

Idiopathic normal pressure hydrocephalus comorbidities

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Certain comorbid conditions are frequently associated with this condition. Here are some common [comorbidities](#) observed in individuals with INPH:

Neurodegenerative diseases: There is a significant overlap between INPH and certain neurodegenerative disorders, including [Alzheimer's disease](#) (AD), [Parkinson's disease](#) (PD), and [Lewy body dementia](#) (LBD). It is estimated that approximately 5-15% of individuals diagnosed with AD or PD may also have INPH. This overlap can complicate the diagnosis and management of these conditions.

Hypertension and cardiovascular diseases: There is a higher prevalence of hypertension and cardiovascular diseases in individuals with INPH compared to the general population. Hypertension can potentially impact cerebral blood flow and contribute to the development or progression of INPH.

Diabetes mellitus: Diabetes mellitus, a metabolic disorder characterized by high blood sugar levels, has been associated with an increased risk of developing INPH. It is hypothesized that diabetes-related microvascular changes and alterations in cerebrovascular dynamics may contribute to the development of INPH.

Urinary and bladder dysfunction: [Urinary incontinence](#) is one of the classic symptoms of INPH. However, urinary dysfunction can also be associated with other conditions, such as benign prostatic hyperplasia (BPH), overactive bladder, or pelvic floor dysfunction. Differentiating between the primary cause of urinary dysfunction and the impact of INPH can be challenging.

Psychiatric disorders: [Depression](#) and anxiety disorders are often observed in individuals with INPH. These conditions can arise due to the impact of INPH symptoms on daily functioning, social interaction, and overall quality of life. Proper management of these comorbid psychiatric conditions is important for comprehensive care.

It is important to note that while these comorbidities are frequently observed in individuals with INPH,

they may not be present in every case.

119 patients with NPH coding at the University Clinic Münster from January 2009 to June 2017 were examined. The study primarily concentrated on examining [symptoms](#), [comorbidities](#), and radiological measurements, including [callosal angle](#) (CA) and [Evans index](#) (EI). To evaluate the progression of symptoms, a novel scoring system was developed to quantitatively assess the course at specific time points: 5-7 weeks, 1-1.5 years, and 2.5 years after the operation. This scoring system aimed to provide a standardized approach for measuring and tracking the development of symptoms over time. Logistic regression analyses were employed to identify [predictors](#) associated with three key outcomes: shunt implantation, surgical success, and the development of [complications](#).

Among the comorbidities observed, [hypertension](#) was the most prevalent. [Gait disturbance](#), in the absence of [polyneuropathy](#), was identified as a predictor of a favorable surgical outcome. [Hygroma](#) development was associated with a combination of vascular factors and cognitive disorders. The presence of spinal/skeletal changes, diabetes, and vascular constellations was found to increase the likelihood of developing complications.

The [evaluation](#) of [comorbidity](#) holds significant importance and necessitates meticulous [observation](#), [expertise](#), and [multidisciplinary](#) care ¹⁾.

[Alzheimer's disease](#) is the most common cause of dementia worldwide and frequent comorbidity in [idiopathic normal pressure hydrocephalus](#) (iNPH). The presence of AD pathology is associated with worse outcomes after a shunt procedure in iNPH. Preoperative diagnosis of AD is challenging in patients with iNPH, which involves reduced concentrations of the [cerebrospinal fluid biomarkers](#).

The aim was to estimate the effect size of iNPH as a factor in CSF levels of AD biomarkers and to test if correction could be used to improve diagnostic value.

The cohort included 222 iNPH patients with data in the Kuopio NPH registry and brain biopsy and CSF samples available. We divided the patients into groups according to AD pathology per brain biopsy. For control cohorts, we had CSF samples from cognitively healthy individuals (n = 33) and patients with diagnosed AD and no iNPH (n = 39).^{*}Results: Levels of all investigated biomarkers differed significantly between groups, with the exception of t-Tau levels between healthy individuals and iNPH patients with AD pathology. Applying a correction factor for each biomarker ($0.842 \cdot A\beta 1 - 42$, $0.779 \cdot t\text{-Tau}$, and $0.610 \cdot P\text{-Tau181}$) for the effect of iNPH yielded a sensitivity of 2.4% and specificity of 100%. The ratio of P-Tau181 to $A\beta 1 - 42$ was moderately effective in aiding the recognition of AD pathology in iNPH patients (sensitivity 0.79, specificity 0.76, area under the curve 0.824).

Correcting for iNPH as a factor failed to improve diagnostic effectiveness, but the P-Tau181/ $A\beta 1 - 42$ ratio showed some utility in the diagnosis of AD in iNPH patients ²⁾

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Last update: **2024/06/07 02:56**

