

PROGRESSIVE PARENCHYMATOUS INTRACRANIAL HYPERTENSION – REVIEW

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The parenchymatous intracranial hypertension is the intracranial pressure increase caused by the intracranial volume modifications due to an intrinsic parenchymatous lesion (expansive intra-parenchymatous lesion, brain edema, etc.) or an extrinsic lesion (tumor, traumatic, infectious extra-parenchymatous compression, etc.). Depending on the location and the development manner, intracranial expansive processes cause the progressive increase in the intracranial pressure and then the occurrence of the ICH syndrome by the development of a supplementary volume. Benign intracranial tumors have a slow volume increasing rate and the neurological syndrome is installed progressively, while the ICH syndrome may occur late. Malign tumors have a rapid development rate and the neurological syndrome occurs precociously. The clinical decompensation represents the aggravation of the symptomatology by the tumor extension or by exceeding the compensating capacities of the intracranial pressure increase.

Keywords: brain gliomas, brain metastases, chronic subdural hematoma, intracranial hypertension, intracranial pressure, decompensation of intracranial hypertension

INTRODUCTION

The parenchymatous intracranial hypertension is the intracranial pressure increase caused by the intracranial volume modifications due to an intrinsic parenchymatous lesion (expansive intra-parenchymatous lesion, brain edema, etc.) or an extrinsic lesion (tumor, traumatic, infectious extra-parenchymatous compression, etc.). The primary cerebral lesion and the secondary endocranial volume changes (expansive or compressive new volume, hypoxic or traumatic brain edema, etc.) cause disorders of the intracranial pressure equilibrium mechanisms. The infraclinical stage includes the compensating mechanisms of the intracranial pressure increase and, if these mechanisms are exceeded by the pressure increase, the characteristic clinical and paraclinical

charts of the intracranial hypertension syndrome are shown. [6, 20, 21]

The parenchymatous intracranial hypertension develops depending on the existing etiology, on how rapidly the parenchymatous lesion extends, on the compensating mechanisms of the pressure increases, and on the efficiency of the medical and/or neurosurgical treatment.

The development of the parenchymatous intracranial hypertension includes:

- an incipient ICH syndrome,
- a compensated ICH syndrome, or
- a complete development till decompensation – the acute form with brainstem ischemia or with cerebral hernia.

The parenchymatous intracranial hypertension can occur in:

- the intracranial space-replacing lesions: cerebral tumors, hematomas, cerebral tumors, intracranial hematomas, cerebral abscesses, intracranial cystic lesions, etc.,

- the traumatic brain edema,
- the hypoxic brain edema through posttraumatic secondary cerebral ischemia or in sub-arachnoid hemorrhage,

- general intoxications with neurotoxins: endogenous or exogenous.

The parenchymatous intracranial hypertension varies in its starting manner, intensity of symptoms and development, which depend on:

- the nature of the lesion, which determines the development speed and the effect of the lesion on the neighbouring cerebral parenchyma (brain edema),

- the location of the lesion, which can involve the sector or integral cerebral parenchyma (encephalitis, generalized brain edema), depending on the involvement of the cerebral vessels or of the anatomic paths of circulation of the cerebrospinal fluid. The vascular compression causes ischemic disorders and the blockage of the cerebrospinal fluid circulation can lead to an obstructive hydrocephalus.

Depending on these characteristics, intracranial disorders may present a specific, focal symptomatology, (epileptic crises, motor and sensorial deficits, etc.) or general symptoms (asthenia, cephalgia, psychic disorders, endocrine disorders, etc.) or they may start as an intracranial hypertension syndrome. There are significant differences from the point of view of evolution, prognosis and therapy between intracranial disorders that start as an intracranial hypertension syndrome and those starting as a focal or non-characteristic symptomatology, and which subsequently present an ICH syndrome. [20, 21, 22]

A distinction is made between:

- the parenchymatous intracranial hypertension with an acute start, and

- the parenchymatous intracranial hypertension with an progressive start.

PATHOGENESIS OF PROGRESSIVE PARENCHYMATOUS INTRACRANIAL HYPERTENSION

Intracranial expansive processes with a progressive development can have the initial clinical manifestation of a neurological focus syndrome or by a non-specific symptomatology, to which the symptomatology of the intracranial hypertension is gradually added afterwards.

Depending on the location and the development manner, intracranial expansive processes cause the progressive increase in the intracranial pressure and then the occurrence of the ICH syndrome by the development of a supplementary volume. [18, 20, 21, 35, 38]

The new endocranial volume may be represented by:

- the lesion that replaces the intracranial space (endocranial tumor, intra-parenchymatous hematoma, subdural chronic hematoma, cerebral abscess, etc.), whose volume has increased progressively;

- the secondary brain edema of a primary lesion having a relatively reduced volume; extended brain edema with a compressive (mass) effect on the fluid spaces and on adjacent parenchymatous structures.

Benign intracranial tumors have a slow volume increasing rate and the neurological syndrome is installed progressively, while the ICH syndrome may occur late.

Malign tumors have a rapid development rate and the neurological syndrome occurs precociously.

The clinical decompensation represents the aggravation of the symptomatology by the tumor extension or by exceeding the compensating capacities of the intracranial pressure increase, and it manifests itself by:

- increase in the frequency of comitial crises,
- occurrence or accentuation of neurological deficits,

- occurrence of symptoms and installation of the ICH syndrome,

- occurrence of consciousness disorders: confusion syndrome, apathy, etc.

- installation of coma.

PROGRESSIVE PARENCHYMATOUS INTRACRANIAL HYPERTENSION

The occurrence of the intracranial hypertension syndrome depends on the characteristics of the endocranial lesions. These lesion characteristics refer to:

- the speed of the increase in the supplementary endocranial volume, which depends on the nature of the lesion (benign or malign tumor, intra-cerebral hematoma, subdural chronic hematoma, etc.):

- benign tumor lesions have a slow increase, with moderate cerebral suffering,

- the sudden occurrence of the intracranial hypertension may suggest a rapid expansion

- at the level of the tumor by producing an intra-tumor hemorrhage or the accelerated development of a tumor cyst

- the biological effect on the adjacent cerebral tissue (direct, compressive edematous effect, etc.):

- malign gliomas lead to the occurrence of the brain edema, they produce

- neoformation vessels, etc.

- cerebral metastases are accompanied by an important brain edema and the intracranial hypertension is frequently present.

- the location of the supplementary volume (direct compression on the vital structures, blockage of the cerebrospinal fluid circulation):

- subtentorial tumors evolve more frequently with ICH compared to supratentorial tumors, because the circulation of the cerebrospinal fluid may be blocked by the peritumoral edema and by the location of the tumor,

- tumors, even the malign ones localized at the frontal, occipital pole, can develop for long periods of time with no ICH syndrome.

The period that includes the changes in the intracranial pressure, reaching and then exceeding the normal pressure limit values is long, with a slow increasing speed of the intracranial pressure, and decompensation can take place rapidly or after a longer period of time. During the infraclinical period, there is a compensation of the pressure increases caused by the newly added volume, but the limit pressure is reached. Once the compensating capacities are exceeded, the intracranial pressure increase is rapid, and the duration of these pathological pressure values is short due to the rapid decompensation.

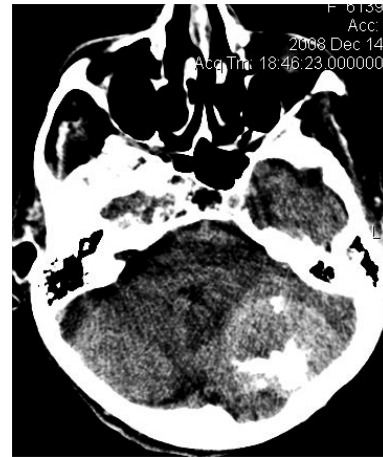


FIG. 1 Subtentorial tumor with blocked fourth ventricle

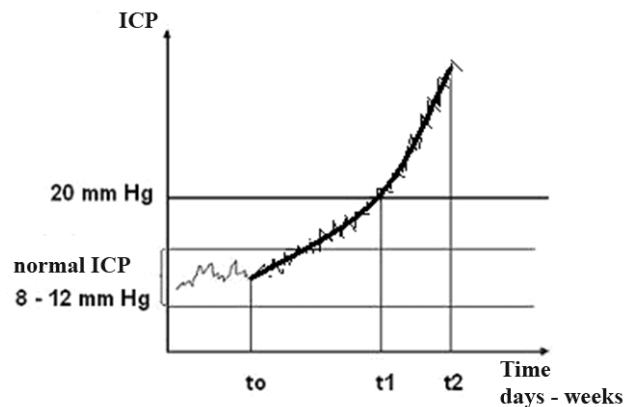


FIG. 2 Acute ICP increase, with a prolonged infraclinical period and a rapid decompensation; the interval to-t1 is prolonged, weeks or even months, and the decompensation may last for a few days

There is also the possibility of a slow increase in intracranial pressure: with a prolonged infraclinical period and a long period of pathologic ICP increase. The intracranial pressure increases slowly and progressively up to the normal limit value, and it continues its slow increase depending on the ICH etiology. The ability to compensate the pressure increase allows a prolonged maintenance of the cerebral sanguine flux, and there is a long period of the pathological pressure values. The ICH syndrome is prolonged and its aggravation is rapidly produced,

surpassing all the compensating methods of pressure increases.

ETIOLOGY OF PROGRESSIVE PARENCHYMATOUS INTRACRANIAL HYPERTENSION

1. Brain gliomas with a reduced degree of malignity

They are cerebral tumors with a slow increasing rate, most frequently they have a supratentorial location, especially frontal and temporal, or they may be developed in the brainstem, and they are more frequent in young adults.

The dominating symptoms are motor deficient, epileptic crises, cephalgia and psychic disorders.

The ICH syndrome with a papillary edema occurs more rarely because the location does not affect the circulation of the cerebrospinal fluid and because the dominating symptoms bring the patient to the physician before the increase in the tumor volume, which produces signs of intracranial hypertension.

2. Brain gliomas with an increased degree of malignity

They are malignant cerebral tumors, usually located deep into the white matter, but sometimes they may also have an intra-ventricular development or, from the deep cerebral hemispheric white matter, they may have a cortical extension.

The mechanisms that lead to the occurrence of the symptomatology are:

- local parenchymatous effects;
- compression on the neighbouring structures: nervous parenchyma, cerebral vessels or the circulation paths of the cerebrospinal fluid,
- the tumor invasion on the neighbouring structures: nervous parenchyma and/or cerebral vessels,
- local tissue destruction with the formation of the cellular degrading products,
- hypoxia of the neighbouring nervous tissue by the direct compression of the sanguine vessels too,
- metabolic and electrolytic disorders
- release of cytokines, of free radicals, etc.
- diffuse intracranial effects caused by:
- the increase in the intracranial pressure by
- the tumor volume

- the secondary effects of the volume increase on the cerebral parenchyma, on the blood circulation and on the dynamics of the cerebrospinal fluid.

The clinics is represented by:

- focal neurological syndromes:
- irritation syndrome: psychic disorders, epileptic crises
- syndrome in a deficit: sensitive or motor.
- syndrome of progressive intracranial hypertension:
- psychic disorders,
- cephalgia,
- vomiting,
- papillary edema, etc.

3. Brain metastases

Cerebral metastases are accompanied by an important brain edema, and intracranial hypertension is frequently present. The single cerebral metastasis may be focalized anywhere in the cerebral parenchyma and it practically has the characteristics of a malignant cerebral tumor; multiple metastases usually exceed the direct surgical therapeutic possibilities.

The symptomatology depends on the location of the lesion/lesions and on the occurrence of the intracranial hypertension through well-known mechanisms.

4. Intra-cerebral hematoma

In the primary intra-cerebral hematomas, the occurrence and development of the intracranial hypertension depends on preexistent factors (elements of hypertensive encephalopathy), on the volume of the hematoma and on the superficial or deep lumbar location, in the oval center, on the communication with the ventricular system, and perhaps on the circulating blockage of the cerebrospinal fluid.

5. Chronic subdural hematoma

The chronic subdural hematoma is a well-known neurosurgical entity; it occurs after a usually minor cranial traumatism, and it is characterized by the slow evolution of the "pre-clinical" period.

Usually, the focal neurological symptomatology is of a first ground interest, but, sometimes, the ICH syndrome may be marked, especially in the case of a cranial-cerebral traumatism, which subsequently produces clinical decompensation.

6. Benign intracranial tumors

PROGRESSIVE PARENCHYMATOUS INTRACRANIAL HYPERTENSION

Benign intracranial tumors are represented by meningiomas, neurinomas, hypophysial tumors, etc. The benign intracranial tumors present a slow increasing rate, and the symptomatology is represented by the focal neurological syndromes, and, usually at a delayed stage, by the intracranial hypertension syndrome. They most frequently cause the occurrence of the ICH syndrome by blocking the circulation of the cerebrospinal fluid, producing an intracranial hypertension by disorders of the cerebrospinal fluid dynamics. Secondly, the benign intracranial tumor induces a peritumoral brain edema, which brings about the occurrence of a parenchymatous intracranial hypertension with a progressive evolution.

7. Other endocranial lesions

Other types of expansive intracranial lesions – cerebral abscesses, dural empyemas, parasitoses (hydatid cyst), etc., present variable evolutions of the intracranial hypertension syndrome based on the same characteristics: location of the lesion, the expansion speed and the edematous character of lesions.

THE RELATIONSHIP BETWEEN LESION LOCATION AND ICH OCCURRENCE

In the case of a parenchymatous ICH with a gradual start, the relation between the location of the lesion and the occurrence of the intracranial hypertension syndrome seems less significant because the blocking mechanism of the cerebrospinal fluid circulation with the ICH occurrence by the compressive lesion of the ventricular system, belongs to the intracranial hypertension by disorders of the cerebrospinal fluid dynamics.

The multiple concomitant regional monitoring of the intracranial pressure has revealed the existence of significant pressure differences between the values of the sector intracranial pressure, especially in the case of temporal lesions, which may evolve towards decompensation by cerebral hernia before the ICP equalization in all the endocranial compartments.

Usually, the occurrence and rapid decompensation of the intracranial hypertension, which is explained by the location of the lesion, frames this type in the ICH by

blocking the circulation of the cerebrospinal fluid and by the occurrence of the acute obstructive hydrocephalus.

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