Original Article

HEALTH SCIENCES
MEDICINE

DOI: 10.32322/jhsm.1681670
J Health Sci Med. 2025;8(5):859-864

Quantitative evaluation of cranial MRI findings in idiopathic intracranial hypertension

Fatma Kökcü
Department of Radiology, Faculty of Medicine, Tokat Gaziosmanpaşa University, Tokat, Turkiye

Cite this article as: Kökcü F. Quantitative evaluation of cranial MRI findings in idiopathic intracranial hypertension. *J Health Sci Med.* 2025;8(5):859-864.

Received: 22.04.2025 ◆ **Accepted**: 17.08.2025 ◆ **Published**: 16.09.2025

ABSTRACT

Aims: Idiopathic intracranial hypertension (IIH) is a syndrome characterized by intracranial pressure. The purpose of this study was to evaluate the accuracy of diagnosis by giving the quantitative values of imaging findings of IIH on cranial MRI.

Methods: This study included 37 patients who were diagnosed with IIH and 22 healthy controls were included by using a match-to-pair technique regarding their sex and age. Optic nerve sheath diameter (ONSD) and optic nerve diameter (OND), transverse diameter of Meckel cave (MCD), superior sagittal sinus area (SSSA), right and left transverse sinus area (RTSA, LTSA), transverse diameter of right lateral ventricle frontal horn (LVFHD), vertical length and A-P diameter of sella cavity, vertical length of pituitary gland (VLPG) were measured. ROC analysis was performed for the cut off values.

Results: There was statistically significant difference between patient group and control group in terms of ONSD, MCD, SSSA, RTSA, LTSA sella cavity A-P diameter, VLPG vertical length of sella and hypoplasia of dural venous sinus. On the other hand, there was no statistically significant difference between the patient and the control groups regarding LVFHD, OND. ONSD showed an AUC of 0.996 with a cutoff value of 6.4 mm >=, it was found to be highest reliable marker in the differential diagnosis of IIH patients from the controls.

Conclusion: We believe that this study can an important contribution to the diagnosis IIH and follow-up period after its treatment by measuring ONSD, MCD, Sella A-P and VLPG, vertical length of sella and SSSA.

Keywords: Idiopathic intracranial hypertension, magnetic resonance imaging, quantitative evaluation

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a syndrome characterized by intracranial pressure increase without any space occupying lesion. There are other definitions like meningitis serosa or pseudotumor cerebri as well. It usually presents by headache. It can be seen more in obese women who are in fertility period, but it can be observed in each age and gender.[1] The underlying pathophysiological mechanism is not known certainly. The incidence of the disease changes. However, its frequency has raised with respect to the increasing incidence of obesity.[2,3] Diagnosis of the disease is usually achieved through clinical findings, imaging findings and lumbar puncture. It is characterized by symptoms of IIH are severe headache in bilateral frontal and retro-orbital areas, temporary visual disorders, dizziness, diplopia, neck and back pain, nausea and vomiting. As to imaging findings,[4] distention of the optic nerve sheath, distention of the Meckel cave, slit like ventricles, flattened posterior optic globe and empty sella turcica, deformated pituitary gland are the main findings.[5]

The purpose of this study was to evaluate the accuracy of diagnosis by giving the quantitative values of imaging findings of IIH on cranial magnetic resonance imaging (MRI).

METHODS

Patients Subjects

This study included 37 patients who were diagnosed with IIH by using Modified Dandy Criteria (including headache, persistent or progressive visual dysfunction, papilledema, pulsatile tinnitus, greater than 250 mmH_2O CSF opening pressure without ventricular enlargement or intracranial mass on imaging, and normal CSF constituents) and examined for their headaches between January 2016 and May 2021. Images of the patients were scanned retrospectively through PACS system. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its lateramendments or comparable ethical standards. This retrospective study was approved by the institutional review board of the Tokat Gaziosmanpaşa University Faculty of Medicine Dean's Office Clinical Researches Ethics Committee (Date: 31.03.2022, Decision No: 22-KAEK-076).

Patients who were younger than 18, pregnant and who had intracranial mass, hydrocephalus or vascular pathology in brain MRI, who had treatment or medication which can

Corresponding Author: Fatma Kökcü, fatmakokcu79@hotmail.com

cause IIH or have systemic diseases, who had current or prior sinus vein thrombosis was not included in the study. The ones with pregnancy and postpartum status, and who had surgical operation which can change CSF circulation were also excluded from the participants group.

Control Subjects

Twenty-two healthy controls were included by using a match-to-pair technique regarding their sex and age. The ones who have any kind of nervous system disease and have primary headache syndromes like migraine and cluster headache were not included in the study.

MRI Protocol

All patients were scanned with 1.5 Tesla MRI device (1.5-Tesla magnet, GE Signa Excite HD; GE Medical Systems, Milwaukee, WI, USA). The patients were examined using 16 channel neurovascular head coil in the routine axial plane, using the sagittal T2W, transvers propeller T2W (TR: 9000 ms, TE: 90 ms, NEX: 1.0, slice thickness: 5.5 mm, slice spacing: 1.5 mm) coronal FLAIR T2W (TR: 4803 ms, TE: 107ms, NEX: 2.0, slice thickness: 7 mm, slice spacing: 1.5 mm), axial 3D BRAVO (TR: 9.25 ms, TE: 3.58 ms, NEX: 1.0, slice thickness: 1 mm, slice spacing: 0.5 mm) DWI (TR6992 ms, TE: 84.4 ms, NEX: 2.0, slice thickness: 5 mm, slice spacing: 5.5).

Image Analysis

All measurements were done by one neuroradiologist, blinded to patient and control groups and to the medical history, with 15 years of neuroradiology experience. Optic nerve sheath diameter (ONSD) (from widest portion) and optic nerve (ON) thickness (4 mm posterior to the globe) were measured on axial T2W series (Figure 1). Transverse diameter of Meckel cave (MCD) was measured on axial T2W series as well from middle part of cave (Figure 2). Furthermore, superior sagittal sinus area (SSSA) was measured from 1 cm superior of torcular herophili on the same series. Area measurements were done with manuel ROI drawing external contour of sinuses (Figure 3). Right transverse sinus area (RTSA) and left transverse sinus area (LTSA) were also measured in a similar way from sagittal T2W images from the middle part of sinuses.

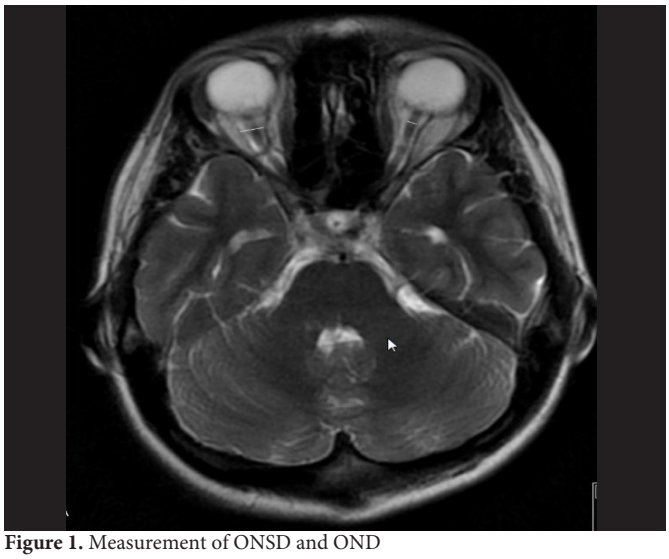

Figure 1. Measurement of ONSD and OND

ONSD: Optic nerve sheath diameter, OND: Optic nerve diameter

Figure 2. Measurement of MCD

MCD: Meckel cave diameter

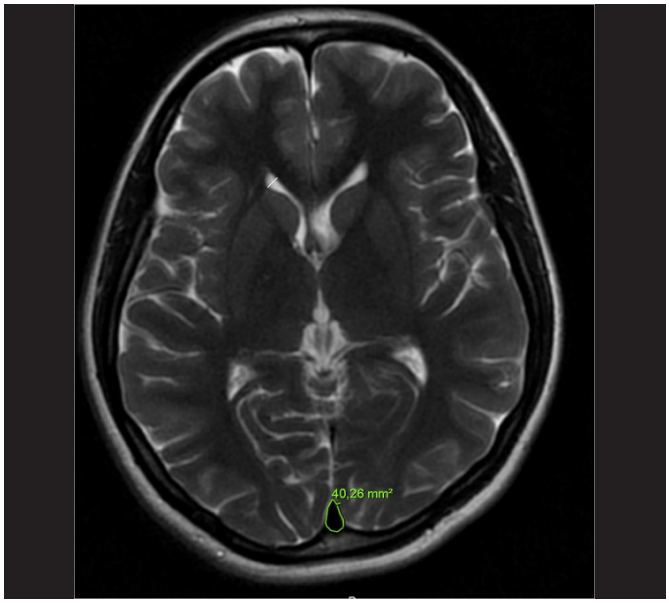

Figure 3. Measurement of LVFHD, area of SSSA

LVFHD: Lateral ventricle frontal horne diameter, SSSA: Superior sagittal sinus area

Transverse sinuses were examined in terms of hypoplasia from MPR images of axial 3D bravo series. Regarding ventricular compression, transverse diameter of right lateral ventricle frontal horn were measured on basal ganglions plane of axial T2W series (Figure 3). The vertical length of sella cavity (VLSC) (from the middle point of baseline to middle point of diaphragma sella) on and A-P diameter of sella cavity (from the tip of anterior clinoid to tip of posterior clinoid) were measured on midsagittal plane of T2W sagittal series (Figure 4). Vertical length of pituitary gland (VLPG) was measured on midsagittal plane.

Statistical Analysis

Statistical analysis was conducted by using SPSS 20 program. As the data did not show normal distribution, a nonparametric Mann-Whitney U test was run to compare the groups. p-value

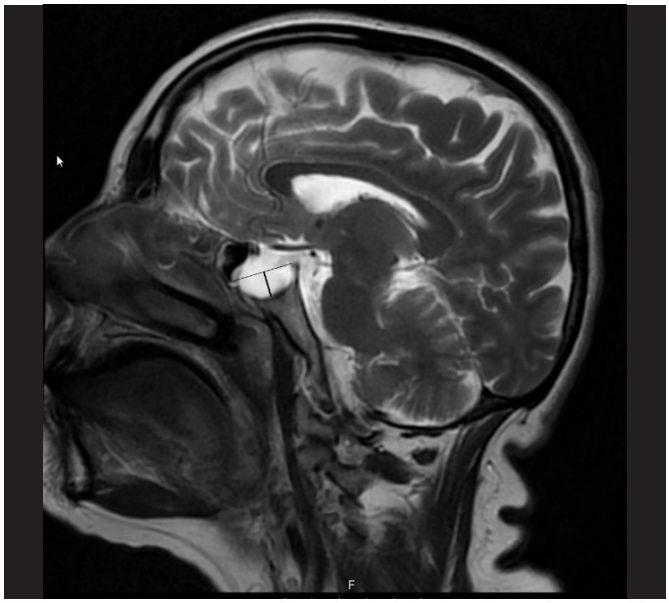

Figure 4. Measurement vertical length (thick line) and A-P diameter (thin line) of sella cavity

was lower than 0.05 and this was interpreted as significant. ROC analysis was performed for the cut off values. ROC curves were used for the comparison of sensitivity and specificity.

RESULTS

In this study total 59 patients consisting of 37 patient group and 22 control group were evaluated. Average age was 39 (18-71) in the patient group and 41 (18-69) in the control group and there was no statistically significant difference. The patient group included 24 female and 13 male patients. As to the control group, there were 13 female and 9 male subjects. Averages and statistical values of the conducted measurements were summarized in Table 1. Here 33 patients reported headaches (89.1%), 24 patients reported visual impairment (64.8%), 18 patients reported dizziness (48.6%), 16 patients (43.2%) reported back and neck pain, 7 patients (18.9%) reported pulsatile tinnitus. Average opening pressure CSF on LP was 380 mmH_2O (280-430). In all patients the CSF constituents was found to be normal.

Table 1. Comparison of measurements in IIH and control group

	IIH (37)		Control (22)		
	Mean	SD	Mean	SD	p
ONSD (mm)	8.0	1.0	5.0	0.6	<0.001
OND (mm)	3.0	0.5	2.9	0.4	0.225
MCD (mm)	7.0	1.7	4.9	1.2	<0.001
LVFHD (mm)	3.5	1.6	4.6	2.5	0.096
Sella A-P diameter (mm)	12.3	2.6	10.7	1.3	0.011
VLSC (mm)	9.2	2.1	7.2	0.9	<0.001
VLPG (mm)	2.7	1.1	5.1	1.9	<0.001
SSSA (mm²)	23.3	11.3	32.1	9.7	0.001
RTSA (mm²)	18.9	14.2	29.1	13.2	0.003
LTSA (mm²)	14.3	15.9	16.0	12.0	0.323

IIH: Idiopathic intracranial hypertension, ONSD: Optic nerve sheath diameter, OND: Optic nerve diameter, MCD: Meckel cave diameter, LVFHD: Lateral ventricle frontal horne diameter, VLSC: Vertical length of sella cavity, VLPG: Vertical length of pituitary gland, SSSA: Superior sagittal sinus area, RTSA: Right transves sinus area, LTSA: Left transves sinus area

It was found that there was statistically significant (p-value <0.005) difference between patient group and control group in terms of ONSD, MCD, SSSA, RTSA, LTSA, A-P diameter of sella cavity, VLSC, VLPG, and hypoplasia of dural venous sinus. On the other hand, there was no statistically significant difference between the patient and the control groups regarding transverse diameter of lateral ventricular frontal horn, transverse diameter of optic nerve. The value of ONSD, MCD, SSSA, RTSA, A-P diameter of sella cavity, VLS and VLPG in the differential diagnosis of IIH positive patients from control subjects was evaluated by the ROC curve. Since ONSD showed an AUC of 0.996 with a cutoff value of 6.4 mm >= (p<0.001), it was found to be highest reliable marker in the differential diagnosis of IIH patients from the controls. Also MCD showed an AUC of 0.850 with a cutoff value of 5.5 mm >= (p<0.001). VLS showed an AUC of 0.826 with a cutoff value of 8,8 mm >= (p<0.001)., A-P diameter of sella showed an AUC of 0.697 with a cutoff value of 11.8 mm >= (p<0.001). VLPG with a cutoff value of <=4mm (p<0.001 AUC:0.888), SSSA with a cutoff value of <=26 mm (p<0.001, AUC:0.750) and RTSA with a cutoff value of <=21mm (p<0.001, AUC:0.729) were reliable markers in the differential diagnosis of IIH patients from the controls too. This relationship was displayed in Figure 5, and Table 2.

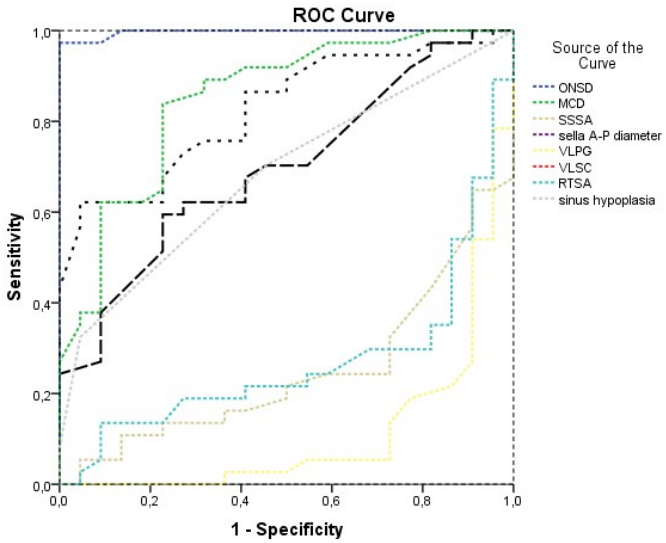

Figure 5. ROC curve of parameters in differential diagnosis of IIH patients for control

ROC: Receiver operating characteristic, IIH: Idiopathic intracranial hypertension

Table 2. ROC of p-value significant parameters in differential diagnosis of IIH patients for control

	Cutoff value	AUC/SD	Specificity	Sensitivity
ONSD (mm)	>=6.4	0.996/0.003	1	0.973
MCD (mm)	>=5.5	0.849/0.052	0.772	0.837
VLSC (mm)	>=8.8	0.826/0.052	0.9545	0.621
VLPG (mm)	<=4	0.887/0.047	0.727	0.945
SSSA (mm²)	<=26	0.750/0.063	0.727	0.756
RTSA (mm²)	<=21	0.729/0.068	0.818	0.702
Sella A-P diameter (mm)	>=11.8	0.697/0.068	0.594	0.772

90-100: Excellent, 80-90: Good, 70-80: Fair, 60-70: Poor, 50-60: Fail, ROC: Receiver operating characteristic, AUC: Area under curve, SD: Standard deviaition, ONSD: Optic nerve sheath diameter, MCD: Meckel cave diameter, VLSC: Vertical length of sella cavity, VLPG: Vertical length of pituitary gland, SSSA: Superior sagittal sinus area, RTSA: Right transves sinus area

When examined in relation to sinus hypoplasia, there was 59% (n: 26) sinus hypoplasia in the patient group and 37% (n: 14) of them were in LTS, 24% (n: 9) were in RTS and 8% (n: 3) were bilateral. 45% (n: 10) rate of sinus hypoplasia was detected in the control group. 40% (n: 9) of them were in LTS 4% (n: 1) were in RTS. There were no patients with bilateral hypoplasia in the control group. Sinus hypoplasia was significantly more in the patient group than it was in the control group (p: 0.012). Rate of LTS hypoplasia was similar in both groups, however RTS hypoplasia was more in the patient group.

DISCUSSION

One of the most common findings related to IIH is the distention of the ONS and tortuosity.[5-7] There has been multiple research investigating this subject and it has been claimed by some studies that distention of the ONS and tortuosity has not changed statistically significantly.[8] In some studies, patients with IIH and healthy people were compared through volume measurement. According to the last study conducted based on volume measurement,[1] when ONS volume is higher than 201 mm^3, it is interpreted as significant for IIH diagnosis. Furthermore, it was emphasized in some studies, which were based on measurement of the ONSD, that increase in the diameter of the ONS is significant for the diagnosis of the IIH and sensitivity and specificity were lower. However, to conduct a volume measurement there should be either an automatic volume measurement program or it should be carried out by conducting semi-automatic volume measurement by drawing ROI through external contour of the ONS on each section. Semi-automatic volume calculation programs are used in the centers with no automatic volume calculation program and this is time-consuming. Therefore, diameters of the ONS and ON were calculated in this study as it was fast and practical. It was claimed in some studies that ON volume does not change in patients with IIH while some assert that it increases in these patients.[1] In this study, there was no difference between two groups in terms of optic nerve diameter. As to the ONSD it was statistically significantly higher in the patient group and it was due to the CSF flow through intraorbital subarachnoid distance as a result of intracranial pressure increase in the patients with IIH. Furthermore, flattening in glob posterior was observed due to the same mechanism.[9] Also, ONSD was the highest reliable marker in the differential diagnosis of IIH patients from the controls with a high AUC. In a patient with suspected intracranial hypertension based on clinical and examination findings when ONSD is higher than 6.4 mm, it can be reported IIH with high sensitivity and specificity.

Compression and decrease in diameter of dural venous sinuses resulting from pressure which is secondary to intracranial pressure increase.[10] Superior sagittal sinus area was measured from 1 cm superior of torcular herophili. When it is compared to the control group, average of superior sagittal sinus area was 23.3 mm^2 while it was 32.1 mm^2 in the control group and there was statistically significant difference. In a similar study Rohr et al.[10] also ran cross-sectional area measurement from superior sagittal sinus. When they compared the situation before and after IIH treatment, they found that sinus area increased after the treatment. In the present study, when SSSA is less than 26 mm^2, it can be reported highly probable IIH according to ROC analysis.

Transverse sinus stenosis is another IIH finding and it has been seen in 30-93% of patients.[11] There is no certain information regarding whether stenosis is a cause or result of IIH. Statistically significant rate of transverse sinus stenosis was found in the study by Horev et al.[1] They measured diameters of transverse sinus before and after lumbar puncture (L-P) and compared the results. It was seen that diameters of transverse sinus significantly increased after L-P. As to the ONS diameter and pituitary gland height obtained after L-P, there was no difference. However our study was based on routine MRI and there was no examination of MR venography of the patients. On the other hand, transverse sinus hypoplasia was significantly more in the patient group (p: 0.012) when an evaluation was conducted in terms of hypoplasia. The kind of hypoplasia which was seen in the patient group was RTS hypoplasia. To get a more optimal examination we conducted measurement of the area instead of diameter, and when we compared the results to the control group's area of RTS was found to be narrower in the patient group. There was no significant difference regarding LTS area. The cause of average area difference in transverse sinuses was actually the fact that RTS hypoplasia was more in the patient group decreased average of area. As LTS hypoplasia was not different in the patient and control groups, average area measurements were not also statistically different.

Distention of the Meckel's cave is another IIH finding.[12,13] The distention is caused by chronic intracranial pressure increase and pouch, which is like meningocele, formed in spaces filled with CSF. On the other hand, there is also literature claiming the narrowing in Meckel's cave in IIH.[14] There is also publications showing that there is no change in MCD.[15] To get a quantitative evaluation, we measured transverse diameters of Meckel's cave at axial section. In this study, it was seen that MCD was statistically significantly wider in the patient group than the control group. In ROC analysis, MCD showed high AUC. If MCD is higher than 5.5 mm in a patient with suspected intracranial hypertension it can be reported highly probable IIH with high sensitivity and specificity

Empty or partial empty sella appearance is a radiological finding supporting IIH. Chronic intracranial pressure increase and increase in CSF pressure cause compression and height decrease in pituitary gland, and remodeling and enlargement in sella bone structure.[16-19] As a result, empty/partial empty sella appearance is formed. When the relation between vertical lengths of pituitary gland and sella were examined in some morphological studies, it was found that statistically significant decrease was seen in the patients with IIH.[18-20] However, Beier et al.[21] claimed in their study that empty sella was not related to IIH. Furthermore, Hoffmann et al.[1] compared normal patients to the patients with IIH by conducting volume measurement of pituitary gland. In this study, we measured vertical length of pituitary gland. Moreover, we measured vertical length of sella cavity and A-P diameter of sella to distinguish it from empty/partial empty sella appearance seen in elderly ages without any relation to IIH. In the patients with IIH, vertical length of pituitary gland

was seen to have decreased, and vertical length of sella cavity and A-P diameter of sella was found to have increased. Both VLSC and VLPG showed good AUC in differentiation of IIH from to control subjects. A-P diameter of sella showed poor AUC.

It has been stated by some studies in the literature that slit like ventricles by ventricular compression is a finding of IIH.[22] On the other hand, some studies claim that ventricular configuration in IIH patients is normal.[23,24] Zheng et al.[25] compared changes of ventricular volume in normal subjects and CVT related IIH. They did not find significant difference in ventricular system volumes between these groups. In this study diameter measurement was conducted at ventricular system to obtain a quantitative evaluation. As in hydrocephalus measurement of diameter was run from lateral ventricle frontal horn. When the patient and control groups were compared, there was no statistically significant difference in terms of diameter of ventricles.

Limitations

Main limitation of this study was that the number of patients was not high and there was no access to detailed clinical information patients such as symptoms duration period. To our knowledge, while there are many studies in the literature on imaging findings of idiopathic intracranial hypertension, the number of studies providing cut-off values is quite limited. However, our study is preliminary, and we believe that cut-off values can be correlated with larger patient groups in larger centers. Furthermore, there was no optimal evaluation in terms of venous stenosis due to the absence of MRI venography.

CONCLUSION

As a result, final diagnosis of IIH is achieved through measurement of CSF pressure after LP although clinical findings and imaging may help the process. We believe that this study can make an important contribution the diagnosis IIH and follow-up period after its treatment by measuring ONSD, MCD, Sella A-P and vertical diameter, vertical length of pituitary gland and SSSA. Sometimes it can be confusing whether it is within normal limits or pathological when reporting IIH cranial MR imaging findings such as increase of ONSD, MCD or decrease of VLPG. According to present study it can be reported highly probable IIH when the measurements are higher than 6.4 mm for optic nerve sheath diameter, 5.5 mm for Meckel cave diameter, 8.8 mm for vertical length of sella and less than 4 mm for vertical length of pituitary gland, 26 mm^2 for superior sagittal sinus area and 21 mm^2 for right transverse sinus area in a patient with suspected intracranial hypertension based on clinical and examination findings.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Tokat Gaziosmanpaşa University Faculty of Medicine Dean's Office Clinical Researches Ethics Committee (Date: 31.03.2022, Decision No: 22-KAEK-076).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

Acknowledgement

Associated Prof. Osman Demir did the statistical analysis.

REFERENCES

1. Hoffmann J, Schmidt C, Kunte H, et al. Volumetric assessment of optic nerve sheath and hypophysis in idiopathic intracranial hypertension. *AJNR Am J Neuroradiol*. 2014;35(3):513-518. doi:10.3174/ajnr.A3694
2. Bénard-Séguin É, Costello F. Idiopathic intracranial hypertension with papilledema. *Neurosurg Clin N Am*. 2025;36(2):281-298. doi:10.1016/j.nec.2024.12.001
3. Zhou C, Zhou Y, Liu L, et al. Progress and recognition of idiopathic intracranial hypertension: a narrative review. *CNS Neurosci Ther*. 2024;30(8):e14895. doi:10.1111/cns.14895
4. Mathew NT, Ravishankar K, Sanin LC. Coexistence of migraine and idiopathic intracranial hypertension without papilledema. *Neurology*. 1996;46(5):1226-1230. doi:10.1212/wnl.46.5.1226
5. Agid R, Farb RI, Willinsky RA, Mikulis DJ, Tomlinson G. Idiopathic intracranial hypertension: the validity of cross-sectional neuroimaging signs. *Neuroradiology*. 2006;48(8):521-527. doi:10.1007/s00234-006-0095-y
6. Brodsky MC, Vaphiades M. Magnetic resonance imaging in pseudotumor cerebri. *Ophthalmology*. 1998;105(9):1686-1693. doi:10.1016/S0161-6420(98)99039-X
7. Manfré L, Lagalla R, Mangiameli A, et al. Idiopathic intracranial hypertension: orbital MRI. *Neuroradiology*. 1995;37(6):459-461. doi:10.1007/BF00600093
8. Gibby WA, Cohen MS, Goldberg HI, Sergott RC. Pseudotumor cerebri: CT findings and correlation with vision loss. *AJR Am J Roentgenol*. 1993;160(1):143-146. doi:10.2214/ajr.160.1.8416612
9. Acheson JF. Idiopathic intracranial hypertension and visual function. *Br Med Bull*. 2006;79-80:233-244. doi:10.1093/bmb/ldl019
10. Rohr A, Bindeballe J, Riedel C, et al. The entire dural sinus tree is compressed in patients with idiopathic intracranial hypertension: a longitudinal, volumetric magnetic resonance imaging study. *Neuroradiology*. 2012;54(1):25-33. doi:10.1007/s00234-011-0850-6
11. Ball AK, Clarke CE. Idiopathic intracranial hypertension. *Lancet Neurol*. 2006;5(5):433-442. doi:10.1016/S1474-4422(06)70442-2
12. Bialer OY, Rueda MP, Bruce BB, Newman NJ, Biousse V, Saindane AM. Meningoceles in idiopathic intracranial hypertension. *AJR Am J Roentgenol*. 2014;202(3):608-613. doi:10.2214/AJR.13.10874
13. San Millán D, Kohler R. Enlarged CSF spaces in pseudotumor cerebri. *AJR Am J Roentgenol*. 2014;203(4):W457-W458. doi:10.2214/AJR.14.12787
14. Degnan AJ, Levy LM. Narrowing of Meckel's cave and cavernous sinus and enlargement of the optic nerve sheath in Pseudotumor Cerebri. *J Comput Assist Tomogr*. 2011;35(2):308-312. doi:10.1097/RCT.0b013e31820d7a70

15. Balık AÖ, Akıncı O, Yıldız S, Hasırcı Bayır BR, Ulutaş C. Role of neuroimaging markers on predicting of idiopathic intracranial hypertension. *Acta Radiol*. 2024;65(8):999-1006. doi:10.1177/02841851241256008
16. Kyung SE, Botelho JV, Horton JC. Enlargement of the sella turcica in pseudotumor cerebri. *J Neurosurg*. 2014;120(2):538-542. doi:10.3171/2013.10.JNS131265
17. Ranganathan S, Lee SH, Checkver A, et al. Magnetic resonance imaging finding of empty sella in obesity related idiopathic intracranial hypertension is associated with enlarged sella turcica. *Neuroradiology*. 2013;55(8):955-961. doi:10.1007/s00234-013-1207-0
18. Yuh WT, Zhu M, Taoka T, et al. MR imaging of pituitary morphology in idiopathic intracranial hypertension. *J Magn Reson Imaging*. 2000;12(6):808-813. doi:10.1002/1522-2586(200012)12:6<808::aid-jmri3>3.0.co;2-n
19. Kesler A, Yaffe D, Shapira M, Kott E. Optic nerve sheath enlargement and reversal of optic nerve head in pseudotumor cerebri. *Harefuah*. 1996;130(7):457-503.
20. Korkmazer B, Karaman AK, Kızılkılıç EK, et al. Efficacy of dural sinus quantitative measurements in idiopathic intracranial hypertension: a practical diagnostic feature. *Clin Neuroradiol*. 2023;33(2):545-554. doi:10.1007/s00062-022-01244-0
21. Beier D, Korsbæk JJ, Bsteh G, et al. Magnetic resonance imaging signs of idiopathic intracranial hypertension. *JAMA Netw Open*. 2024;7(7):e2420138. doi:10.1001/jamanetworkopen.2024.20138
22. Weisberg LA. Computed tomography in benign intracranial hypertension. *Neurology*. 1985;35(7):1075-1078. doi:10.1212/wnl.35.7.1075
23. Agid R, Farb RI, Willinsky RA, Mikulis DJ, Tomlinson G. Idiopathic intracranial hypertension: the validity of cross-sectional neuroimaging signs. *Neuroradiology*. 2006;48(8):521-527. doi:10.1007/s00234-006-0095-y
24. Arkoudis NA, Davoutis E, Siderakis M, et al. Idiopathic intracranial hypertension: imaging and clinical fundamentals. *World J Radiol*. 2024;16(12):722-748. doi:10.4329/wjr.v16.i12.722
25. Zheng YM, Chen J, Yuan MG, Wu ZJ, Dong C. Does a change in ventricular size predict a diagnosis of cerebral venous thrombosis-related acute intracranial hypertension? Results of a retrospective imaging study. *Acta Radiol*. 2019;60(10):1308-1313. doi:10.1177/0284185118823346