Emergency treatment principles in intracranial hypertension

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As general principles the treatment of intracranial hypertension depends on the type of intracranial hypertension and on the stage of the illness. First the treatment is an etiologic one in order to remove the cause that has caused the intracranial pressure increase: intracranial masses producing elevated ICP should be removed when possible. Simultaneously, there is an attempt to stop the pathogenic mechanisms that impact on the nervous structures, and a symptomatic treatment is applied in order to reduce the intensity of the clinical syndrome.

In the case of a patient with a suspicion of decompensating intracranial hypertension, one must perform and assess:

• the complete evaluation of the patient with the acute ICH syndrome: one must assess the consciousness level, the conscience state, the aspect of the pupils must be monitored and the Glasgow score must be established,

• the vital conditions: free aerial ways, respiration and the circulatory function; resuscitation operations are performed if needed, with oro-tracheal intubation, etc. In case of traumatisms, a cervical vertebral-medullar lesion must be excluded, and possible lesions with a vital risk must be revealed if they require an immediate solution.

• the emergency exploration by a cranial-cerebral computer tomography establishes the diagnosis and the therapeutic option is decided. (2, 8, 12, 14, 16, 30)

Thus the first therapeutic actions are determined depending on the lesional characteristics:

- the expansive intracranial process (tumor, traumatic process), which has induced the ICH syndrome is surgically extirpated in order to remove the supplementary pathogenic volume with a compressive effect (mass effect), and which leads to a brain edema.

- the expansive intracranial process that blocks the CSF circulation paths with the production of an obstructive hydrocephalus and the ICH syndrome requires at first the performance of a ventricular drainage and then, based on the characteristics of the lesion, there will be an intervention for its extirpation.

- in the blood blockage of the ventricular system in intracranial hemorrhages, with the occurrence of the ICH syndrome due to obstructive hydrocephalus, the ventricular drainage is performed, and then the exploration and the treatment of the intracranial hemorrhage.

- the presence of a brain edema without any other lesion requires the anti-edematous treatment in order to reduce the intracranial pressure, at the same time as maintaining the a cerebral perfusion pressure within normal limits by the

administration of mannitol, furosemide, dexamethasone, vasopressors, etc.

- in non-surgical multiple traumatic lesions, a pathogenic therapy is applied in order to decrease the intracranial pressure and stop the evolution of secondary traumatic lesions.

• an appropriate cerebral perfusion pressure must be maintained in order to prevent the cerebral ischemia. PCP must be higher than 60 – 80 mm Hg.

• avoidance of the situations when the cerebral metabolism increases:

- avoidance of stimuli: aspiration strictly when needed, patient's transportation, etc.

- hyperthermia must be vigorously discouraged: antithermic medication, muscular relaxing substances, cold packing up; the moderate hypothermia is an efficient procedure to decrease the cerebral metabolism and for cerebral protection.

• the evaluation of the biological parameters with the urgent control of the sanguine glycemia; in the case of the neurosurgical patient with a normal carbohydrate metabolism, it is not recommended to use glucose solutions as this may worsen the brain edema or the cerebral ischemic lesions

• the decrease in the arterial and intracranial venous sanguine volume by:

- head raising, which facilitates the venous drainage and increases the CSF removal

- the hyperventilation decreases PCO2 leading to an arterial vasoconstriction that decreases the cerebral sanguine volume

- AT monitoring, in order to hinder AT variations: correction of the arterial hypotension (decrease in the cerebral sanguine perfusion), as well as of the high blood pressure(increase in the intracranial pressure)

- pharmacologic methods to decrease the metabolism and the O2 needs with the reduction of the cerebral sanguine volume (thiopental 4-6 mg/kg or propofol 2-3 mg/kg), but with the cerebral perfusion pressure maintained within normal limits

• the signs of ICH decompensation – unilateral mydriasis – (the cerebral compliance has been exceeded) require urgent measures of ICP decrease and in order to prevent a further ICP increase:

- the occurrence of acute hydrocephalus (by any mechanism) imposes the CSF drainage with a ventricular catheter, which rapidly decreases the intracranial pressure,

- in order to prevent the cerebral herniation, a hemi-craniectomy can be used with a partial resection of the temporal lobe,

- in cases of malign brain edema, which is resistant to medicine therapies, a hemi-craniectomy can be performed with the removal of the cranial bone and the sectioning of the dura mater in order to allow the expansion of the cerebral parenchyma during the acute stage,

- in brainstem compressions, at the same time as the aggressive medication treatments, the decompression of the posterior cerebral fosse is performed by means of a sub-occipital craniectomy, completed by a ventricular drainage if there is also hydrocephalus.

• in the case of an operated patient, with a post-surgery ICP monitoring, the normalization of the intracranial pressure values is monitored, as well as the improvement of the cerebral circulation:

- if the cerebral perfusion pressure is below 70 mm Hg, the correction treatment of the intracranial sanguine contribution is started, even if the ICP is still high, by means of the administration of colloidal

solutions, intravenous vasopressors, etc.

- if the intracranial pressure has values of less than 20 mm Hg and the cerebral perfusion pressure is above 70 mm Hg, the patient is supervised, and the evolution of the ICP and PCP values are monitored.

- if the intracranial pressure is higher than 20 mm Hg, the agitated patient is sedated and the permeability of the respiratory paths is checked; the head is raised at 25 - 30 degrees from horizontal, which leads to a moderate ICP decrease through an increase in the venous drainage. The ICP normalization during these maneuvers does not require other therapeutic actions regarding the intracranial pressure.

• in the case of the patient who has not been in a surgery, but who has an ICP monitoring indication, one must survey and correct the intracranial pressure increases and the cerebral perfusion pressure decreases. The ICP values of more than 20 mm Hg require the agitated patient's sedation and the check of the respiratory path permeability. The decreased value of the cerebral perfusion pressure requires the specific therapy for the maintenance of the cerebral circulation within normal limits.

Treatment scheme in traumatic acute intracranial hypertension

The therapeutic stages for patients with traumatic brain injury and an acute ICH syndrome aim to reduce the ICP increases and to maintain the PCP within normal limits. The treatment is performed progressively, depending on the monitoring values of ICP, PCP and of the other parameters. (2, 7, 8, 12, 14)

• sedation and perhaps a moderate hyperventilation; this is applied as long as ICP < 20 mm Hg for the first 12 hours:

- sedatives

- PaCO2 is maintained at ≈ 35 mm Hg in the case of mechanically ventilated patients

- when the ICP value is above 20 mm Hg, one must verify whether the increased pressure values are caused by the agitated state or if there is a mechanical obstruction that may cause the pressure increase. The patient who manifests a psycho-motor agitation is sedated, and, if ICP decreases below 20 mm Hg, the monitoring, as well as the pathogenic and symptomatic therapy in the intensive care ward are continued.

• CSF drainage with the ventricular catheter: if ICP > 20 mm Hg for more than 5 minutes, CSF drainage is used whenever needed, as long as the operation proves to be efficient.

• use of diuretics: if ICP is maintained high above 20 mm Hg for more than 5 minutes, the following procedures are used:

- osmotic diuresis: mannitol administration.

- ansa diuretics: furosemide is used

The combination of these two diuretics is more efficient and is performed while monitoring of osmolarity and of the sanguine electrolytes.

• hyperventilation with the maintenance of PaCO2 at values of 25 – 30 mm Hg. Hyperventilation causes cerebral vasoconstriction and the ICP decrease by decreasing the cerebral sanguine flux. Hyperventilation is applied intermittently and it is recommended on the second day after a severe cranial-cerebral traumatism.

• hypertensive therapy for the increase in the systemic blood pressure, which can ensure a normal cerebral perfusion pressure if the intracranial pressure is increased. If cerebral contusion lesions are evident (areas of cerebral hemorrhage – hemorrhagic contusion), the systemic arterial pressure

can increase up to 150 – 170 mm Hg; if there are no areas of cerebral contusion, the blood pressure can increase to 180 mm Hg.

• controlled hypothermia, maintaining an approximate temperature of 35 ° C.

• administration of a hypertonic NaCl solution with a concentration of 7.5 %.

• surgical decompression: if the cerebral CT exploration shows that the cerebral edema is very important without revealing a compressive intracranial lesion, performing a large decompressive craniectomy, unilateral or bilateral, must be considered.

• the anesthetic administration is performed with an electro-encephalic monitoring up to doses that diminish the EEG activity, using:

- non-barbituric hypnotic substances: etomidate, propofol.

- barbituric substances: phenobarbital, thiopental (barbituric coma).

• Trometamol administration (THAM), which generates a partial decrease in CO2 pressure and produces a cerebral vasoconstriction. The condition is for the reactivity of the cerebral vessels to CO2 to be intact

• the lumbar puncture of CSF drainage is applied after the exhaustion of the other therapeutic means. The cerebral computer tomography exploration must reveal the presence of the basal cisterns and of the lateral ventricles. The risk of inducing a cerebral hernia by this operation, which is not recommended in intracranial hypertension, is considered to be smaller than the unfavorable evolution by the accentuation of the intracranial pressure increase. (12, 31)

Treatment scheme in intracranial hypertension of ischemic stroke

The emergency treatment of the massive ischemic cerebral or cerebellar stroke, which may lead to an ICH syndrome, includes:

• securing the vital conditions: free aerial ways, respiration and circulatory function, in the circumstances of a critical condition or of a coma,

• the intravenous administration of a recombined activator of tissue plasminogen (rTPA, rtPA) during the first three hours from the beginning of the stroke in a dose of 0.9 mg/kg; maximum 90 mg. (1, 13, 17, 18, 24)

The intravenous administration of streptokinase or of other thrombolytic agents does not have the same effects as the rtPA administration.

• if it is necessary, a progressive decrease in the systemic blood pressure is performed,

• administration of osmotic diuretics (mannitol) if there are any signs of intracranial hypertension decompensation,

• hyperventilation when intracranial hypertension decompensation and cerebra hernia occurrence are imminent,

• cortico-therapy is not recommended in the treatment of the cerebral edema in cerebral ischemic stroke

• surgical intervention if the ICH decompensation occurs:

- decompression and the evacuation of a cerebellar stroke with a compressive effect on the brainstem, perhaps a ventricular drainage too,

- decompression and the evacuation of a massive cerebral hemispheric stroke, which can reduce the intracranial hypertension, but the surviving patients are left with major neurologic deficits.

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