Intracranial hypotension: step by step

Poster No.: C-2550
Congress: ECR 2018
Type: Educational Exhibit
Keywords: Neuroradiology brain, Neuroradiology spine, CT, MR, Myelography, Diagnostic procedure, Education, Cerebrospinal fluid, Diverticula, Fistula
DOI: 10.1594/ecr2018/C-2550

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Learning objectives

- Description of intracranial hypo and hypertension and its main causes.

- Approach to the diagnostic management of intracranial hypotension in the different clinical situations, understanding of radiological findings and physiopathological substrate.
Background

**THE INTRACRANIAL HYPOTENSION** is defined as a lower opening pressure (OP) than 6 cmH$_2$O. It is a relatively rare disorder with an annual incidence of 5 per 100,000 inhabitants/year, with a female to male ratio 2:1.

Intracranial pressure (ICP) increases with Valsalva maneuvers, decreases with standing and equals that of the medullary canal with lateral decubitus.

This can be estimated by measuring the OP of the cerebrospinal fluid (CSF) by lumbar puncture (LP) and is determined by the sum of volumes of brain, CSF and intracranial blood, which remains constant under normal conditions.

Approximately two thirds of the **CSF is produced and secreted** by the choroid plexus.

The **main clinical feature** is orthostatic headache which is defined as a development or aggravation of headache when patients move from a supine to an upright position and disappears or relieves after lay down.

In addition to headaches, a wide variety of other symptoms have also been reported.

**The etiology** is a decrease in the amount of the CSF, usually by loss of it, either spontaneous or secondary (post-traumatic, post-surgical, systemic disease, post-LP...) and rhinorrhea and / or otorrhea may exist. The pathophysiological mechanisms of hypotension and CSF hypertension are interrelated, as a situation of hypertension may lead to hypotension secondary to spontaneous CSF leakage.

In **spontaneous intracranial hypotension** there is no known cause. There may be situations in which there is a weakness of the preexisting dural sac or there are clear traumatic antecedents, iatrogenic (as in the case of a LP) or post-surgical.

CSF fistulas can be located:

- **Cribriform lamina**: is the most frequent site and it can be associated with meningocele, arachnoid hernia and arachnoid cyst. The signs are unilateral mass, opacification of the olfactory recess and descending gyro-rectum. (Fig. 1 on page 8, Fig. 28 on page 30, Fig. 32 on page 29).
- **Sphenoids**: perisellar region (sphenoid roof, sellar floor), transclival and lateral recess. (Fig. 2 on page 8, Fig. 3 on page 9).

- **Transphenoidal** or **intrarsphenoidal**. It is believed that the persistence of the Stenberg canal can cause CSF fistulas.

- **Temporal and middle ear**: tegmen tympani and mastoideum. (Fig. 4 on page 10, Fig. 32 on page 29).

- **In the petrosal apex**, dura mater or arachnoid can be herniated from the Meckel cave.

Several imaging tests are available to support the diagnosis of intracranial hypotension, including head CT / paranasal sinuses, head MRI, spinal MRI, MR and CT Cisternography, radionuclide cisternogram and myelography.

Knowing the pathophysiology of this disease we will understand the different typical manifestations that can be found in imaging tests.

- The decrease in volume and intracranial pressure will lead to a descent of the brain and traction on its anchoring structures, producing an **obliteration of the subarachnoid cisterns and ventricular collapse**, evident in both CT and MRI.

- The rupture of the small lymphatic vessels located in the thickness of the dura-arachnoid interface and bridge veins conditions the formation of **subdural or hygromas collections**. (CT, MRI). (Fig. 5 on page 11).

- The increase in intracranial blood volume, which compensates the decrease in CSF volume, is responsible for the **dilation of meningeal vessels and cortical veins**. (CT, MRI). (Fig. 6 on page 12, Fig. 7 on page 13).

- **Pachymeningeal diffuse enhancement** (MRI) produced by dilatation of the vessels and hyperemia, this being the most recognized image finding. (Fig. 8 on page 14, Fig. 7 on page 13).

- **Pituitary hyperemia** (MRI) is a cardinal sign and occurs due to congestion of the hypophyseal venous plexus which can be mistaken for an adenoma. (Fig. 9 on page 15).
-At the spinal level we can find extra-axial CSF collections that cause compression in the medullary cord or nerve roots, dilated epidural and intradural veins, dural enhancement, meningeal diverticula, liquid collection in the soft tissues after C1-C2 or epidural or subdural collections. The epidural space allows the CSF to move easily vertically, so the location of the epidural collection doesn't necessarily correlate with the site of the fistula. MRI of the spine may be useful when there are no intracranial findings or are equivocal. (Fig. 10 on page 16, Fig. 11 on page 17, Fig. 12 on page 18).

Regarding the treatment of intracranial hypotension, there are conservative measures, such as bed rest, pharmacological or surgical (epidural blood patches or direct closure of the fistula).

IDIOPATHIC INTRACRANIAL HYPERTENSION (IIH) is defined as the elevation of ICP over 250 mmH20 with normal composition of the CSF without being justified by other causes.

It has an incidence of 1.2 / 100,000 inhabitants/year, which is increasing and is more prevalent in young women with obesity, reaching an incidence of 21 / 100,000 inhabitants/year. Obesity or rapid weight gain are considered risk factors to develop it.

The typical clinical symptoms are headache, transient vision loss (bilateral or unilateral), pulsatile tinnitus, photopsias or permanent loss of vision. The papilledema is a characteristic sign and is typically bilateral and symmetrical.

The pathogenesis of this entity is not clear. There are several theories that support that the increase in intracranial venous pressure is due to stenosis of the venous sinuses or that this stenosis is produced by the IIH. Also it may be due to an increase in the production of CSF with an increase in resistance in its absorption.

The typical findings in the imaging tests are:

1) Skull base:

-Empty sella secondary to herniation of the arachnoid space into the pituitary fossa through the diaphragm sellae and enlargement of the Meckel's cave (sensitivity (SN) 80%, specificity (SP) 83% estimated by Samuel Bidot et al). It is the most frequent finding but is also relatively frequent in the general population. (Fig. 13 on page , Fig. 14 on page 19, Fig. 15 on page 20, Fig. 16 on page 21, Fig. 20 on page 25, Fig. 33 on page 27).
- **Foramen ovale enlargement** (SN 50%, SP 80%).

2) Orbital findings:

- **Distension of the perioptic subarachnoid space** (SN 58%, SP 89%). (Fig. 17 on page 22, Fig. 20 on page 25, Fig. 21 on page 26).

- **Increase of the optic nerve tortuosity** (SN 43%, SP 90%). (Fig. 17 on page 22).

- **Posterior scleral flattening** (SN 66%, SP 98%): essential in the diagnosis, although it is not specific. (Fig. 21 on page 26).

- **Intraocular protrusion of the optic disc** (SN 36%, SP 99%).

- **Optical disc enhancement** (SN 16%, SP 99%): not very sensitive. (Fig. 18 on page 23, Fig. 21 on page 26). This finding is recognized in the post-contrast enhancement FLAIR sequences.

3) Vacular findings:

- **Transverse narrow sinus** (SN 97%, SP 93%). (Fig. 19 on page 24).

4) Parenchymal findings:

- **Herniation of the cerebellar tonsils** through the foramen magnum > 5 mm (SN 16%, SP 95%) and **meningoceles** (SN 11%, SP 0%) (Fig. 15 on page 20). The brain sagging is also present in the Chiari malformation. In 15% of general population there is a downward displacement of the cerebellar tonsils between 2 and 5 mm.

- **Small size of the ventricles**, although currently it is not given a great value.

- **CSF fistulas**: IIH may be a cause of fistula formation. There are theories that spontaneous fistulas are a variant of IIH because they act as a "natural" derivation of the CSF. In these cases there is usually no pachymeningeal enhancement or tonsillar herniation as there is no low CSF pressure. (Fig. 20 on page 25).
IIH is a diagnosis of exclusion and these imaging findings may be useful when the major criteria are not fully met.

**Treatment** will consist of weight loss in obese patients, pharmacological, serial LP or surgery.
**Images for this section:**

**CSF LEAK: CRIBRIFORM PLATE**

**Fig. 1:** A-Cisternography-CT, sagittal view and B-coronal view, with intrathecal contrast administration: CSF leak by the cribriform lamina.

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Fig. 2: CSF leak through the sphenoid. A-CT, supine decubitus, sagittal view: bone defect in clivus (blue arrow). B-CT-cisternography with intrathecal contrast, prone position: extravasation of CSF to the sphenoidal sinus.

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**Fig. 3:** A 40 years old male with a macroadenoma. He suddenly started with rhinorrhea. A CT-cisternography was perform. A-Coronal view, B-sagittal view and C-coronal view at the level of the nostril: CSF fistula through the left nostril, which seems to come from the sella turcica, through the sphenoid sinus.

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Fig. 4: A-CT axial view and B-CT cisternography with intrathecal contrast administration: bone erosions in the tegmen tympani and petrous apex with contrast extravasation from the cisterns to the left middle ear and apex petrous defect, possibly through several points. Findings compatible with CSF fistula through the left tegmen tympani and apex petrous.

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Fig. 5: Patient with spontaneous orthostatic headache and signs of intracranial hypotension in imaging tests; A-Brain MRI: sagittal view, T2-weighted, B-sagittal view FLAIR and C-coronal view, contrast-enhanced T1-weighted: bilateral frontal subdural collections.

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Fig. 6: Clinic of spontaneous intracranial hypotension. Brain MRI, axial view, contrast enhanced T1W: venous ingurgitation.

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**Fig. 7:** Brain MRI, coronal view, T1W post-contrast enhancement: universal dural enhancement and engorgement of venous plexus in the floor of the sella turcica.

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**Fig. 8:** IC hypotension after lumbar puncture. A- Brain MRI: coronal view, T2W: subdural collection vs dural thickening B-Brain MRI, coronal view, contrast-enhanced T1-weighted: confirmation of diffuse supra and infratentorial pachymeningeal enhancement.

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Fig. 9: Patient with intracranial hypotension data. A- Brain MRI: sagittal view, contrast-enhanced T1-weighted: engorged pituitary gland and collapse of the suprasellar cistern. B- Brain MRI, sagittal view, contrast-enhanced T1-weighted: normal pituitary and suprasellar cistern size after treatment.

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Fig. 10: A 33-year-old woman with suspected multiple sclerosis due to optic neuritis. Brain and spinal MRI (not shown) was performed and demyelinating lesions were present. She began with orthostatic headache after a LP. Complete spinal MRI was performed without and with IVC. The headache resolved after a blood patch. A- Spinal MRI: sagittal view, T2- weighted: posterior epidural hyperintense collection (arrow). B- Sagittal view T1- weighted :posterior epidural collection. C- contrast-enhanced T1- weighted: enhancement of the posterior epidural space.

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**Fig. 11**: A 42-year-old woman with clinical signs of spontaneous intracranial hypotension. Spinal MRI T2W with fat suppression was performed: prominent epidural space with venous plexus engorgement.

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Fig. 12: Patient with signs of SIH. Spinal MRI, axial view, T2W fat suppression: circumferential epidural collection.

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Fig. 14: 60-year-old patient with enlargement of Meckel’s caves and partially empty sella. A- Coronal view of a neck CT scan with contrast: dilated Mekel cavum. B-Sagittal section: partially empty sella.

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Fig. 15: A 60-year-old patient with CSF fistula and signs of intracranial hypertension. A - MRI cisternography, sagittal view and B - T1-weighted with intrathecal contrast: partially empty sella, meningocele in the cribriform plate and CSK leak to the nasal fossa.

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Fig. 16: An 80-year-old woman who progressively begins with headache, nausea and vomiting. Baseline brain CT scan shows signs of IIH. Conservative treatment was performed and 10 months later, a follow-up CT scan shows disappearance of the findings. A-Increase in size of the Meckel's caves. With bone window multiple bony defects at the skull base with the presence of pseudomeningocele (not shown). B- Control CT: disappearance of the findings.

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**DISTENSION OF THE SUBARACHNOID PERIOPTIC SPACE**

*Fig. 17:* Male of 60 years with signs of intracranial hypertension. Brain MRI, A-coronal view, fat-suppressed T2-weighted and B-sagittal reconstruction: distension of the perioptic subarachnoid space and tortuosity of the optic nerve.

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**Fig. 18:** A-Brain-MRI: axial view, T1W and B-contrast enhanced T1W: enhancement of the both optic discs in the postcontrast T1W sequences.

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Fig. 19: Stenosis of the left sigmoid sinus, left transverse and right sigmoid-transverse union. A- Venous angiography-MRI PC 3D contrast enhanced. B and C- Venous angiography-MRI PC 2D.

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Fig. 20: A 37-year-old woman with IIH and rhinorrhea through the right nostril. A-Brain MRI, sagittal view, fat-suppressed T2W: distension of the optic nerve sheaths. B- Sagittal view, fat-suppressed T2W: increase of Meckel's cavum and empty sella. C-coronal view, T1W: descent of frontal convolutions. D- Intrathecal Gadolinium-Enhanced MRI Cisternography, coronal view, T1W: leakage through the right cribriform lamina.
**Fig. 21:** A 40 years old female with signs of IIH and CSF leak in the cribriform plate (not shown). A- Brain-MRI, axial view, T2W: prominent subarachnoid space around the optic nerves with papilledema and posterior scleral flattening. B- axal FLAIR and C-axial FLAIR post-contrast: subtle enhancement of the papilla.

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**Fig. 33:** A 50-year-old man with a spontaneous CSF fistula on the left cribriform plate. A- MRI Cisternography, coronal view, T1W with intrathecal contrast: CSF leak at the cribriform plate. B-Brain MRI, sagittal view, T2W: empty sella.

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Fig. 31: A 55-year-old male operated in the right ear for cholesteatoma, since then he had rhinorrhea and has had three episodes of meningitis. A cisternography-MRI was performed where a continuity solution was observed in the anterior cranial fossa in the left cribiform plate with CSF output and herniation of the parenchyma through it. He was operated with a satisfactory result. A-MRI cisternography with intrathecal contrast, coronal view, T1W: contrast leakage by left cribrosa plate. B-Brain MRI, sagittal view cisternography: herniation of cerebral content and empty sella. C-MRI with fat suppression: distension of the subarachnoid perioptic space and tortuosity of the optic nerve. D- MRI cisternography with intrathecal contrast, sagittal view T1W: accumulation of contrast in the nasal fossa.

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**Fig. 32:** Various patients: A- MRI cisternography with intrathecal contrast, T1W coronal view: leakage of LCR left cribriform plate (spontaneous). B- T1W coronal view and C-axial contrast: fistula between internal auditory canal and geniculate ganglion, the contrast extavasates to the right tympanic cavity and soft parts of the neck. D- MRI cisternography with intrathecal contrast T1W, coronal view: right temporal fistula.

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Fig. 28: Patient with meningocele and CSF leak through the cribriform plate. CT cisternography without and with intrathecal contrast administration A-coronal view, B-axial view and C-sagittal view.

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Findings and procedure details

In this review, we present an approach to diagnostic management in these two situations with different imaging techniques: we describe the guidelines to follow, the different tests available and the typical findings, and we will focus on intracranial hypotension.

**IDIOPATHIC INTRACRANIAL HYPERTENSION:**

There are several criteria that make us suspect the presence of IIH, including the **modified Dandy criteria:**

- Symptoms and signs of increased ICP.
- Absence of other neurological anomalies or altered level of consciousness.
- Elevation of the ICP with normal composition of the CSF.
- No other apparent causes of intracranial hypertension.
- Neuroimaging studies do not reveal causes of intracranial hypertension.

The main image techniques are:

**Brain MRI:** the usual protocol in our hospital includes: axial FLAIR, axial diffusion, axial T1W and T2W, sagittal T2W (3 mm slice thickness), coronal T2W of orbits, axial FLAIR (2 mm slice thickness) pre and post contrast enhancement of orbits and venous angiography 2D PC of the sagittal and transverse sinus.

There are several findings that are typical of IHH (which have already been explained in the previous section) that should be searched.

**The head CT** observing some of these signs such as the empty sella, the tortuosity of the optic nerve and the small size of the ventricles.

**The ophthalmic ultrasound** would be useful to identify the intraocular protrusion of the optic disc and the distention of the perioptic subarachnoid space.
INTRACRANIAL HYPOTENSION (IH)

There are different clinical situations and therefore we will have several diagnostic possibilities at our disposal. We expose three situations:

1-Patient with symptoms of IH without evidence of CSF loss.
2-Presence of rhinorrhea and / or otorrhea.
3-Antecedent of LP and appearance of IH symptoms. (Fig. 22 on page 37).

The International Classification for Headache Disorders proposes this criteria for the diagnostic of SIH. (Fig. 23 on page 37).

The management of intracranial hypotension includes, in the case of being spontaneous and not existing rhinorrhea and/or otorrhea, brain CT and MRI, spinal MRI and cisternography-MRI / CT according to the need to establish the diagnosis, cause and place of the CSF-leak.

A proposed diagnostic algorithm would be the performance of head and spinal MRI. In the case of being positive, a blood patch can be used. If it doesn't work, this procedure can be repeated again. If it still doesn't work, it will be necessary to find the exact leakage site: if an extensive leak that affects more than two spinal segmentis is suggested, the myelogram or CT-myelogram may be useful, and if a small leak is suspected, a MRI with gadolinium could be performed. Site-directed epidural blood patch could be useful. If all this does not work, surgery would be necessary.

When brain and spinal MRI are negative, but there is a high clinical suspicion, a Intrathecal Gadolinium-Enhanced MR or CT Cisternography could be performed. If the findings are negative and there are still symptoms, the study could be repeated in 3-6 months.

If the patient has symptoms of intracranial hypotension accompanied by rhinorrhea / otorrhea, we can obtain a sample of them for the detection of b2-transferrin, which confirms the diagnosis, and then performe a paranasal sinus CT and brain MRI. If necessary, studies of cisternography are carried out to locate the exact point of leakage.
We review the studies in patients with suspected CSF fistula or hypotension of unknown cause in two centers, during the last 10 years.

Below we develop the different tools that exist for the diagnosis of IH:

1-Take sample of nasal / otological secretion: measure the activity of beta-2 transferrin (2-Tr) and trace protein Beta (p-T).

It is preferable to use p-T since it is measured by a fast method with good analytical performance. In case of doubtful values (0.64 and 1.14 mg / L) or that could not be made, 2-Tr would be carried out.

If it is negative, we must reevaluate clinically and if it is positive we can use an imaging test to locate the fistula. (Fig. 24 on page 38).

2-Image techniques

-Brain- CT/paranasal sinuses: this test is usually used in emergencies, as an aid to the initial diagnosis, but it is not used routinely. It is especially useful to locate bone defects at the skull base, making 1 mm slice thinkness and sagittal and coronal reconstructions. Although it is a nonspecific finding, in the appropriate clinical setting, a liquid-air level could suggest the presence of fistula at that level. (Fig. 25 on page 39, Fig. 26 on page 40, Fig. 27 on page 41).

-Cisternography and myelo-CT: needs intrathecal contrast administrated by LP (iodinated contrast, 20 cc) and active fistula. It most cases it can be perform in prone position, so that the contrast reaches the anterior and middle cranial fossa.

It has to be done in the early and late phase (2-4h later). It has the disadvantage of having ionizing radiation and is contraindicated in meningitis and intracranial hypertension (because a LP mus be done) which are pathologies that interest us. Now they are only used in complex cases. Small contrast extravasations are sometimes difficult to differentiate from bone because of the high density they both have. (Fig. 28 on page 42, Fig. 29 on page 43).

-Brain MRI: with and without intravenous gadolinium, using sequences highly enhanced in T2 with suppression of adjacent tissues. As advantages, it has a lot of contrast resolution and the possibility of performing PROPELLER sequences that reduces artifact by movement.
Some qualitative and quantitative findings suggestive of IH has been proposed by some studies:

Qualitative:

- Subdural collections (frequency: 36% -50%).
- Pachymeningeal enhancement (F 56% -83%).
- Venous engorgement (F 31% -93%).
- Pituitary gland enlargement (50% -63%).
- Brain sagging.
- Normal: 4% -20%.

Quantitative:

There are a few studies that have performed quantitative measurements via an MRI to support the diagnosis of IH and to provide more objective criteria (by Kerim Aslan et al).

Among these measures we can find the pontomesencephalic angle (PMA), mamillopontine distance (MPD) and mesencephalon anterior-posterior/medial-lateral diameter ratio, which have high sensitivity and specificity. They are difficult to measure. (Fig. 30 on page 44).

Taken together, the most suggestive findings of this pathology would be the dural enhancement along with venous engorgement and the decrease of the mamilopontine distance.

-MRI of the spine: may be useful when there are no intracranial findings or are equivocal. Sequences are made without and with intravenous gadolinium including T1W and T2W sequences without and with sagittal fat suppression, axial T1W and T2W. The typical findings are: extratecal CSF collections, epidural and dilated intradural veins, dural enhancement, meningeal diverticula, liquid collection in the soft tissues after C1-C2 or epidural or subdural collections.

-MR Cisternography: imaging study of the cerebrospinal system after administration of contrast material in the subarachnoid space.
The study protocol in hour hospital:

1- T1W sagittal and coronal+ fat saturation with the patient in prone position.

2- Intrathecal contrast administration by LP.

3- Trendelenburg position: 20-25 min (so that the contrast reaches the subarachnoid space of the middle and anterior cranial fossa).

3- Prone position with fat suppression.

If it is not clear we can perform axial and coronal sequences with Valsalva maneuver. If the point of leakage is still not clear we can repeat more sequences in 30-40 minutes, and if necessary, late sequences can be made.

In MRI, fistulas are seen as hyperintense tracts from the subarachnoid space to the sinuses. It can also be associated with alterations in the parenchyma and encephaloceles.

It is important to highlight the utility of CT / MRI cisternography to establish the diagnosis and location of CSF leakage, which is essential for therapeutic planning (whether surgical or not) in patients with more than one lesion or doubts in the baseline tests.

(Fig. 32 on page 46, Fig. 33 on page 47).

-Radionuclide cisternogram: it allows identifying the site where the loss of CSF occurs. The absence of radioactive activity in the cerebral convexity 24 hours after the administration of the radioisotope is the most commonly observed abnormality and also the early appearance of the radioisotope (<4 hours) in the bladder and kidneys.

-Digital subtraction myelography: for fast fistulas.
Fig. 22: A 56-year-old man with myelodysplastic syndrome. He was hospitalized for transplant of allogeneic hematopoietic progenitors. He started with symptoms of hypotension 10 days after lumbar puncture. Conservative treatment was performed and symptoms and radiological signs were resolved in a month. A-Brain CT axial view: bilateral fronto-parietal subdural hygromas. Left paratrial porencephalic cavity due to old infarction. B-Brain MRI, axial view, contrast enhanced-T1 weighted: diffuse pachymeningeal enhancement. C-Brain MRI coronal view, T2-weighted: bilateral subdural hygromas. D- Brain CT axial view one month later: resolution of subdural hygromas.

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• A-Any headache fulfilling Criterion B.

• B-Headache has developed in temporal relation to low CSF pressure or CSF leakage, or has led to its discovery.

• C-Low CSF pressure (<6 cm H2O) and/or evidence of CSF leakage on imaging.

• D-No better accounted for by another ICHD-3 diagnosis.

Fig. 23: The International Classification for Headache Disorders.

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The p-T is a protein with enzymatic activity, synthesized mainly in the choroid plexuses and leptomeninges. It is one of the most abundant proteins of CSF. It is also found, although in small concentrations, in other secretions such as aqueous humor and perilymph. In serum, the concentration range is 0.12-1.44 mg/L and in the CSF of 11.50-32.60 mg/L. The concentration ratio between CSF and serum varies between 30-40, and is the highest among CSF-specific proteins. In the nasal secretions of healthy people the concentration range is 0.22-1.69 mg/L.

A p-T value ≥ 1.14 mg/L indicates CSF fistula; A value ≤ 0.64 mg/L discards it. Values between 0.64 and 1.14 mg/L are not conclusive, it would be necessary to perform 2-Tr detection.

2-Tr is the desialized form of transferrin. It is found mainly in the CSF, although it is also present in the perilymph and in the aqueous and vitreous humors. It is not usually detected in serum or other secretions (except chronic and high alcohol consumption, and in certain low frequency genetic variants). It is considered a CSF reference marker, with a reported diagnostic sensitivity of 73-93% and a specificity of 97-100%. The main disadvantage of its use is that it requires a laborious electrophoretic method for its detection. As an advantage over the measurement of the pT it has been observed that the test has been perform in a higher number of samples, since the method requires a very small volume of sample and allows the application of the samples in the gel although they have a high viscosity grade.

Renal insufficiency and bacterial meningitis, which produce an increase and decrease, respectively, in the concentrations of p-T in the CSF. The qualitative detection of 2-Tr is carried out directly when the sample can not be processed for the measurement of p-T (high viscosity or low volume) or when the patient has renal insufficiency or suspicion of bacterial meningitis.

**Fig. 24:** Measure the activity of 2-Tr and p-T.

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Fig. 25: A 58-year-old woman with paresthesias in the right lower limb after a traffic accident. Lumbosacral MRI was performed and showed no alterations (not shown). Brain CT was also performed where a possible meningocele was observed in the right tegmen tympani. The study was completed with paranasal-sinuses CT and MRI temporal bone where a meningoencephalocele was observed in the right tegmen tympani. Axial CT: bone defect of tegmen tympani with soft tissue image on the roof of the mastoid antrum. B-CT coronal view and and C-MRI sagittal view FIESTA: cerebral parenchyma herniation.

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Fig. 26: A 73-year-old male with frontal traumatic brain injury (TBI) due to a syncope 5 years previously. Years later he began with rhinorrhea, analyzing and confirming that it was CSF. We performed a temporal bone CT and brain MRI. A-CT coronal view with bone window and B- with soft tissue window: bone defect with soft tissue herniation. C-Brain MRI coronal view, T2 weighted: fluid content in the right sphenoid sinus suggesting dural disruption. D-Head MRI fast imaging employing steady-state acquisition (FIESTA): Meningoencephalocele projecting from the middle cranial fossa to the pterygoid recess of the right sphenoid.

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**Fig. 27:** A 48-year-old woman with aqueous rhinorrhea and headache. She started with symptoms of meningitis. Head CT, head MRI and paranasal sinus CT were performed, where signs of benign intracranial hypertension were observed and an enlargement of the right olfactory sulcus on its anterior side. A-MRI, sagittal view T2-weighted: distention of the perioptic subarachnoid space and B- empty sella and meningocele of 6 -7 mm in cribriform plate. C-CT of the paranasal sinus, coronal view, bone window: enlargement of the right olfactory sulcus. D- Head MRI, coronal view T2 weighted: meningocele in the left olfactory sulcus.

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Fig. 28: Patient with meningocele and CSF leak through the cribriform plate. CT cisternography without and with intrathecal contrast administration A-coronal view, B-axial view and C-sagittal view.

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**Fig. 29:** Dorso-lumbar CSF leak: A-Spinal CT-cisternography, A-coronal view and B-axial view: contrast leakage to the epidural space the right level D10-D11. The leaky contrast column ascends along the entire dorsal column, reaching cervical levels and caudally to lumbar levels. The contrast goes to the right retroperitoneum.

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• **Abnormal measures:** MPD 4.86 mm, PMA 50.1 degrees and AP / ML ratios 0.89.

• **Normal measures:** MPD 7.22 mm, PMA 65.65 degrees and AP / ML ratios 0.76.

**Fig. 30:** Quantitative measures

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Fig. 31: A 55-year-old male operated in the right ear for cholesteatoma, since then he had rhinorrhea and has had three episodes of meningitis. A cisternography-MRI was performed where a continuity solution was observed in the anterior cranial fossa in the left cribriform plate with CSF output and herniation of the parenchyma through it. He was operated with a satisfactory result. A-MRI cisternography with intrathecal contrast, coronal view, T1W: contrast leakage by left cribrosa plate. B-Brain MRI, sagittal view cisternography: herniation of cerebral content and empty sella. C-MRI with fat suppression: distension of the subarachnoid perioptic space and tortuosity of the optic nerve. D- MRI cisternography with intrathecal contrast, sagittal view T1W: accumulation of contrast in the nasal fossa.

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**Fig. 32:** Various patients: A- MRI cisternography with intrathecal contrast, T1W coronal view: leakage of LCR left cribriform plate (spontaneous). B- T1W coronal view and C- axial contrast: fistula between internal auditory canal and geniculate ganglion, the contrast extavasates to the right tympanic cavity and soft parts of the neck. D- MRI cisternography with intrathecal contrast T1W, coronal view: right temporal fistula.

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**Fig. 33**: A 50-year-old man with a spontaneous CSF fistula on the left cribriform plate. A- MRI Cisternography, coronal view, T1W with intrathecal contrast: CSF leak at the cribriform plate. B-Brain MRI, sagittal view, T2W: empty sella.

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Conclusion

The imaging techniques help to diagnose intracranial hypotension and hypertension and cisternography is a useful tool in the topographic location and diagnosis of CSF leakage.
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