelberg, Germany; software Heidelberg eye explorer software version 6.9a) adhering to the OSCAR-IB quality control criteria and describing findings in accordance with the APOSTEL criteria.^{12,13} For peripapillary retinal nerve fiber layer (pRNFL) thickness, a 12° (3.4 mm) ring scan centred on the optic nerve head was used (1536 A-scans, automatic real-time tracking [ART]: 100 averaged frames).¹⁴ Ganglion cell layer (GCL) volume was measured by means of a

20°×20° macular volume scan (centred on the macula with 512 A-scans and 25 B-scans aligned vertically with 16 averaged frames). GCL volume was derived from the 6-mm circular area centered on the foveola according to the Early Treatment Diabetic Retinopathy Study (ETDRS) grid.¹⁵ Image processing was semiautomated using the built-in proprietary software for automated layer segmentation with manual correction of obvious errors. Measurements of worse eyes were used for statistical analysis, i.e., higher pRNFL thickness as a marker of papilledema and lower GCL volume as a marker of neuronal cell loss.

Transbulbar sonography (ABSolu, Quantel Medical, Cournon d'Auvergne, France) was performed after topical anesthesia with oxybuprocaine eye drops. Optic nerve sheaths were qualitatively assessed by transverse and longitudinal B-scans using a 20 MHz annular probe placed temporally on the eyeball to verify the presence of a bat sign indicating perineural CSF congestion. Arachnoid optic nerve sheath diameter (AONSD) was quantified using standardized amplitude-modulated (A-scan) ultrasonography with tissue sensitivity settings. Standardized A-scan echography provides more accurate measurements of AONSD than caliper-based measurements obtained from B-scan ultrasonography, which lacks standardized settings and is affected by the applied gain. The 8-MHz parallel-beam A-scan probe was placed on the temporal equator of the eye while the participant maintained primary gaze.^{16,17} At least two measurements were taken within 3 mm of the posterior bulb wall, and the highest diameter was documented.

Magnetic Resonance Imaging

An experienced neuroradiologist (WM) blinded to diagnosis systematically evaluated all neuroimaging scans applying the four MRI criteria as defined by Korsbaek et al.: (i) suprasellar herniation graded from I-V with cut-off set at $\geq$ grade III, i.e. $>1/3$ of the sella height, in sagittal plane (ES), (ii) uni- or bilateral distension of the perioptic nerve sheath $> 2\text{mm}$ in the coronal plane of T2 weighted images, measured 3 mm behind the globe (POSD), (iii) uni- or bilateral PGF by qualitative evaluation on axial T2 weighted images and (iv) uni- or bilateral TSS by index of TSS (ITSS) ≥ 4 .^{3,18} Minimal requirements for brain MRI sequences to evaluate pituitary and orbital morphology were set as defined by Korsbaek et al. (1.5T or 3T, at least T1 and T2w sequences in two different planes for excluding structural lesions, with or without thin-cut, fat-suppressed orbital sequences, venous non-contrast MR angiography or T1 post gadolinium sequence).³

PLIHS Score

The previously published Pre-LP Intracranial Hypertension Scale (PLIHS) was calculated for each patient according to the original description.⁷ The PLIHS is a 7-point clinical prediction score derived from multivariable logistic regression and incorporates five variables available before LP: papilledema (Frisén grade ≥ 2 ; 2 points), presence of pulsatile tinnitus (2 points), ES (1 point), ONSD (1 point), and obesity (BMI $\geq 30 \text{ kg/m}^2$; 1 point). In accordance with the original publication, a score ≥ 3 was considered indicative of a high probability of elevated CSF-OP ($\geq 25 \text{ cmH}_2\text{O}$).⁷

The study was approved by the ethics committee of the Medical University Vienna (ethical approval number: 2216/2020). As this was a retrospective study, the need for written informed consent was waived by the ethics committee. This study adheres to the reporting guidelines outlined within the 'Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement'.

Data Availability Statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request by a qualified researcher and upon approval by the ethics committee of the qualified researcher's institution and the data-clearing committee of the Medical University Vienna.

Statistical analysis

Statistical analysis was performed using R (Version 4.4.2).¹⁹ Categorical variables were expressed in absolute frequencies and percentages, continuous parametric variables as mean and standard deviation (SD) and continuous non-parametric variables as median with interquartile range (IQR), as appropriate. Continuous variables were tested for normal distribution by the Shapiro-Wilk normality test.

Patients were stratified according to CSF OP into two groups: non-elevated ($<25\text{cmH}_2\text{O}$), and elevated CSF OP ($\geq 25\text{cmH}_2\text{O}$). Univariable comparisons were performed using the chi-square test, one-way analysis of variance (ANOVA), and the Kruskal-Wallis test as appropriate.

To identify predictors of elevated CSF-OP, univariable logistic regression analyses were performed for demographic, clinical, ophthalmological, and radiological variables. Multivariable logistic regression models were constructed with elevated CSF-OP ($\geq 25\text{cmH}_2\text{O}$) as the dependent variable, and variables associated with CSF-OP at $p < 0.20$ in univariable analyses as independent variables. Odds ratios (ORs) with 95% confidence intervals were calculated. A clinical prediction score (VIHS) was derived from the final multivariable model by assigning integer point values proportional to the β coefficients of the retained predictors. Point values were rounded to the nearest integer to obtain a simplified bedside scoring system. The discriminative performance of the VIHS was assessed using ROC curve analysis. AUC with 95% CI, sensitivity, specificity, PPV and NPV were calculated for clinically relevant score thresholds. As a secondary exploratory analysis, the distribution of normal ($\leq 20\text{cmH}_2\text{O}$), borderline (>20 to $<25\text{cmH}_2\text{O}$), and elevated ($\geq 25\text{cmH}_2\text{O}$) CSF-OP was assessed across VIHS score categories to evaluate its ability to discriminate across the clinically relevant spectrum of CSF-OP. The discriminatory performance of the VIHS and PLIHS scores was compared using DeLong's test. Separate multivariable models were fitted using pRNFL thickness and AONSD in place of Frisén grading, and an OCT-enhanced VIHS (VIHS-OCT) was derived. Prespecified thresholds for pRNFL thickness ($115\mu\text{m}$, $120\mu\text{m}$, and $125\mu\text{m}$) and AONSD (4.5mm , 5.0mm , 5.5mm) were evaluated.

Finally, exploratory subgroup analysis examined combinations of ophthalmological biomarkers to assess whether concordant abnormalities provided greater discrimination than isolated findings. The significance level was set at a two-sided p-value < 0.05.

3. Results

In the final analysis, 148 patients were included (median age 31.5 years [range 18–81], 87.2% female, median BMI 32.4 kg/m² [16.6–71.1]). Median CSF-OP was 27cmH₂O (range 10.5–50), with elevated ICP observed in 86 (58.1%) patients. The inclusion/exclusion process is shown in Fig. 1. Characteristics of the study cohort are described in Table 1. Ring scans were available in 147 (99.3%), macular scans in 142 (95.3%), and transbulbar sonography in 139 (93.9%) patients.

Table 1
Demographics and clinical characteristics of the study cohort.

Age (years) ^a	All (n = 148)	< 25cmH ₂ O (n = 62)	≥ 25cmH ₂ O (n = 86)	p-value
	31.5 (18–81)	32.5 (18–81)	31 (18–70)	0.171
Female ^b	129 (87.2)	52 (83.9)	77 (89.5)	0.391
BMI (kg/m ²) ^a	32.4 (16.6–71.1)	29.5 (16.6–46.7)	34.6 (20.4–71.1)	< 0.001
Headache ^b	118 (79.7)	52 (83.9)	66 (76.7)	0.392
Visual disturbances ^b	91 (61.5)	38 (61.3)	53 (61.6)	> 0.999
Pulsatile tinnitus ^b	62 (41.9)	24 (38.7)	38 (44.2)	0.619
Frisén scale ^c	1 (1–2)	0 (0–1)	2 (1–3)	< 0.001
Visual acuity of worse eye ^c	1.0 (1.0–1.2)	1.0 (1.0–1.2)	1.0 (1.0–1.2)	0.447
Visual field mean deviation of worse eye (dB) ^c	-1.95 (-4.31, -0.70)	-1.77 (-3.55, -1.01)	-2.12 (-5.69, -0.46)	0.95
pRNFL thickness of worse eye (μm) ^c	127 (105–206)	109 (98–124)	163 (118–250)	< 0.001
GCL volume of worse eye (mm ³) ^c	1.08 (1.01–1.14)	1.08 (1.01–1.12)	1.09 (1.02–1.16)	0.289
AONSD of worse eye (mm) ^d	5.5 (0.7)	5.3 (0.8)	5.6 (0.7)	0.011
ES	84 (63.6)	34 (58.6)	50 (67.6)	0.38
ONSD	121 (93.1)	54 (93.1)	67 (93.1)	> 0.999
ONT	70 (53.4)	29 (50.0)	41 (56.2)	0.599
PGF	46 (35.1)	13 (22.4)	33 (45.2)	0.011
TSS	69 (65.7)	22 (47.8)	47 (79.7)	0.001
≥ 1 MRI sign	124 (93.9)	54 (93.1)	70 (94.6)	> 0.999

^aMedian (range), ^bNumber (percentage), ^cMedian (IQR), ^dMean (SD)

AONSD: arachnoid optic nerve sheath diameter, BMI: body mass index, CI: confidence interval, CSF-OP: cerebrospinal fluid opening pressure, ES: empty sella, GCL: ganglion cell layer, MRI: magnetic resonance imaging, ONSD: optic nerve sheath distension, ONT: optic nerve tortuosity, OR: odds ratio, PGF: posterior globe flattening, pRNFL: peripapillary retinal nerve fibre layer, TSS: transverse sinus stenosis

Age (years) ^a	All (n = 148)	< 25cmH ₂ O (n = 62)	≥ 25cmH ₂ O (n = 86)	p-value
	31.5 (18–81)	32.5 (18–81)	31 (18–70)	0.171
≥ 2 MRI signs	91 (69.7)	38 (65.5)	54 (73.0)	0.463
≥ 3 MRI signs	35 (26.5)	9 (15.5)	26 (35.1)	0.02
^a Median (range), ^b Number (percentage), ^c Median (IQR), ^d Mean (SD)				
AONSD: arachnoid optic nerve sheath diameter, BMI: body mass index, CI: confidence interval, CSF-OP: cerebrospinal fluid opening pressure, ES: empty sella, GCL: ganglion cell layer, MRI: magnetic resonance imaging, ONSD: optic nerve sheath distension, ONT: optic nerve tortuosity, OR: odds ratio, PGF: posterior globe flattening, pRNFL: peripapillary retinal nerve fibre layer, TSS: transverse sinus stenosis				

Insert Fig. 1.

Insert Table 1.

In univariable logistic regression models, age ≤ 40 years, BMI ≥ 30kg/m², papilledema (Frisén scale ≥ 2), pRNFL thickness ≥ 125µm, PGF, TSS and the presence of ≥ 3 MRI signs were associated with increased risk of CSF OP ≥ 25cmH₂O (Table 2). In a multivariable model, age ≤ 40 years, BMI ≥ 30 kg/m², pRNFL thickness ≥ 125µm and TSS remained significantly associated with CSF OP ≥ 25cmH₂O, whereas ≥ 3 MRI signs only showed a non-significant trend (Table 2).

Table 2

Univariable and multivariable logistic regression analyses identifying predictors of elevated CSF-OP ($\geq 25\text{cmH}_2\text{O}$).

	Univariable		Multivariable	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age ≤ 40 years	3.20 (1.52–6.92)	0.002	5.20 (1.56–19.84)	0.010
Female sex	1.65 (0.62–4.41)	0.313	n.a.	n.a.
BMI $\geq 30\text{kg/m}^2$	2.78 (1.41–5.55)	0.003	5.46 (1.65–19.84)	0.008
Headache	0.62 (0.26–1.45)	0.290	n.a.	n.a.
Visual disturbances	1.01 (0.52–1.98)	0.967	n.a.	n.a.
Tinnitus	1.25 (0.65–2.45)	0.506	n.a.	n.a.
Frisén scale ≥ 2	4.99 (2.42–10.84)	< 0.001	0.40 (0.08–1.72)	0.239
pRNFL thickness $\geq 125\mu\text{m}$	8.67 (4.15–19.1)	< 0.001	15.56 (3.62–87.11)	0.001
AONSD $\geq 5.00\text{mm}$	1.69 (0.74–3.94)	0.216	2.06 (0.53–8.58)	0.304
Migraine as comorbidity	1.09 (0.37–3.42)	0.876	n.a.	n.a.
Any comorbidity	0.73 (0.37–1.43)	0.357	n.a.	n.a.
ES	1.47 (0.72–3.02)	0.290	n.a.	n.a.
ONSD	0.99 (0.24–3.93)	0.991	n.a.	n.a.
ONT	1.28 (0.64–3.57)	0.483	n.a.	n.a.
PGF	2.86 (1.35–6.33)	0.008	0.19 (0.02–1.62)	0.131
TSS	4.27 (1.83–10.36)	0.001	8.28 (2.48–33.28)	0.001
≥ 3 MRI signs	2.95 (1.29–7.26)	0.013	7.29 (0.76–79.77)	0.088
AONSD: arachnoid optic nerve sheath diameter, BMI: body mass index, CI: confidence interval, CSF-OP: cerebrospinal fluid opening pressure, ES: empty sella, MRI: magnetic resonance imaging, ONSD: optic nerve sheath distension, ONT: optic nerve tortuosity, OR: odds ratio, PGF: posterior globe flattening, pRNFL: peripapillary retinal nerve fibre layer, TSS: transverse sinus stenosis				

Insert Table 2.

Because fundoscopy remains the clinical standard and OCT and transbulbar sonography are not universally available, Frisén grading was retained in the primary model, while pRNFL thickness and AONSD were evaluated in separate models (Supplemental Table 1).

In the primary model, age ≤ 40 years (OR 3.37, 95% CI 1.21–10.0, $p = 0.023$), BMI $\geq 30\text{kg/m}^2$ (OR 3.44, 95% CI 1.27–9.98, $p = 0.018$), Frisén ≥ 2 (OR 2.78, 95% CI 1.03–7.85, $p = 0.046$) and TSS (OR 4.61, 95% CI 1.67–13.9, $p = 0.004$) were associated with CSF-OP $\geq 25\text{cmH}_2\text{O}$, whereas PGF and ≥ 3 MRI signs were not. Based on these findings, a 5-point clinical prediction score (VIHS) was developed, with TSS contributing 2 points, and each of the other three covariates contributing 1 point. The VIHS demonstrated good discrimination for CSF-OP $\geq 25\text{cmH}_2\text{O}$ (AUC 0.79, 95% CI 0.71–0.87). A score ≥ 4 identified a high-risk group with a sensitivity of 61.0%, specificity of 89.1%, PPV of 87.8%, and NPV of 64.1%. Lower thresholds increased sensitivity to 91.5% at ≥ 2 and 98.3% at ≥ 1 , but provided only moderate NPV (75.0% and 83.3%, respectively) and markedly reduced specificity (32.6% and 10.9%). When CSF-OP was further categorized as normal ($\leq 20\text{ cmH}_2\text{O}$), borderline (> 20 to $< 25\text{cmH}_2\text{O}$), or elevated ($\geq 25\text{cmH}_2\text{O}$), increasing VIHS categories were associated with a progressive shift toward elevated CSF-OP. Among patients classified as high risk (VIHS ≥ 4), 87.8% had elevated CSF-OP and only 4.9% had normal CSF-OP, compared with 33.3% and 48.5%, respectively, among patients classified as low risk (VIHS ≤ 2) (Fig. 2).

Insert Fig. 2.

In the external validation, the PLIHS score ≥ 3 had a sensitivity of 83.1% and specificity of 34.8% for the identification of CSF-OP $\geq 25\text{cmH}_2\text{O}$, with a PPV of 62.0% and NPV of 61.5% (AUC 0.67, 95% CI 0.57–0.77). The VIHS demonstrated significantly better discrimination than PLIHS score ($p = 0.020$). The distribution of CSF-OP categories across individual VIHS and PLIHS scores is shown in Fig. 3.

Insert Fig. 3.

When Frisén grade was replaced by pRNFL thickness, a threshold of $\geq 125\mu\text{m}$ was associated with an almost 11-fold increased risk of elevated CSF-OP (OR 10.49, 95% CI 3.40–39.05, $p < 0.001$). We therefore created an OCT-enhanced 6-point risk score (VIHS-OCT), in which pRNFL thickness $\geq 125\mu\text{m}$ was assigned 2 points. VIHS-OCT demonstrated a good discrimination for CSF-OP $\geq 25\text{cmH}_2\text{O}$ (AUC 0.86, 95% CI 0.79–0.93), significantly exceeding that of the VIHS in a paired comparison ($p = 0.025$). A score ≥ 5 identified a high-risk group with a sensitivity of 49.2%, specificity of 93.3%, PPV of 90.6%, and NPV of 58.3%. When CSF-OP was further categorized as normal ($\leq 20\text{cmH}_2\text{O}$), borderline (> 20 to $< 25\text{cmH}_2\text{O}$), or elevated ($\geq 25\text{cmH}_2\text{O}$), increasing VIHS-OCT categories were associated with a progressive shift toward elevated CSF-OP. Among patients classified as high risk (VIHS-OCT ≥ 5), 90.6% had elevated CSF-OP and only 3.1% had normal CSF-OP, compared with 17.2% and 58.6%, respectively, among patients classified as low risk (VIHS-OCT ≤ 2) (Supplemental Fig. 1).

Exploratory analysis suggested that concordant ophthalmological abnormalities were associated with a substantially higher probability of elevated CSF-OP than isolated findings. Isolated AONSD enlargement ($\geq 5.00\text{mm}$) was associated with a 28.6% (12/42) probability of elevated CSF-OP, compared with 77.4% and 79.0% when accompanied by papilledema or pRNFL thickening, respectively. In sensitivity analyses,

this finding remained consistent using both a lower ($\geq 4.50\text{mm}$; 14/47 [29.8%]) and a more stringent AONSD threshold ($\geq 5.50\text{mm}$; 7/23 [30.4%]).

4. Discussion

In the present study, we developed the VIHS, a pragmatic prediction model integrating age, obesity, papilledema severity, and TSS, which demonstrated good discrimination for elevated CSF-OP. Replacing fundoscopic grading with pRNFL thickness further improved discrimination in the VIHS-OCT, although neither score reliably excluded elevated CSF-OP at low values. As a secondary analysis, external validation of the previously proposed PLIHS demonstrated only modest discriminative performance.

While the incidence of IIH continues to rise in parallel with the global obesity epidemic, only a minority of individuals with obesity develop this disorder.^{1,20} Similarly, although migraine-like headache is a common manifestation of IIH, most patients with migraine do not have IIH.²¹ Consequently, many patients with obesity and migraine-like headache undergo MRI, where findings traditionally associated with IIH, such as partial ES, ONSD, or TSS, may raise diagnostic suspicion. However, these MRI signs are not uncommon in the general population – particularly (partial) ES when observed in isolation – and therefore lack specificity for IIH, often representing incidental findings rather than evidence of IIH.^{22,23} Together with variability in MRI acquisition, interpretation, and reporting practices, this underscores the need for multimodal pre-LP risk stratification.²⁴

Although LP with measurement of CSF-OP remains indispensable for confirming the diagnosis of IIH in patients with a high clinical suspicion and may even provide temporary symptomatic relief in some patients, it is an invasive procedure requiring specialized expertise and is associated with patient discomfort and potential complications, including post-lumbar puncture headache, back pain, infection, bleeding, and, rarely, cerebral herniation.^{25,26} Consequently, routine LP in patients referred because of isolated or non-specific radiological or ophthalmological findings is neither desirable nor practical. Improved pre-LP risk stratification may therefore help estimate the diagnostic yield of invasive testing and prioritize patients with a high probability of elevated CSF-OP.

Against this background, Raggi et al. developed the PLIHS score as a pragmatic tool to stratify patients according to their probability of elevated CSF-OP before LP.⁷ Although the score incorporates well-established markers of IIH, it also includes less specific features, such as tinnitus and ES, while omitting quantitative ophthalmological biomarkers. In our cohort, the diagnostic performance of the PLIHS score was substantially lower than originally reported, driven primarily by a marked reduction in specificity (34.8% vs. 65.5%), whereas sensitivity remained relatively preserved (83.1% vs. 87.4%). In multivariable analyses, obesity and papilledema retained their predictive value, whereas tinnitus and ES contributed little to risk discrimination. While the prevalence of empty sella was comparable between both cohorts (63.6% vs. 66.0%), pulsatile tinnitus was nearly twice as frequent in our cohort (41.9% vs. 22.2%), which may have contributed to the reduced specificity of the PLIHS.

Conversely, quantitative pRNFL thickness together with TSS emerged as the strongest predictors of elevated CSF-OP. Notably, TSS outperformed other MRI biomarkers, including ES and the presence of ≥ 3 MRI signs.⁶ This observation is biologically plausible given the increasingly recognised role of impaired cerebral venous outflow in the pathophysiology of IIH. Although it remains uncertain whether TSS is a primary driver or consequence of raised ICP, its strong association with elevated CSF-OP suggests that it reflects clinically relevant disturbances in CSF dynamics.²⁷ By contrast, aggregate MRI scores did not improve prediction, suggesting that MRI biomarkers differ in their diagnostic value.

Although pRNFL thickness was one of the strongest predictors of elevated CSF-OP, fundoscopy remains the cornerstone of the initial ophthalmological assessment; consequently, Frisén grading was retained in the clinically applicable VIHS. Nevertheless, an OCT-enhanced version (VIHS-OCT) provided more pronounced risk stratification, reducing the probability of elevated CSF-OP in the low-risk group while maintaining a similarly high probability in the high-risk group. Beyond its incremental predictive value, OCT offers several additional advantages over fundoscopy, including objective quantification of papilledema, better differentiation from pseudopapilledema (e.g., optic disc drusen or crowded discs), as well as simultaneous assessment of neuroaxonal injury. Whereas pRNFL measures the thickness of the unmyelinated retinal ganglion cell axons, which increases because of axoplasmic flow stasis in papilledema, GCL represents retinal ganglion cell bodies and serves as a structural biomarker of irreversible neuronal loss. Consequently, combined assessment of pRNFL and GCL thickness distinguishes true resolution of papilledema from atrophy-related pseudonormalisation while simultaneously providing prognostic information regarding permanent neuroaxonal injury.⁹

Importantly, the clinical utility of the VIHS may extend beyond prediction of the conventional $\geq 25\text{cmH}_2\text{O}$ diagnostic threshold. When CSF-OP was considered across normal, borderline, and elevated ranges, increasing VIHS values were associated with a progressive shift toward elevated CSF-OP. This may provide clinically relevant risk stratification across the CSF-OP spectrum, particularly because borderline values require interpretation in the context of the overall clinical presentation. Although lower VIHS thresholds achieved high sensitivity, their NPV remained insufficient to reliably exclude elevated CSF-OP and was accompanied by a substantial loss of specificity. Thus, the VIHS appears primarily suited for risk stratification and rule-in rather than rule-out, with high scores identifying patients with a high probability of elevated CSF-OP, while low scores do not support safe omission of LP.

Exploratory analyses suggested that concordance across ophthalmological biomarkers may provide greater diagnostic information than isolated findings. Patients with concomitant papilledema, increased pRNFL thickness, and enlarged AONSD were more likely to have elevated CSF-OP, whereas AONSD enlargement alone was associated with a considerably lower probability. Although these subgroup analyses were limited by small sample sizes, the findings support interpreting ophthalmological biomarkers in combination. Importantly, the low probability of elevated CSF-OP associated with AONSD enlargement alone remained consistent across different AONSD thresholds, suggesting that this finding was not merely attributable to the selected cut-off. Conversely, discordant findings, such as increased pRNFL thickness without AONSD enlargement, should prompt careful evaluation for alternative

ophthalmological diagnoses, including optic disc drusen, crowded disc, and other causes of pseudopapilledema. Although exploratory, these findings further emphasize the importance of interpreting AONSD in conjunction with structural evidence of papilledema rather than as a stand-alone marker of elevated CSF-OP.

Several limitations should be acknowledged. First, the retrospective single-centre design introduces the possibility of selection bias and may limit generalisability to other healthcare settings. However, this is mitigated by the consecutive inclusion of patients and our centre covering a large and representative catchment area of patients suspected for IIH.²⁸ Second, although relatively large for a deeply phenotyped cohort of patients undergoing evaluation for suspected IIH, the sample size remains modest for prediction modelling, resulting in wide confidence intervals for some estimates and raising the possibility of model overfitting. Third, MRI examinations were acquired across different scanners using routine clinical protocols, which may have introduced variability in image acquisition. Fourth, OCT and transbulbar sonography were not available in all patients, reducing statistical power and inducing further potential for selection bias. Fifth, the newly generated VIHS score has undergone only internal evaluation and therefore requires prospective external validation before clinical implementation. Finally, patients with IIH without papilledema were not included, limiting the applicability of the proposed model to this diagnostically challenging subgroup. Despite these limitations, the validity of our findings is substantially strengthened by independent external validation of a previously proposed prediction model, the availability of comprehensive multimodal clinical, ophthalmological, and radiological phenotyping, systematic MRI assessment by an experienced neuroradiologist, and the evaluation of all candidate predictors against directly measured CSF-OP.

5. Conclusions

In conclusion, the VIHS provides a simple, clinically applicable prediction model incorporating age, obesity, papilledema severity, and TSS for pre-LP risk stratification in patients evaluated for suspected IIH. High VIHS values demonstrated strong rule-in performance, whereas low values did not reliably exclude elevated CSF-OP, underscoring that the score should complement rather than replace direct CSF-OP measurement when IIH remains clinically suspected. External validation of the previously proposed PLIHS demonstrated only modest overall discrimination in our cohort. Future prospective multicentre studies are warranted to externally validate the VIHS and determine whether additional biomarkers further improve pre-LP risk stratification.

Abbreviations

AONSD

arachnoid optic nerve sheath diameter

AUC

area under the curve

BMI

body mass index
CI
confidence interval
CSF
cerebrospinal fluid
CSF-OP
cerebrospinal fluid opening pressure
ES
empty sella
ETDRS
Early Treatment Diabetic Retinopathy Study
GCL
ganglion cell layer
ICP
intracranial pressure
IIH
idiopathic intracranial hypertension
IIH-WOP
idiopathic intracranial hypertension without papilledema
IQR
interquartile range
ITSS
index of transverse sinus stenosis
LP
lumbar puncture
MD
mean deviation
MRI
magnetic resonance imaging
NPV
negative predictive value
OCT
optical coherence tomography
ONSD
optic nerve sheath distension
ONT
optic nerve tortuosity
OP
opening pressure
OR

odds ratio
PGF
posterior globe flattening
PLIHS
Pre-Lumbar Puncture Intracranial Hypertension Scale
POSD
perioptic nerve sheath distension
PPV
positive predictive value
pRNFL
peripapillary retinal nerve fiber layer
ROC
receiver operating characteristic
SD
standard deviation
TSS
transverse sinus stenosis
VIIH
Vienna Idiopathic Intracranial Hypertension
VIHS
Vienna Intracranial Hypertension Score
VIHS-OCT
optical coherence tomography-enhanced Vienna Intracranial Hypertension Score

Declarations

Author contributions

NK: acquisition of data, data management, statistical analysis and interpretation of data, drafting of manuscript. JN: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. MB: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. NM: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. CS: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. SZ: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. SM: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. WM: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. KN: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. CW: acquisition of data, interpretation of data, critical revision of manuscript for intellectual content. BP: acquisition of data, interpretation of data, study supervision, critical revision of manuscript for intellectual content. GB: study concept and design, acquisition of data, interpretation of data, study supervision, critical revision of manuscript for intellectual content.

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Conflicts of interest

Nik Krajnc: has participated in meetings sponsored by, received speaker honoraria or travel funding from Alexion, BMS/Celgene, Janssen-Cilag, Merck, Neuraxpharm, Novartis, Roche and Sanofi-Genzyme and held a grant for a Multiple Sclerosis Clinical Training Fellowship Programme from the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS).

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Christoph Stapf: declares no conflict of interest relevant to this study.

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Stefan Macher: declares no conflict of interest relevant to this study.

Wolfgang Marik: declares no conflict of interest relevant to this study.

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Figures

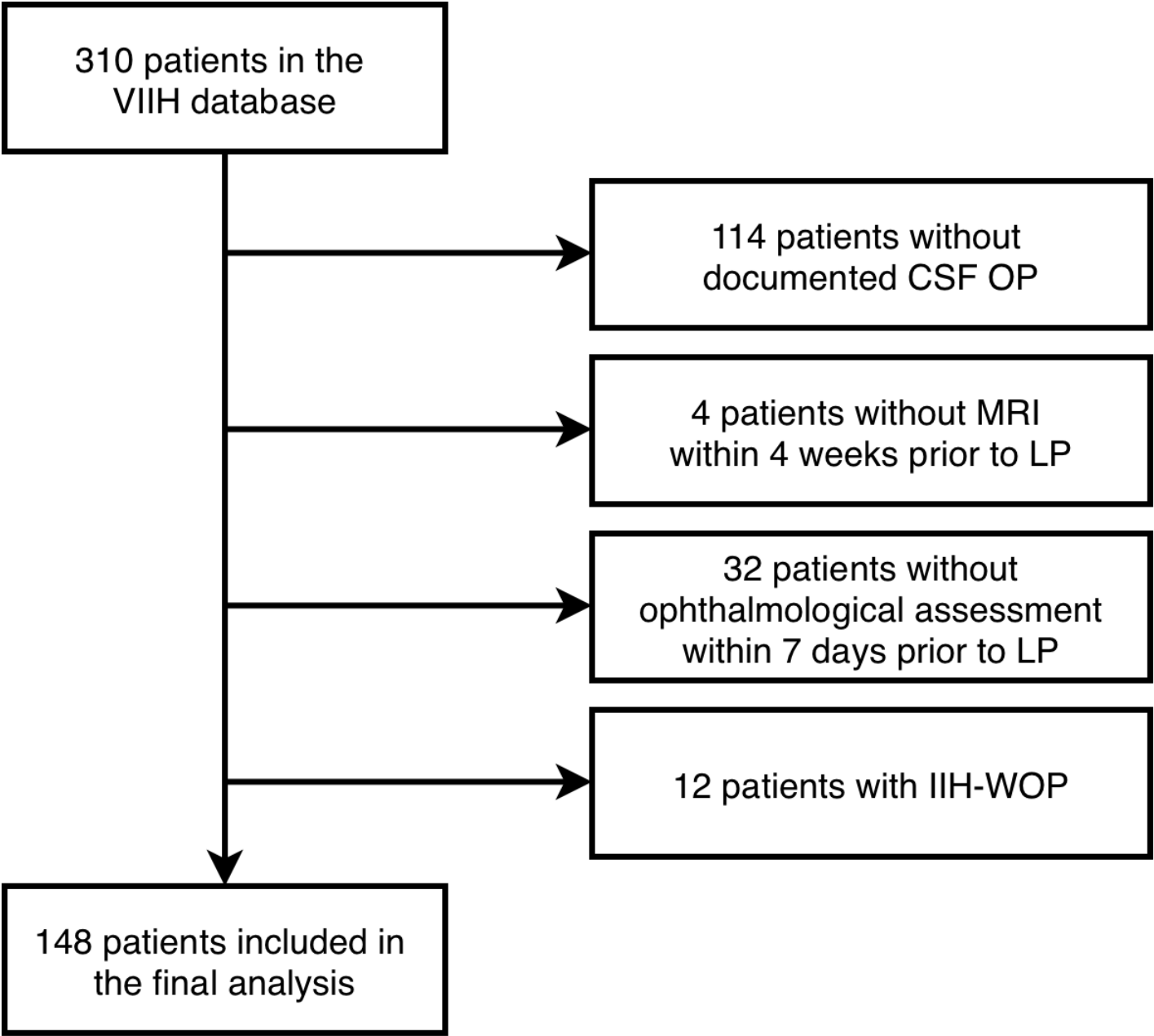


Figure 1

Flowchart of the inclusion/exclusion process.

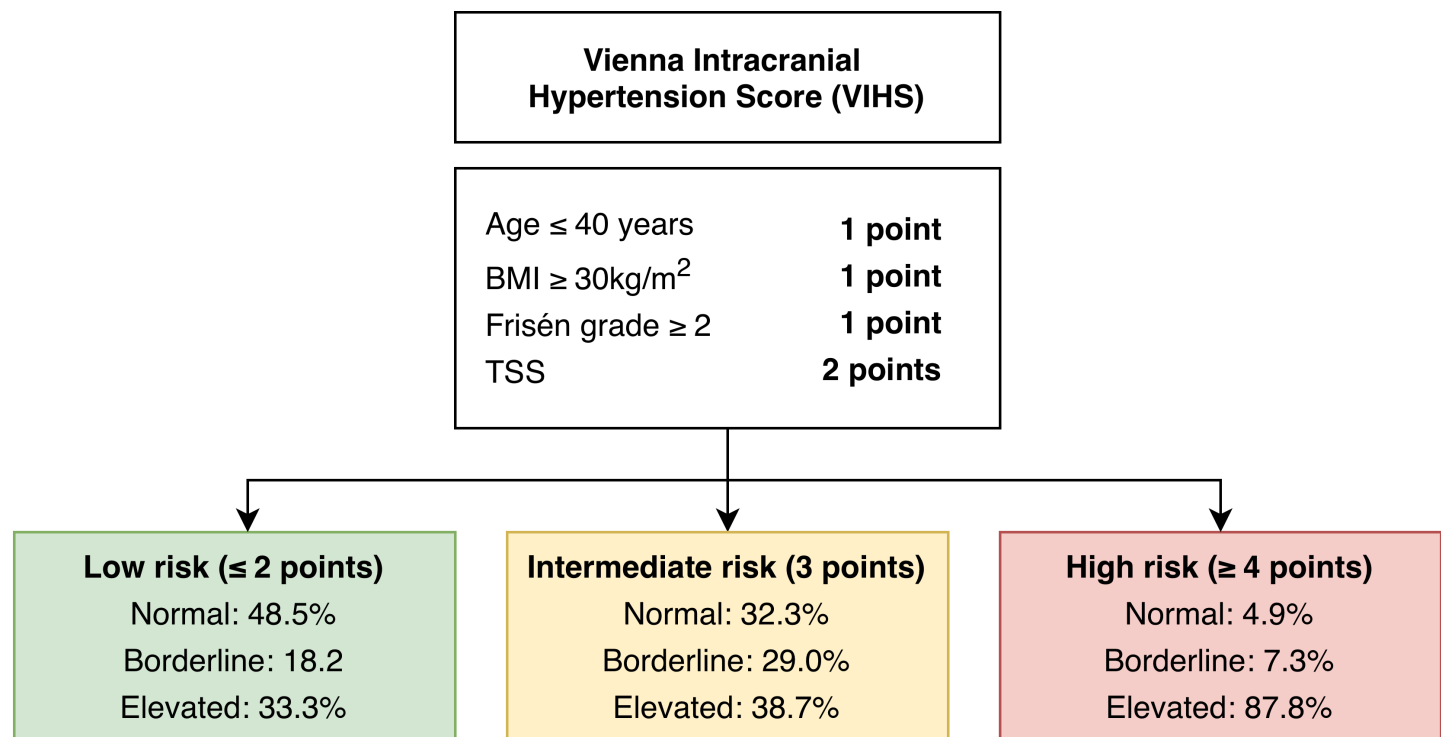


Figure 2

Clinical application of the Vienna Intracranial Hypertension Score (VIHS) for pre-lumbar puncture risk stratification. Distribution of normal ($\leq 20\text{cmH}_2\text{O}$), borderline (>20 to $<25\text{cmH}_2\text{O}$), and elevated ($\geq 25\text{cmH}_2\text{O}$) CSF-OP across low- (≤ 2 points), intermediate- (3 points), and high-risk (≥ 4 points) VIHS categories. BMI: body mass index, CI: confidence interval, CSF-OP: cerebrospinal fluid opening pressure, TSS: transverse sinus stenosis.

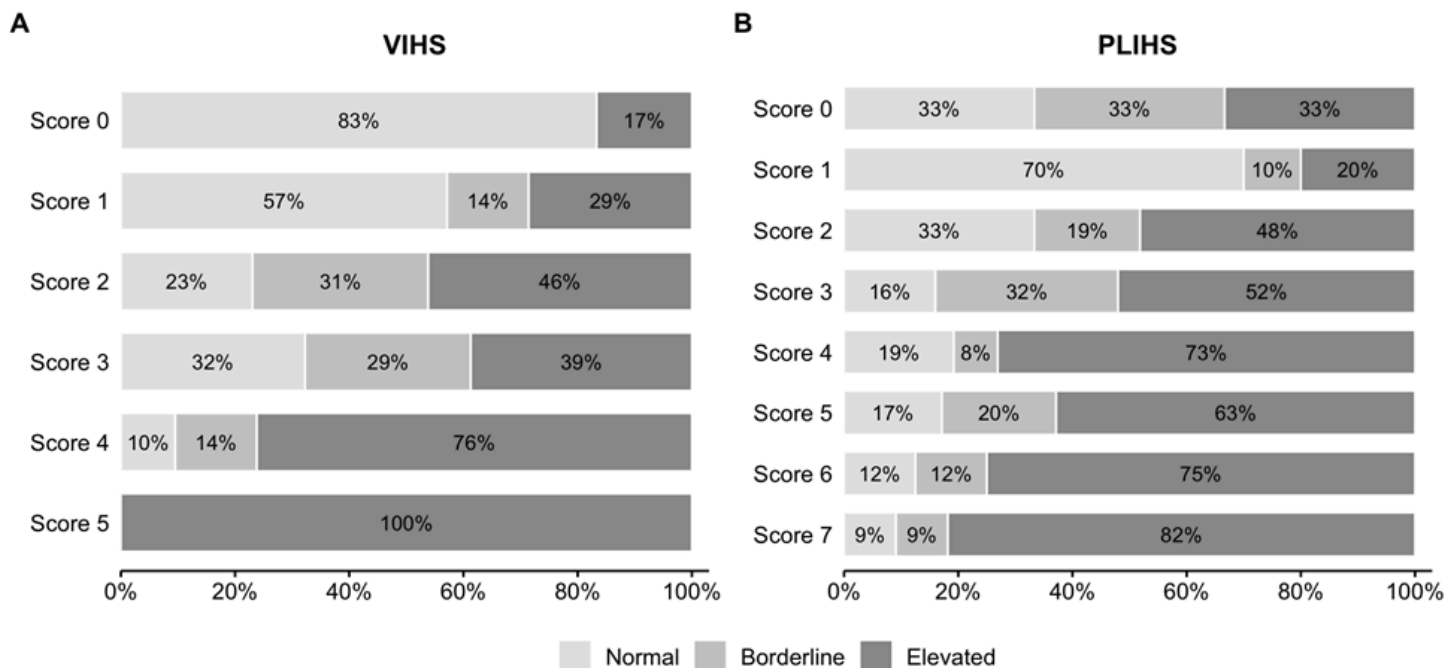


Figure 3

Distribution of CSF-OP categories across individual VIHS (A) and PLIHS scores (B). Proportions of patients with normal ($\leq 20\text{cmH}_2\text{O}$), borderline (>20 to $<25\text{cmH}_2\text{O}$), and elevated CSF-OP ($\geq 25\text{cmH}_2\text{O}$) are shown for each score value. CSF-OP: cerebrospinal fluid opening pressure, PLIHS: Pre-Lumbar puncture IIH scale.

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