SECTION 8

CSF Dynamics and Intracranial Pressure Disorders

Case 73

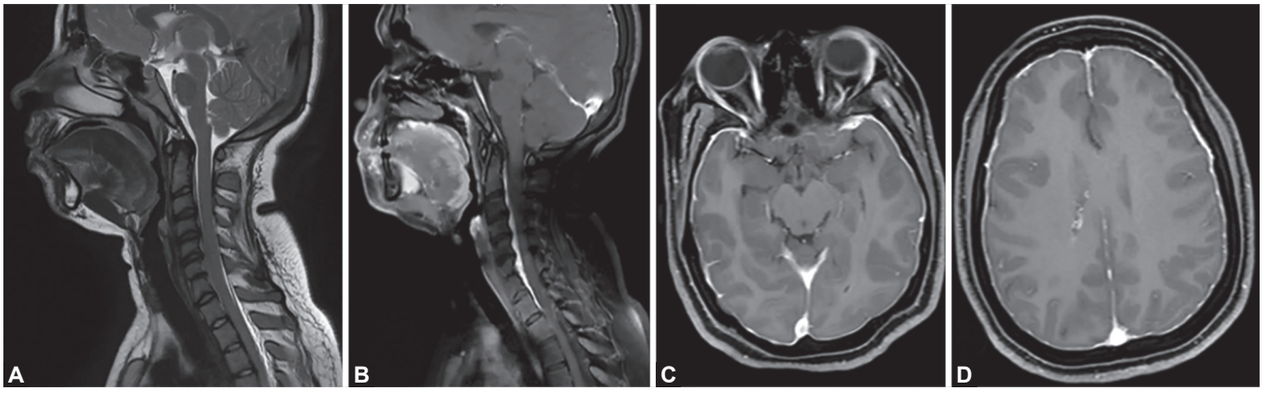
HISTORY

A middle-aged patient presented with a two-week history of severe orthostatic headache, described as worsening upon standing and improving when supine. Symptoms have been unresponsive to analgesics and are now associated with intermittent neck stiffness and mild nausea. No recent trauma, lumbar puncture, or infectious symptoms were reported, prompting MRI evaluation for underlying structural or cerebrospinal fluid abnormalities.

Intracranial hypotension, or craniospinal hypotension, is a condition caused by cerebrospinal fluid (CSF) leakage, usually from the spine, into the epidural space or directly into veins via CSF-venous fistulas.

CLASSIFICATION OF INTRACRANIAL HYPOTENSION

- **Primary (spontaneous) intracranial hypotension (SIH):** Occurs without an obvious cause, often related to spinal CSF leaks.
- **Secondary intracranial hypotension:** Osteophytes at cervicodorsal spine, iatrogenic causes (e.g., lumbar punctures, surgeries), traumatic injuries, or excessive shunting.
- Notably, not all CSF leaks lead to intracranial hypotension. Skull base leaks rarely cause the syndrome, and leaks above the hydrostatic indifference point (usually the lower cervical or upper thoracic spine) are less likely to result in clinical hypotension.



Case description

Sagittal T2-weighted images (A) demonstrate classical features of intracranial hypotension, including distension of the torcular Herophili, upward convexity of the pituitary gland, reduction of the mammillo-pontine angle, and posterior sagging of the corpus callosum. Sagittal (B) and axial (C and D) post-contrast images show diffuse pachymeningeal enhancement along the bilateral cerebral convexities, further supporting the diagnosis.

In addition, spinal imaging reveals an anterior epidural thickening extending from C4 to C7 levels, without evidence of significant spinal canal compromise or cord compression. These findings support a diagnosis of spontaneous intracranial hypotension, likely secondary to a spinal dural leak.

DIAGNOSIS

Intracranial hypotension (pachymeningeal enhancement, sagging brain).

INTRODUCTION

Intracranial hypotension is a condition caused by a reduction in cerebrospinal fluid (CSF) volume, often due to a CSF leak through a dural defect. It can occur spontaneously (spontaneous intracranial hypotension, SIH) or as a result of trauma, surgery, or lumbar puncture. The hallmark clinical symptom is an orthostatic headache that improves when lying down. If untreated, it can lead to complications such as subdural hematomas, cerebellar tonsillar herniation, and cerebral venous sinus thrombosis. Early recognition and treatment, including epidural blood patches or surgical repair of the leak, can prevent long-term sequelae.

DETAILED IMAGING FINDINGS

Imaging, particularly MRI, plays a crucial role in diagnosing intracranial hypotension. Key findings are given as follows.

Cranial MRI Findings

- **Diffuse pachymeningeal enhancement:** Smooth, non-nodular enhancement of the dura on post-contrast T1-weighted images.
- **Brain sagging:** Downward displacement of the brainstem and cerebellar tonsils with effacement of the suprasellar cistern.
- **Subdural fluid collections:** Hygromas or hematomas due to stretching and rupture of subdural veins.
- **Pituitary enlargement:** Reactive hyperemia causing reversible pituitary gland enlargement.
- **Venous engorgement:** Rounded appearance of dural venous sinuses (venous distention sign).
- **Flattening of the pons:** Decreased pontomesencephalic angle (<40°) and shortened mamillopontine distance (<6.5 mm).

Spinal Imaging Findings

- **Epidural fluid collections:** High T2 signal intensity in the spinal epidural space.
- **Dural enhancement:** Thickened and enhancing dura along the spinal canal.
- **Dilated epidural veins:** Secondary to reduced CSF pressure.
- **Active CSF leak:** Visible contrast extravasation on CT or MR myelography.

KEY FEATURES

The most significant imaging characteristics of intracranial hypotension include:

- **Diffuse pachymeningeal enhancement:** The most common cranial MRI finding.
- **Brain sagging and tonsillar herniation:** Indicative of reduced intracranial buoyancy.
- **Subdural collections:** Hygromas or hematomas secondary to low CSF pressure.
- **Venous distention sign:** Rounded dural venous sinuses on sagittal imaging.
- **Pituitary hyperemia:** Enlarged pituitary gland with increased height (>6.9 mm).

DIFFERENTIAL DIAGNOSIS

Intracranial hypotension must be differentiated from other conditions with overlapping imaging features:

Condition	*Imaging features*
Aseptic meningitis	Pachymeningeal enhancement but with inflammatory signs (e.g., nodularity)
Chiari malformation type I	Cerebellar tonsillar herniation without pachymeningeal enhancement or subdural collections
Pituitary adenoma	Pituitary enlargement but without brain sagging or pachymeningeal enhancement
Subdural hematoma (trauma)	Subdural collections without other signs of low CSF pressure (e.g., brain sagging)
Cerebral venous sinus thrombosis (CVST)	Venous engorgement with thrombosis but no pachymeningeal enhancement or brain sagging

Clinical history and advanced imaging techniques like myelography are essential for accurate differentiation.

RECENT ADVANCES: Intracranial hypotension demonstrate

Contrast-enhanced MRI reveals diffuse, smooth dural enhancement and venous engorgement (e.g., "venous distension sign" in dural sinuses), with MR elastography (MRE) detecting reduced brain stiffness and altered damping ratio as novel biomarkers. T2/FLAIR sequences exhibit subdural hygromas (CSF-intensity collections) and brainstem sagging, while pituitary enlargement correlates with chronicity. Diffusion-weighted imaging shows no characteristic restriction; diagnosis relies on structural/contrast features rather than diffusion changes.

PEARLS IN NEURORADIOLOGY WITH SALIENT FEATURES

Intracranial hypotension is a potentially debilitating condition that is often underdiagnosed due to its variable clinical presentation and imaging findings. Key imaging features include diffuse pachymeningeal enhancement, brain sagging, subdural collections, venous engorgement, and pituitary enlargement. Early diagnosis using MRI and targeted spinal imaging is critical for effective management, including epidural blood patches or surgical repair of CSF leaks. Recognizing these hallmark features ensures timely intervention and

prevents complications such as subdural hematomas or cerebellar herniation.

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Case 74

HISTORY

An elderly patient presents with gradually worsening gait disturbance, cognitive decline, and new-onset urinary incontinence over several months. Family reports frequent falls and increasing forgetfulness despite stable medical management of comorbidities. These symptoms have progressed insidiously, prompting neuroimaging to evaluate for reversible structural or neurodegenerative causes.

DIAGNOSIS

Normal pressure hydrocephalus (NPH).

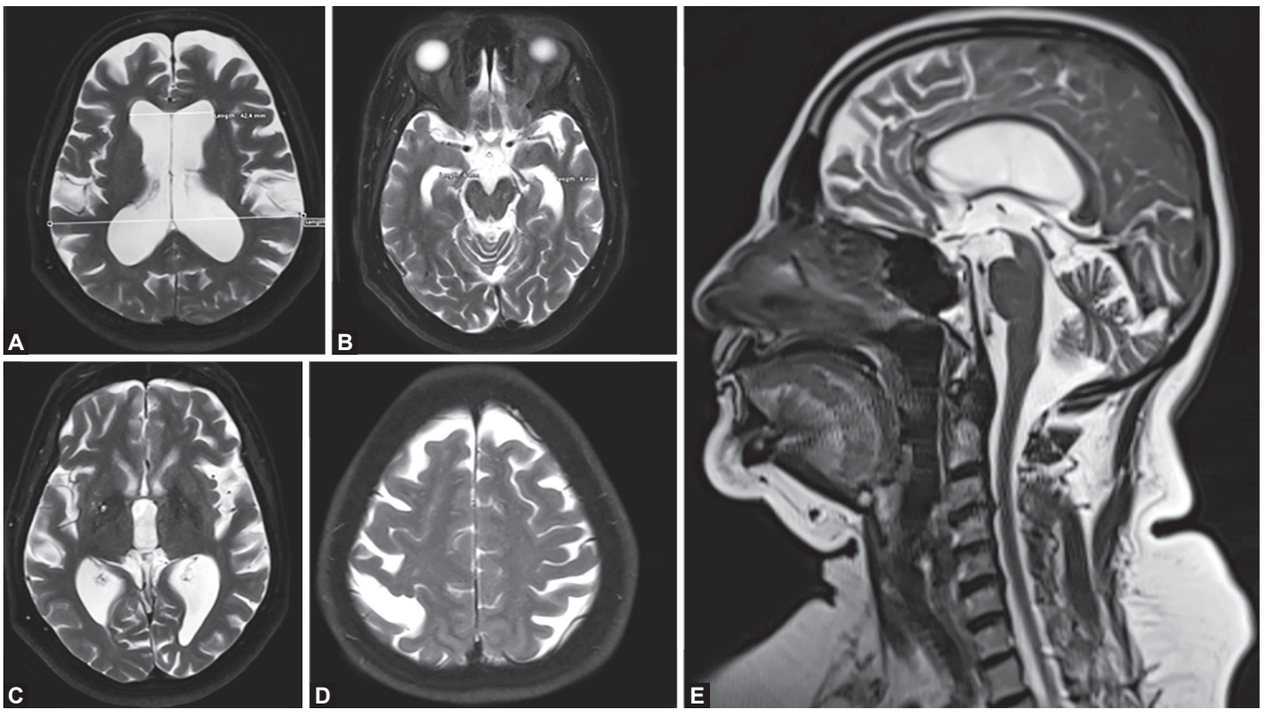
INTRODUCTION

Normal pressure hydrocephalus (NPH) is a treatable neurological disorder characterized by ventriculomegaly due to impaired cerebrospinal fluid (CSF) absorption, typically affecting adults over 65. It presents with the classic triad of gait disturbance, cognitive decline, and urinary incontinence, often mimicking neurodegenerative diseases. Early diagnosis through imaging is critical, as surgical CSF diversion (e.g., ventriculoperitoneal shunt) can reverse symptoms in 70–80% of cases.

DETAILED IMAGING FINDINGS

Structural MRI/CT Features

- **Ventriculomegaly:**
  - Evans index >0.3 (maximal frontal horn width divided by inner skull diameter).
  - Temporal horn width ≥6 mm, disproportionate to hippocampal atrophy.
- **Callosal angle:** ≤90° on coronal MRI (vs. >90° in atrophy).
- **DESH sign:** Disproportionately enlarged subarachnoid spaces with tight high-convexity sulci and widened Sylvian fissures.
- **Periventricular hyperintensities:** T2/FLAIR hyperintense bands along lateral ventricles (70% of cases).



Case description

Neuroimaging demonstrates ventriculomegaly with disproportionate dilatation of the bilateral lateral ventricles and third ventricle, associated with mild periventricular signal change consistent with transependymal CSF seepage. The Evans' index is increased (> 0.3) measured in (A). There is widening of the temporal horns of the lateral ventricles (> 6 mm) (B), not attributable to hippocampal atrophy. Associated features of disproportionately enlarged subarachnoid space hydrocephalus (DESH) are present, characterized by enlarged Sylvian fissures (C) with relative narrowing of the high-convexity and parafalcine cortical sulci (D), out of proportion to generalized cortical atrophy. Mild upward bowing of the corpus callosum is also demonstrated on T2w sagittal(E).

- **Aqueductal flow void:** Hyperdynamic CSF flow on T2-weighted MRI (50–70% specificity).

Advanced Imaging Techniques

- **Phase-contrast MRI:** Elevated aqueductal CSF stroke volume (>42 µl).
- **Diffusion tensor imaging (DTI):** Reduced fractional anisotropy in periventricular white matter.
- **Magnetization transfer ratio (MTR):** Decreased values in periventricular regions.

Post-shunt Changes

- Reduction in ventricular size (30–50% within 6 months).
- Resolution of periventricular hyperintensities and DESH features.

KEY FEATURES

- **Clinical triad:** Gait apraxia (80–95% cases), subcortical dementia (50–75%), and urinary incontinence (60%).
- **Imaging biomarkers:**
  - Evans index >0.3 + DESH sign: 89% positive predictive value for shunt responsiveness.
  - Callosal angle ≤90°: 86% sensitivity for NPH *vs* Alzheimer's disease.
- **Reversibility:** 60–80% show gait improvement within 3 months post-shunt.

DIFFERENTIAL DIAGNOSIS

Condition	*Key imaging features*	*Distinguishing factors*
Alzheimer's disease	Medial temporal lobe atrophy, preserved ventricles	Amyloid PET positivity, no DESH sign
Parkinson's disease	Normal ventricles, nigrosomal iron deposition on SWI	Tremor-dominant symptoms, DaTSCAN abnormalities
Vascular dementia	Confluent white matter hyperintensities, microbleeds	Stepwise cognitive decline, hypertension history
Chronic hydrocephalus	Persistent transependymal edema, aqueductal stenosis	Elevated ICP symptoms (headache, papilledema)

RECENT ADVANCES: Normal pressure hydrocephalus (NPH) demonstrate

Structural MRI reveals ventriculomegaly with disproportionately enlarged subarachnoid space hydrocephalus (DESH), characterized by high-convexity tight sulci and enlarged sylvian fissures, aiding differentiation from neurodegenerative disorders. Diffusion tensor imaging (DTI) shows microstructural changes detectable via mean diffusivity histogram analysis, distinguishing NPH from Alzheimer's/Parkinson's with 86% sensitivity and 96% specificity. MR elastography identifies reduced brain stiffness as a novel biomarker, while contrast enhancement and T2/FLAIR signal abnormalities are absent in uncomplicated NPH.

PEARLS IN NEURORADIOLOGY WITH SALIENT FEATURES

NPH is characterized by **ventriculomegaly (Evans index >0.3)**, **DESH sign**, and **narrowed callosal angle** on MRI/CT. The presence of ≥2 imaging biomarkers (e.g., aqueductal flow void, temporal horn dilation) increases diagnostic specificity to 85%. While DESH has 77% positive predictive value for shunt response, combining clinical triad assessment with CSF tap-test improves sensitivity to 90%.

DIFFERENTIAL DIAGNOSIS

Requires exclusion of neurodegenerative disorders via amyloid PET or DaTScan. Early shunt placement halts progression in 70% of cases, with gait improvement often preceding cognitive recovery.

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Case 75

HISTORY

A 40-year-old female presents with a chronic, worsening headache over several months, now accompanied by intermittent episodes of transient visual obscurations. Symptoms persist despite analgesic use, and she reports occasional pulsatile tinnitus. There is no history of trauma, recent infection, or prior similar complaints.

DIAGNOSIS

Idiopathic intracranial hypertension (IIH).

INTRODUCTION

Idiopathic intracranial hypertension (IIH), also known as pseudotumor cerebri, is a condition characterized by elevated intracranial pressure (ICP) without an identifiable cause. It predominantly affects young, overweight women and presents with symptoms such as headache, pulsatile tinnitus, transient visual obscurations, and papilledema. If untreated, IIH can lead to permanent visual impairment. Diagnostic criteria include clinical signs of increased ICP, normal cerebrospinal fluid (CSF) composition, and exclusion of secondary causes through neuroimaging.

DETAILED IMAGING FINDINGS

Imaging findings in IIH are crucial for diagnosis and exclusion of secondary causes. Key observations include:

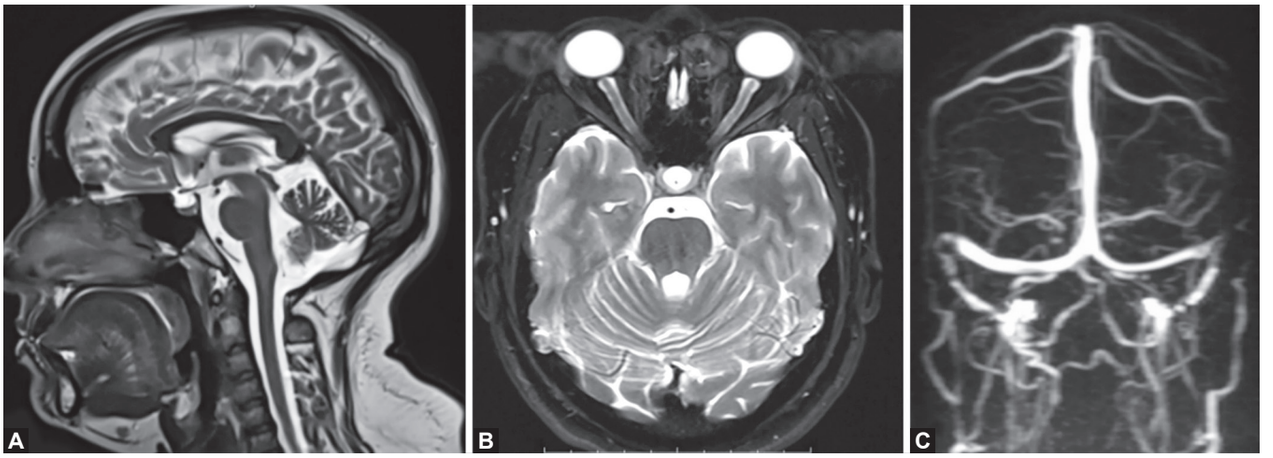
- **MRI findings:**
  - *Empty sella turcica:* Partial or complete flattening of the pituitary gland due to increased ICP.
  - *Posterior globe flattening:* Flattening of the posterior sclera observed on orbital imaging.
  - *Optic nerve sheath distension:* Enlargement of the perioptic subarachnoid space with or without optic nerve tortuosity.
  - *Optic nerve protrusion:* Intraocular protrusion of the optic nerve head due to papilledema.
  - *Transverse venous sinus stenosis:* Smooth-walled venous narrowing in one or both transverse sinuses.
  - *Slit-like ventricles:* Narrowing of the ventricular system without hydrocephalus.
- **MR venography (MRV):** Identification of transverse sinus stenosis or collapse.
- **CT findings:** Normal brain parenchyma with no evidence of hydrocephalus or mass lesions.

These imaging features are non-specific but strongly suggestive when combined with clinical findings.

KEY FEATURES

The most significant imaging characteristics of IIH include:

1. Empty sella turcica, often seen on sagittal MRI.
2. Posterior globe flattening, indicative of elevated ICP.
3. Optic nerve sheath distension, a hallmark sign of raised CSF pressure.



Case description

Sagittal T2-weighted MRI (A) demonstrates a partially empty sella, reflecting downward remodeling of the sella turcica secondary to chronically elevated intracranial pressure. Axial T2-weighted MRI (B) also shows a partially empty sella, along with prominent perioptic nerve sheath fluid bilaterally, suggestive of increased cerebrospinal fluid pressure along the optic nerve sheaths. 3D MR venography (C) reveals bilateral transverse sinus stenosis, involving the lateral-most segments, a typical venous abnormality associated with IIH.

4. Transverse venous sinus stenosis, detected via MRV.
5. Papilledema-related optic nerve protrusion, visible on orbital imaging.

These features are essential for differentiating IIH from other conditions causing increased ICP.

DIFFERENTIAL DIAGNOSIS

Differentiating IIH from other causes of increased ICP and papilledema is critical. Common differential diagnoses include:

Condition	*Imaging features*
Brain tumor	Mass lesion with edema; contrast enhancement
Venous sinus thrombosis	Irregular narrowing or occlusion of venous sinuses on MRV
Hydrocephalus	Enlarged ventricles; obstructive flow patterns
Choroid plexus papilloma	Enhancing lesion in ventricles with increased CSF production
Meningitis	Meningeal enhancement; CSF abnormalities on lumbar puncture
Pseudopapill-edema	Normal optic disc structure without signs of ICP elevation

Careful evaluation of neuroimaging findings combined with clinical criteria ensures accurate diagnosis.

RECENT ADVANCES: Idiopathic intracranial hypertension (IIH) demonstrate

T2/FLAIR MRI reveals optic nerve sheath distension (≥5.7 mm), posterior globe flattening, and empty sella (pituitary flattening >50%), while transverse sinus stenosis correlates with papilledema severity. Diffusion-weighted imaging (DWI) shows no parenchymal restriction; optic nerve DTI reveals reduced fractional anisotropy indicating axonal injury in chronic papilledema. Contrast enhancement is absent in brain parenchyma; subtle leptomeningeal enhancement may occur in severe cases but lacks diagnostic specificity.

PEARLS IN NEURORADIOLOGY WITH SALIENT FEATURES

Idiopathic intracranial hypertension is a syndrome characterized by elevated ICP without an identifiable cause, primarily affecting young overweight women. Imaging plays a pivotal role in diagnosis by identifying features such as empty sella turcica, optic nerve sheath distension, posterior globe flattening, and transverse venous sinus stenosis.

KEY TAKEAWAYS

- IIH is a diagnosis of exclusion requiring neuroimaging to rule out secondary causes.
- MRI and MRV are the preferred modalities for detecting characteristic imaging markers.
- Prompt recognition and treatment are essential to prevent permanent visual loss.

Understanding the imaging findings and differential diagnoses ensures effective management and improved patient outcomes.

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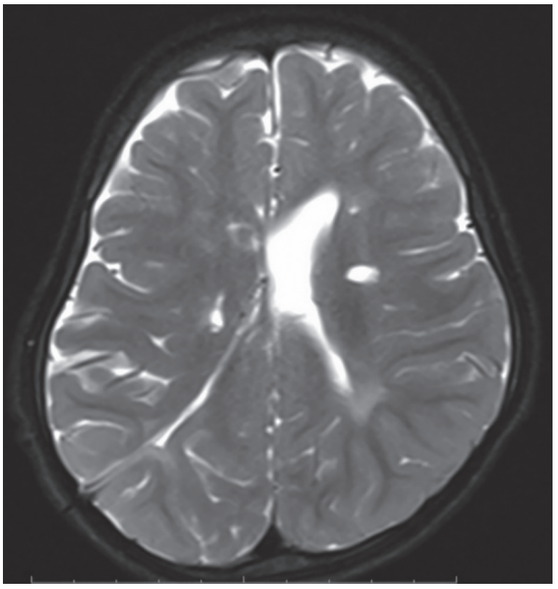
Case 76

HISTORY

A 10-year-old child with a history of ventriculoperitoneal shunt revision 8 months prior now presents with 2 weeks of recurrent, positional headaches and episodic giddiness triggered by sudden movements. Symptoms intensify upon standing and are accompanied by transient visual blurring, though neurological exam remains non-focal.



Case description

Axial T2-weighted, there is collapse of the body of the right lateral ventricle, giving it a slit-like appearance. A linear T2 hyperintense tract is seen extending from the collapsed right lateral ventricle to the right parietal cortex, consistent with post-VP shunt status and suggestive of chronic CSF overdrainage. There is mild periventricular ooze adjacent to the left lateral ventricle, likely due to altered regulation of CSF pressures. Gliosis is noted in the bilateral corona radiata.

DIAGNOSIS

Shunt overfunction (slit ventricle syndrome).

INTRODUCTION

Shunt overfunction, or overdrainage, occurs when a ventriculoperitoneal (VP) shunt excessively drains cerebrospinal fluid (CSF), leading to intracranial hypotension and complications. It affects 10–30% of shunted patients, often causing posture-dependent symptoms and requiring urgent imaging to prevent irreversible neurological damage. Early recognition through characteristic neuroimaging patterns is critical for timely intervention.

DETAILED IMAGING FINDINGS

Structural Features

- **Ventricular collapse:**
  - Slit-like lateral/third ventricles (axial width ≤2 mm).
  - Loss of periventricular CSF spaces on T2-weighted MRI.
- **Intracranial hypotension signs:**
  - Venous distension sign (convex inferior transverse sinus margin on sagittal MRI).
  - Pachymeningeal enhancement (68% sensitivity).
  - Brain sagging with tonsillar descent (42% of chronic cases).

Shunt-related Changes

- Catheter migration into brain parenchyma (21% of overdrainage cases).
- Subdural hygromas/hematomas (30% prevalence).

Advanced Techniques

- *Phase-contrast MRI:* Reduced aqueductal CSF flow (≤5 ml/min vs 10–15 ml/min normal).
- *SWI:* Prominent cortical veins with increased susceptibility artifact.

KEY FEATURES

Diagnostic Triad

1. Postural headaches (worse upright, relieved supine).
2. Slit ventricles + venous distension sign on MRI.
3. Shunt valve refill delay (>5 seconds after compression).

High-risk Populations

- Pediatric patients with congenital hydrocephalus (4 higher risk).
- Shunts using low-pressure valves (70% of overdrainage cases).

DIFFERENTIAL DIAGNOSIS

Condition	*Imaging differentiation*	*Key clinical distinction*
Shunt underdrainage	Ventricular enlargement, transependymal edema	Headaches worsen when supine
CSF leak	Spinal epidural fluid collections, dural tears	Beta-2 transferrin positivity in nasal discharge
Migraine	Normal ventricular size, absence of shunt signs	Response to triptans
Infection	Perishunt enhancement, leptomeningeal inflammation	Fever, elevated CSF WBC count

RECENT ADVANCES: Shunt overfunction

The available studies focus on unrelated topics: Structural neuroimaging in idiopathic normal-pressure hydrocephalus prognosis, contrast-enhanced T2-FLAIR for meningitis detection, and pediatric central tegmental tract hyperintensity markers. None describe imaging correlates of shunt overfunction such as diffusion restriction, T2/FLAIR changes, or contrast enhancement.

PEARLS IN NEURORADIOLOGY WITH SALIENT FEATURES

Shunt overfunction should be suspected when imaging reveals:

1. **Slit-like ventricles** with concurrent venous distension sign.
2. **Posture-dependent symptoms** correlating with intracranial pressure changes.
3. **Subdural collections** without trauma history.

Definitive management involves valve adjustment (programmable systems) or anti-siphon device placement. Emerging technologies like smart shunt sensors show promise for real-time flow monitoring, potentially reducing revision rates by 40%. Serial MRI remains essential for tracking ventricular compliance and preventing chronic complications like craniosynostosis.

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Case 77

HISTORY

A 28-year-old male presents with persistent clear nasal discharge and intermittent headaches following minor head trauma one week ago. He reports no fever or visual changes, and symptoms have gradually worsened despite conservative management. Clinical concern arises for a post-traumatic complication involving the anterior skull base.

DIAGNOSIS

CSF rhinorrhea (cribriform plate defect, meningocele).

INTRODUCTION

Cerebrospinal fluid (CSF) rhinorrhea results from an abnormal communication between the subarachnoid space and sinonasal cavity, often presenting as intermittent clear nasal discharge. Early diagnosis is critical due to a 19% lifetime risk of meningitis if untreated. Imaging plays a pivotal role in localizing skull base defects and guiding surgical repair.

DETAILED IMAGING FINDINGS

High-resolution CT (HRCT)

- **Bone defects**: Detects 88–95% of skull base fractures or dehiscences, commonly at the cribriform plate (33%), ethmoid roof (28%), or sphenoid sinus (22%).
- **Multiplanar reconstructions**: Essential for assessing defect size and surgical planning.

CT Cisternography

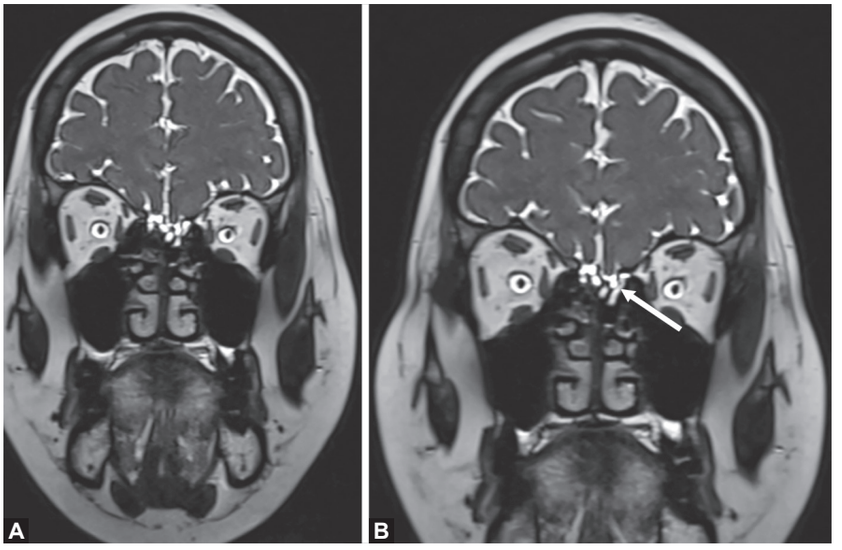
- **Contrast extravasation**: Identifies active leaks with 95% sensitivity when performed during active CSF flow.
- **Limitations**: Requires lumbar puncture and real-time leakage, missing intermittent cases.

MRI/MR Cisternography

- **T2-weighted sequences**: Demonstrates CSF tracts as hyperintense fluid connecting subarachnoid space to sinonasal cavities.
- **Contrast-enhanced MR cisternography**: Achieves 99% sensitivity and 100% specificity using intrathecal gadolinium (off-label).
- **Heavily T2 3D imaging**: Visualizes CSF leaks without radiation, ideal for pediatric cases.

Ancillary Signs

- **Intracranial hypotension**: Brain sagging, venous distension, or pachymeningeal enhancement in chronic cases.
- **Encephaloceles**: Associated with 40% of spontaneous leaks, particularly in idiopathic intracranial hypertension.



Case description

(A) Coronal FIESTA focal hyperintensity extending into the left ethmoid air cells, suggestive of CSF signal intensity in the region. Mild inferior displacement (sagging) of the insulated olfactory bulb is noted. (B) Zoomed coronal FIESTA red arrow highlights a CSF signal tract extending into the left ethmoid sinus confirming a CSF leak.

KEY FEATURES

Diagnostic Triad

1. Positional clear rhinorrhea (worsens with Valsalva).
2. HRCT-confirmed skull base defect.
3. Beta-2 transferrin positivity (100% specific for CSF).

Etiologic Patterns

- **Traumatic** (44%): Ethmoid roof defects with comminuted fractures.
- **Spontaneous** (28%): Cribriform plate or lateral sphenoid recess defects with meningoceles.
- **Tumor-related**: Osteolytic lesions with enhancing soft tissue masses.

DIFFERENTIAL DIAGNOSIS

Condition	*Imaging features*	*Key differentiators*
Allergic rhinitis	Normal skull base, mucosal thickening	Negative beta-2 transferrin
Sinusitis	Air-fluid levels, no bone defects	Purulent discharge, fever
Migraine-related rhinorrhea	Unremarkable HRCT/MRI	Responsive to triptans
Skull base tumors	Osteolytic lesions with mass enhancement	Biopsy-proven histopathology

RECENT ADVANCES: CSF rhinorrhea

CSF rhinorrhea highlight the efficacy of contrast-enhanced FLAIR subtraction techniques, which improve detection of leak sites by visualizing T2/FLAIR signal changes and contrast enhancement at osteodural defects. This method demonstrates heightened sensitivity to gadolinium leakage compared to conventional T1-weighted imaging, enabling precise localization of small fistulas.

PEARLS IN NEURORADIOLOGY WITH SALIENT FEATURES

CSF rhinorrhea requires urgent evaluation when:

1. Intermittent clear rhinorrhea correlates with positional changes.
2. HRCT reveals skull base defects >3 mm, particularly in the ethmoid or sphenoid regions.
3. MR cisternography confirms active leakage in equivocal cases.

First-line management combines HRCT for bony detail and beta-2 transferrin testing for biochemical confirmation. Spontaneous leaks warrant MRI to exclude idiopathic intracranial hypertension, as 70% recur without acetazolamide or shunt

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