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Vascular Intracranial Hypertension

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Intracranial hypertension is one of the most important syndromes in neurology and neurosurgery; raised intracranial pressure is the most common cause of death in neurosurgery. A recent classification of intracranial hypertension is based upon the etiopathogenesis of intracranial hypertension: a) parenchymatous intracranial hypertension, b) vascular intracranial hypertension, c) meningeal intracranial hypertension and d) idiopathic intracranial hypertension. Vascular etiologies can individualize vascular types of intracranial hypertension: (1) cerebral venous thrombosis reduces venous outflow and determines low cerebrospinal fluid drainage and brain edema; (2) hypertensive encephalopathies cause brain swelling, both brain edema and congestive brain swelling with raised Intracranial Pressure (ICP); (3) ischaemic stroke induces an increased capillary permeability with open brain-blood barrier, brain edema and severe ICP increase. The main features of ICP increase are: the speed of ICP increase up to and respectively above, the normal limit of ICP, the pathological value of ICP and the duration that the pathological values of ICP are maintained. These features of ICP increase depend on its etiology: a low speed of ICP increase in cerebral venous thrombosis or a high speed in hypertensive encephalopathies or in ischaemic strokes. Also the periods when ICP stays at high values are different depending on the etiologies of ICH: a long period in cerebral venous thrombosis and a short period in ischaemic strokes.

Key words: Brain edema, cerebral venous thrombosis, hypertensive encephalopathy, intracranial pressure, ischaemic stroke, vascular intracranial hypertension

INTRODUCTION

A proposed classification of intracranial hypertension (ICH)^[1,2] is based upon the etiopathogenesis of ICH:

- a. Parenchymatous ICH which has intrinsic brain causes. This form appears in expansive intracranial processes (tumours, haematomas, cerebral abscesses etc.), in traumatic brain edema, intoxications with neural toxins.
- b. Vascular ICH-this has the etiology in disorders of the blood circulation (brain or general vascular injury)
- c. Meningeal ICH caused by the decrease in the absorption of Cerebrospinal Fluid (CSF) in meningitis^[12], subarachnoid haemorrhage and
- d. Idiopathic ICH.

Brain swelling in vascular ICH is of two types:

- brain edema-this is an increase in the water content of the brain tissue and
- congestive brain swelling, in which the volume that increases is the intravascular volume.

Brain edema or/and congestive brain swelling cause the increase of Intracranial Pressure (ICP). The increase of the cerebral blood volume can be caused by an important in flow of blood or by a reduction or a stopping of cerebral blood outflow. There is also a reduction of the CSF absorption when the cerebral blood outflow lessens.

The causes of vascular ICH can be:

- A. Different vascular cerebral diseases: cerebral thrombophlebitis, cerebral venous thrombosis and superior sagittal sinus thrombosis, mastoiditis with transverse or sigmoid sinus thrombosis (the "otic hydrocephalus" described by Symmonds) and
- B. Different extracerebral diseases: hypertensive encephalopathies such as acute hypertensive encephalopathy, cases of malignant hypertension from any cause (glomerulonephritis, eclampsia or phaeochromocytoma) and chronic hypertensive encephalopathy (Binswanger's encephalopathy) or cerebral venous outflow reduction in congestive cardiac failure or intrathoracic mass lesions.
- C. The acute stroke which is a cerebrovascular disease with different mechanisms and two resultant effects on the brain: ischaemia (85%) or haemorrhage (15%). The origin of primary lesion in ischaemic stroke can be an intra- or extra cranial vascular disease (stenosis, embolization). Brain edema occurred after the increase of cerebral blood flow around infarction is an oncotic extracellular brain edema^[1] and thereby the increased ICP occurs.

Clinical acute symptomatology is due to elevated ICP in vascular ICH, but many symptoms are different depending on the etiology.

Etiologies of vascular intracranial hypertension

Cerebral venous thrombosis: Cerebral venous thrombosis comprises: superficial cortical veins thrombosis, dural sinus thrombosis, deep venous system thrombosis and cavernous sinus thrombosis^[3].

Thrombosis occlusion of the venous system occurs in: infection (usually local as: otitis, sinusitis etc. and also meningitis); head trauma; pregnancy and puerperium etc.

Frequencies of involvement of dural sinuses and other veins are: superior sagittal and lateral sinus thrombosis in 75-85% of cases, superficial cortical veins thrombosis in 10-15% of cases, deep cerebral venous thrombosis in 5-10% of cases and cavernous sinus thrombosis in less than 5% of cases.

Cerebral venous thrombosis reduces venous outflow from the brain and the venous engorgement causes brain edema. Also the venous sinuses are important in cerebrospinal fluid absorption and sinus thrombosis causes a diminution of the CSF drainage^[4].

An increased vascular permeability or open blood-brain barrier appears because of the venous engorgement and therefore a vasogenic brain edema occurs. The diminishing of CSF drainage determines an increase in the pressure of intraventricular CSF and then a hydrocephalic extracellular brain edema occurs^[5,6].

These processes may all elevate intracranial pressure and the clinical symptomatology is due to increased ICP.

Hypertensive encephalopathy: Arterial hypertension is the most important factor predisposing to cerebro-vascular diseases. A group of encephalopathies may be related to disordered vascular autoregulation caused by excessive blood pressure^[7,8].

1. Acute hypertensive encephalopathy due to acute blood pressure elevations in malignant hypertension, or uncontrolled hypertension in pregnancy, glomerulonephritis or phaeochromocytoma. The acute blood pressure increase produces a breakthrough of cerebral vascular autoregulation with forced vascular dilatation and/or increased vascular permeability. Therefore brain swelling occurs: either brain edema or congestive brain swelling. Hydrostatic extracellular brain edema (mechanism of ultrafiltration) is caused by pronounced increases of intravascular brain pressure in severe arterial hypertension^[1,7,8]. This type of brain edema occurs when the brain-blood barrier is intact. There is an open blood-brain barrier in cases of congestive brain swelling and a vasogenic brain edema occurs.

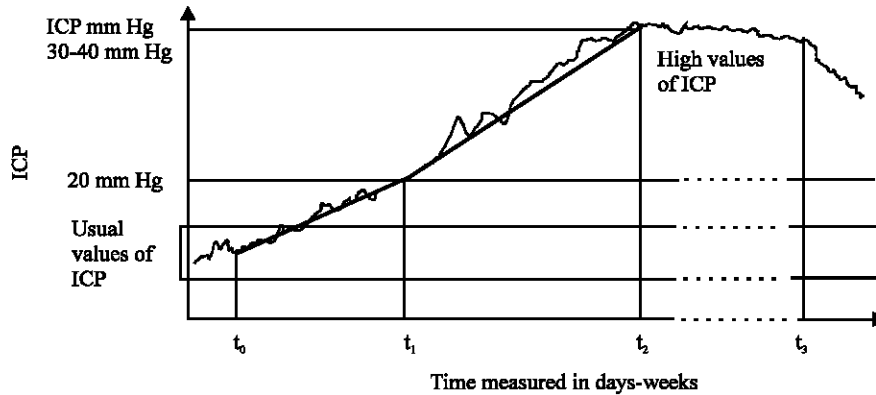


Fig. 1: ICP increase in cerebral venous thrombosis

ICP is measured in mm Hg; Time is measured in days to weeks;

t_0 = the start of cerebral venous thrombosis

t_1 = the moment of reaching the limit of normal value of ICP

t_2 = the moment of reaching the maximum value of ICP usually 30-40 mm Hg

t_3 = the moment when the high ICP starts to decrease

$\Delta t_1 = t_1 - t_0$: the period of increase of ICP up to the normal limit; it lasts for several days

$\Delta t_2 = t_2 - t_1$: the period of increase of ICP up to the maximum value; it may last several hours up to several days

$\Delta t_3 = t_3 - t_2$: the period when ICP stays at high values; it usually lasts several weeks

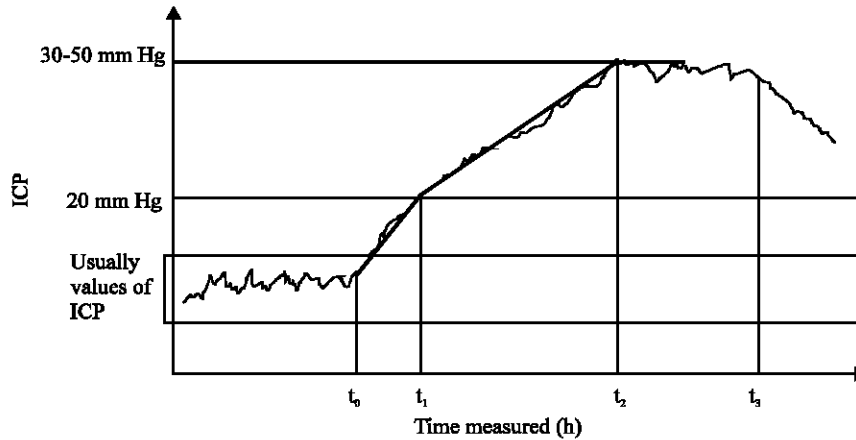


Fig. 2: ICP increase in hypertensive encephalopathies

ICP is measured in mm Hg; Time is measured in hours; t_0 = the start of acute blood pressure elevation, causing brain swelling and an increase in ICP

t_1 = the moment of reaching the limit of normal value of ICP

t_2 = the moment of reaching the maximum value of ICP usually 30-50 mm Hg

t_3 = the moment when the high ICP starts to decrease

$\Delta t_1 = t_1 - t_0$: the period of increase of ICP up to the normal limit; it lasts for several hours

$\Delta t_2 = t_2 - t_1$: the period of increase of ICP up to the maximum value; it may last for a few hours

$\Delta t_3 = t_3 - t_2$: the period when ICP stays at high values; it may last several hours, seldom a few days.

The treatment for decreasing high blood pressure improves the clinical state quickly

- Chronic hypertensive encephalopathy (Binswanger's encephalopathy) is a rare cerebrovascular disease with chronic brain edema as an extracellular brain edema: hydrostatic brain edema combined with oncotic brain edema.

Ischaemic cerebrovascular disease: Ischaemic stroke comprises 85% of all cases of cerebrovascular diseases. The large lesions accompanied by brain edema and brain shifts are associated with intracranial pressure (ICP) increase.

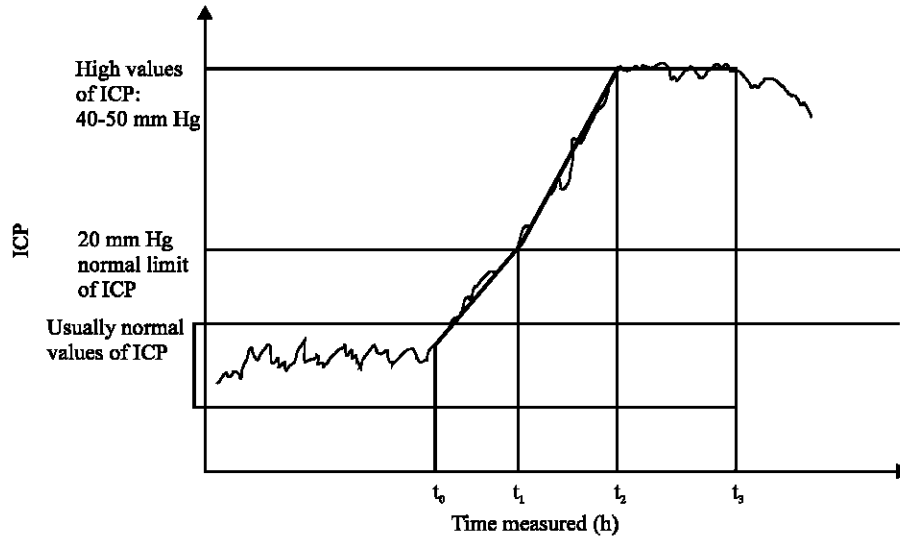


Fig. 3: ICP increase in ischaemic strokes

ICP is measured in mm Hg; Time is measured in minutes; t_0 = the start of cerebral vessel occlusion (eg-occlusion of middle cerebral artery)

t_1 = the moment of reaching the limit of normal value of ICP

t_2 = the moment of reaching the maximum value of ICP usually 40-50 mm Hg

t_3 = the moment when the high ICP starts to decrease; the decrease of high ICP seldom occurs in malignant middle artery territory infarction

$\Delta t_1 = t_1 - t_0$: the period of increase of ICP up to the normal limit; it lasts from half an hour up to a couple of hours (usually a few hours)

$\Delta t_2 = t_2 - t_1$: the period of increase of ICP up to the maximum value; it lasts from several minutes up to half an hour to an hour

$\Delta t_3 = t_3 - t_2$: the period when ICP stays at high values; the duration varies from several hours to a few days; in malignant middle artery territory infarction Δt_3 is the period of clinical deterioration leading to death in 80% of cases

The causes of ischaemic strokes with increased ICP are large cerebral vessel occlusion or stenosis: the internal carotid artery or the middle cerebral artery^[9,10].

The large ischaemic stroke of the middle cerebral artery occurs in up to 10% of the stroke patients and is known as malignant middle artery territory infarction, because of the high mortality (80% of cases). In the ischaemic hemisphere there is a metabolic ischaemic cascade produced by the fall in cerebral blood flow. The capillary permeability increases (open blood-brain barrier) and the extracellular brain edema (or vasogenic brain edema) occurs^[1]. There is a severe post ischaemic brain edema with an increased ICP and the brain shift can also occur (Fig. 5).

Characteristics of increased ICP in vascular ICH: There are differences in increased ICP in cases of intracranial hypertension caused by the cerebral venous thrombosis, hypertensive encephalopathy or ischaemic stroke depending on the etiology^[2,11].

The main features of ICP increase are:

- The speed of the ICP increase up to the normal value of ICP, of 20 mm Hg,
- The speed of ICP increase above the normal limit value,
- The highest value of pathological ICP,
- The duration that the pathological values of ICP are maintained,
- The recurrence of the pathological ICP values.

Depending on the above features there are three main patterns for the ICP increase in vascular ICH (Fig. 1-3):

- a. A low speed of ICP increase up to the normal limit value of ICP (20 mm Hg) and then a low speed of ICP increase over 20 mm Hg in cerebral venous thrombosis (Fig. 1). The period of increase of ICP usually lasts several days and the period when ICP stays at high values lasts several weeks.

Table 1: Etiological characteristics of vascular intracranial hypertension

Cerebral venous thrombosis	Hypertensive encephalopathy	Ischaemic stroke
Involved cerebral vessels: -dural sinus thrombosis -cortical veins thrombosis	Involved cerebral vessels: -cerebral arteries dilatation	Involved cerebral vessels: -middle cerebral artery infarction
Cerebral blood flow: Reduced venous outflow	Cerebral blood flow: increased arterial inflow	Cerebral blood flow: reduced arterial inflow
Pathogenesis: -venous engorgement, open BBB and vasogenic brain edema and -low CSF drainage with hydrocephalic edema	Pathogenesis: -vascular dilatation; closed BBB and hydrostatic brain edema and -increased vascular permeability with open BBB and vasogenic edema	Pathogenesis: -ischaemic increased capillary permeability, open BBB and vasogenic brain edema
ICP increase: -low speed up to normal limit -low speed over normal limit	ICP increase: -high speed up to normal limit -low speed over normal limit	ICP increase: -high speed up to normal limit -high speed over normal limit
Chronic evolution, Not usually decompensation	Acute and subacute evolution Rarely decompensation Usually decompensation	Acute evolution
Pathogenic treatment	Etiologic and pathogenic treatment	Pathogenic treatment, also hemicraniectomy

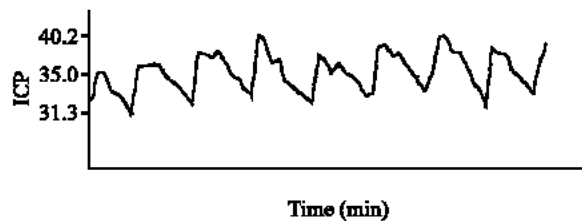


Fig. 4: Monitoring of ICP (screen capture of ICP monitoring) with high values of ICP and pathological types of waves.



Fig. 5: Malignant left middle artery territory infarction with severe ischaemic brain edema and the brain shifts to right

- c. A high speed of ICP increase up to the normal limit and also a high speed of ICP increase over 20 mm in ischaemic stroke (Fig. 3). The period of increase of ICP up to the normal limit lasts from half an hour up to a couple of hours and the increasing of ICP up to the maximum value lasts from several minutes up to half an hour to an hour.

Figure 4 shows the monitoring of ICP with a pathological types of ICP waves.

A vascular type of intracranial hypertension can occur because of different vascular etiologies. The main vascular etiologies are: cerebral venous thrombosis, hypertensive encephalopathy and ischaemic stroke. Each etiology determines a characteristic pathogenesis and a specific type of ICP increase:

- Cerebral venous thrombosis reduces venous outflow and causes brain edema and low CSF drainage;
- Hypertensive encephalopathy determines brain swelling: both brain edema and congestive brain swelling;
- Ischaemic stroke causes an ischaemic open brain-blood barrier and vasogenic brain edema occurs. In malignant middle cerebral artery territory infarction there is a severe ICP increase and brain herniation can occur.

- b. A high speed of ICP increase up to the normal limit and a lower speed of ICP increase over the normal limit of ICP, in hypertensive encephalopathy (Fig. 2). The period of increase of ICP lasts for a few hours and the period when ICP stays at high values may last several hours, seldom a few days. The treatment for reducing the high blood pressure improves the clinical state quickly.

Etiological characteristics of vascular intracranial hypertension are presented in Table 1. Treatment is usually pathogenetical; etiological treatments can be applied in hypertensive encephalopathies; a hemicraniectomy may reduce mortality in cases of acute and complete internal carotid artery or middle cerebral artery territory infarction.

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