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Original Article

Patients of idiopathic normal-pressure hydrocephalus have small dural sac in cervical and upper thoracic levels: A supposed causal association

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ABSTRACT

Background: Idiopathic normal pressure hydrocephalus (iNPH) is a neurological disorder presenting a triad including dementia and ventricular enlargement. The mechanism causing excessive cerebrospinal fluid (CSF) accumulation in the ventricles in iNPH is poorly understood. We hypothesized that the age-related degradation of the spinal shock-absorbing system composed of a spinal dural sac (SDS) and surrounding soft tissue, preventing ventricular enlargement caused by wide CSF pulsation driven by heartbeats, may be involved in the ventricular enlargement observed in iNPH.

Methods: Sixty-four patients with iNPH in their seventies who underwent a lumboperitoneal shunt and a control group of 79 people in the same age group who underwent brain check-ups were included in the study. We compared the sizes of the cervical and upper parts of the thoracic SDS using magnetic resonance imaging between the two groups.

Results: The anterior-posterior distances of the dural sac at C5 were shorter in patients with iNPH of both sexes than those in the control group ($P = 0.0008$ in men and $P = 0.0047$ in women). The number of disc levels with disappeared CSF space surrounding the cervical cord was more in iNPH ($P = 0.0176$ and $P = 0.0003$). The midsagittal area of the upper part of the spinal sac, C2-Th4, was smaller in iNPH ($P = 0.0057$ and $P = 0.0290$).

Conclusion: Narrowing of the cervical dural sac and midsagittal area in the upper part of the SDS in patients with iNPH may reflect the degradation of the shock-absorbing mechanism for CSF pressure pulsations, which may cause iNPH or at least aggravate iNPH by other unknown causes.

Keywords: Cervical spondylosis, Idiopathic normal pressure hydrocephalus, Shock absorber, Spinal dural sac

INTRODUCTION

Idiopathic normal pressure hydrocephalus (iNPH) is a neurological disorder characterized by nonobstructive ventricular enlargement without a precedent causative event and mainly presents with disturbances in gait, micturition control, and cognition. As it primarily affects older adults, 60–80 years old, its prevalence is increasing in aging societies.[1,5] However, the mechanism of

ventricular enlargement without a particular cause, such as subarachnoid hemorrhage or obstruction of cerebrospinal fluid (CSF) flow, has yet to be elucidated.[4,6,11-13,16,17,24,26]

Through the treatment of a large number of iNPH patients, 40–60 patients a year, at our Normal Pressure Hydrocephalus Center, Atsuchi Neurosurgical Hospital, Kagoshima, Japan, we frequently encounter iNPH patients with spinal column disorders, including cervical spondylosis, leading to narrowing of the spinal dural sac (SDS). Due to the high incidence of such an accompaniment, we hypothesized that the narrowing of the SDS is not a mere coincidence associated with iNPH but a causative or aggravating factor. In addition, our previous study on spontaneous intracranial hypotension (SIH) showed that the SDS shrinks even in the early stages of the disease, suggesting that the SDS acts as a shock absorber for intracranial CSF pressure change.[7] Therefore, we hypothesized that the degradation of this physiological shock-absorbing mechanism of the SDS for intracranial CSF pressure enhances the intracranial CSF pressure pulsations driven by heartbeats, eventually resulting in the enlargement of the ventricles and some parts of the cisterns. To test this hypothesis, we compared the size of the SDS on magnetic resonance imaging (MRI) in iNPH patients in their seventies and examinees of brain check-ups (BCU) in the same age range as controls.

MATERIALS AND METHODS

At our normal pressure hydrocephalus center, Atsuchi Neurosurgical Hospital, 96 patients underwent lumbar-peritoneal shunt surgery for iNPH between January 2021 and July 2022. Among them, 64 consecutive patients (36 men and 28 women) in their seventies, the largest age group, were selected for this study. For the controls, 79 participants (50 men and 29 women) in the same age group were selected from the 661 people who underwent BCU in our hospital during the same period. In the patient group, in addition to brain scans, cervical-level magnetic resonance (MR) images and upper thoracic-level MR images were routinely obtained to rule out cryptic causes of hydrocephalus. In the control group, in addition to whole brain scans, T2-weighted midsagittal cervical MR images were routinely obtained in all 79 examinees, while T2-weighted midsagittal MR images at the upper thoracic level were obtained in 55.

As a surrogate for the total volume of the SDS, we evaluated the above mentioned size of the upper part of the SDS, cervical, and upper thoracic vertebral levels in both groups.

Neuroradiologic features, including the Evans index, distances from the vertebral body to the spinous process at the mid-C5 level, number of disc levels with vanished CSF space surrounding the cervical cord, and midsagittal cervical and upper thoracic intradural area (C2 bottom-Th4 bottom), were compared between the two groups for each sex [Figure 1]. We chose the C2 bottom instead of the C1 top for the upper margin of the area because the latter was frequently obscured in our routine scans.

Statistical analyses

Statflex® version 6.0 software program (Artech Japan) was used for statistical analysis of the results. Data were analyzed using the Mann–Whitney U-test. Differences were considered statistically significant at $P < 0.05$.

Ethical consideration

This noninterventional study was approved by the hospital's medical ethics committee (R4-2, October 2022). This study was conducted according to the principles of the Declaration of Helsinki, as revised in 2000, and the ethical guidelines for medical and health research involving human subjects (effective February 9, 2015) promulgated by the Ministry of Health, Welfare and Labor, Japan. The data were analyzed in a de-identified manner to protect patient privacy.

RESULTS

The Evans index was significantly higher in the iNPH group than that in the BCU group in both sexes: men (median 0.34 vs. 0.28, $P < 0.001$) and women (median 0.34 vs. 0.25, $P < 0.001$) [Table 1].

The distances from the midvertebral body to the spinous process at the C5 level were significantly shorter in the iNPH group than those in the BCU group in both sexes: men (median 10.2 vs. 11.4 mm, $P = 0.0008$) and women (median 10.2 vs. 11.0 mm, $P = 0.0047$) [Figure 2a].

The number of disc levels with vanished CSF space surrounding the cervical cord was significantly more in the

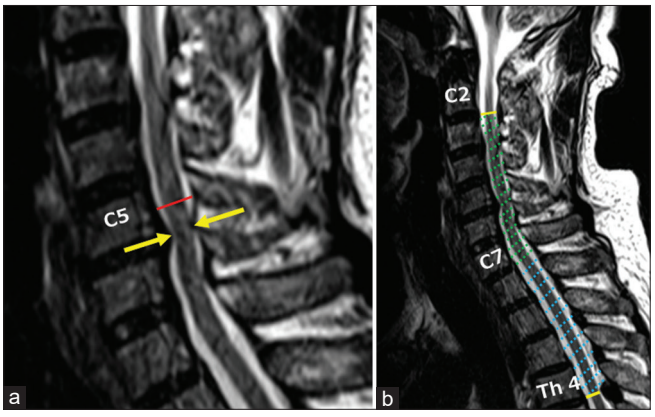

Figure 1: Measurements of magnetic resonance imaging features at cervical and upper thoracic levels. (a) Red line: Distance (mm) from the mid-C5 vertebral body to the mid-C5 spinous process. Yellow arrows: Number of disc levels at which the CSF space surrounding the cervical spinal cord vanishes, one in this case. (b) Dotted area: Midsagittal area (mm^2) from C2 bottom to Th4 bottom.

Table 1: Features on Magnetic Resonance Imaging

			Age (year)	Evans index	Distance at C5 (mm)	Disappeared CSF around cervical spinal cord (number of level)		Cervical+Upper thoracic mid-sagittal area[#] (mm^2)
iNPH	Total=64	Median (IQR)	75 (72-77)	0.34(0.31-0.35)	10.2 (9.4-11.1)	1 (0-2)		1798 (1709-1986)
		Mean	74.7	0.33	10.2	1.30		1842
	Men=36	Median (IQR)	74 (71-76)	0.34 (0.31-0.36)[※]	10.2 (9.4-11.2)[※]	1 (0-2)[※]		1898 (1783-2052)[※]
		Mean	74.1	0.34	10.2	1.44		1912
	Women=28	Median (IQR)	77 (74-78)	0.34 (0.31-0.35)[※]	10.2 (9.4-11.0)[※]	0 (0-1)		1754 (1676-1819)[※]
		Mean	75.5	0.33	10.2	1.11		1740
Brain Check-up (control)	Total=79	Median (IQR)	73 (71-75)	0.27 (0.25-0.28)	11.1(10.3-12.2)	0 (0-1)	*n*=55[§]	1946 (1804-2077)
		Mean	73.1	0.27	11.6	0.51		1936
	Men=50	Median (IQR)	73 (71-76)	0.28 (0.26-0.29)	11.4 (10.2-12.2)	0 (0-1)	*n*=30[§]	2016 (1936-2151)
		Mean	73.5	0.27	11.2	0.56		2019
	Women=29	Median (IQR)	72 (70-74)	0.25 (0.24-0.27)	11.0 (10.4-11.8)	0 (0-1)	*n*=25[§]	1824 (1742-1921)
		Mean	72.4	0.26	11.1	0.41		1832

Values are presented with median (IQR: interquartile range) and mean, CSF: Cerebrospinal fluid, iNPH: idiopathic normal pressure hydrocephalus, EI: Evan's index on axial scan of brain magnetic resonance imaging, [※]: Statistically significant difference between iNPH patients and control of same gender ($p<0.05$), [#]: Mid-sagittal area from C2 bottom to Th4 bottom level, [§]: Number of the measured area was smaller than total due to the lack of imaging of upper thoracic level in some brain check-up examinee

iNPH group than in the BCU group in both sexes: men (mean 1.44 vs. 0.56; median one vs. 0, $P = 0.0176$) and women (mean 1.11 vs. 0.41; median one vs. 0, $P = 0.0003$) [Figure 2b].

The midsagittal cervical to the upper thoracic intradural area (C2 bottom-Th4 bottom) was significantly smaller in the iNPH group than that in the BCU group in both sexes; men (median 1898 vs. 2016 mm^2, $P = 0.0057$) and women (median 1754 vs. 1824 mm^2, $P = 0.0290$) [Figure 3].

DISCUSSION

In this study, as only the sagittal MR images of the upper part of the spine that are included in routine imaging for iNPH and BCU were available, we measured the size of the upper part of the SDS as a surrogate of the whole volume of the SDS. We found a significantly narrow subarachnoid space surrounding the cervical spinal cord and a smaller SDS in patients with iNPH compared to examinees of the BCU. These results are consistent with our previous observation of the high prevalence of a narrow spinal canal in patients with iNPH.[9,15,23]

The mechanism of hydrocephalus development without a particular cause remains unclear. Some researchers have argued that age-related decay in the CSF absorption mechanism involving arachnoid granules may play a role in developing iNPH.[4,6,11,12,16,17,24,26] However, the reason for the "normality of CSF pressure" is not explained by this theory.

Before discussing the mechanism underlying the development of iNPH, we consider the following facts:

First, the brain expands and shrinks due to changes in the intracerebral blood volume according to the cardiac beat. These brain volume changes cause wide CSF pulsations, providing wide-pressure pulsations on the brain surface.[20,25]

Second, an animal model of a unilateral large craniotomy of the normal brain (decompressed brain) showed enlargement of the ventricle on the decompressed side, suggesting that a large movement of the brain results in enlargement of the ventricles.[14] Recent research has found that large CSF pulsation pressure on the ventricular wall causes enlargement of the ventricles in cases of obstructed CSF flow, noncommunicating hydrocephalus.[18]

Third, the CSF volume in the SDS is larger than that in the cranium, at approximately 75 mL versus 25 mL.[2,3,10,13,19,21,22] Intracranial CSF can enter and exit the SDS quickly through the foramen magnum (FM) and subarachnoid space surrounding the cervical spine.

Fourth, the spinal epidural space between the two layers of the dura mater contains soft tissue, including fat and venous plexus without valves, functioning like a cushion. Therefore, the SDS may act as a shock absorber that lessens the intracranial CSF pressure pulsations driven by the cardiac beats. Our previous study also suggested the existence of this mechanism; the SDS shrinks before changes in intracranial structures due to low CSF

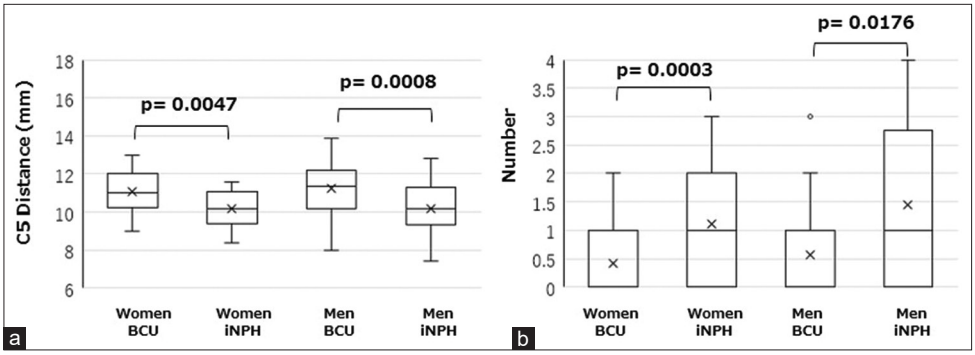

Figure 2: Comparison of cervical magnetic resonance imaging findings between patients with idiopathic normal-pressure hydrocephalus (iNPH) and controls (brain check-up (BCU), examinees of brain check-up). (a) The distance from the mid-C5 vertebral body to the mid-C5 spinous process in patients with iNPH was significantly shorter than in controls of both sexes. (b) The number of disc levels with vanished cerebrospinal fluid space surrounding the cervical spinal cord was significantly higher in patients with iNPH than in the controls of both sexes. The values are presented by box plot.

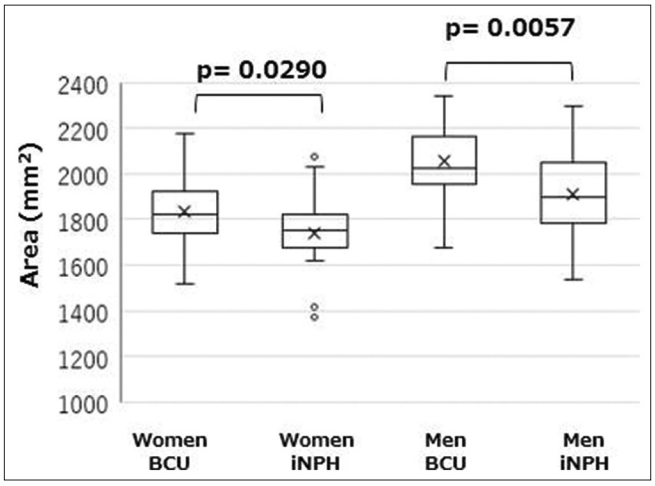

Figure 3: Midsagittal area of the upper level of spinal dural sac (from C2 bottom to Th4 bottom). The area was significantly smaller in patients with idiopathic normal-pressure hydrocephalus than in controls (brain check-up) of both sexes. The values are presented by box plot.

pressure in patients with SIH. In contrast, the two layers of the dura mater in the cranium are almost inseparable and firmly attached to the skull; thus, they do not act as shock absorbers.[7,8]

Therefore, if the cervical subarachnoid space becomes narrow enough to prevent the CSF from moving freely or the whole SDS smaller, the spinal shock-absorbing mechanism cannot cancel the large intracranial CSF pressure pulsations. This degradation of the shock-absorbing mechanism may enhance CSF pressure pulsations on the inner and outer brain surfaces, eventually resulting in the ventricular and subarachnoid space enlargement typically seen in iNPH patients. This speculation seems supported by the fact that iNPH occurs primarily in older adults in their 60s to 80s, in whom degenerative spondylosis is common.[1,16]

The development mechanism of iNPH uses a dual-chamber model, intracranial CSF space, and spinal CSF space [Figure 4]. Changes in the intracerebral blood volume driven by cardiac beats can cause wide pulsations of the CSF pressure (black arrow on B1 in [Figure 4a]). However, under normal conditions, intracranial CSF can quickly move into the large spinal CSF space in the SDS through the FM and cervical subarachnoid space during the systolic phase and move out during the diastolic phase (green arrow in [Figure 4a]). The SDS and epidural soft tissue can absorb the wide pulsations of the CSF pressure (blue arrows in [Figure 4a]). Due to this pressure-absorbing mechanism, the pressure pulsations on the brain surface, ependyma, and pia mater are largely canceled (red arrow on B2 in [Figure 4a]).

When spinal degenerative changes occur, the narrowing of the cervical subarachnoid space causes a decrease in flow velocity. It limits the movement of the CSF into and out of the SDS (green arrow in [Figure 4b]). The degenerative changes may also decrease the volume of the SDS and surrounding epidural soft tissue. These changes result in a diminished CSF pressure-absorbing mechanism (blue arrows in [Figure 4b]) and increased CSF pressure pulsations on the brain surface (red arrow on B2 in [Figure 4b]).

As a result, the increased CSF pressure pulsations increase the ventricle sizes and some parts of the cisterns [Figure 4c], which is typically observed in patients with iNPH. Disfigurement of the cerebrum may impair its function. The increase in the size of the ventricular and cisternal spaces results in weakened CSF pressure pulsations on the enlarged brain surface (red arrow on B2 in [Figure 4c]), resulting in the steadiness of the size of the ventricles and cisterns.

Limitations

There were many inherent limitations in this retrospective survey.

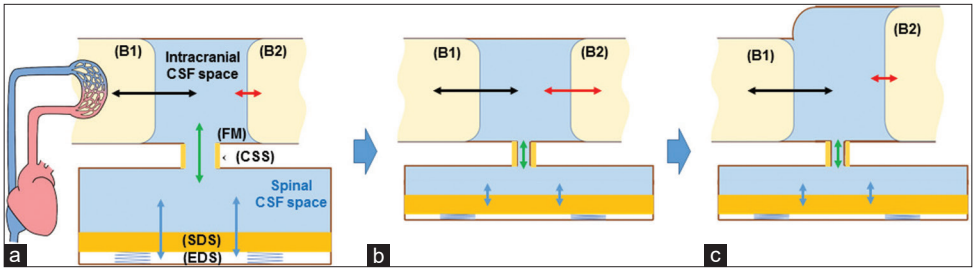

Figure 4: Conceptual illustration of causative relationship between narrow spinal dural sac (SDS) and idiopathic normal pressure hydrocephalus (iNPH). (a) The Physiological pressure-absorbing mechanism that prevents the brain surface (B2) from receiving large cerebrospinal fluid (CSF) pressure pulsations (black arrow) driven by intracerebral blood volume changes (B1). The pulsations are largely canceled (red arrow) by large spinal dural sac (SDS) pulsations (blue arrows) surrounded by the epidural space (EDS) including soft tissue, owing to a quick CSF shift (green arrow) through the foramen magnum (FM) and cervical subarachnoid space (CSS). B1: Brain model generating CSF pressure pulses; B2: Brain model receiving CSF pressure pulses. (b) When the cervical part of the dural sac or the total dural sac becomes smaller due to vertebral spondylosis, the cancelation mechanism by CSF shift (green arrow) through foramen magnum will be diminished (blue arrows), and the brain surface (B2) will directly receive large CSF pressure pulsations (red arrow). (c) Large CSF pressure pulsations cause enlargement of the inner and outer brain surfaces (B2), leading to iNPH. The enlargement of the brain surface itself lessens the CSF pulsations (red arrow), leading to a steady width of the brain surface. Black arrow: CSF pressure driven by intracerebral blood volume changes, Green arrow: diminished CSF shift through foramen magnum.

First, we did not measure the volume of the entire SDS or the cervical subarachnoid space volume.

Second, an association between iNPH and various patient clinicopathological features, such as cerebral white matter disease, small vessel disease, arteriosclerosis, hypertension, hypercholesterolemia, diabetes, and excessive alcohol consumption, has been reported.[4,6] In addition, CSF production-absorption imbalance, abnormal cerebrovascular blood flow reactivity increased intracranial pressure wave amplitude, and several genetic mutations have been suggested as causative of the disease.[11,12] However, the present study did not analyze these factors or validate the accuracy of previous suggestions. Therefore, these factors may play a major role in the development of iNPH, and the narrow SDS mechanism may play an ancillary role, only aggravating the disease.

Third, the exact mechanism by which wide CSF pressure pulsations cause enlargement of the brain surface and impair brain function is not well understood.

Therefore, longitudinal prospective follow-up of patients with vertebral spondylosis using modern imaging techniques and elaborate cognitive function tests may help to understand the mechanism of iNPH development.

CONCLUSION

Our retrospective comparison between patients with iNPH and examinee of BCU, both in their seventies, revealed a narrow CSF cervical subarachnoid space and upper SDS in patients with iNPH. These changes in patients with iNPH may indicate degradation of the shock absorber, composed of the SDS and surrounding epidural tissue, compensating for the wide intracranial pressure pulsations. The shock absorber decay may play an important role in developing iNPH. Future prospective studies on patients with spondylosis must validate our findings.

Ethical approval

The author(s) declare that they have taken the ethical approval from IEC.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The author confirms that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Andersson J, Rosell M, Kockum K, Lilja-Lund O, Söderström L, Laurell K. Prevalence of idiopathic normal pressure hydrocephalus: A prospective, population-based study. PLoS One 2019;14:e0217705.
2. Cushing H. Studies on the cerebro-spinal fluid: I. Introduction. J Med Res 1914;31:1-19.
3. Edsbagge M, Starck G, Zetterberg H, Ziegelitz D, Wikkelso C. Spinal cerebrospinal fluid volume in healthy elderly individuals. Clin Anat 2011;24:733-40.
4. Engel DC, Pirpamer L, Hofer E, Schmidt R, Brendle C. Incidental findings of typical iNPH imaging signs in asymptomatic subjects with subclinical cognitive decline. Fluids Barriers CNS 2021;18:37.
5. Hiraoka K, Meguro K, Mori E. Prevalence of idiopathic normal-pressure hydrocephalus in the elderly population of a Japanese rural community. Neurol Med Chir (Tokyo) 2008;48:197-99.
6. Israelsson H, Larsson J, Eklund A, Malm J. Risk factors, comorbidities, quality of life, and complications after surgery in idiopathic normal pressure hydrocephalus: Review of the INPH-CRasH study. Neurosurg Focus 2020;49:E8.
7. Kawahara T, Arita K, Fujio S, Hanaya R, Atsuchi M, Moinuddin FM, *et al.* Dural sac shrinkage signs on magnetic resonance imaging at the thoracic level in spontaneous intracranial hypotension-its clinical significance. Acta Neurochir (Wien) 2021;163:2685-94.
8. Kawahara T, Atsuchi M, Arita K, Fujio S, Higa N, Moinuddin FM, *et al.* Dural sac shrinkage signs on spinal magnetic resonance imaging indicate overdrainage after lumboperitoneal shunt for idiopathic normal pressure hydrocephalus. Surg Neurol Int 2022;13:269.
9. Kawahara T, Atsuchi M, Arita K, Fujio S, Higa N, Hanaya R. Paravertebral cerebrospinal fluid exudation in young women with postdural puncture headache: A hypothetical interpretation based on anatomical study on intervertebral foramen. Asian J Neurosurg 2023;18:117-24.
10. Last RJ, Tompsett DH. Casts of the cerebral ventricles. Br J Surg 1953;40:525-43.
11. Leinonen V, Vanninen R, Rauramaa T. Cerebrospinal fluid circulation and hydrocephalus. Handb Clin Neurol 2017;145:39-50.
12. Leinonen V, Kuulasmaa T, Hiltunen M. iNPH-the mystery resolving. EMBO Mol Med 2021;13:e13720.
13. Matsumae M, Kikinis R, Mórocz I, Lorenzo AV, Albert MS, Black PM, *et al.* Intracranial compartment volumes in patients with enlarged ventricles assessed by magnetic resonance-based image processing. J Neurosurg 1996;84:972-81.
14. Matsumoto T, Nagai H, Fukushima T, Mase M. Analysis of intracranial pressure pulse wave in experimental hydrocephalus. Childs Nerv Syst 1994;10:91-5.
15. Nakajima M, Kuriyama N, Miyajima M, Ogino I, Akiba C, Kawamura K, *et al.* Background risk factors associated with shunt intervention for possible idiopathic normal pressure hydrocephalus: A nationwide hospital-based survey in Japan. J Alzheimers Dis 2019;68:735-44.
16. Nakajima M, Yamada S, Miyajima M, Ishii K, Kuriyama N, Kazui H, *et al.* Guidelines for management of idiopathic normal pressure hydrocephalus (Third Edition): Endorsed by the Japanese society of normal pressure hydrocephalus. Neurol Med Chir (Tokyo) 2021;61:63-97.
17. Oreskovic D, Klarica M, Vukić M. The formation and circulation of cerebrospinal fluid inside the cat brain ventricles: A fact or an illusion? Neurosci Lett 2002;327:103-6.
18. Preuss M, Hoffmann KT, Reiss-Zimmermann M, Hirsch W, Merkenschlager A, Meixensberger J, *et al.* Updated physiology and pathophysiology of CSF circulation--the pulsatile vector theory. Childs Nerv Syst 2013;29:1811-25.
19. Sakka L, Coll G, Chazal J. Anatomy and physiology of cerebrospinal fluid. Eur Ann Otorhinolaryngol Head Neck Dis 2011;128:309-16.
20. Stoquart-ElSankari S, Balédent O, Gondry-Jouet C, Makki M, Godefroy O, Meyer ME. Aging effects on cerebral blood and cerebrospinal fluid flows. J Cereb Blood Flow Metab 2007;27:1563-72.
21. Telano LN, Baker S. Physiology, cerebral spinal fluid. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2023.
22. Tsunoda A, Mitsuoka H, Sato K, Kanayama S. A quantitative index of intracranial cerebrospinal fluid distribution in normal pressure hydrocephalus using an MRI-based processing technique. Neuroradiology 2000;42:424-9.
23. Tucker A, Kajimoto Y, Ohmura T, Ikeda N, Furuse M, Nonoguchi N, *et al.* Fluoroscopic-guided paramedian approach for lumbar catheter placement in cerebrospinal fluid shunting: Assessment of safety and accuracy. Oper Neurosurg (Hagerstown) 2019;16:471-7.
24. Weller RO, Djuanda E, Yow HY, Carare RO. Lymphatic drainage of the brain and the pathophysiology of neurological disease. Acta Neuropathol 2009;117:1-14.
25. Yamada S, Tsuchiya K, Bradley WG, Law M, Winkler ML, Borzage MT, *et al.* Current and emerging MR imaging techniques for the diagnosis and management of CSF flow disorders: A review of phase-contrast and time-spatial labeling inversion pulse. AJNR Am J Neuroradiol 2015;36:623-30.
26. Yamada S, Mase M. Cerebrospinal fluid production and absorption and ventricular enlargement mechanisms in hydrocephalus. Neurol Med Chir (Tokyo) 2023;63:141-51.

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Commentary

This is an interesting clinical study where the investigators performed upper spine MRI studies on iNPH patients and an age-matched "control" cohort. They hypothesized that iNPH had a more narrowed cervical-upper thoracic spinal thecal sac compared to non-iNPH subjects. By multiple measures, they found statistically significant differences supporting the hypothesis.

The authors acknowledge that a methodological limitation is that the analysis was limited to midline sagittal T2 imaging rather than volumetric CSF axial studies. I agree. This is just one of many unanswered questions. Is the spinal stenosis acquired (degenerative) or congenital, or both? Why was the analysis limited to the upper spine? Should not the same hypothesis pertain to the entire spine?

The authors also acknowledge that the etiology of iNPH is unknown and almost certainly multifactorial. Stated otherwise, it is likely a "multihit" phenomenon. Their data rather convincingly suggest that spinal stenosis may be one of the "hits." However, they do not acknowledge that there are millions of patients with cervical spinal stenosis, much worse than demonstrated here, who do not have iNPH.

In the discussion, the authors present a series of "facts." I would argue that none of them are "facts" but instead speculations and observations. For example, the statement that the brain expands and contracts with changes in CBV is misleading. Most of the arterial vasculature runs in the subarachnoid and cisternal spaces. These spaces likely contribute to the expansion/contraction much more than the brain parenchyma itself, where only tiny perforators run. Therefore, the next statement as to the source of CSF pulsations is in question as well.

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