VASCULAR INTRACRANIAL HYPERTENSION PATHOGENESIS AND TREATMENT

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Intracranial hypertension can occur in cerebral-vascular illnesses due to sanguine, cerebral or extra-cerebral circulatory disorders, which modify the dynamics of the intracranial fluids and cause the intracranial pressure increase. The volume of the cerebral parenchyma increases due to the modifications occurred at the level of the cerebral sanguine capillaries, which leads to: the occurrence of the extracellular brain edema and cerebral congestive edematization with an increase in the volume of the cerebral parenchyma by vascular dilatation. The vascular types of intracranial hypertension have characteristic etiologies and they occur in thrombophlebites and cerebral venous thrombosis, in hypertensive encephalopathy and during the ischemic stroke.

Keywords: cerebral venous thrombosis, hypertensive encephalopathy, intracranial hypertension, ischemic stroke

INTRODUCTION

Intracranial hypertension can occur in cerebral-vascular illnesses due to sanguine, cerebral or extra-cerebral circulatory disorders, which modify the dynamics of the intracranial fluids and cause the intracranial pressure increase. There are disorders in the auto-regulation of the cerebral hemodynamics and the cerebral parenchyma volume continues to increase due to the brain edema or to the increase in the cerebral sanguine volume (brain swelling) with the secondary increase in the intracranial pressure. [4, 15, 26]

The volume of the cerebral parenchyma increases due to the modifications occurred at the level of the cerebral sanguine capillaries, which leads to:

- the occurrence of the extracellular brain edema due to the increased quantity of interstitial fluid:
- extracellular edema produced by a hydrostatic mechanism (ultra-filtration) in severe arterial hypertension,
- extracellular edema with oncotic induction (vasogenic edema) due to an increased permeability of the brain blood barrier (open brain-blood barrier)
- cerebral congestive edematization with an increase in the volume of the cerebral parenchyma by vascular dilatation.

The vascular types of intracranial hypertension have characteristic etiologies and they occur by:

- slowing down or decreasing the intracranial venous flux in thrombophlebites and cerebral venous thrombosis, the decrease in the venous flux at the level of the superior longitudinal sinus (SLS) directly in compressive lesions (hollowing fracture, etc.) or in SLS shunting by an intracranial arterial-venous malformation, or the extra-cranial illnesses that block the returning venous circulation at the cervical level, reduce the cerebral venous drainage and cause the decrease in the absorption of the cranial-spinal fluid and then the occurrence of the brain edema.

- in hypertensive encephalopathy, when the hydrostatic brain edema occurs (by ultra-filtration), as well as a brain swelling (by vasodilatation).

- the cerebral ischemia or the ischemic stroke reduces the arterial sanguine contribution and causes an ischemic brain edema, which is a mixed brain edema, both a cellular edema (cytotoxic) and an extracellular brain edema with oncotic induction (vasogenic). [26, 27, 46, 49]

CEREBRAL VENOUS THROMBOSIS

The cerebral venous drainage is slowed-down or even stopped in illnesses that influence the intracranial venous circulation or on extra-cranial conditions that may interest the great vessels at the level of the throat, usually by compression from vicinity. [5, 6, 7, 8, 26]

The cerebral venous circulation is reduced in:

- cerebral thrombophlebites and superficial venous thrombosis,
- thrombosis of dural sinuses,
- thrombosis of the profound venous system, and
- thrombosis of the cavernous sinus. [11, 12, 16]

The thrombotic venous occlusion is more frequent in the following etiological situations:

- infections (usually, the local infections are: otitis, sinusitis, etc., also in the case of meningitis). Mastoiditis may produce the thrombosis of the venous sinuses with a syndrome of secondary intracranial hypertension, an entity that Symonds describes as “otitic hydrocephalus”,
- tumor lesions at the level of the sinus, with its infiltration and obstruction (especially in meningiomas), when the tumor development is not responsible for the occurrence of ICH,
- traumatic brain injury,
- pregnancy and puerperium, etc.
- the calcifications of the sinus extended scythe have a reduced incidence (figure).

The frequency of the impacts on the dural sinuses and of other cerebral veins is as follows:

- the thrombosis of the sagittal sinus and of the lateral sinuses is happens in 75 – 85 % of cases
- the thrombosis of the superficial cortical veins occurs in approximately 10 – 15 % of cases,
- the thrombosis of the profound cerebral veins occurs in approximately 5 – 10 % of cases, and
- the thrombosis of the cavernous sinus occurs in less than 5 % of cases.

Cerebral venous thrombosis reduce the returning venous circulation from the brain and the skull, a venous stasis is produced and the cerebral sanguine circulation is slowed down. There are areas of cerebral hypo-anorexia concomitantly to areas of venous congestive edematization, and the cellular cerebral (cytotoxic) edema occurs, as well as the oncotic extracellular (vasogenic) edema, which evolves to a mixed brain edema. [20, 21, 22, 32, 33]

The venous sinuses also ensure the resorption of the cerebrospinal fluid, and the thrombosis of the venous sinuses leads to a diminished drainage of the cerebrospinal fluid. Therefore, a progressive intra-ventricular accumulation of cerebrospinal fluid occurs, with a pressure increase in the ventricular system and the occurrence of the hydrocephalic brain edema.

These phenomena happen slowly, in varied successions, but the evolution is progressive towards an intracranial pressure increase.

The iatrogenic thrombosis of the internal jugular veins is quoted in cases of prolonged use of the jugular catheters for the intravenous administration of medication. In such cases, the same pathogenic processes occur, and the ICH syndrome may appear.

The symptomatology is caused by the initial causal lesion, after which neurological focal symptoms may occur related to the progression of the venous thrombosis, as well as symptoms caused by the intracranial pressure increase. A venous infarct often happens, which is associated to a cerebral hemorrhage, which also aggravates the neurological clinical presentation.

Usually, the clinic evolves to an incomplete or complete syndrome of intracranial hypertension. [34, 37, 39, 41, 42, 52]

The main characteristics of the intracranial pressure increase in cerebral venous thrombosis are:

-a slow increase in the intracranial pressure up to the normal limit value of 20 mm Hg, usually during a period of a few days,

-over the value of 20 mm Hg, the ICP increase continues to be progressive, and it may reach maximum values of approximately 30 mm Hg in a few hours or days.

This gradual increase allows the compensating mechanisms to act more efficiently, and also for the applied therapy to encourage the intracranial pressure decrease and the improvement of the cerebral sanguine circulation.

- the maximum values that may be reached in cases of intracranial hypertension syndrome are of approximately 30- 35 mm Hg (sometimes the maximum values may be of 40 mm Hg) and

- the pathological pressure values may last for several weeks, with a slow return to normal pressure values and period of intermittent increases,

- usually, there is a recurrence to intracranial pressure values of about and above 20 mm Hg, which causes the persistency of a prolonged attenuated symptomatology.

The treatment of the venous thrombosis with an ICH syndrome is as follows:

- etiological and pathogenic for the vascular disorder, when possible,

- pathogenic for the intracranial hypertension syndrome.

A particular pathogenic mechanism is the reduction of the venous flux at the level of the superior longitudinal sinus (SLS) with an important blockage of the cerebrospinal fluid resorption:

- in the case of arterial-venous malformations with the excitation of the Galien vein (Galien vein aneurisms), when there is a SLS shunting by malformation and the cerebrospinal fluid resorption is diminished by the occurrence of the hydrocephalus. In infants and small children, the dominating symptomatology is the cardiac disorder due to the increased venous return, while the ICH syndrome also occurs in older children.

- in the case of a median intrusive cranial fracture, which interests the third posterior part of the SLS.

- at children with craniostenoses, there may be anomalies of the venous drainage, which interests the sigmoid sinus and the jugular vein, which may cause a venous hypertension, with a diminished drainage of the cerebrospinal fluid and the increase in the intracranial pressure. Usually, the phenomenon occurs up to the age of 6 years old, after which a collateral venous drainage is developed by the stylomastoid plexus, leading to the normalization of the intracranial pressure.

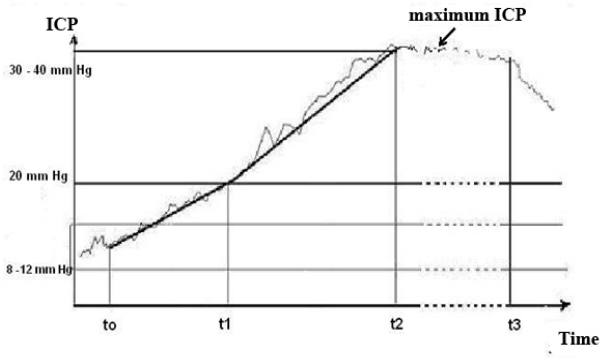


FIG. 1 Cranial pressure increase in cerebral venous thrombosis:

- to = start of the cerebral venous thrombosis

- t1 = the moment when the normal limit value of 20 mm Hg is reached

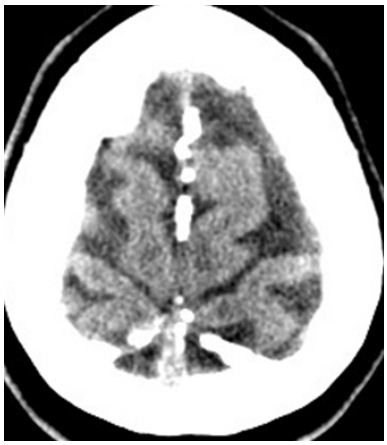
- t2 = after the progressive increase in the intracranial pressure, the maximum values of approximately 30 – 35 mm Hg are reached

- t3 = the moment when, after a varied period of increased pressure values, the ICP begins to decrease progressively, usually after treatment

Δ t1 = t1 – to: the period of intracranial pressure increase up to the normal limit value of 20 mm Hg, which usually lasts for a few days

Δ t2 = t2 – t1: the ICP increase period up to the maximum value, which may last for several hours to several days

Δ t3 = t3 – t2: the period of time of maximum intracranial pressure values, which usually last for several weeks.



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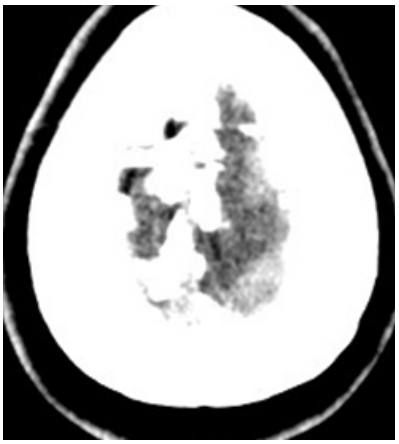

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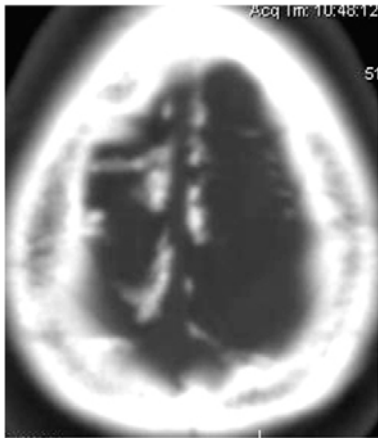

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FIG. 2 Calcifications at the level of the brain scythe (a), extended towards the superior longitudinal sinus (b), bilateral (c), with a slowing-down of the venous drainage, a decreased resorption of the cerebrospinal fluid and an ICH syndrome

HYPERTENSIVE ENCEPHALOPATHY

High blood pressureis the most important predisposing factor for cerebral-vascular illnesses, and the most frequent complication is the cerebral hemorrhage. The exaggerated increase in the values of the systemic blood pressure also causes disorders of the cerebral circulation auto-regulation, with other secondary cerebral suffering.

Hypertensive encephalopathy is defined in the clinical presentationof induced intracranial hypertension by an acute episode of arterial hypertension. [4, 15, 26]

1. The acute hypertensive encephalopathy is caused by the acute blood pressure increase in:

- severe high blood pressure,
- uncontrolled/untreated high blood pressurein pregnancy (eclampsia),
- high blood pressure in glomerulonephritis, pheochromocytoma, etc.

The acute increase in the sanguine pressure values leads to the inefficiency of the cerebral vascular auto-regulation, a generalized cerebral vascular dilatation occurs and/or there is an increased permeability of the cerebral capillaries. The increased permeability in the brain blood barrier has been constant more frequently at the level of the gray matter.

Therefore, the increase in the volume of the cerebral parenchyma is caused by:

- brain swelling by vasodilatation,
- hydrostatic extracellular brain edema, by ultra-filtration when the brain blood barrier is intact (close brain-blood barrier)
- oncotic (vasogenic) extracellular brain edema by an injury of the brain blood barrier (open brain-blood barrier).

The posterior reversible encephalopathy syndrome (PRES) or the reversible posterior leuco-encephalopathy syndrome (RPLS) with a hypertensive etiology is included in the acute form of vascular etiopathogeny ICH. The clinical presentationis typical and the DWI exploration shows an extracellular brain

edema by the increase in the water mobility with a posterior bilateral location, and a sub-cortical interest in the white matter too. The treatment consists of decreasing the systemic blood pressure. [26, 27, 31, 53]

2. The chronic hypertensive encephalopathy (Binswanger encephalopathy) is a rare cerebral-vascular illness with a chronic extracellular brain edema: hydrostatic brain edema combined with the oncotic brain edema.

The intracranial pressure increase in acute hypertensive encephalopathies are characterized by:

- the relatively high speed with which the intracranial pressure reaches the normal threshold value in approximately a few hours
- the ICP continues to increase above the normal values for a period that is usually shorter than the previous interval, of few hours only
- the maximum values that the ICP may reach are of 30 - 50 mm Hg and
- the period with pathologic intracranial pressure values is usually of several hours, rarely of several days. The anti-hypertensive treatment improves the clinical condition.
- The unmonitored hypertensive patients, or those who are incompletely treated may present repeated episodes of hypertensive encephalopathy, or they may suffer from the most frequent complication, which is the cerebral hemorrhage.

The clinical evolution of hypertensive encephalopathy is up to an incomplete syndrome of intracranial hypertension, and it has a regressive aspect. In the pathogeny of the syndrome, there is an auto-limiting mechanism: the intracranial pressure increase caused by the increase in the sanguine blood pressure and by occurrence of the cerebral vasodilatation generated the collapse of the walls of the intracranial sanguine vessels, and, to a certain extent, to a diminished cerebral sanguine volume. The mechanism consists of the direct action of the increased intracranial pressure over a functional disorder that is secondary to the exceeded auto-regulation of the cerebral circulation, and it has a limited value.

The treatment of the hypertensive encephalopathy is both etiologic and pathogenic:

- the treatment of the hypertensive episode, as an etiologic aspect, and
- the pathogenic treatment of the intracranial hypertension syndrome.

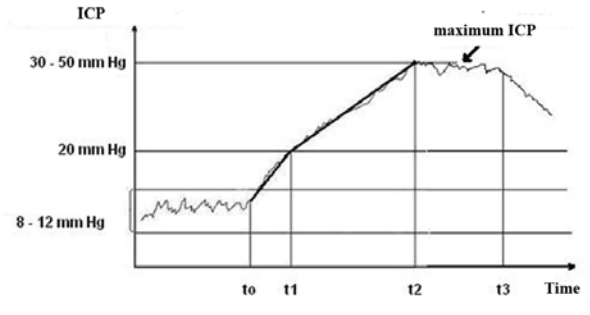

FIG. 3 Intracranial pressure increase in hypertensive encephalopathy:

to = beginning of the increase in the sanguine blood pressure

t1 = the moment when the normal ICP limit value is reached

t2 = the moment when the ICP has the maximum value

t3 = beginning of the decrease in the intracranial pressure values

Δ t1 = t1 – to: period of ICP increase up to the normal limit; it lasts for several hours

Δ t2 = t2 – t1: the ICP increase period to the maximum values, lasting for several hours

Δ t3 = t3 – t2: the period when the pathologic ICP values are maintained, usually of a few hours; it is rarely prolonged to a few days; the causal treatment of the high blood pressureis efficient.

ISCHAEMIC STROKE

The cerebral ischemia is caused by the cerebral circulatory insufficiency, which may be chronic or acute. [1, 2, 3, 9, 11]

The acute cerebral circulatory insufficiency or the ischemic stroke can manifest itself as:

- transitory ischemic stroke,
- progressive ischemic stroke,
- regressive ischemic stroke,

- complete ischemic stroke or the cerebral infarct. The cerebral infarct can be of a thrombotic or embolic nature.

The ischemic stroke represents 85 % of the cerebral-vascular illnesses. The large ischemic cerebral lesions are accompanied by the brain edema with cerebral herniation (sub-falciform) and by the intracranial pressure increase. [13, 14, 15, 17]

There are multiple causes of the ischemic cerebral vascular accidents:

- vascular illnesses: carotid atherosclerosis, infectious arteriopathies, posttraumatic occlusions of internal carotid arteries, of vertebral arteries or of middle cerebral arteries, arterial compressions at the cervical level, various vasculopathies,

- cerebral embolism of a cardiac nature – which represents approximately 60 - 70 % of the cerebral embolism cases: in the case of a mitral illness with atrial fibrillation, coronary thrombosis, paradoxical embolisms, infectious endocardites, etc.

- there may be rare cases of: hyper-coagulability, policitemia vera, etc.

The extended cerebral ischemic infarct with phenomena of intracranial hypertension is caused by the occlusion or stenosis of a great cerebral artery: the internal carotid artery or a terminal branch that irrigates a vast territory, such as the middle cerebral artery.

The extended ischemic infarct of the Sylvian artery occurs in approximately 10 % of the patients with acute cerebral circulatory insufficiency, and it has been designated as the malign infarct of the Sylvian artery due to the increased mortality, of up to 80 % of cases, despite the therapeutic means used. [1, 17, 18, 19, 24, 25]

The massive cerebellum ischemic infarct can cause the collapse of the 4th ventricle with the occurrence of an acute obstructive hydrocephalus and an acute ICH syndrome, and it has a direct compressive effect on the brainstem with the manifestation of vegetative disorders.

In the case of the cerebral hemispheric ischemic stroke, the decreased sanguine flow in the territory of the middle cerebral artery leads to the occurrence of certain ischemic metabolic disorders at the level of the affected cerebral parenchyma. The permeability of the cerebral capillaries increases (open brain-blood barrier) and the extracellular oncotic (vasogenic) edema occurs. The evolution is usually a rapid one, with the extension of the brain edema, the increase in the intracranial pressure and the occurrence of the sub-falciform cerebral hernia (median line movement towards the unaffected cerebral hemisphere). Although the intracranial pressure increasing mechanism is based on the cerebral ischemia with a hypoxic brain edema, which is characteristic for the parenchymatous lesions, while the etiology is represented by the impacts on a great cerebral artery, and it includes the ischemic stroke on vascular intracranial hypertension. [26, 28, 29, 30, 35]

Since the moment of the arterial occlusion, the intracranial pressure increase is:

- rapid until it reaches the normal pressure limit of 20 mm Hg, by the progression of the brain edema and the surpassing of the pressure compensating possibilities, with a duration of up to several hours

- above the normal pressure values, the ICP increase is also a rapid one, and the maximum values are reached within a short interval of time: half an hour – several hours

- the maximum values of the intracranial pressure are of approximately 40 – 50 mm Hg and

- the duration of these pathological values is of several days and it corresponds to the intensive care period.

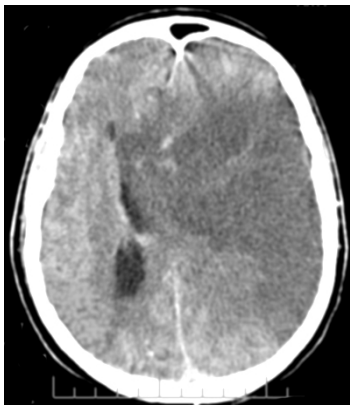

FIG. 4 Malign infarct of left Sylvian artery with important ischemic brain edema and sub-falciform cerebral herniation (by cardiac rhythm disorder)

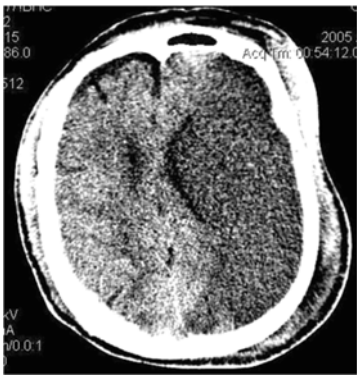

FIG. 5 Complete infarct of left Sylvian artery (traumatic occlusion at the level of the throat, strangulation)

The evolution is rapid towards the decompensation of the intracranial hypertension with almost 80% unfavorable results despite the applied treatments.

There is an attempt in using the etiological treatment of the arterial obstruction and the pathogenic treatment for the ICH syndrome. During the first three hours from the beginning, there may be an intravenous administration of recombined tissue plasminogen activator (rtPA) in a dose of 0.9 mg/kg, maximum 90 mg; the administration of streptokinase or of other thrombolytic agents does not have the same efficiency as rtPA. The brain edema receives a pathogenic treatment with osmotic diuretics (mannitol), and hyperventilation if there is an imminent decompensation of intracranial hypertension and the production of a brain herniation etc. [1, 35, 36, 38, 40, 43, 47].

Sometimes, there is an attempt of a surgical intervention:

- decompressive craniectomy of posterior cerebral fosse and of evacuation of a cerebellum infarct with a compressive effect on the brainstem, perhaps with a ventricular drainage,

- decompressive craniectomy and the evacuation of a cerebral hemispheric massive infarct, which may diminish the intracranial hypertension, but the surviving patients is left with major neurological deficits.

A particular case of generalized cerebral ischemia is met in the post-resuscitation syndrome when the sanguine flux disorder includes the entire brain, with a complete ischemia throughout the stroke, followed by reperfusion disorders.[26, 48, 50, 51, 54, 55]

The consequence of this cerebral circulatory failure, primary – before and during cardiopulmonary resuscitation, and secondary ischemic damage, during reperfusion is the development of the mixed brain edema: both cytotoxic and vasogenic, concomitantly to the production of the glial-neuronal necrosis. The hyperemic reperfusion may exacerbate the brain edema.

The mixed brain edema accentuates the elevated intracranial pressure and it exacerbates the brain injury.

The treatment is complex and the results do not compensate the efforts.

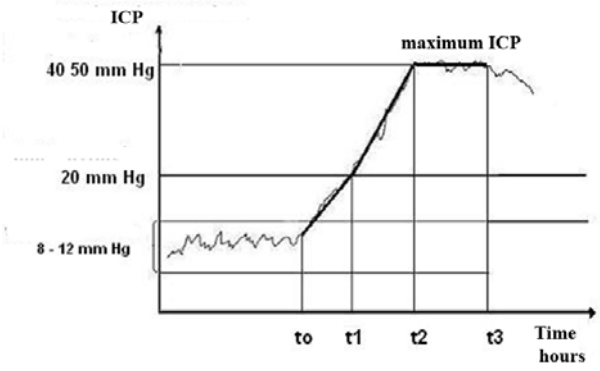

FIG. 6 Characteristics of the intracranial pressure increase in the ischemic stroke:

to = the moment when the arterial occlusion happens

t1 = the moment when the normal ICP limit value is reached

t2 = the moment when the ICP value is at maximum, of approximately 40 – 50 mm Hg

t3 = the moment when the ICP begins to decrease; the ICP value diminishing is rare in the Sylvian malign ischemic stroke

Δ t1 = t1 – to: the ICP increasing period up to the maximum normal value; it lasts for several hours

Δ t2 = t2 – t1: the period when ICP reaches the maximum values, lasting for a half an hour – one hour

Δ t3 = t3 – t2: the period when the pathological ICP values are maintained; it may last for several days.

The table below includes a comparative presentation of the three forms of vascular intracranial hypertension.

Table 1 Etio-pathogenic and evolutionary characteristics of the various forms of vascular ICH

Cerebral venous thrombosis	Hypertensive encephalopathy	Ischemic stroke
Cerebral vascular pathology: - thrombosis of dural sinuses - thrombosis of cortical veins	Cerebral vascular pathology: - dilatation of cerebral arteries	Cerebral vascular pathology: - infarct of Sylvian artery - massive cerebellum infarct
Cerebral blood flow : Reduced venous outflow	Cerebral blood flow : Increase arterial inflow	Cerebral blood flow : Reduced arterial inflow
Pathogenesis: - venous dilatation; open BBB and vasogenic brain edema and - diminished CSF drainage with hydrocephalic brain edema	Pathogenesis: - dilatation of cerebral vessels; closed BBB and hydrostatic brain edema, and - increased vascular permeability with open BBB and vasogenic brain edema	Pathogenesis: - ischemic increased capillary permeability; open BBB and vasogenic brain edema
ICP increase: - slow to the normal limit - slow above the normal limit	ICP increase: - rapid to the normal limit - slow above the normal limit	ICP increase: - rapid to the normal limit - rapid above the normal limit
Sub-acute and chronic evolution Possible decompensation	Acute and sub-acute evolution Rarely decompensation	Acute evolution Usually decompensation
Pathogenic treatment	Pathogenic and etiologic treatment	Etiologic and pathogenic treatment, Decompressive craniectomy

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