IMAGING OF NORMAL PRESSURE HYDROCEPHALUS

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ABSTRACT

Normal pressure hydrocephalus (NPH) is hydrocephalus without an increase in intracranial pressure. The term Idiopathic Normal Pressure Hydrocephalus (INPH) has been used to describe individuals presenting with ventriculomegaly of unexplained etiology, accompanied by the classic triad of symptoms (gait disturbance, urinary incontinence, and dementia). CT-scan is more practical, cheaper, widely available, and can assess the anatomical condition of the brain and ventricles, but MRI is the best modality and superior to CT. It can assess the anatomic conditions better, changes in white matter, and the presence of flow-void sign. Radiological signs of INPH are the presence of ventriculomegaly with Evan's index > 0.3, z-EI > 0.42, the presence of DESH (Disproportionately Enlarged Subarachnoid Space Hydrocephalus), cingulate sign, callosal angle <90°, widening of the temporal horn unrelated to hippocampal atrophy, no obvious obstruction to CSF flow, periventricular white matter changes, and flow-void sign in the aqueduct or 4th ventricle on MRI.

Keywords: CT-scan, imaging modality, MRI, normal pressure hydrocephalus, radiographic feature

INTRODUCTION

Normal pressure hydrocephalus (NPH) is hydrocephalus without an increase of intracranial pressure. The prevalence of NPH increases with age, which is about 3.3 per 100,000 people aged 50-59 years, 49.3 per 100,000 people aged 60-69 years, and 181.7 per 100,000 people aged 70-79 years (1). The classic clinical triad of NPH, also called as Hakim's triad, includes gait disturbance, urinary incontinence, and dementia, accompanied with ventricular dilation and normal cerebrospinal fluid pressure. NPH is classified into idiopathic NPH (INPH) of unknown etiology and secondary NPH (SNPH) of known etiology, such as meningitis, trauma, and subarachnoid bleeding (2). INPH is more common than SNPH (1).

For many years, the term INPH has been used to describe individuals presenting with ventriculomegaly of unexplained etiology, accompanied by the classic triad of symptoms. Ventriculomegaly can be detected through brain imaging including computed tomography (CT) or magnetic resonance imaging (MRI) (3). NPH is often misdiagnosed because it is similar to the picture of senile brain atrophy. This review will help in identifying the features of NPH based on the last criteria that are most often used, especially the radiological features that can be found on CT scans

or MRIs.

PATHOPHYSIOLOGY

NPH is estimated to occur in up to 10% of cases of dementia and can be treated with a ventriculoperitoneal shunt (4). NPH is often underdiagnosed, even though as many as 70%-90% of patients undergoing ventriculoperitoneal shunt procedures experience clinical improvement. This may be due to the difficulty of diagnosing NPH because its symptoms overlap with other neurodegenerative diseases, especially Alzheimer's disease (5). In addition, the pathophysiology of idiopathic NPH is still not clearly understood (1).

Although the pathophysiology of idiopathic NPH is not clearly known, there are several theories proposed as the pathophysiological mechanism for the occurrence of INPH, SNPH, pressure that remains normal in the dilated ventricle and the mechanism of the classic triad of symptoms. The pathophysiology of INPH involves decreased craniospinal compliance and impaired CSF pulsation, while the pathophysiology of SNPH involves a disproportion of CSF absorption secretion and chronic obstruction of CSF pathways which will then lead to ventricular dilatation. Normal pressure in NPH is caused by ventricular dilation leading to decreased subependymal resistance, efficient subependymal absorption to compensate for absorption in the sagittal sinus, increased capillary pulsation, and the presence of alternative connections in CSF pathways. Ventricular dilatation causes periventricular edema, compression of the periventricular brain parenchyma and decreased cerebral blood flow leading to lesions in the frontal and temporal lobes. These lesions cause the classic triad of symptoms of NPH in the form of gait disturbances, dementia, and urinary incontinence (1).

IMAGING MODALITY OF CHOICE

CT Scan

CT scan is a commonly used modality to evaluate hydrocephalus. The advantages of CT are relatively fast and simple examination and easy reformatting of data in various fields (multiplanar). Periventricular interstitial edema can be visible on CT, and calcifications are more pronounced on CT than on MRI (6). Head CT scan is a sensitive imaging modality for identifying NPH, but MRI can provide additional information such as aquaductal stenosis, white matter changes or the presence of an underlying etiology such as Alzheimer's disease (2). In addition, CT uses ionizing radiation with a significant dose of radiation (6).

Magnetic Resonance Imaging

MRI is the best modality for diagnosing hydrocephalus, looking for its causes, and its complications. The MRI approach to hydrocephalus can be seen from various aspects, namely specific morphological features, location of obstruction, effects of hydrocephalus on the brain, recognition of the cause of the disease with its specific impact on brain tissue, and CSF dynamics (2).

RADIOGRAPHIC FEATURE OF NORMAL PRESSURE HYDROCEPHALUS

The most frequently used radiological diagnostic criteria for hydrocephalus include ventriculomegaly (Evan index > 0.3), enlargement of the third ventricular recess and lateral ventricular horns, decreased mamillopontine distance and frontal horn angle, thinning and elevation of the corpus callosum, narrowed cortical sulci, hyperintensity in periventricular (interstitial oedema), and “flow void” phenomenon in the sylvian aqueduct on T2WI sequences (7).

The diagnosis of INPH relies on the findings of hydrocephalus on brain imaging. Hydrocephalus is not synonymous with ventriculomegaly. Although ventriculomegaly is commonly found in the elderly population, this does not imply the presence of NPH. In NPH, ventriculomegaly is usually disproportionate to the amount of atrophy present, as seen in **Figure 1**(2). Head CT scan is a sensitive imaging modality for identifying NPH but MRI provides additional information such as aqueductal stenosis, white matter changes, or the presence of an underlying etiology (eg. Alzheimer's disease) (2).

There are two guidelines that are most often used to establish the diagnosis of INPH, namely the international guidelines and the Japanese guidelines. The aim of the INPH diagnostic guidelines is to identify patients who are most likely to benefit from shunt surgery. The two guidelines have some similarities but also some important differences. The terms 'possible' and 'probable' INPH are used in the respective guidelines, with diagnostic criteria based

on clinical and imaging features. However, Japanese guidelines use the term 'probable INPH' for those who improve after removal of CSF. Japanese guidelines also refer to cases that respond well after shunt surgery as 'definite INPH' (8). International guidelines do not mention cases that have a good response after CSF shunting in their diagnostic criteria. The neuroimaging criteria are also different. **Table 1** shows a comparison of the diagnostic neuroimaging features used in these two guidelines (2).

Other radiological features that can support the diagnosis of INPH but are not required to determine probable criteria based on international guidelines are the ventricular size before symptom onset appears smaller and there are no signs of

Table 1. Comparison of international and Japanese guidelines for the diagnosis of INPH (2)

Features	International guidelines	Japanese guideline
Size of Ventricles	Ventricular enlargement not entirely attributable to cerebral atrophy or congenital hydrocephalus (Evan's ratio >0,3 or equivalent)	Evan's ratio >0,3
Additional imaging features	• No obvious obstruction to CSF flow • And at least one of the following: 1. Enlargement of temporal horns not solely due to hippocampal atrophy 2. Callosal angle of 40° or more 3. Evidence of altered brain water content, including periventricular signal changes on CT and MRI not attributable to microvascular ischemic changes or demyelination 4. An aquaductal or fourth ventricle flow void on MRI	• Dilated subarachnoid spaces in the Sylvian fissures and narrowed spaces over the high cerebral convexity and medial surface (DESH) • One or more elliptically dilated sulci over the medial surface and convexity in isolation • A callosal angle of less than 90° on coronal section perpendicular to anterior commissure-posterior commissure plane
DESH = Disproportionately Enlarged Subarachnoid Space Hydrocephalus. These feature are supportive but not essential for a diagnosis of possible INPH		

hydrocephalus, radionuclide cisternogram shows delayed clearance of the radiotracer above the cerebral convexity after 48-72 hours, cine MRI examination showed an increase in ventricular flow rate (9).

Evan's Index

An objective way to assess whether the ventricles are enlarged is to use the Evan's Index or Evan's ratio. It is the ratio of the maximum width of the frontal horn of the lateral ventricle and the transverse diameter of the skull, measured at the same level on both axial CT and MRI images. Values above 0.30 are considered significant, and the higher the value, the more specific it is for NPH. Evan's index is a rough marker of hydrocephalus and can vary depending on the location and angle of the images (2). Measurement of Evan's Index can be seen in **Figure 2** (10).

Lateral ventricular enlargement in INPH is usually seen vertically on a coronal (z-axis) section as opposed to an axial (x-axis) view. The Evan's Index on the z-axis (z-EI) can be calculated by comparing the height of the lateral ventricular frontal horn on the coronal section divided by the midline

diameter of the skull, with a limit of 0.42. A value > 0.42 indicates ventriculomegaly and this assessment is said to be superior to the Evan's Index as measured on an axial section with a value > 0.3. In the same coronal section, the Brain per Ventricle Ratio (BVR) can be calculated. The coronal sections used to calculate z-EI and BVR are those that pass through the anterior commissure and are perpendicular to the line between the anterior and posterior commissures. Measurement of z-EI and BVR can be seen in **Figure 3**(8). In the section through AC, the BVR value for the diagnosis of INPH is <1.0 and in the section through the PC the value is <1.5 (8).

Disproportionately Enlarged Subarachnoid Space Hydrocephalus (DESH)

Findings of DESH (Disproportionately Enlarged Subarachnoid Space Hydrocephalus), such as ventriculomegaly, dilatation of the Sylvian fissure, and narrowing or increased convexity of the midline subarachnoid space are specific findings of INPH with high positive predictive value but low negative predictive value (8). In addition, the cingulate sulcus sign can be found, which is narrower of posterior part of the cingulate sulcus than the anterior part (10). The MRI images of DESH can be seen in **Figure 4** (12).

Callosal Angle

Sharpening of the callosal angle can be measured as an indirect sign of DESH and is very useful for diagnosing INPH and predicting the effect of shunt intervention (8). Using MPR, a coronal section was obtained at the level of the posterior commissure with the orientation of the section perpendicular to the anterior-posterior commissure line. At this level section, the callosal angle is measured as the angle between the superior border of the right and left lateral ventricles.11 CA < 90° is found in most cases of INPH. When combined with an Evan index > 0.3, INPH can be distinguished from AD with a sensitivity and specificity of 97 and 94%, respectively (2). Measurement of callosal angle can be seen in **Figure 5**(10).

Widening of Temporal Horns

One of the signs of hydrocephalus is dilatation of the temporal horn. International guidelines state that one of the signs of NPH on imaging examination is a widening of the temporal horn that is not caused by hippocampal atrophy (9).

There are no standard values in existing guidelines, but a diameter of more than 2 mm in adults is considered pathological. The temporal horn diameter limit of 4 mm showed a sensitivity of 0.92 and a specificity of 0.78 for the diagnosis of INPH, minimizing the risk of false negative cases. The temporal horn diameter limit of 6 mm shows a sensitivity of 0.32 and a specificity of 0.98, maximizing the ability to detect true positive cases (12). Widening of temporal horns in head CT scan can be seen in **Figure 6**(13).

White Matter Changes

MRI can detect changes in periventricular white matter that indicate changes in the water content of the brain. This can occur in communicating and non-communicating hydrocephalus. Changes in the deep periventricular white matter can also be seen in INPH. This is not an essential finding, but is associated with ischemic complications (9).

Periventricular white matter changes can be caused by transependymal extravasation of the CSF due to increased pressure, and can be seen on CT as hypodense lesions in the anterior and posterior horn regions. On MRI, these changes can be detected on T2 or FLAIR sequences. CSF extravasation must be distinguished from age-related white matter changes, which are less than 10 mm in diameter on axial section and decrease in thickness from anterior to posterior, as shown in **Figure 7**(14).

Leukoaraiosis (LA) and cerebral amyloid angiopathy (CAA) are common in the elderly and often accompany INPH. Because the clinical and MR imaging are very similar in that they can detect periventricular white matter changes, an accurate diagnosis of INPH is important to predict the patient's reaction and responsiveness to VP shunt surgery. INPH without LA and CAA will have good responsiveness to VP shunt surgery. To distinguish whether an INPH is accompanied by LA, CAA, or not, the SWI sequence on an MRI plays an important role (14). MRI images in INPH, LA, and CAA cases can be seen in **figure 8**(15).

CSF Flow Void

The advantage of MRI is that the T2WI sequence can show the CSF flow void sign which is related to the pulsation rate of the CSF flow. CSF flows back and

forth through the aqueducts during the cardiac cycle in response to arterial blood flow to the brain. This was observed as a flow void, ie decreased signal on MRI especially in the aqueduct on T2WI on MRI of patients with communicating hydrocephalus. These CSF flow voids can be seen in normal individuals, but are more prominent in patients with INPH. Initially this increased CSF flow void was thought to be predictive of patients responding well to shunting. In NPH that responds well to shunts, the CSF flow to the aqueduct is increased, but the significance of this finding is unclear and in further studies the correlation between the degree of CSF flow void and post-surgical outcome was low (2). Image of the flow void sign on the MRI can be seen in **Figure 9**(13)

Differential Diagnosis

Clinical signs and symptoms of INPH can be confused with normal aging processes or other neurodegenerative diseases such as Alzheimer's disease (AD) or Parkinson's disease (PD) or even vascular diseases such as vascular dementia (VD). In 2005, an international committee of hydrocephalus investigators published the extensive "Guidelines for the Diagnosis and Management of Idiopathic NPH," categorizing patients into 2 groups. (1) Probable: the age of onset was kept at more than 40 years, with a symptom duration of at least 3 to 6 months. A clinical diagnosis in the "probable" category requires gait or balance disturbance plus impairment in cognition or bladder control or both. (2) Possible: onset can occur at any age after childhood, with symptoms lasting less than three months. The patient presented with incontinence or cognitive impairment but no observable gait or balance disturbance (15).

Gait changes are the most prominent clinical feature in the early stages of INPH and are believed to be the most responsive to shunting. Dementia without gait disturbance can be safely excluded from the diagnosis. The INPH gait is described as "glued on the floor", magnetic gait, gait apraxia, or frontal ataxia, in which the steps are short, with decreased stride length and height, with outwardly rotated feet, diminished cadence, and a broadened base as opposed to a narrow base in PD. Patients tend to turn around, their posture is disturbed, and a history of falls can be reported. Sometimes, patients complain of vague pain in their legs after walking a moderate distance. Cognitive impairment NPH has prominent subcortical and frontal features with psychomotor slowing, decreased attention and concentration, and apathy, whereas other forms of dementia such as VD. Vascular dementia is a more likely diagnosis when there is a history of gradual cognitive decline with asymmetric signs. The lack of delusions or visual hallucinations or the presence of non-fluctuating cognitive status distinguishes NPH from Lewy-body dementia. Cortical features (aphasia, agnosia, and apraxia) are less prominent in NPH than in VD or AD. INPH can be distinguished from frontotemporal dementia (FTD) by the lack of personality changes, impulsivity, or aphasia. Urinary urgency may appear early in INPH, which may progress to urinary incontinence at a later stage (15).

Management

The management of INPH can be divided into conservative and surgery. As conservative management, diuretics and osmotic acetazolamide are sometimes used in patients with INPH. Memantine has shown some positive effects in those with neuropsychiatric symptoms. However, prospective cohort studies comparing surgical treatment with surgical treatment in patients with INPH have shown moderate to marked improvements in cognition, balance, urinary function, or activities of daily living in the majority of the shunted population, while the majority of unshunted patients either had marked worsening of symptoms or had no change from their baseline levels (15).

Shunting is used as standard surgical management of normal pressure hydrocephalus. The VP shunt is the most popular, whereas the ventriculo-atrial (VA) shunt is rarely implanted because of its more frequent long-term complications. Lumbo-peritoneal (LP) shunts are also increasingly being tried. Gait disturbance is the symptom most responsive to shunting. Cognitive impairment may improve with surgery if it is not too severe at the time of intervention, while urinary incontinence improves in 36% to 90% of patients (8, 15).

Complication

According to the Japanese Guideline, several complications that can occur after shunt intervention in the management of NPH patients include shunt dysfunction due to shunt tube obstruction, headache,

shunt infection, and subdural hematoma, which are associated with excessive CSF drainage (8). This is in line with another study stated that the incidence associated with VP shunt interventions was quite high in the past. Failure, infection, obstruction, over- or under-drainage, or subdural hematoma are examples of complications (15). However, with the introduction of newer materials for shunts and valves, complications have decreased by 20%. Complications not related to shunts, such as seizures and intracerebral hemorrhage, have also decreased significantly in recent times. Meanwhile, the intervention with LP had a lower incidence of infection than the intervention with VP shunt (8).

Prognosis

Symptoms of INPH usually improve after surgical intervention. Outcomes after surgical intervention have been reported for various periods, from 3 months to 6 years. Short-term outcomes (at 1 year after shunt intervention) may be affected by complications associated with the surgical procedure. In addition, the outcome was also influenced by the duration and severity of the disease, the response to the knock test, and the status of typical imaging findings (DESH). Regarding the rate of symptom improvement after shunt intervention, gait disturbance showed the highest improvement rate. The long-term outcome of INPH is influenced by the presence of comorbidities. For example, stroke affects functional prognosis, cancer affects life prognosis, Alzheimer's disease affects cognitive function, and Parkinson's disease affects motor function (8).

CONCLUSION

The guidelines most often used for the diagnosis of INPH are the international guidelines and the Japanese guidelines, where radiological examination is one of the diagnostic criteria in addition to clinical symptoms and other supporting examinations.

Radiologic examinations that can be used for the diagnosis of INPH are CT scan and MRI of the head. CT scan is more practical, cheaper, more widely available, and can assess the anatomical condition of the patient's brain and ventricles, but MRI is the best radiological examination and is superior to CT scan because it can assess anatomic conditions better, assess changes in white matter, and assess the presence of a flow void sign.

Signs that can be found on radiological examination of INPH cases are the presence of ventriculomegaly with Evan's index >0.3, z-EI> 0.42, the presence of DESH (Disproportionately Enlarged Subarachnoid Space Hydrocephalus), Cingulate sign, Callosal angle <90°, widening of the temporal horn. unrelated to hippocampal atrophy, no obvious obstruction to CSF flow, the presence of periventricular white matter changes, and the presence of a flow void sign in the aqueduct or 4th ventricle on MRI.

STATEMENT OF ETHICS

All data and images used for the publication of this case were sourced from the literature review with the original source acknowledged and no written informed consent was obtained from the patient.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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DATA AVAILABILITY STATEMENT

No additional data than the one presented in this article was used.

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FIGURE LEGENDS

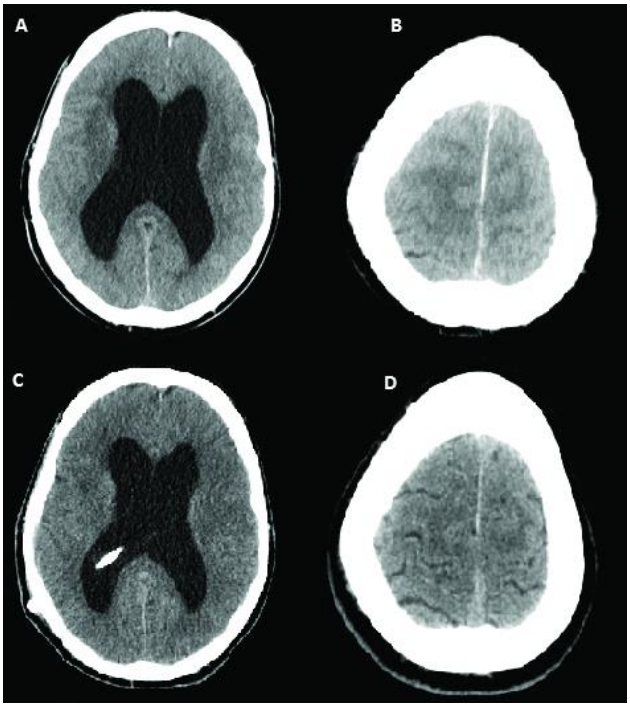

Figure 1. Head CT scan of a patient with INPH before and after surgery (2). A and B: Ventriculomegaly and narrowed sulci on the vertex of a patient with INPH;C and D: postoperative imaging showed improvement.

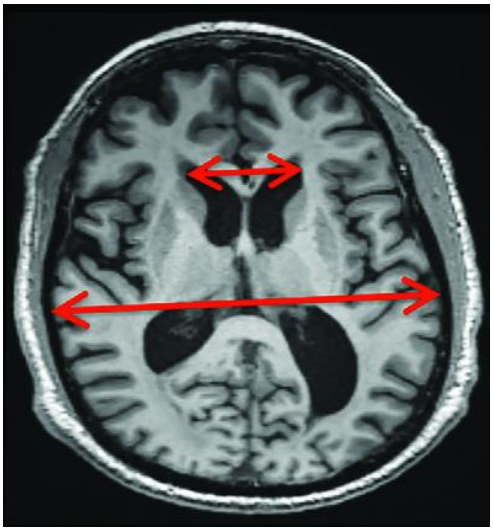

Figure 2. Measurement of Evan's Index (10). EI was determined on the axial view by measuring the greatest width of the right and left lateral ventricular frontal horn divided by the largest cranial width at the same level.

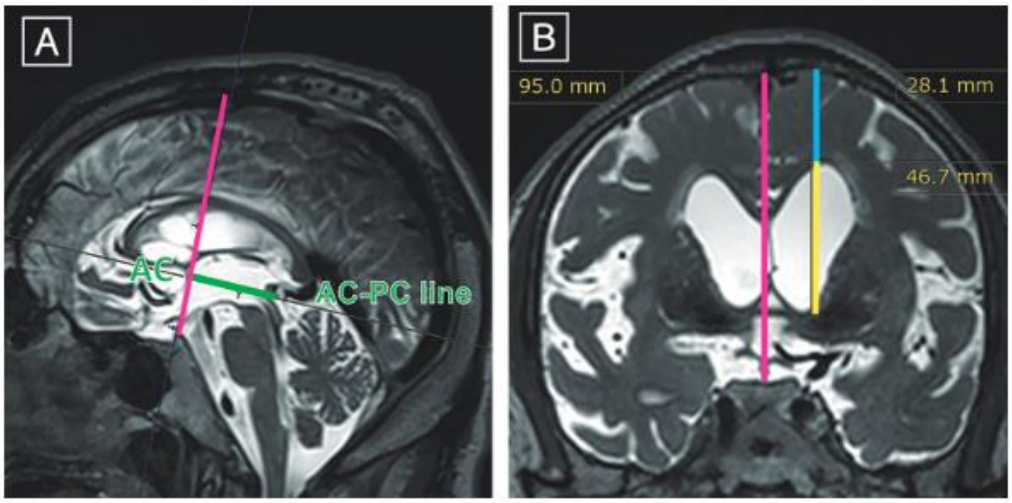

Figure 3. Evaluation of z-EI and BVR (8). (A) Evaluation of z-EI and BVR was performed on coronal sections obtained on sections that pass through the anterior commissure and are perpendicular to the AC-PC line (green line).(B) The height of the lateral ventricular frontal horn (yellow line) on the z-axis divided by the midline diameter of the skull (magenta line) is the z-EI, with a cut-off value of 0.42.The BVR at AC level is obtained by calculating the maximum length of the brain on the z-axis just above the lateral ventricle (yellow line) divided by the maximum length of the lateral ventricle (cyan line).In the slice through AC, the value is < 1.0 and in the piece through PC the value is < 1.5.This figure shows z-EI 0.49 (>0.42), BVR AC level 0.6 (<0.1).

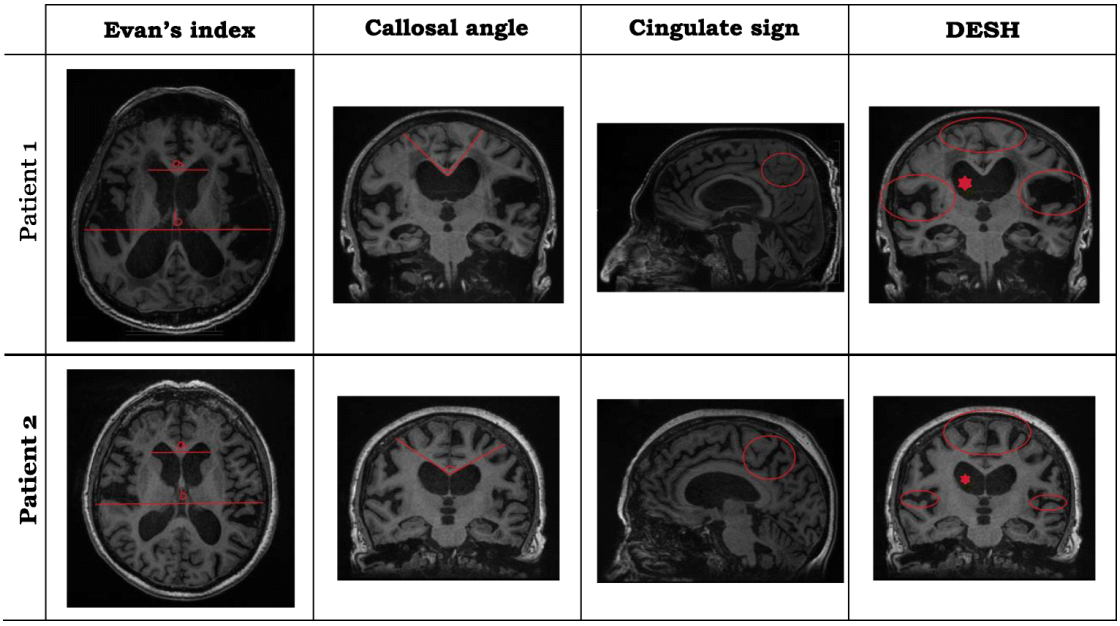

Figure 4. The MRI findings (T1WI) in two patients with Hakim's triad were positive (12). Both patients showed ventriculomegaly (Evan's index = 0.35). Patient 1, 75 years old male, had narrow callosal angle (85°), positive cingulate sign, and DESH. All of these signs indicate an INPH. Patient 2, a 77-year-old woman, had a blunt callosal angle (115°), negative cingulate sign, and no DESH. Patient 1 had positive results on the lumbar infusion test (LIT) and external lumbar drainage (ELD), and was treated with a VP shunt. Patient 2 was negative on both functional tests and a VP shunt was not indicated in this patient.

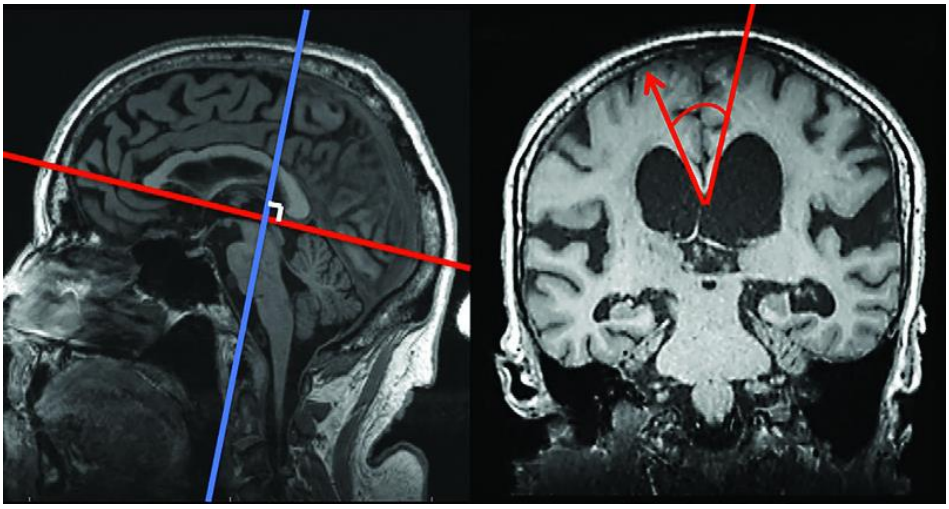

Figure 5. Callosal angle measurement (10).

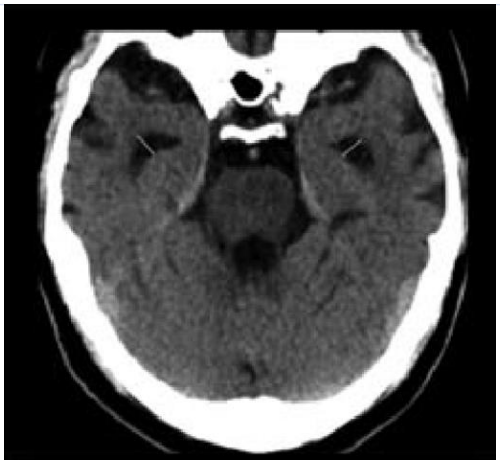

Figure 6. Widening of temporal horns in head CT scan (13).

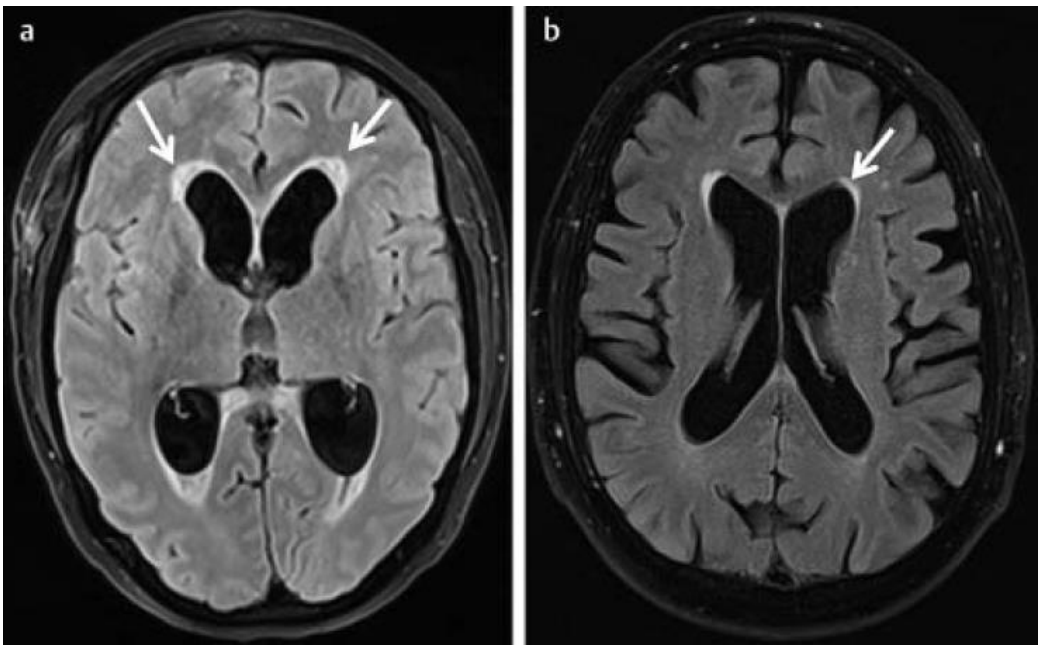

Figure 7. MRI appearance of periventricular white matter changes (14). (A) Periventricular white matter changes in hydrocephalus;(B) Age-associated periventricular white matter changes.

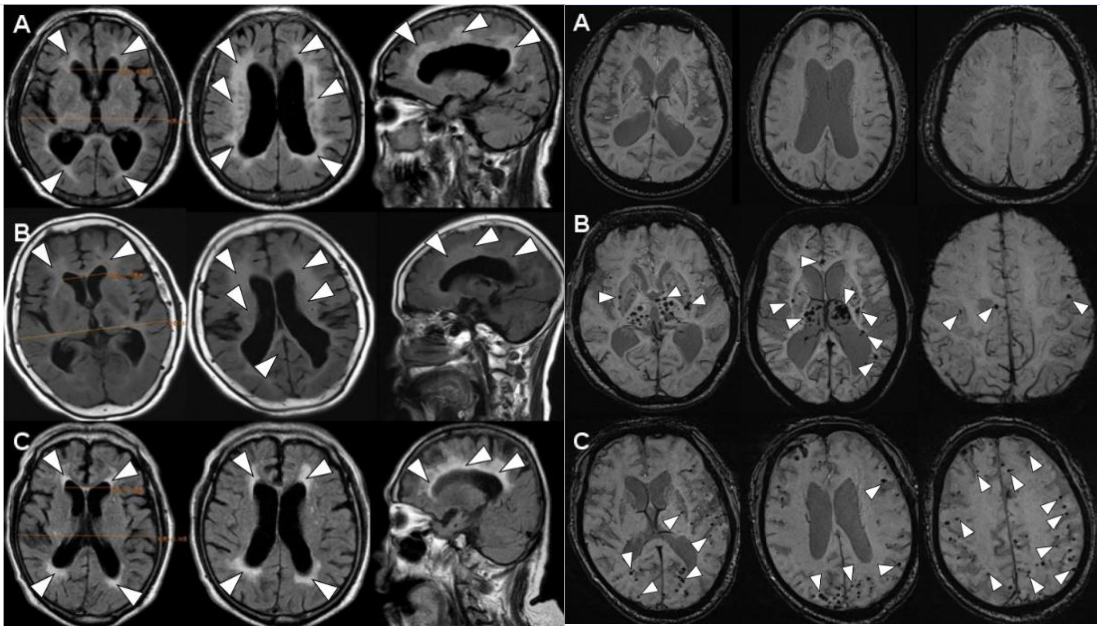

Figure 8. MRI images in INPH, LA, and CAA cases (15).Left: FLAIR Sequence.There is a similar picture, namely ventricular dilatation (Evan's index > 0.3, periventricular and deep white matter changes were seen in all three cases. (A) INPH case; (B) LA; (C) CAA.Right: SWI sequence. Threedifferent cerebral microbleed (CMB) phenomena (A) INPH, no CMB was seen (B) LA, CMB was seen with distribution in deep brain structures (basal ganglia, thalamus, corpus callosum, internal and external capsules); (C)CAA: multiple CMB seen in the lobar cerebral area (cortex and sub cortical white matter).

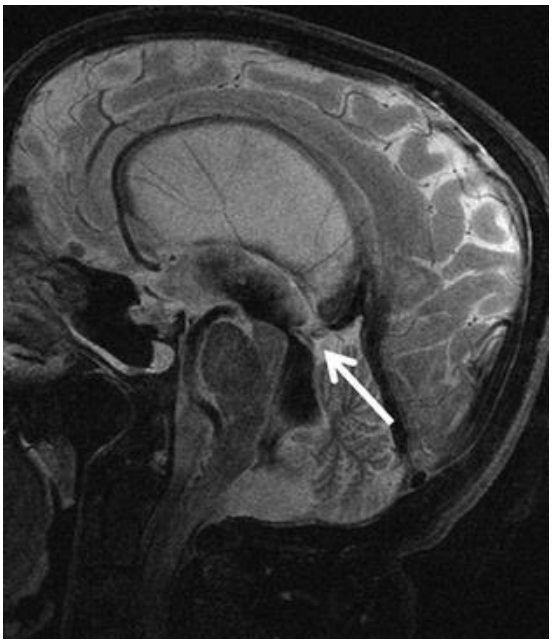

Figure 9. Image of the flow void sign on a sagittal T2WI MRI sequence (14).T2WI is a flow-sensitive sequence, and the sagittal section in the image shows a strong flow void in the aqueduct which is an indirect sign of INPH.