

Intracranial hypotension

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Abstract

The intracranial hypotension is the decrease in the intracranial pressure caused by the decrease in the volume of the cerebrospinal fluid secondary to the CSF loss. The intracranial hypotension is characterized by an orthostatic cephalgia, not calmed by analgesics accompanied by nausea, vomiting, vertigos, diplopia, etc. The diagnosis is confirmed by a lumbar manometry and by the performed paraclinical explorations (cerebral CT, cerebral MRI).

The intracranial hypotension treatment is - etiologic, and it consists in closing the CSF fistula; pathogenic, which consists in recreating the normal CSF volume, and symptomatic, which refers to the treatment of the symptoms related to intracranial hypotension.

Keywords: cerebrospinal fluid, CSF fistula, intracranial hypotension, intracranial pressure

Introduction

The intracranial hypotension represents the intracranial pressure decrease; it may be generated by the decrease in the volume of any of the intracranial components: the cerebral parenchyma, the cerebrospinal fluid, and the sanguine content and/or by the rapid removal of a pathologic volume under the circumstances of a pressure equilibrium achieved by the efficient compensating mechanisms (especially in

the case of chronic subdural hematomas).

The decrease in the volume of the cerebral parenchyma, by various mechanisms – senile cerebral atrophy, surgical resection, etc., does not generate first of all an intracranial hypotension syndrome, but there may be focal neurological symptoms depending on the suffering cerebral areas. The volume of the diminished cerebral parenchyma is progressively diminished by gliosis and/or by CSF; therefore, the intracranial pressure is maintained at normal values. The decrease in the intracranial sanguine volume by arterial hypotension leads to the decrease in the cerebral perfusion pressure with ischemic phenomena; this may also lead to a decreased CSF secretion, but the intracranial pressure decrease seems to be less important than ischemia. The surgical removal of a pathologic volume produces a sudden decrease in the endocranial volume and in the intracranial pressure, also known as the surgical cerebral-ventricular collapse, and which is rapidly compensated from a therapeutic point of view. Once the neurosurgical intervention has been performed, the endocranial cavity is no longer a close environment, and there is no correspondence between the intracranial hypotension and the surgical cerebral collapse. Therefore, of the three intracranial components, only the diminished volume of the cerebrospinal fluid intervenes in the generation of the intracranial hypotension. (Figure 1).

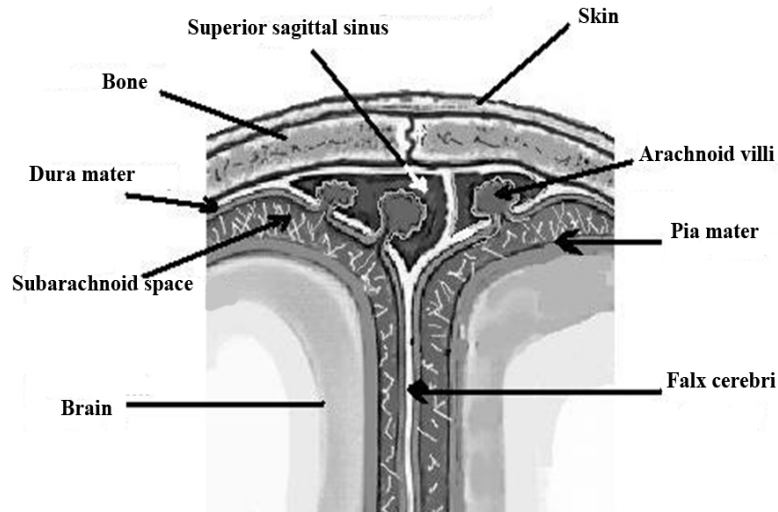


Figure 1 Scheme of the meningeal layers

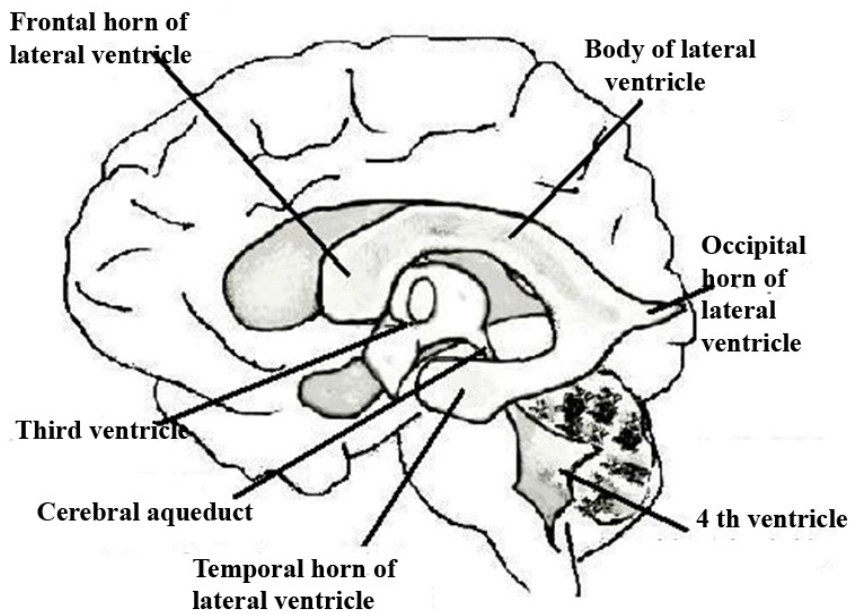


Figure 2 The ventricular system – schematic drawing

The intracranial hypotension is, therefore, the decrease in the intracranial pressure caused by the decrease in the volume of the cerebrospinal fluid secondary to the CSF loss (Figure 2). The intracranial hypotension is characterized by an orthostatic cephalgia, exacerbated by

coughing, laughing, the Valsalva maneuver, which is not calmed by analgics and is accompanied by nausea, vomiting, vertigos, diplopia, etc. The diagnosis is confirmed by a lumbar manometry and by the performed paraclinical explorations (cisternography, cerebral CT, cranial-cerebral RMN).

From a clinical perspective, the intracranial hypotension may be represented as:

- an acute form, with a violent symptomatology present in clinostatism, and which may evolve towards psychic disorders and the modification of the conscience state,
- a sub-acute form with absent symptoms or of a reduced intensity in clinostatism; they occur or they are exacerbated in orthostatism,
- a chronic, frequent form, with a symptomatology occurred in orthostatism, of smaller, bearable intensity and a tiresome feeling that may allow the development of an activity, but with a reduced efficiency though.

Etiology and pathogeny

The decrease in the normal CSF quantity in the cranial-spinal space may be generated by:

- the persistence of a dural continuity solution and the most frequent causes are the following ones:
 - operations involving the lumbar puncture: the exploratory lumbar puncture, myelography, myelo-CT, rachianesthesia,
 - cranial-spinal traumatism with the generation of a CSF fistula,
 - cranial-cerebral neurosurgical or spinal interventions,
 - extremely rarely, by a spinal anterior-lateral CSF fistula in thoracic surgery,
- decrease in the CSF secretion by various mechanisms (after radiotherapy, senile, etc.)
- reduced CSF quantity by a ventricular-peritoneal drainage,
- the so-called “spontaneous” intracranial hypotension, when none of the above-mentioned causes is underlined, but

the explorations prove the existence of several arachnoid fissures at the level of the spinal radicular sheath, or the case of the occult nasal CSF fistula.

- There have been cases of intracranial hypotension without the identification of a dural lesion in situations of dehydration, diabetic coma, uremia, or severe systemic illnesses.

There are two theories concerning the mechanism leading to the intracranial hypotension symptomatology:

- the decrease in the volume of cerebrospinal fluid leads to movements of the endocranial structures with the traction of the formations that contain pain receptors: meninx, draining veins to sinuses, trigeminus, glossopharyngeal and vague nerves, as well as the first cervical spinal nerves. Orthostatism increases the traction of these structures, emphasizing the symptomatology.

- the decrease in the CSF volume leads to the intracranial pressure decrease and, therefore, according to the Monro–Kellie theory, a dilatation of the intracranial vascular structures is produced, which generated the accentuated cephalgia in orthostatism. Moreover, there is a dilatation of the spinal epidural veins, explaining the spinal radicular pains.

It is currently considered that these two mechanisms are concomitant, they act together and they justify the symptomatology.

Clinical presentation

The main symptom of the intracranial hypotension is the orthostatic cephalgia: a cephalgia that occurs or is emphasized in orthostatism. The characteristics of the cephalgia vary from one patient to another, but the common element is the fact that it

is exacerbated in the orthostatic position, at a coughing effort, when laughing, etc., and it is not influenced by analgics.

Vertigo is the next symptom from the frequency point of view, vertigos that occur or are accentuated in orthostatism too. There may also be feelings of nausea, vomiting, tingles, facial paresthesias, and radicular pains at the level of the superior limbs, all of them being emphasized in orthostatism.

The establishment of how severe the symptomatology is and of the etiology makes the distinction between the clinical forms: acute, sub-acute and chronic.

In the chronic form of intracranial hypotension, symptoms are accentuated in orthostatism, and they diminish or disappear in clinostatism. Sometimes, symptoms have a reduced intensity with the presence of an accentuated feeling of asthenia. Based on the dominating symptom, there are forms which are mainly cephalalgic, vertiginous, paresthetic, with vegetative disorders, etc.

In the sub-acute form, symptoms are manifested in the horizontal position, and they are exacerbated in orthostatism with an invalidating effect.

The acute form is characterized by an accentuated symptomatology, which is present in clinostatism, with vomiting, to dehydration, photophobia, sometimes even convulsive crises. Moreover, there may be psychic disorders or even modifications of the conscience state. The acute form is very rarely registered nowadays.

Explorations

The intracranial hypotension diagnosis implies the corroboration of the clinical data with the anamnesis, as well as the performance of certain explorations that

may exclude other pathology and confirm the intracranial pressure decrease. In the context of certain maneuvers that include the lumbar puncture (myelography, myelo-CT, rachianesthesia), the occurrence of a clinical syndrome that announces the intracranial hypotension does not require the performance of other investigations in the case of a patient who has already undergone a cranial-cerebral exploration.

In the case of cranial-cerebral or spinal traumatism, the investigations performed in order to establish the traumatic lesions underline the dural lesion.

The occurrence of the symptomatology at temporal distance from a particular situation, which suggests the existence of a persistent dural fistula, requires the performance of certain explorations that may establish the diagnosis. It is considered that the testing of a patient with chronic cephalgia by arranging him or her in a Trendelenburg position, followed by the improvement of the cephalgia, suggests a possible occult CSF fistula and it requires supplementary explorations.

The intracranial pressure measurement establishes the diagnosis, but it must be performed after the cranial-cerebral explorations that exclude another pathology. The cerebrospinal fluid has a quasi-normal composition; sometimes, there may be an increase in the cerebrospinal fluid proteins, lymphocytosis; erythrocytes may be present, with a xanthochromic aspect.

The cranial-cerebral computer tomography may prove the erasure of the basal cisterns without the existence of a cerebral lesion.

The cerebral nuclear magnetic resonance with a contrast substance may reveal:

- dura mater thickening,

- venous sinus dilatation,
- presence of certain subdural fluid collections,
- increase in the volume of the hypophysis gland,
- the movement of the brain downwards: the basal cisterns become smaller, there is an inferior motion of the optic chiasm, and sometimes the cerebellar amygdales descend. (Figure 3)

The presence and the intensity of these signs are in direct relation to the clinical gravity, and the improvement of the symptomatology is accompanied by the attenuation and erasure of the MNR modifications.

The radioactive isotope cisternography allows the outlining of the CSF fistulas:

- directly, by noticing the radio-isotopic accumulation outside the sub-arachnoid space, or

- indirectly, by the lack of outlining regarding the radio-isotope at the level of the cerebral convexity and the notification of the latter in urine or in the adipose tissue.

The computer myelography (myelo-CT) allows the outlining of the CSF spinal fistulas right at the level of the cranium base.

Treatment

The intracranial hypotension treatment is:

- etiologic, and it consists in closing the CSF fistula,
- pathogenic, which consists in recreating the normal CSF volume, and
- symptomatic, which refers to the treatment of the symptoms related to intracranial hypotension.

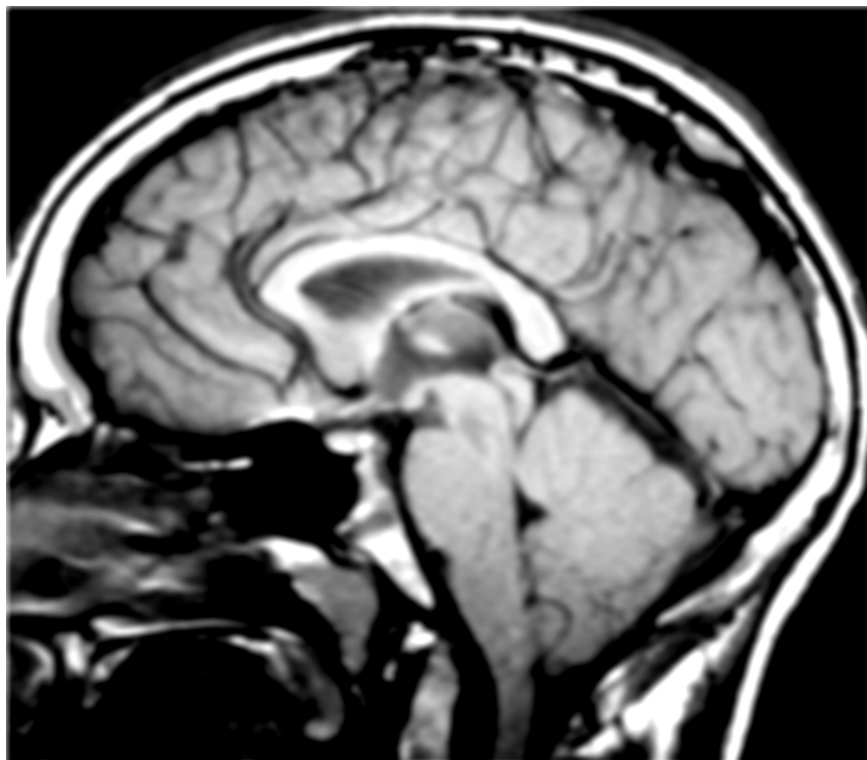


Figure 3 A T1-weighted sagittal MRI with general descent of the brain and sagging of the cerebellar tonsils through the foramen magnum and expansion of the sellar contents

As the intracranial hypotension syndrome frequently occurs after the lumbar puncture, the pathogenic and symptomatic treatment is often in the foreground, waiting for the dural orifice produced by the lumbar puncture to become obstructed.

The symptomatic treatment of cephalgia consists in avoiding the orthostatism, having the patient resting in bed, and by the oral or intravenous administration of caffeine or theophylline. Xanthines (theophylline) also increase the vascular resistance in the cerebral territory, reducing the circulation and diminishing the venous dilatation. Bed rest decreases the CSF pressure in the spinal dural sac and at the level of the remaining lumbar puncture orifice.

The CSF volume is recovered by oral and intravenous hydration, as well as by the administration of mineral corticoids.

Based on the registered evolution, one may appreciate whether the dural orifice of the lumbar puncture is maintained and if the CSF loss persists. In this case, a "patch" applying operation is performed at the level of the dural puncture orifice. The epidural blood patch is achieved by an autologous blood injection in the epidural space at the level of the previously performed lumbar puncture, with very good results; this operation needs to be repeated only very rarely.

The surgical solution of the CSF fistula is applied from the very beginning in traumatic dural lesions (cranial-cerebral wound, vertebral-medullar wound, cranium base fracture with CSF fistula, etc.), or the surgery may be temporized based on the intensity of the CSF loss and on the fistula location, with therapeutic

protection, supervising its spontaneous closure (in the CSF fistula from the cranium base fracture). Therefore postoperative CSF leaks after brain surgery are initially managed conservatively with bed rest, stool softeners and a lumbar drain and persistence of the leak beyond 1 week indicates need for surgical repair. Patients with postoperative CSF leaks after endoscopic approaches are taken back to surgical repair immediately.

There may also be surgical solutions for the spinal dural lesions remaining after the lumbar puncture only if none of the previously presented procedures has proven to be efficient.

Conclusions

1. The intracranial hypotension diagnosis implies the corroboration of the clinical data with the anamnesis, as well as the performance of certain explorations that may exclude other pathology and confirm the intracranial pressure decrease.

2. The intracranial pressure measurement establishes the diagnosis, but it must be performed after the cranial-cerebral explorations that exclude another pathology.

3. The treatment of intracranial hypotension is etiologic, and consists in closing the CSF fistula; pathogenic, which consists in recreating the normal CSF volume and symptomatic, which refers to the treatment of the symptoms related to intracranial hypotension.

The surgery for CSF fistula is applied from the very beginning in traumatic dural lesions or the surgery may be temporized with therapeutic protection, supervising its spontaneous closure.

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