KOZIKOWSKA, Marta, POCIECHA, Agnieszka and KMAK, Bożena. Diagnosis, clinical symptoms and treatment of Hakim-Adams syndrome. Quality in Sport. 2024;17:53920. eISSN 2450-3118.
https://dx.doi.org/10.12775/QS.2024.17.53920
https://apcz.umk.pl/QS/article/view/53920

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 30.07.2024. Revised: 06.08.2024. Accepted: 11.08.2024. Published: 16.08.2024.

Diagnosis, clinical symptoms and treatment of Hakim-Adams syndrome

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ABSTRACT

Introduction and purpose

Normal pressure hydrocephalus (NPH) is a chronic and progressive neurological disorder that affects the accumulation of cerebrospinal fluid (CSF) in the brain's ventricles. The disease is not limited by age, but it is more prevalent in older adults. The accumulation of excess CSF in the brain's ventricles is caused by the obstruction of normal CSF flow, leading to an enlargement of the ventricles and pressure on brain structures. NPH manifests as cognitive disorders, gait disorders, and urinary incontinence. NPH is considered a potentially reversible cause of dementia. This study aims to discuss the etiology, symptoms, differential diagnosis, and treatment methods associated with normotensive hydrocephalus.

Materials and methods

A comprehensive literature review was conducted through an extensive bibliographic search, with a primary focus on original research articles obtained from reputable databases such as PubMed, BioMed Central, Polish Medical Platform, and Google Scholar. The search was specifically targeted towards articles pertaining to Normal Pressure Hydrocephalus (NPH).

Conclusions

Normotensive hydrocephalus (NPH) is a complex neurological condition that is characterized by an imbalance between the production and absorption of cerebrospinal fluid (CSF). NPH is classified into idiopathic and secondary types, which helps to determine the possible underlying causes and mechanisms that contribute to the development of the condition. The clinical manifestations of NPH are highly variable, and the diagnostic process can be complicated due to the need to differentiate it from other neurodegenerative disorders. Underdiagnosis or misdiagnosis is common, leading to a decrease in patients' quality of life and, in many cases, disability.

The prevalence of NPH is increasing, highlighting the need for further research and the development of novel therapeutic approaches to improve patient outcomes.

Keywords:

normotensive hydrocephalus, Hakim-Adams syndrome, cerebrospinal fluid

INTRODUCTION

Normotensive hydrocephalus is a clinical syndrome that is characterized by the classic triad of symptoms, also known as Adams' triad or Hakim's triad [1,2]. This triad consists of gait disturbances, cognitive disturbances, and urinary incontinence or frequent urination. This form of hydrocephalus is a type of communicating hydrocephalus, where excess cerebrospinal fluid accumulates in the ventricles, leading to an enlargement of the chambers and an increase in pressure inside the skull. This, in turn, compresses surrounding tissues, resulting in neurological complications [2,4].

It is important to note that communicating hydrocephalus is distinguished from obstructive or non-communicating hydrocephalus by the lack of structural obstruction to the flow of cerebrospinal fluid, such as stenosis of the aqueduct of Sylvius. Normotensive hydrocephalus can occur idiopathically (known as iNPH) or can be caused by a disturbance in the circulation of cerebrospinal fluid due to the presence of scars caused by previous haemorrhage or trauma, as well as after meningitis or infection [3].

History

Normotensive hydrocephalus, a condition first described by the Colombian neurosurgeon Salomon Hakim in 1956, is often characterized by the classic triad of symptoms: gait disturbance, cognitive impairment, and urinary incontinence. The syndrome was initially identified in a 16-year-old male patient who had suffered a severe head injury in a car accident. Following surgical treatment for subdural hematoma, the patient remained semi-comatose, prompting tests that revealed ventricular enlargement and intracranial pressure within the upper normal range. A ventricular-atrial shunt implantation procedure was then performed, resulting in significant improvement. This case was subsequently documented in The New England Journal of Medicine in 1964. Since then, Hakim's work has been instrumental in furthering our understanding of normotensive hydrocephalus and its clinical manifestations [4,5].

Epidemiology

The incidence of normotensive hydrocephalus is a phenomenon that has been studied extensively with varying results. The prevalence ranges from 2 to 20 million on an annual basis, depending on the source. Of these cases, approximately 50% are classified as primary or idiopathic NPH, while secondary NPH may occur across all age groups. The incidence of idiopathic NPH is estimated to be 0.3-3% in patients over the age of 60 and increases with age. The risk of NPH does not seem to differ significantly between genders or ethnic groups. However, in individuals diagnosed with dementia, the incidence of normotensive hydrocephalus ranges from 2-6%. The highest risk of this disease is typically observed in individuals in their 70s and 80s, as reported in the literature [5,6].

Pathophysiology

Hydrocephalus is a medical condition that arises from an imbalance between the production and absorption of cerebrospinal fluid (CSF) in the brain. This results in an accumulation of CSF in the ventricles, causing an increase in intracranial pressure (ICP) [7]. The enlarged ventricles exert significant pressure on the adjacent cortical tissue, causing various effects on the patient, including distortion of fibers in the radial coronary artery (corona radiata). Normal production of CSF in the body is around 600-700 ml, which is then absorbed into the bloodstream [7,8]. However, in the case of hydrocephalus, an abnormality arises in the balance between the production and absorption of CSF. This abnormality can lead to the development of two types of the disorder: idiopathic and secondary [8].

The exact pathogenesis of idiopathic normal pressure hydrocephalus (iNPH) has not been determined in detail, but there are some consistent mechanisms of the disorder. These mechanisms include a disturbed balance of production and reabsorption of CSF, increased resistance to the outflow of CSF, no overproduction of CSF, and an undisturbed flow of CSF in the ventricles. Studies have shown that patients with hypertension or coronary heart disease have a higher risk of developing idiopathic NPH than healthy people. Impaired CSF absorption is the primary mechanism in most cases of secondary NPH. The most common causes are intraventricular or subarachnoid hemorrhage (due to trauma or aneurysm) and

previous acute or chronic meningitis as a result of infection, cancer, or inflammatory disease [9].

These conditions cause inflammation and subsequent fibrosis of the brain base and/or arachnoid granules, which is associated with reduced CSF resorption and leads to the gradual accumulation of CSF in the ventricular system. Although the increase in pressure may not be measurable at lumbar puncture, it is assumed to occur locally in the periventricular white matter tracts, causing the described disorder and clinical symptoms [9,10].

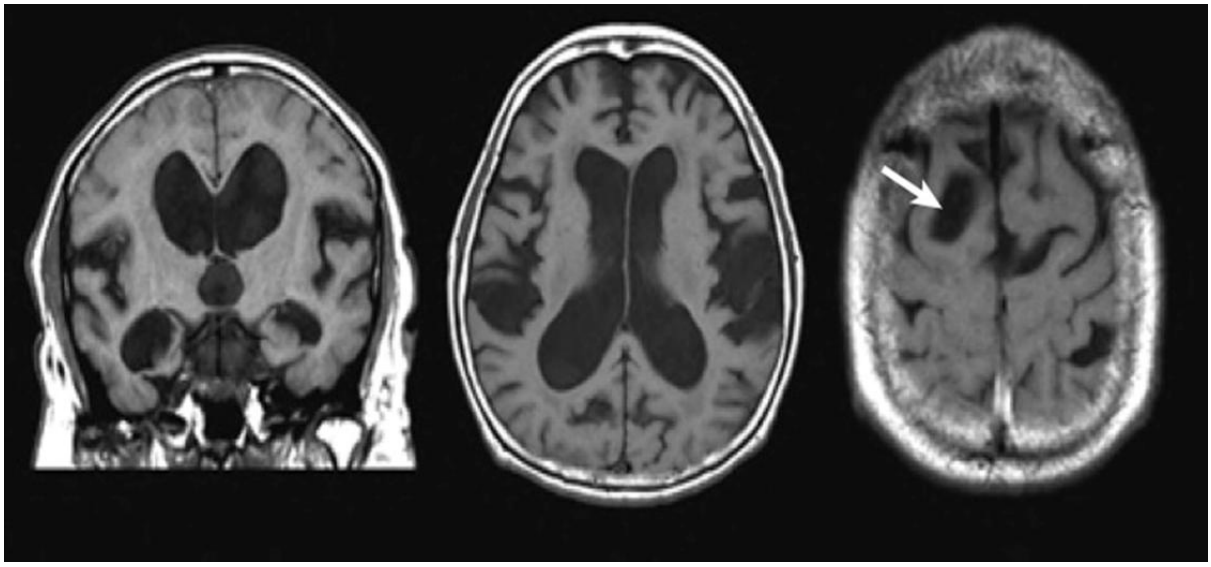

Image 1. Typical MRI of a patient with iNPH [9]

Symptoms

Normotensive hydrocephalus is a neurological disorder that is characterized by the classic symptoms of Hakim's triad, also known as Adams' triad, which includes gait disturbances, cognitive disorders, and frequent urination or urinary incontinence. The symptoms of NPH appear unexpectedly, usually within a time frame of 3-6 months, and are believed to result from dysfunction of the motor areas of the frontal lobe and periventricular white matter tracts [11,13].

Gait/balance disorders occur in almost all people with normotensive hydrocephalus, and is typically the first symptom of the disease. Enlargement of the lateral ventricles is believed to be the cause of this symptom, which may affect the motor fibres of the corticospinal tract [12].

A characteristic gait abnormality in NPH is wide-based, slow movement, taking short steps, and a gait "glued to the floor" that may resemble the gait specific to Parkinson's disease. Patients with NPH have difficulty turning their torso, so they take several steps to be able to turn 180 or 360 degrees. Postural instability, as demonstrated by the pull test, is typical. Gait deviations can be divided into mild, significant, or severe, depending on the extent of difficulty that the patient experiences while walking. Approximately 40% of people diagnosed with NPH also experience tremors in their arms, legs or feet [12,13].

Urinary incontinence is a symptom that appears in the advanced stage of the disease, occurring in 50% of patients at the time of treatment. Initially, the symptom is manifested by increased frequency of urination, usually at night, progressing to permanent, urgent urinary incontinence. It is worth noting that the earliest reported problem is often pathological pressure on the bladder and not incontinence itself [14].

Cognitive disorders are a hallmark of normotensive hydrocephalus, and usually develop over months or years, occurring much later than gait dysfunction. Patients usually have features of subcortical and frontal disorders, such as psychomotor retardation, loss of attention and concentration, impaired executive function, and apathy. Executive function, the ability to conceptualize aspects of action and translate them into appropriate and effective behavior, is impaired early on and may be more resistant to treatment. Cortical features, such as apathy, are much less common [15, 21, 22].

Diagnostic criteria

As per the criteria proposed by Relkin, the diagnosis of normal pressure hydrocephalus (NPH) requires the presence of certain radiological features. These include an Evans index of at least 0.3, indicating the ratio of maximum ventricular width to the largest biparietal dimension between the internal laminae of the skull [15]. In addition, the imaging must reveal enlargement of the temporal horn, changes in the periventricular signal, and the absence of a flow signal in the water supply/chamber four (evidence class III) [15,16].

Furthermore, the guidelines now suggest that a corpus callosum angle of more than 40° be considered in the diagnosis of NPH. The identification of pathological ventricular

enlargement as a result of brain atrophy can be challenging. This often leads to misdiagnosis or a failure to diagnose up to 80% of hydrocephalus cases. While all patients with normotensive hydrocephalus have ventricular enlargement, not all cases of ventricular enlargement in older adults are indicative of primary NPH [17].

Magnetic resonance imaging is the preferred imaging technique in the diagnosis of NPH. Furthermore, a narrow fluid space in areas high on the fornix or midline to the fissure of Sylvius has been associated with the diagnosis of probable or definite NPH (class III evidence) [18,20].

A commonly used diagnostic approach for normal pressure hydrocephalus (NPH) is the Miller-Fisher test. This method entails a large-volume lumbar puncture (LP) whereby approximately 30-50 ml of cerebrospinal fluid is removed [19].
Subsequently, the patient's cognitive functions and gait are assessed before and 2-3 hours after the procedure. The Miller-Fisher test has been adopted as a valuable tool in the diagnosis of NPH and is frequently used in clinical settings [21].

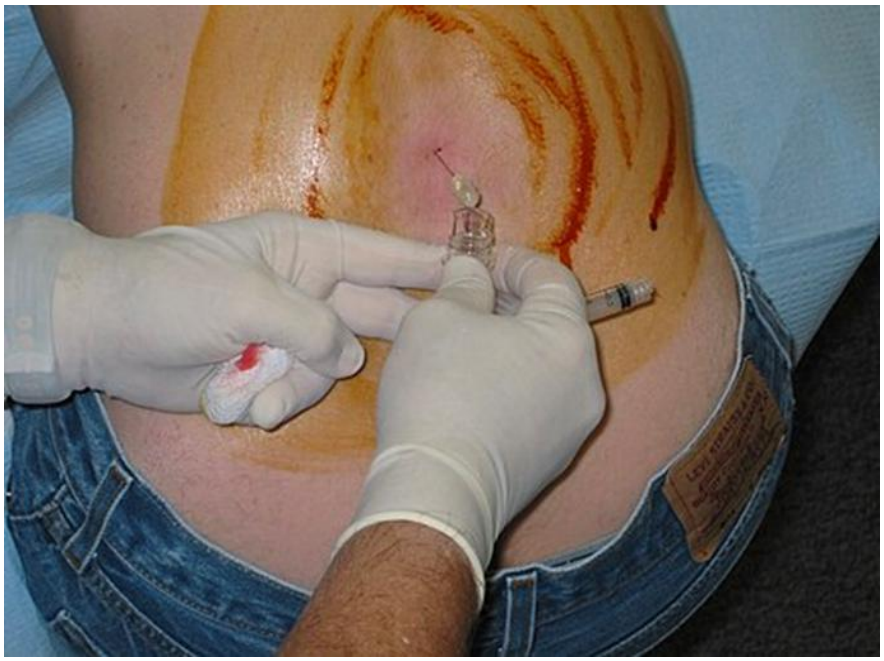

Image 3. Photo of a patient undergoing a lumbar puncture (LP). Cerebrospinal fluid (CSF) obtained from LP may be examined to aid in diagnosing NPH.

Treatment

Recent advances in diagnostic and therapeutic techniques have revolutionized the management of normal pressure hydrocephalus (NPH), with clinical improvement rates reaching 80-90%. Neurosurgical intervention plays a significant role in reducing the severity of symptoms associated with NPH [22]. The most common surgical procedure involves establishing a connection between the ventricular system and the peritoneal cavity through ventriculoperitoneal (VP) shunt placement or by creating an alternative cerebrospinal fluid circulation route via ventriculostomy. Although ventricular-atrial (VA) shunt implantation was once a popular option, it is no longer preferred due to higher rates of long-term complications. Lumboperitoneal (LP) shunts have also been employed in English-speaking countries to treat NPH, with surgical success rates ranging from 20-80% depending on the study. However, elderly patients exhibiting the complete triad of Hakim's symptoms are less likely to experience significant improvement following surgical intervention compared to those diagnosed earlier in life. It should be emphasized, however, that age alone should not serve as a contraindication for surgical intervention in patients without additional surgical risk factors. Older age only predicts a lower likelihood of improvement after valve implantation in the presence of the complete triad of symptoms [21, 22, 23].
Therefore, early and accurate diagnosis of NPH is paramount in achieving successful outcomes following surgical intervention.

The treatment of normotensive hydrocephalus (NPH) involves various practices, one of which is the removal of cerebrospinal fluid (CSF) through lumbar puncture. The diagnosis of idiopathic NPH (iNPH) was initially made through large-volume lumbar puncture (>30 ml). However, external lumbar drainage (ELD) is now gaining acceptance as a more sensitive predictor in patients. This method involves the insertion of a catheter into the lumbar spine and the drainage of CSF at a rate of 10-15 cm3 per hour for 72 hours [24].

In clinical trials, this treatment exhibited a positive impact on 66% of patients. Partial improvement in gait and perception was observed, although this is not a definite predictor of postoperative improvement. It is important to note that surgery is associated with risks such as

infection, valve damage, headache, or the occurrence of hydrocele or subdural hematoma resulting from excessive CSF drainage [25].

Volumetric MRI neuroimaging, including cerebral ventricular and per cerebral CSF volume ratios, has uncertain value in predicting the response of patients to ventricular shunt implantation, as per Class IV evidence. The success of the procedure depends on rigorous qualifications. Patients with symptoms of dementia lasting less than two years and those with marked gait disorders, or isolated gait disorders, have a better prognosis after the procedure [25, 26]. To date, there have been no reports in the literature about the efficacy of pharmacological treatment for primary normotensive hydrocephalus. Acetazolamide has not demonstrated any sustained reduction in ICP, and only a transient decrease in ICP after acetazolamide has been shown to be a positive predictor of good response to VP valve implantation [26].

Current research is aimed at finding alternative treatments for NPH. Studies conducted on rabbits and dogs have demonstrated that steroids can reduce the production of cerebrospinal fluid. However, further research is required in this direction to determine the efficacy of this treatment option in humans [15,27].

Guidelines for surgical treatment

The following are the suggested neurosurgical practice guidelines for determining the eligibility of a patient with idiopathic normal pressure hydrocephalus (iNPH) for surgery:

- In the case of high cerebrospinal fluid (CSF) pressure, an investigation of a secondary cause of NPH is warranted.
- A lumbar puncture of 40 to 50 ml (large volume) can potentially indicate a benefit from manoeuvring, and should be considered in the evaluation process.
- People who are unresponsive to high-volume touches can be evaluated using external lumbar drainage (ELD).
- MRI cerebrospinal fluid flow studies have been found to have no significant predictive value in the context of determining the eligibility of a patient with iNPH for surgery [13,28].

Clinical outcome of NPH treatment

It has been estimated that surgical intervention yields a lasting clinical benefit for 70 to 80% of patients diagnosed with normal pressure hydrocephalus (NPH), including idiopathic NPH (iNPH), as compared to their preoperative state, with observation periods that range from one to seven years. These findings are a testament to the remarkable advancement in the efficacy of treatment as compared to the therapeutic outcomes achieved in the past [29]. In addition, over 80% of patients demonstrated improvement in all areas of Hakim's triad, as compared to their preoperative state, after a follow-up of seven years. However, some patients, despite experiencing initial improvement, may subsequently experience regression in aspects of cognitive function and urinary incontinence over time. This occurrence has been attributed to the progression of a comorbid neurodegenerative disease [29,30].

Complications

Recent advancements in surgical treatment methods and materials used in valve replacement have led to a significant reduction in the occurrence of postoperative complications. The rate of valve-related complications, such as over/under drainage or subdural hematoma, is typically between 3% to 4%, with an infection rate of less than 1%. Consequently, the overall long-term incidence of such complications is less than 20%. This figure indicates a marked improvement in outcomes compared to the past, where the incidence of complications was as high as 35-40%. Additionally, non-procedure-related complications, such as seizures and intracerebral hemorrhage, occur in less than 5% of cases. When treating normotensive hydrocephalus with cerebrospinal fluid (CSF) transfusion, the perioperative morbidity rate is less than 5%, with a mortality rate of less than 0.1% [30,32].

Prognosis

The prognosis of patients diagnosed with normal pressure hydrocephalus (NPH) is dependent on several variables including the underlying etiology of the disease, severity of symptoms,

time of diagnosis, and implemented treatment [30]. Neglecting to address symptoms of cognitive impairment, gait disturbances, and urinary incontinence in NPH patients may result in dire consequences, ultimately leading to mortality. However, undergoing neurosurgical intervention has been shown to significantly enhance patients' comfort and facilitate their return to daily activities [31,32].

Summary

Nowadays, normotensive hydrocephalus (NPH) represents a pressing and socially significant issue. Despite extensive research, the aetiology and pathogenesis of this condition remain poorly understood. The clinical manifestations of NPH are highly variable, and the diagnostic process is complicated by the need to differentiate it from other neurodegenerative disorders. Consequently, underdiagnosis or misdiagnosis is common, leading to a decrease in patients' quality of life and, in many cases, disability. The prevalence of NPH continues to rise, highlighting the need for further research and the development of novel therapeutic approaches.

Disclosures

Author's contribution:

Conceptualization- Marta Kozikowska, Agnieszka Pociecha

Formal analysis- Bożena Kmak, Agnieszka Pociecha, Marta Kozikowska

Investigation- Marta Kozikowska, , Agnieszka Pociecha, Bożena Kmak

Writing-rough preparation- Bożena Kmak, Agnieszka Pociecha

Writing-review and editing- Marta Kozikowska, Bożena Kmak, Agnieszka Pociecha,

Visualization- Agnieszka Pociecha, Bożena Kmak, Marta Kozikowska

All authors have read and agreed with the published version of the manuscript.

Conflict of interest: The author declare no conflict of interest.

Funding statement: No external funding was received to perform this review

Statement of institutional review committee: not applicable

Statement of informed consent: not applicable

Statement of data availability: not applicable

Bibliography

1. Gavrilov GV, Gaydar BV, Svistov DV, et al. Idiopathic Normal Pressure Hydrocephalus (Hakim-Adams Syndrome): Clinical Symptoms, Diagnosis and Treatment. Psychiatr Danub. 2019;31(Suppl 5):737-744.
2. Kot-Bryćko K, Kot M, Pudło R. Wodogłowie normotensyjne - zapomniane, ale groźne. Psychiatra po dyplomie. 2014.
3. Kuriyama N, Miyajima M, Nakajima M. Nationwide hospital-based survey of idiopathic normal pressure hydrocephalus in Japan: Epidemiological and clinical characteristics. Brain Behav. 2017;7(4). doi:10.1002/brb3.635.
4. Tisell M, Höglund M, Wikkelsø C. National and regional incidence of surgery for adult hydrocephalus in Sweden. Acta Neurol Scand. 2005;112(2):72-75. doi:10.1111/j.1600-0404.2005.00441.x.
5. Jasińska-Myga B. Zespoły pozapiramidowe u osób w wieku podeszłym. Polski Przegląd Neurologiczny. 2010;6(4).
6. Jaraj D, Rabiei K, Marlow T, et al. Prevalence of idiopathic normal-pressure hydrocephalus. Neurology. 2014;82(16):1449-1454. doi:10.1212/WNL.0000000000000342.
7. Marmarou A, Young HF, Aygok GA. Diagnosis and management of idiopathic normal-pressure hydrocephalus: a prospective study in 151 patients. J Neurosurg. 2005;102(6):987-997. doi:10.3171/jns.2005.102.6.0987.
8. Graff-Radford NR, Godersky JC. Symptomatic congenital hydrocephalus in the elderly simulating normal pressure hydrocephalus. Neurology. 1989;39(12):1596-1600. doi:10.1212/WNL.39.12.1596.
9. Krauss JK, Regel JP, Vach W, et al. Vascular risk factors and arteriosclerotic disease in idiopathic normal-pressure hydrocephalus of the elderly. Stroke. 1996;27(1):24-29. doi:10.1161/01.STR.27.1.24.
10. Kuriyama N, Tokuda T, Miyamoto J, et al. Retrograde jugular flow associated with idiopathic normal pressure hydrocephalus. Ann Neurol. 2008;64(2):217-221. doi:10.1002/ana.21431.
11. Hebb AO, Cusimano MD. Idiopathic normal pressure hydrocephalus: a systematic review of diagnosis and outcome. Neurosurgery. 2001;49(5):1166-1184. doi:10.1097/00006123-200111000-00027.

12. Lenfeldt N, Larsson A, Nyberg L, et al. Idiopathic normal pressure hydrocephalus: increased supplementary motor activity accounts for improvement after CSF drainage. Brain. 2008;131(11):2904-2912. doi:10.1093/brain/awn220.
13. Sudarsky L, Simon S. Gait disorder in late-life hydrocephalus. Arch Neurol. 1987;44(3):263-267. doi:10.1001/archneur.1987.00520150065016.
14. Stolze H, Kuhtz-Buschbeck JP, Drücke H, et al. Comparative analysis of the gait disorder of normal pressure hydrocephalus and Parkinson's disease. J Neurol Neurosurg Psychiatry. 2001;70(3):289-297. doi:10.1136/jnnp.70.3.289.
15. Iddon JL, Pickard JD, Cross JJ. Specific patterns of cognitive impairment in patients with idiopathic normal pressure hydrocephalus and Alzheimer's disease: a pilot study. J Neurol Neurosurg Psychiatry. 1999;67(6):723-732. doi:10.1136/jnnp.67.6.723.
16. Graff-Radford NR, Godersky JC, Jones MP. Variables predicting surgical outcome in symptomatic hydrocephalus in the elderly. Neurology. 1989;39(12):1601-1604. doi:10.1212/WNL.39.12.1601.
17. Sorbi S, Hort J, Erkinjuntti T, et al. EFNS-ENS Guidelines on the diagnosis and management of disorders associated with dementia. Eur J Neurol. 2012;19(9):1159-1179. doi:10.1111/j.1468-1331.2012.03812.x.
18. Lezak MD, Howieson DB, Bigler ED, Tranel D. Neuropsychological Assessment. 5th ed. Oxford University Press; 2012.
19. Shprecher D, Schwalb J, Kurlan R. Normal Pressure Hydrocephalus: Diagnosis and Treatment. Curr Neurol Neurosci Rep. 2008;8(5):371-376. doi:10.1007/s11910-008-0058-1.
20. Mori E, Ishikawa M, Kato T, et al; Japanese Society of Normal Pressure Hydrocephalus. Guidelines for management of idiopathic normal pressure hydrocephalus: second edition. Neurol Med Chir (Tokyo). 2012;52(11):775-809. doi:10.2176/nmc.52.775.
21. Pyykkö OT, Nerg O, Niskasaari HM, et al. Incidence, comorbidities, and mortality in idiopathic normal pressure hydrocephalus. World Neurosurg. 2018;112. doi:10.1016/j.wneu.2018.01.177.
22. Kuriyama N, Miyajima M, Nakajima M, et al. Nationwide hospital-based survey of idiopathic normal pressure hydrocephalus in Japan: Epidemiological and clinical characteristics. Brain Behav. 2017;7(3) doi:10.1002/brb3.635.

23. Liew BS, Takagi K, Kato Y, et al. Current Updates on Idiopathic Normal Pressure Hydrocephalus. Asian J Neurosurg. 2019;14(4):1096-1103. doi:10.4103/ajns.AJNS_230_19.
24. Williams MA, Malm J. Diagnosis and Treatment of Idiopathic Normal Pressure Hydrocephalus. Continuum (Minneap Minn). 2016;22(2 Dementia):579-599. doi:10.1212/CON.0000000000000305.
25. Shprecher D, Schwalb J, Kurlan R. Normal pressure hydrocephalus: diagnosis and treatment. Curr Neurol Neurosci Rep. 2008;8(5):371-376. doi:10.1007/s11910-008-0058-1.
26. Das JM, Biagioni MC. Normal Pressure Hydrocephalus. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.
27. Damasceno BP. Neuroimaging in normal pressure hydrocephalus. Dement Neuropsychol. 2015;9(4):350-355. doi:10.1590/1980-57642015dn94000002.
28. Kiefer M, Unterberg A. The differential diagnosis and treatment of normal-pressure hydrocephalus. Dtsch Arztebl Int. 2012;109(1-2):15-25. doi:10.3238/arztebl.2012.0015.
29. Saldarriaga-Cantillo A, Yepes-Gaviria V, Rivas JC. Normal pressure hydrocephalus: Diagnostic delay. Biomedica. 2020;40(4):656-663. doi:10.7705/biomedica.5382.
30. Godoy Hurtado A, Barstchi P, Brea Salvago JF, et al. Low- and Negative-Pressure Hydrocephalus: New Report of Six Cases and Literature Review. J Clin Med. 2023;12(12):4112. doi:10.3390/jcm12124112.
31. Orlandi R, Vasilache CG, Mateo I. Palliative ventriculoperitoneal shunting in dogs with obstructive hydrocephalus caused by tumors affecting the third ventricle. J Vet Intern Med. 2020;34(4):1556-1562. doi:10.1111/jvim.15818.
32. Kolecka M, Farke D, Failling K, Kramer M, Schmidt MJ. Intraoperative measurement of intraventricular pressure in dogs with communicating internal hydrocephalus. PLoS One. 2019;14(9)doi:10.1371/journal.pone.0222725.