

Idiopathic normal pressure hydrocephalus

Magnetic resonance imaging

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1. prerequisite: ventricular enlargement without block (i.e., [communicating hydrocephalus](#)). MRI excels at ruling out [obstructive hydrocephalus](#) due to [aqueductal stenosis](#)

2. features that correlate with favorable response to [shunt](#). These features suggest that the [hydrocephalus](#) is not due to [atrophy](#) alone. Note: atrophy / [hydrocephalus ex vacuo](#), as in conditions such as [Alzheimer's disease](#), lessens the chance of, but does not preclude responding to a [shunt](#) ([cortical atrophy](#) is a common finding in healthy individuals of advanced [age](#) ¹⁾)

a) periventricular low density on CT or high intensity on T2WI MRI: may represent [Transependymal edema](#). May resolve with shunting

b) compression of convexity sulci (as distinct from dilatation in atrophy). Note: focal [sulcal dilation](#) may sometimes be seen and may represent atypical reservoirs of CSF, which may diminish after shunting and should not be considered as atrophy ²⁾.

c) rounding of the [frontal horns](#)

Other helpful findings in iNPH that require MRI

1. Japanese guidelines ³⁾ for iNPH also identify the following features:

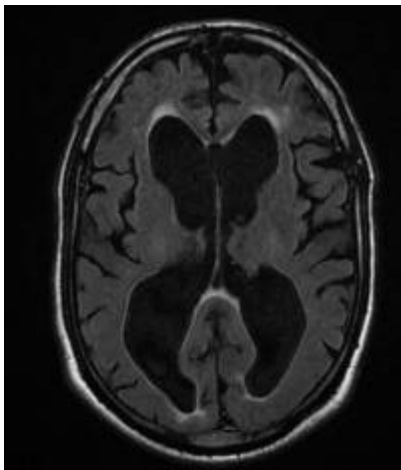
a) **DESH** hydrocephalus with enlarged **subarachnoid spaces** primarily in the **Sylvian fissure** and **basal cisterns** and effacement of the subarachnoid space over the **convexity** (so-called “tight high convexity”).

In comparison, dilated **subarachnoid space** in the high convexity is suggestive of **atrophy**

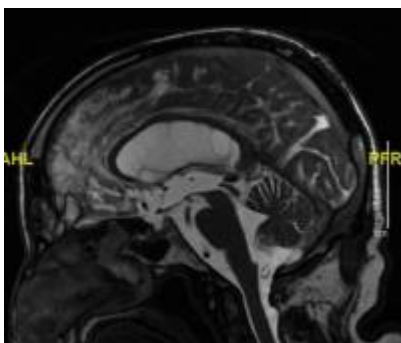
b) ventricular enlargement in iNPH deforms the **corpus callosum**, including:

- upward bowing and thinning (best appreciated on sagittal MRI)
- impingement on the **falx**, producing an acute **callosal angle** ($\leq 90^\circ$, demonstrated on a coronal MRI perpendicular to the AC-PC line, passing through the **posterior commissure** (PC))

2. phase-contrast MRI may demonstrate hyperdynamic flow of CSF through the **aqueduct**. Although some patients improve with no change in ventricles, clinical improvement most often accompanies reduction of ventricular size.



Marked hydrocephalus affecting the **lateral ventricles**, although without clear signs of **transependymal edema**, but with loss of volume of the **brain parenchyma** from chronic chronology.



In the sagittal volumetric T2 sequence, there is no clear occupation of the **cerebral aqueduct**, also observing the passage of **cerebrospinal fluid** through the aqueduct. He also appreciates flow artifact on T2 in the 3rd ventricle. The 3rd ventricle presents a slight increase in caliber with the 4th ventricle

of normal size, however, the increase in the 3rd ventricle is much less than the lateral ventricular dilatation. Signs of chronic small vessel ischemic zone distributed in both cerebral hemispheres. A small area of encephalomalacia and right occipital gliosis due to sequelae of an old ischemic infarction.

NPH is characterized by an ongoing periventricular neuronal dysfunction seen on MRI as [periventricular hyperintensity](#) (PVH). Clinical improvement after shunt surgery is associated with CSF changes indicating a restitution of axonal function. Other biochemical effects of shunting may include increased monoaminergic and peptidergic neurotransmission, breakdown of [blood brain barrier](#) function, and [gliosis](#) ⁴⁾.

An MRI-based diagnostic scheme used in a multicenter prospective study (Study of [Idiopathic Normal Pressure Hydrocephalus](#) on Neurological Improvement [SINPHONI]) appears to suggest that features of disproportionately enlarged subarachnoid-space hydrocephalus (DESH) are meaningful in the evaluation of NPH ⁵⁾.

Phase contrast magnetic resonance imaging

see [Phase contrast magnetic resonance imaging for idiopathic normal pressure hydrocephalus](#).

Diffusion-weighted imaging

[Diffusion-weighted magnetic resonance imaging](#) (DWI) ⁶⁾ is used generally in the diagnosis and treatment of various [neurodegenerative diseases](#). The [apparent diffusion coefficient](#) (ADC) of the brain, calculated from DWI data, is overestimated because of the effect of bulk motion (rigid body motion caused by the brain pulsation).

Arterial spin labelled imaging

see [Arterial spin labelled imaging for idiopathic normal pressure hydrocephalus](#).

Resting-state functional magnetic resonance imaging

[Resting-state functional magnetic resonance imaging for idiopathic normal pressure hydrocephalus](#).

Magnetic resonance elastography

[Magnetic resonance elastography for normal pressure hydrocephalus](#).

1)

Schwartz M, Creasey H, Grady CL, et al. Computed Tomographic Analysis of Brain Morphometrics in 30 Healthy Men, Aged 21 to 81 Years. *Ann Neurol*. 1985; 17:146–157

2)

Holodny AI, George AE, de Leon MJ, et al. Focal Dilation and Paradoxical Collapse of Cortical Fissures and Sulci in Patients with Normal-Pressure Hydrocephalus. *J Neurosurg*. 1998; 89: 742–747

3)

Mori E, Ishikawa M, Kato T, et al. Guidelines for management of idiopathic normal pressure hydrocephalus: second edition. *Neurol Med Chir (Tokyo)*. 2012; 52:775–809

4)

Tullberg M, Blennow K, Månsson JE, Fredman P, Tisell M, Wikkelsö C. Ventricular cerebrospinal fluid neurofilament protein levels decrease in parallel with white matter pathology after shunt surgery in normal pressure hydrocephalus. *Eur J Neurol*. 2007 Mar;14(3):248-54. PubMed PMID: 17355543.

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6)

Moseley ME, Cohen Y, Mintorovitch J, Chileuitt L, Shimizu H, Kucharczyk J, Wendland MF, Weinstein PR. Early detection of regional cerebral ischemia in cats: comparison of diffusion- and T2-weighted MRI and spectroscopy. *Magn Reson Med*. 1990 May;14(2):330-46. doi: 10.1002/mrm.1910140218. PMID: 2345513.

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