Chapter-14

RAISED INTRA CRANIAL PRESSURE, EFFECTS ON BRAIN AND MANAGEMENT

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ABSTRACT

Normal intracranial pressure is exerted by the cerebrospinal fluid on the cerebral tissue which produces a pressure in the cranium. This is maintained in the range of 7-15 mm Hg. The cranium is formed as a protecting framework to the brain tissues through eight bones 2 temporal, 2 parietal, frontal, occipital, sphenoid, and ethmoid bones. The neurocranium contains three components the brain Tissues 80%, Blood 10 % and CSF 10%. These are responsible for ICP, if any one of these increases in volume, then other of one or two compresses and adjusts a limited time to avoid increasing ICP but after some instant, brain is exhausted and increase ICP, So that one increased →Volume increased → ICP increased → Brain shrank → Brain Injury. There are elastance and compliance mechanism which stable the ICP in some instant, because the brain 100–150 ml CSF additionally absorb without changes in the ICP. In adult, normally CSF volume is 150 ml, the 125ml within subarachnoid spaces and 25ml in the ventricles. Whenever CSF volume increases then the compensatory mechanism is started so, low pressure of vein is collapsed and CSF egress into the lumbar subarachnoid space, when the compensatory mechanism is exhausted then increased of additional volume cause sudden ICP rise. The cerebrospinal fluid is circulating through the neuroaxis from the site of secretion to the site of absorption due to rhythmic systolic pulse wave in choroid arteries. The CSF circulation is depending on frequency of respiration, jugular venous pressure, postures and the individual physical activity. The mechanism of compliance is changing the ICV and ICP. It means, the ICP is elevated, then ICV becomes low, when ICP is normal, the ICV is become high. So, small changes in the ICV that changes in the ICP.

Keywords: *ICP-Intracranial pressure, CBF-Cerebral blood flow, CPP-Cerebral perfusion pressure, MAP- Mean arterial pressure, and PP- Pulse pressure.*

1. INTRODUCTION

Monroe-Kellie Doctrine theory is a fundamental to understanding of the negative effects of the increased intracranial pressure of the brain. The Monro-Kellie doctrine or hypothesis states that the sum of volumes of brain, cerebrospinal fluid (CSF) and Intracerebral blood are constant. An increase in one should cause a reciprocal decrease in either one or both of the remaining two. (Mokri, B, 2013).

So, we tell about ICP that pressure inside the cranial vault that helps circulation in the brain tissue. The normal value is 07–15 mm Hg in adult in rest condition in supine position.

(Steiner, L. A., & Andrews, P. J. D, 2006).

The brain function will be normal in the adults when ICP is between 10-20 mmHg, 3 -7mmHg in children, and in newborns 1.5-6 mmHg. When ICP value 20-25

mm of Hg is required for proper treatment to save cerebral injury and ICP 40 mm Hg or more which cause severe intracranial hypertension that damage cerebral tissues which indicates a serious illness. (COSNAROVICI, R. V., & CERNEA, D. M.)

ICP depends on the following factors: (Piccinini et al., 2017)

CSF production volume. CSF reabsorption resistance. Intracranial space.

Venous pressure, or superior longitudinal sinus pressure are equivalent.

2. PHYSIOPATHOLOGICAL STUDY OF CBF, CMRO2 CBV AND CPP

Cerebral blood flow (CBF): The total blood supply of brain is 800 ml / min, alogwith CBF of 40 to 50 ml / 100gm of brain tissue / min. The cerebral metabolic rate of oxygen consumption ($CMRO_2$) is about 4-6 ml / 100gm of brain tissue/min. So, hypoxic condition and anaemia ***decrease*** arterial oxygen content in the brain. (Rodríguez-Boto et al., 2015) The CMRO2 is an indicator to determine the values of CBF. In which it is determined by the cerebral auto regulation by means of cerebral vascular resistance (CVR) and cerebral perfusion pressure (CPP). MAP and ICP 90% of the $CMRO_2$ correspond to neural tissue and 10% to the supporting tissue or Glial cells (about more than 50% of the total brain volume. (Shin et al., 2020)

CBV: The blood in the brain refers to both cerebral blood volumes (CBV) and cerebral blood flow (CBF). CBV is the constant volume of blood in the brain, approximately 10% of the total intracranial volume. CBV directly contributes to ICP, whereas the effect of CBF is indirect, through cerebral auto regulation. The position changing such as elevation of head up to ***30 degrees especially*** in hypertension, decrease ICP via changing of MAP, pressure of airway, central venous pressure and increase absorption of CSF. (Le Du et al., 1998) The position above ***30-degree,*** standing or decubitus ***fluctuate*** the systemic arterial pressure and defaecation or coughing which increase intrathoracic or intra-abdominal pressure that up rise ICP. So, the maintenance of CPP up to 70 - 80 mmHg is requiring improving the life. (Schwarz et al., 2002)

CPP: The oxygenation of brain tissues is depending on CPP that supplies through blood (O2 and Nutrients). The ICP is directly responsible to cerebral perfusion pressure and CPP is depending on MAP and ICP. There are three situations.

1. Increased Intracranial volume is not effects on ICP due to CSF compensatory mechanism.
2. The compensatory mechanism is exhausted due to overloading so, ICP is later increased.

3. The compensatory system lost their function. So, mild increase of volume may increase ICP. (Rodríguez-Boto et al., 2015)

3. RELATIONSHIP BETWEEN CPP, MAP, ICP and PP

CPP = 83.33 mmHg. (Adelson et al., 2005)

CPP = MAP – ICP. = 93.33 – 10 = 83.33 mm Hg.

Average ICP = About10 mmHg.

Mean Arterial Pressure (MAP): The average pressure is in the arteries in one cardiac cycle. It is best indicator of perfusion to the vital organs that is calculating through SBP and DBP. One cardiac cycle = Systolic blood pressure and 2 diastolic blood pressure.

$$MAP = \frac{SBP + 2\,DBP}{3} = \frac{120 + 2 \times 80}{3} = \frac{280}{3} = 93.33.$$

Pulse pressure: (Homan et al., 2018)

It is a pressure between systolic blood pressure (SBP) and diastolic blood pressure (DBP). The normal value of PP is 40 mm Hg. The SBP is a maximum pressure of the aorta during heart contraction and rejection of blood from left ventricle into aorta about 120 mm Hg. while DBP is a minimum pressure of aorta during relaxation of heart before ejecting of blood from left ventricle to aorta about 80mm Hg. If, SBP is increased about 150 mm Hg, then 150 – 80 = 70. The PP is wide about 70 mm Hg. The widen pulse indicates several diseases like iron deficiency anaemia, aortic regurgitation, arteriosclerosis, aortic sclerosis and hyperthyroidism.

So, we can calculate; PP = SBP - DBP = 120 – 80 = 40.

Aetiological factors effecting on ICP: The study about causes of ICP mainly found the four factors that responsible to increase ICP;

1. Hydrocephalus due to blockage in the ventricle.
2. Cerebral oedema due to infection e. g. meningitis, encephalitis or hypoxemia.
3. Brain haemorrhage due to severe trauma such as sub dural, epidural, intra parenchymal haematoma, Intra ventricular, subarachnoid haemorrhage and ruptured aneurysm.
4. Mass like tumour, blood clots, infarct and abscess.

Clinical finding of raised ICP

In adult: Mental abilities decreased → Headache → The breathing becomes shallow→ Lethargic condition → weakness of muscles, slowing of speech, numbness, Confusion, seizure → Systolic BP increased →If, does not receive emergency management then go to Coma. Lastly the brain shifted anywhere that cause herniation. (Riveros Gilardi et al., 2019)

In children: Vomiting and mostly symptoms as adult.

In infant: The shape of head is affected, bulging of fontanel, separated skull suture, Drowsiness, and vomiting. Lastly the brain shifted anywhere that cause herniation.

The compression of optic nerve results→ The vision of one or both eyes loses gradually. The eye may be red, dyschromatopsic, exhaphthalmos or photophobic. The compression of oculomotor nerve is produced →ocular sign → Double vision (diplopia) and blurred vision. (Joyce et al., 2022) due to the compression of midbrain, loses all reflexes such as blinking, gagging, no reacting of pupil with the light, dilated and fixed in mid position. Iris becomes wide, ptosis, ocular deviation. The compression involves the pons, breathing status disturb and become irregular. (Knight, J., & Rayi, A., 2020).

4. LATE SIGN

1. **The Cushing response** (Cushing triad) that includes hypertension, bradycardia and bradypnoea. (Dinallo, S., & Waseem, M., 2022)

   In hypertension: Raised ICP, the Systolic blood pressure is increased to manage CBF. So, the para sympathetic nervous system acts as a inhibitory action on heart →decreased the heart beat → Slow the conduction from the sinus node via atrioventricular node → Decrease pulse rate → increase pulse pressure → Ominous sign → The pulse becomes wide that indicates life collapse.

   Bradycardia: Blood supply to the lungs decreased→O2 demand in the lung decreased→ respiration become shallow → Bradypnoea → CO2 volume increased → Vasodilatations → Brain tissue compression increased → ICP increased → Hypoxic - anoxic brain injury.

   Bradypnoea /Abdominal respiration: The respiration is become chain stroke respiration → Apnoea → Hyperpnoea.

2. **Motor changes:** The abnormal motor movement produces three Posture sign.

- **Decerbate Sign-** The body is extended, the muscles are tightened, arms, legs are straightened out ward, toes pointed downward, head and neck being backward. It indicates sever brain injury. ***(Dietz et al., 2022).***
- **Flaccid Sign-** The person can't initiate any voluntary movement due to hypotonia or complete loss of tone.
- **Decorticate Posture Sign-** Abnormal flexion in which the person is stiff, arm bent toward the body, wrists and fingers are bent on the chest, fists are clenched and legs held straighten out ***(Rodriguez-Beato, F. Y., & De Jesus, O., 2020).***

3. **Seizure:** It is an abnormal electrical activity of the brain in which muscles function is increases and metabolism is also increases. The damages anywhere in the corticospinal tract give result of presence of Babinski sign. In which the stimulation of the lateral planter aspect of the foot, it leads to extension / dorsiflexion or upward movement of the big toes (hallux). (Fodstad et al., 2006) In infant, the CST is not fully myelinated, so the positive Babinski sign is normal up to 24 months of age. ***(Seehusen et al., 2003).***

Diagnosis and management depend on radiological and hematological outcomes

The diagnosis of acute neurological condition and therapeutic measurement are by analysis of CSF, CBC, blood glucose level, RFT, Electrolytes and Osmolarity, X-Ray and HCT / MRI head.

5. EMERGENCY CARE

1. Checking of urine output, if, urine output increased then mannitol infusion is working properly, otherwise not working.
2. Give reverse Trendelenburg position - The head and chest are elevated about at 30°than abdomen and legs while hip and knee are not flexed. (Adeloye, A., 1998).
3. Maintain airway, avoid shivering, coughing, sneezing and avoid suctioning more than 10 seconds. The suctioning should be done under hyperventilation with 100 % of oxygen.
4. Avoid extreme hip flexion otherwise increases abdominothoracic pressure that increase ICP. 5. In case of dehydration, the fluid is given as required until CSF increased.

6. FIRST PREFERENCE MANAGEMENT

i. **Promote outflow of jugular vein –** The head of the patient in neutral position and the head of the bed is elevated to 30°. The pregnant women lying in left lateral position because activity of cardiac vagus nerve is suppressed while activity of cardiac sympathetic nerve is enhanced. Abdominal pressure may reduce by using muscle relaxants, laxatives, or decompression in cases of abdominal hypertension. ***(Gleason et al., 2011).***

ii. **Use prophylactic:** (Wakerley et al., 2020)

- **Antipyretic -** Paracetamol 1000 mg infusion through IV as required.
- **Anti seizure drugs –** Inj. phenytoin or Phenobarbitone is recommended when increase both ICP and tissue oxygen demand.
- **Muscular relaxant –** Chlorpromazine is given as required.
- Sedative drug is recommended e. g. midazolam, propofol or etomidate intravenously.
- Anti Inflammatory drugs like dexamethasone that reduce cerebral oedema.
- Glycerin liquid through NG tube introduced into the stomach.

iii. **Osmotic diuretics:** Mannitol infusion is required, the fluid flow increases from low concentration to high concentration which mix into blood then pass to the ***kidney's*** urine output is increased and decreased blood volume, decreased ICP, Increased CPP, O2 and nutrient ***reaches through*** blood to brain and finally function of brain is corrected.

iv. **Repeat cranial CT scan:** This needs to surgical evacuation, as well as appearance of massive cerebral oedema.

Cerebral metabolic rate of oxygen consumption should be decreased through sedation, analgesic such as benzodiazepine reduce CMRO2 and CBF but no effects on ICP while narcotics increase ICP and no support CMRO2 and ***CBF. (Kinoshita,*** K., 2016). Maintain temperature to normal level because it ***increases*** metabolic rate about 10-13% C° that cause vasodilatation which increase CBF and may increase IVP. ***(Rangel-Castillo et al., 2008).***

v. **Improve cerebral oxygenation:**

- Achieve moderate hyperventilation with carbon dioxide pressure (pCO_2) between 25 – 30 mmHg and intense hyperventilation is contraindication in the first 24 hours.
- Maintain oxygen level (spo2) 90-100% and oxygen pressure over 80mmHg.
- In normal perfusion pressure the systolic blood pressure higher than 90mmHg, haematocrit levels of 30% to 33% and haemoglobin levels of 8 to 10 g/dL.

vi. **Decrease cerebral oedema:** Drainage of CSF that decreases ICP.

vii. **Osmotherapy:** About 20% mannitol or hypertonic saline (3%, 7.2%, 20%) should be given as a 20 minutes bolus with the interval of 4 hrs, maximum 1 liter / day. The serum Osmolarity will be maintained below 320 mOsm/kg and serum sodium below 155 mEq /L. (Rodríguez-Boto et al., 2015).

Second-line Treatment:

- ✓ When previous treatment is failed or minimal effects and ICP 20 mmHg and then second-line treatment should be applied as a hyperventilation that reduces ICP.
- ✓ Moderate hypothermia (23-33°C) is a safe therapy after sever injury of brain up to 24 hours.
- ✓ Barbiturate in high dose reduces refractive intracranial hypertension and withdrawal of barbiturate gradually to avoid risk of rebound intracranial hypertension.
- ✓ Catheterization into the ventricle through anterior fontanels for CSF drainage to reduce ICP.
- ✓ Ventriculoperitoneal shunts → The CSF drain from ventricle to peritoneum.
- ✓ Craniectomy for decompression is useful when maximum medical treatment is failed. This procedure is applied in head injury, cerebral infarction and removes the clots or tumour. (Albanese et al., 1999).

7. CONCLUSIONS

The management of ICP is requiring physiopathological knowledge of brain and expert monitoring and understand raised ICP phenomenon because the current diagnosis of ICH and appropriate treatment can help to manage raised ICP and other

destruction but, in late, it develops iatrogenic effects that produce serious illness.

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