INTRACRANIAL HYPERTENSION MANAGEMENT

MANEJO DA HIPERTENSÃO INTRACRANIAL

David Gonçalves Nordon[1], Sandro Blasi Espósito[2]

RESUMO

Hipertensão intracraniana é uma situação comum e perigosa no serviço de emergência hospitalar. Todos os esforços devem ser direcionados para impedir ou reduzir as lesões secundárias. O ATLS na primeira avaliação é importante, junto com tomografia computadorizada do crânio, que é útil para identificar casos cirúrgicos ou lesões anatômicas, e monitoramento da pressão intracraniana. Manitol é a droga mais usada para diminuir a pressão intracraniana, mas deve ser usada com atenção ou pode causar um choque hipovolêmico. Coma barbitúrico e craniectomia descompressiva são reservados para os casos refratários como procedimentos de segunda linha.

Descritores: hipertensão intracraniana, pressão intracraniana, cuidados críticos.

ABSTRACT

Intracranial hypertension is a hazardous and common situation in the emergency room. Every effort must be made in order to avoid or reduce its secondary lesions. Advanced Trauma Life Support in the first evaluation is important, along with head computerized tomography, which is useful to identify surgical cases or anatomical lesions, and intracranial pressure monitoring. Mannitol is the mostly used drug to reduce intracranial pressure, but it must be used attentively, otherwise it may cause hypovolemic shock. Barbiturate coma and decompressive craniectomy are reserved to the refractory cases, as second tier procedures.

Key-words: intracranial hypertension, intracranial pressure, critical care.

BACKGROUND

The brain is an extremely complex and delicate system; everything in it must work smoothly and perfectly, for life to proceed. The skull is a non-extendable recipient, which contains brain tissue (80%), cerebrospinal fluid (CSF) (10%) and blood (10%). The cerebrospinal fluid is contained within the ventricles; the blood, within the blood vessels. Both of them work as first regulators of the intracranial pressure (ICP).

Younger people have more brain tissue than older people and are, therefore, more prone to develop intracranial hypertension. The brain tissue needs a minimum perfusion rate to work properly (60ml/100g/min), which is maintained accordingly to the body's arterial pressure. Therefore, the brain perfusion pressure (BPP) can be estimated by subtracting the IPC from the medium arterial pressure (MAP), and stands, usually, around 70mmHg, whereas normal IPC is expected to be around 10mmHg.

There are many different ways through which the skull contents may exert more pressure: the increase in the amount of CSF (due to hyper-production or obstruction of any draining point); blood increase (haemorrhage, blood vessels dilation), known as bloating; or tissue increase (tumors, for instance). Also, any inflammatory reaction, especially because of an ischemic and hypoxic injury (which may be primary or secondary) or a trauma, can cause an increase in the blood vessels permeability and liquid overflow (edema). In order to compensate the IPC raising, the brain uses several mechanisms: stops the production of CSF; expels as much CSF as it can; contracts as many blood vessels as it can, in order to prevent the inflow of more blood.

Nevertheless, this mechanism may not be enough; thus, the vasomotor center in the medulla oblongata produces a systemic blood vessels contraction in order to increase the MAP (and, therefore, the PPC). This contraction, however, generates an ischemia, which leads to blood vessels dilation and consequent increase in the ICP, causing a vicious cycle, that may be worsened by inadequate volume repletion.

This cycle must be broken by the physician even before it starts.

The intracranial hypertension follows a general development of four phases, as seen in the Langfitt curve (figure 1). In the first phase, the volume expelled is enough to compensate the ICP increase and it remains unchanged; in the second phase, the brain runs out of homeostatic mechanisms, so recurrent and transitory rises in the ICP appear in the continuous monitoring of the ICP as pathological pressure waves. In the third phase, minimal increases in the volume are proportional to big rises in the ICP; in the fourth phase, there is a complete vasomotor arrest, in whose apex the ICP equals the MAP.[1]

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1 - Acadêmico do curso de Medicina - PUC-SP

2 - Professor do Depto. de Medicina - PUC-SP

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Contato: d-nordon@uol.com.br

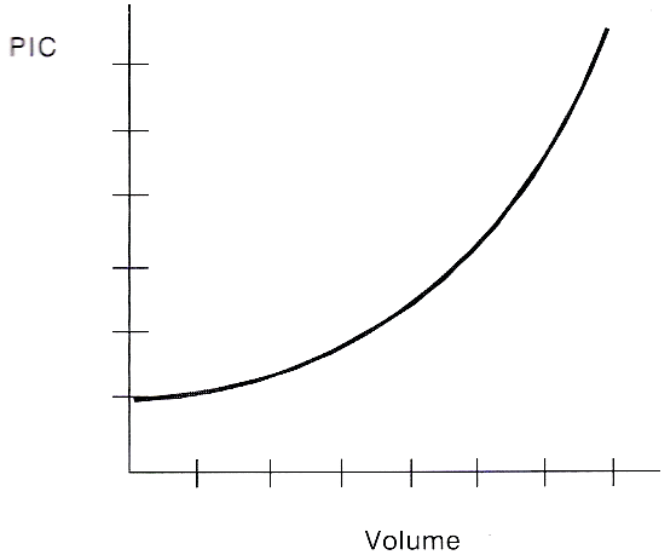

Figure 1. Langfitt Curve

CLINICAL PRESENTATION AND DIAGNOSIS

The classical presentation is a triad: headache, vomiting and papilar edema, which are not always present at the same time. The headache is persistent, from moderate to severe in intensity, and usually is worse in the morning. The vomiting is not preceded by nausea, and the papillary edema is secondary to the blood stasis of the veins that drain the orbits. Visual impairment tend to appear later, up to complete blindness. Abducens nerve paralysis may occur, along with oculomotor nerve lesion (and ipsilateral mydriasis), psychic blunting, behavioral changes and convulsion.[1,2,3]

During the unbalance phase, conscience level and papillary alteration appear, and the Cushing triad occur (hypertension (to balance the increase in the ICP), bradycardia (vasomotor center compression) and Cheyne-Stokes Respiration (respiratory center compression)). This triad is not commonly seen in younger children, whose skull is still not completely grown and is able to increase in order to contain the ICP raise. The brain stem lesion occurs when internal hernias start to develop, either through the Paccione's foramen or the magnum foramen. It is important to observe that the interhemispheric fissure may restrict the increased ICP to only one side of the brain, at least, in the beginning; sometimes, the bloated hemisphere starts to herniate, and the medium line suffers a deviation. This compresses and compromises a so far sound area of the brain. Bilateral mydriasis is serious and mostly shows an irreversible lesion.[2,4,5]

The most effective method of diagnosis is the ICP monitoring. Neurological exam alterations are sensitive but not specific; papilar edema, on the other hand, is specific but not sensitive. The computerized tomography (CT) is also specific, but not sensitive, and thus a normal CT shall not exclude the intracranial hypertension diagnosis. The ICP monitoring catheter can be made of optic fiber, polyethylene or silicon, and has greater advantages when inserted in the ventricles, what allows it to drain CSF. Normal ICP is lower than 10mmHg; ICP higher than 20mmHg for longer than 10 minutes in adults needs evaluation and, probably, treatment; head CT can be used to exclude surgical cases.[1]

MANAGEMENT

Many of the cases of intracranial hypertension will be secondary to trauma; thus, following the ATLS is the first basic procedure. These patients must be treated to avoid or reduce secondary lesion, due to hypoxemia, hypercapnia, hypocapnia, etc. The treatment algorithm is shown in figure 2.

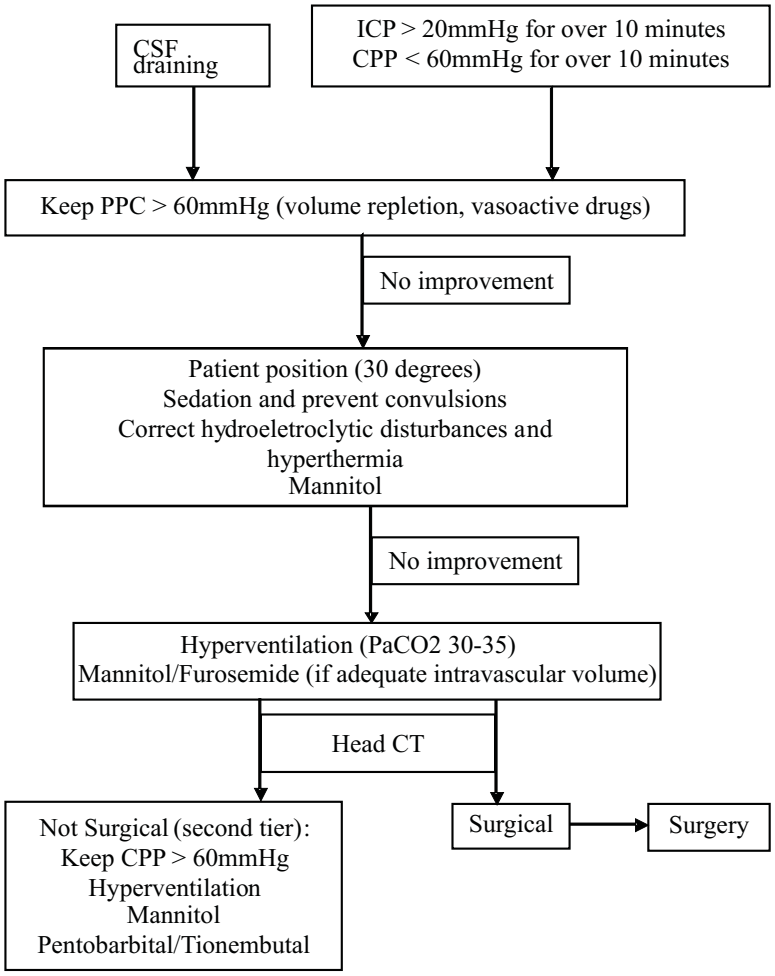

Figure 2. Treatment Algorithm

A. Orotracheal intubation: must be performed in every patient whose Glasgow Coma Scale (GSC) result is lower than nine.

B. Ventilation: mechanical ventilation must be used, but prophylactic hyperventilation (PaCO2 lower than 35mmHg) must be avoided in the first 24 hours of treatment. The oxygen saturation must be kept above 92% to avoid hypoxemia.

C. Hemodynamic stabilization: the blood pressure must be kept at normal range, otherwise the brain is in danger of suffering ischemia; firstly crystalloids are indicated; if necessary, vasoactive drugs can be used. Blood pressure must be attentively observed, especially if hyperosmolar or diuretic drugs are used.

ICP management:

1. The CPP must be kept above 60mmHg; the ICP, below 20mmHg. MAP must be invasively monitored and be used to guide the treatment and volume repletion (which is essential to keep the CPP).
2. Patients position: a 15 to 30 degrees inclination is interesting in hemodynamically stable patients, due to the increase in venous return. Unstable patients, however, may develop hypotension and subsequent brain blood vessels dilation and rise in the ICP.
3. Normothermia: hyperthermia worsens ischemic injuries. Hypothermia (32-34°C) may be useful to lower ICP, as it causes a vasoconstriction and subsequent lowering of brain blood flow.
4. Sedation: when the patient is still conscious, it is useful to avoid movements and actions that would increase the ICP.
5. CSF draining: effective, causes minimal side effects.
6. Hyperventilation: the hypocapnia causes a vasoconstriction and quickly lowers the blood flow in 4% each 1mmHg below normal. Hyperventilation, however, when long-lasting (over a day) loses its effects and, when below 25, causes lactate production and more edema. Therefore, it must be used for initial control of ICP or control of sudden raising.
7. Hyperosmolar and diuretic agents: mannitol (10-25% solution): 0,75-1g/kg, in fast administration. Depending on the patient's response, extra doses of 0,25-0,5g/kg each four to six hours follow the initial bolus. Blood osmolarity must be periodically measured (and must be kept around 300-320mOsm/L). Low doses of diuretic may be associated (furosemide, 20mg), especially when there is risk of complication due to cardiac insufficiency. Nowadays, studies are being made on 23,4% saline administration, which supposedly is eight times more effective than mannitol concerning intracranial hypertension control.[6]
8. Head CT: indicated if the intracranial hypertension cannot be controlled, in order to rule out other possible causes.

Second tier: unconventional maneuvers to refractory intracranial hypertension, includes:

1. Barbiturates: halves brain activity and neuronal energy consumption, thus, lowering the ICP. Nevertheless, barbiturate cause systemic vasodilatation, miocardic depression, arterial hypotension and pulmonary secretions accumulations. And, as the brain blood flow lowers, encephalic ischemia may develop. Pentobarbital: attack dose: 3-10mg/kg (30 infusion), followed by 5mg/kg/h for three hours. The maintenance dose shall be adjusted in order to prevent suppressions in the EEG.
2. Decompressive craniectomy: reserved for the surgical and the most refractory cases; its results being better as soon as it is performed,[4,7-9] and most survivors develop severe sequelae, especially those older than 45. Better results come out in younger patients; on the other hand, older patients usually present co-morbidities that may worsen the prognosis. Subdural collections, hydrocephaly, convulsions and infections are some of the complications that might occur.

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