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Increased Intracranial Pressure Syndrome

Kafa İçi Basınç Artışı Sendromu

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Abstract

Increased intracranial pressure syndrome (IIPS) is a condition in which intracranial pressure (ICP) rises above the upper limit of normal for age, leading to the development of symptoms and signs. Under normal conditions, the average ICP is 1.5-6 mmHg in infants, 3-7 mmHg in children, and 5-15 mmHg in adults. IIPS is defined as ICP remaining above 20 mmHg for more than 5 minutes. ICP may increase in the presence of increased blood, cerebrospinal fluid and brain tissue volume within the skull, or in the presence of space-occupying lesions. The purpose of this protocol is to provide pediatricians with general information on the diagnosis, monitoring, and treatment of ICP. This protocol does not replace the professional opinion of the physician.

Keywords: Cerebral edema, increased intracranial pressure, traumatic brain injury, pediatric intensive care

Öz

Kafa içi basınç artışı sendromu (KİBAS); kafa içi basıncının yaşa göre normal değerin üst sınırının üstüne çıkması, buna bağlı semptom ve bulguların gelişmesi durumudur. Normal koşullarda ortalama intrakranial basınç (İKB) infantlarda 1,5-6 mmHg, çocuklarda 3-7 mmHg, yetişkinlerde 5-15 mmHg'dır. İKB'nin 20 mmHg'nın üzerinde 5 dakikadan daha uzun seyretmesi durumunda artmış İKB'den söz edilir. Kafatası içinde; kan, BOS ve beyin dokusu hacminin artması veya yer kaplayıcı lezyon varlığında İKB yükselebilir. Bu protokolün amacı KİBAS'ın tanısı, takibi ve tedavisi konusunda çocuk hekimlerine genel bilgi vermektir. Bu protokol, hekimin profesyonel görüşünün yerine geçemez.

Anahtar Kelimeler: Serebral ödem, kafa içi basınç artışı, travmatik beyin hasarı, çocuk yoğun bakım

Introduction

Increased intracranial pressure syndrome (IIPS) is a condition characterized by an increase in intracranial pressure (ICP) above the upper limit of the age-specific normal range, accompanied by the development of associated symptoms and findings.[1,2] Under normal conditions, the average ICP is 1.5-6 mmHg in infants, 3-7 mmHg in children, and 5-15 mmHg in adults.[1] A sustained ICP above 20 mmHg for more than 5 minutes is considered elevated ICP.[2] However, measuring and interpreting ICP in children is challenging. Numerous factors must be considered, including the child's age, the method of measurement, the child's position during measurement, whether the child is crying, whether sedation was administered, and the child's body mass index.[3] The upper limit of ICP measured via lumbar puncture (LP) is generally accepted as 20 mmHg. Although cerebrospinal fluid (CSF) pressure measured during LP correlates with ICP, it may not always provide an accurate indication. This is because

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numerous factors—such as the patient's position, whether sedation was administered, the type of medication used if sedation was given (ketamine may increase CSF production and thereby raise pressure), pain perception, and whether the patient performs a Valsalva maneuver—can influence the CSF pressure measured during LP.[3] The general consensus is that it is more appropriate to evaluate the patient not solely based on ICP but in conjunction with other clinical findings.[2]

Although it is connected to the neck via the foramen magnum and other small foramina, the intracranial space can be conceptualized as a closed box. Approximately 80% of the intracranial space is occupied by brain tissue, 10% by CSF, and 10% by blood (Figure 1).

According to the Monro-Kellie doctrine, the cranium is not flexible. The combined volumes of blood, CSF, and brain tissue maintain a constant intracranial volume. An increase in one of these components or the presence of a space-occupying lesion is compensated for by a decrease in the others (Figure 2).

Within the skull, an increase in the volume of blood, CSF, and brain tissue or the presence of a space-occupying lesion leads to an elevation in ICP. Cerebral venous sinus thrombosis, masses in the upper mediastinum, and conditions compressing the jugular veins result in cerebral venous stasis by impairing venous return. An increase in cerebral blood volume results in increased ICP (Figure 3). ICP may also develop in cases of meningitis and subarachnoid hemorrhages, where obstruction of CSF flow or impaired absorption leads to CSF accumulation and increased ICP. It has been demonstrated that CSF production rate in adults and older children is approximately 20 mL/hour or 500 mL/day.[4] On the other hand, a study by Page et al.[5] reported that CSF production in children under 8 years of age is approximately 15 mL/hour.

Diffuse cerebral edema develops in conditions such as ischemic or anoxic events, acute liver failure, hypertensive encephalopathy, and Reye's syndrome, whereas brain tumors, cerebral infarction, hematoma, and cerebral abscess typically cause localized cerebral edema. Cerebral edema results in an increase in brain tissue volume and in IIPS.

IIPS Reasons

Intracranial space-occupying lesions (tumor, cyst, hematoma, abscess), increased CSF volume (hydrocephalus, meningitis), vasogenic cerebral edema (trauma, tumor, abscess, hypertension, pseudotumor cerebri), cytotoxic cerebral edema (cerebral infarction/ischemia, hypoxia), and osmotic cerebral edema (electrolyte imbalance, inappropriate antidiuretic hormone secretion) are mechanisms that contribute to increased ICP. These mechanisms can cause ICP, alone or in combination (Table 1). However, temporary increases in ICP

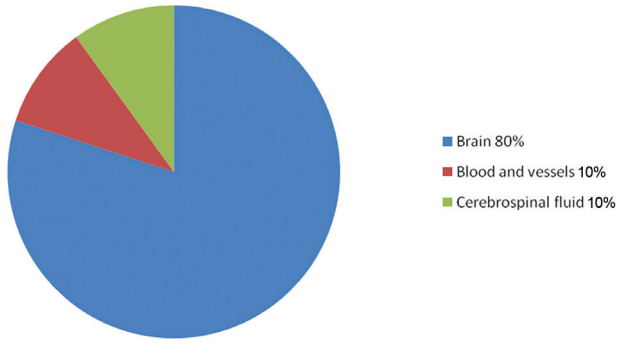

Figure 1. Components filling the intracranial space

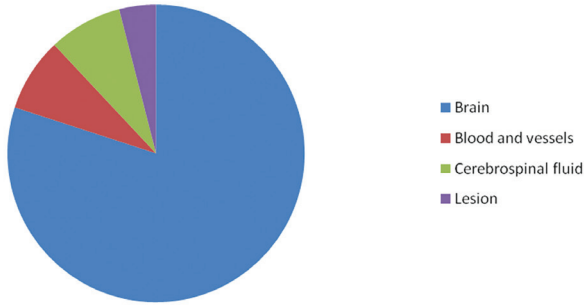

Figure 2. Intracranial components in the compensation phase

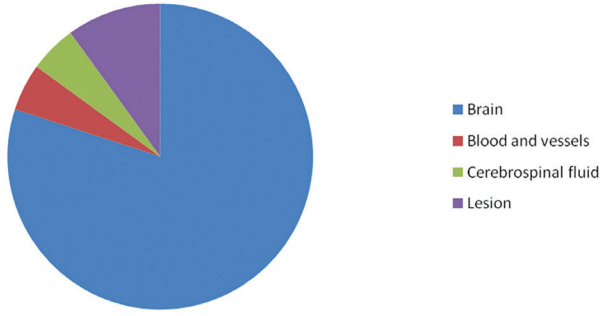

Figure 3. Intracranial components in the decompensation phase

Table 1. IIPS reasons

Head trauma (subdural, epidural, subarachnoid, intraparenchymal hemorrhages)
Cerebral edema (infarction, ischemia, hypoxia)
Diffuse axonal injury
Aneurysm rupture
Arteriovenous malformations
Increased cerebrospinal fluid production or impaired cerebrospinal fluid circulation (hydrocephalus)
Central nervous system infections (encephalitis, meningitis, abscess)
Intracranial space-occupying lesions (tumor, cyst, hematoma, abscess)
Vasculitides (microscopic polyangiitis)
Hypertensive encephalopathy (posterior reversible encephalopathy syndrome, etc.)

may occur during physiological events such as sneezing, coughing, straining, or the Valsalva maneuver.

Physiopathology

One or more of the following mechanisms may cause an increase in ICP. An increase in ICP can lead to compression of brain tissue, impaired perfusion, and brain cell damage.

1. Cerebral edema resulting from cellular damage and disruption of the blood-brain barrier.
2. Decreased vascular tone due to impaired autoregulation.
3. Impaired CSF circulation.
4. Increased CSF production in response to cerebral inflammation.
5. Vasodilation and increased cerebral blood flow (CBF) secondary to hypercapnia or hypoxia.

Inappropriate head and neck positioning, improper administration of sedation and analgesia, electrolyte imbalance (hyponatremia), and low blood oncotic pressure (hypoalbuminemia) also contribute significantly to cerebral edema. However, cerebral edema is the primary mechanism underlying the development of IIPS. There are three types of cerebral edema.

• Cytotoxic or cellular edema is intracellular edema resulting from mechanical damage to the cell membrane. This edema, which appears within minutes of trauma, is primarily caused by dysfunction of the Na-K ATPase. The uncontrolled influx of sodium into the cell, followed by the entry of water, leads to intracellular edema. This form of edema is common in patients with brain injuries such as traumatic brain injury, traumatic axonal injury, or hypoxic-ischemic injury. In these injuries, brain cells can sustain severe damage.

• Vasogenic edema: Increased permeability of capillary endothelial cells causes fluid to pass into the extracellular space. Neurons are not primarily injured. Vasogenic edema is observed in tumors, intracranial hematomas, infarcts, abscesses, and central nervous system infections. Since neurons are not primarily injured, treatment aimed at reducing edema may prevent secondary ischemic injury to the surrounding brain tissue. In this context, steroid therapy may be beneficial in treating vasogenic edema that forms around mass lesions.

• Interstitial edema is characterized by increased fluid in the periventricular white matter. As in hydrocephalus, increased CSF volume is the most common cause of elevated hydrostatic pressure. Interstitial edema responds to treatments that reduce CSF pressure.

Brain Elasticity and Compliance

Brain elasticity represents the increase in ICP per unit increase in intracranial volume and is denoted by "dP/dV". High elasticity indicates that a small change in volume will result in a significant change in ICP. Compliance, on the other hand, is the inverse and is denoted by "dV/dP". Compliance reflects the skull's elasticity-in other words, its ability to absorb pressure changes. Intracranial elasticity (dV/dP) is non-linear. In the early stages of the pathological process, increases in parenchymal volume minimally affect ICP. Once compensatory mechanisms are exhausted, even small increases can significantly elevate ICP.

Pressure-volume Index (PVI)

The PVI is defined as the increase in pressure observed when 1 mL of normal saline is infused into the ventricle via an intraventricular catheter. In children, the normal PVI values vary with age but average 0-2 mmHg/mL. Values of 4 mmHg/mL or higher indicate low cerebral compliance.

Cerebral Perfusion Pressure (CPP)

The perfusion pressure of any tissue depends on the difference between systemic arterial pressure and venous pressure within the vascular bed of that tissue. The intracranial venous pressure within the dural sinuses is approximately equal to the mean arterial pressure (MAP). In practice, CPP is calculated using the following formula:

CPP=MAP-ICP

(MAP=2/3 diastolic pressure+1/3 systolic pressure)

In an ischemic brain region, a decrease in CPP increases brain damage, while an increase in CPP protects brain tissue from the effects of ischemia. However, an excessive increase in CPP may raise the risk of hemorrhage. The lower limit of CPP is generally accepted as 40 mmHg, and pressures below this level have been associated with increased mortality.[6,7] In contrast, there is not a full consensus regarding the upper limit of CPP. Different threshold values have been established across various studies based on age groups.[6,7] Although the optimal CPP is not definitively known, a range of 50-60 mmHg is generally accepted as normal. To maintain CPP, the MAP must be between 50 and 150 mmHg. At pressures >150 mmHg, hydrostatic edema may develop, leading to increased ICP. Conversely, in various diseases of the central nervous system, increased mortality and morbidity have been reported at values below this threshold.[6] Studies suggest that using CPP rather than ICP monitoring is more appropriate for patient follow-up and treatment planning.[8]

CBF

CBF refers to the volume of blood reaching the brain within a given time interval. Under normal conditions, the blood flow to the brain in a healthy adult is 750 mL/min. This volume accounts for approximately 15% of the blood pumped by the heart in one minute. In this case, the average blood flow is 50-

54 mL/min per 100 mg of brain tissue (50-54 mL/min/100 g). CBF depends on many factors. One of these is blood viscosity. In addition, factors such as blood vessel diameter and CPP affect blood flow.

The partial pressure of oxygen (PaO_2) in arterial blood affects cerebral perfusion. An increase in PaO_2 causes vasoconstriction, while a decrease causes vasodilation. Conversely, an increase in the partial pressure of carbon dioxide ($PaCO_2$) in arterial blood (hypercapnia) leads to cerebral vasodilation and increased CBF, while a decrease in $PaCO_2$ (hypocapnia) causes vasoconstriction.

Autoregulation

CBF is regulated by a physiological adaptation mechanism that maintains constant blood flow in response to changes in perfusion pressure, independently of metabolic increases. Cerebral circulation is primarily regulated by metabolic and chemical stimuli, perfusion pressure, and neural stimuli. Thus, CBF is maintained, and the brain's active regions are protected with increased blood supply.[9]

Symptoms and Findings

The clinical presentation of elevated ICP varies depending on the child's age and whether the pressure increase is gradual or acute. For example, in infants, if IIPS rises gradually, macrocephaly and bulging fontanelles may be prodromal signs, whereas in acute IIPS, severe headache, vomiting, and altered mental status are the prominent clinical features. Additionally, certain symptoms and findings specific to the underlying condition causing IIPS may accompany the syndrome. The approach to IIPS is outlined in the algorithm.

The three key findings of IIPS are bradycardia, hypertension (increased pulse pressure), and irregular breathing. This is referred to as the "Cushing triad". Additional symptoms and findings include headache, nausea, vomiting, altered consciousness, papilledema, and symptoms related to cranial nerves (most commonly diplopia, anisocoria, and impaired eye movements associated with pathologies of the 2nd and 6th cranial nerves). Other common findings may include abnormal gait and coordination disorders. Additionally, papilledema on fundoscopic examination may be a specific indicator of elevated ICP.

In children with suspected high ICP, LP carries a risk of cerebral herniation. Therefore, in children with clinical suspicion (cerebral edema, space-occupying lesions, or obstructive hydrocephalus, etc.), an LP should not be performed until neuroimaging is conducted to identify conditions that may constitute contraindications.[10]

Radiological Imaging in IIPS

The diagnosis of IIPS is established based on the clinical presentation and neuroimaging. However, radiological findings may take time to become apparent. Even if the patient develops an acute increase in ICP, the appearance of radiological findings may be delayed by up to 24 hours.[11] Due to its ease of access in emergency settings, computed tomography (CT) of the brain is the preferred imaging method in the initial phase. CT is also the preferred imaging modality for the initial assessment of intracranial injuries caused by head trauma. However, magnetic resonance imaging (MRI) is superior to CT in identifying signs of increased ICP. Its disadvantages include reduced accessibility and a longer procedure time compared to CT.

The following findings may be detected in cranial imaging:

- Midline shift
- Obliteration of the basal cisterns
- Blurring of the sulci
- Enlargement of the optic nerve sheath diameter (ONSD)
- Findings related to the primary pathology causing ICP elevation (brain edema, intracranial mass, hemorrhage, etc.).

Repeat brain imaging (CT or MRI within 12 to 24 hours). If there is no change in the patient's condition, routine repeat imaging is not necessary. However, if there is a deterioration in the patient's level of consciousness (decrease on the Glasgow Coma scale), changes in pupil size (anisocoria, strabismus), new neurological findings, focal deficits, if the patient has a history of head trauma, if a space-occupying lesion is present on the initial CT, or if ICP monitoring cannot be performed, repeat central imaging may be appropriate.[12,13]

Monitoring of IIPS

Even with appropriate treatment, If IIPS is not properly monitored, it may lead to herniation. In such cases, the following symptoms and signs may appear:

- Worsening headache
- Altered level of consciousness
- Unilateral pupil dilation (anisocoria)
- Third cranial nerve palsy
- Decerebrate posture
- Respiratory irregularities
- Cardiac arrest

Various Methods are Used for ICP Monitoring

- To detect increases in ICP, pressure should be monitored using a probe placed in the lateral ventricle by a neurosurgeon (evidence level III).[6] The goal is to maintain ICP below 20

mmHg. In cases where ICP monitoring is not possible, the patient can be monitored using repeated brain CT scans.

• The ONSD can be measured using ultrasound. There are limited studies supporting age-based normal values.[14,15] The normal ONSD value is 2.1-4.0 mm in children under one year of age and 2-3 mm in children over one year of age.[12] Although there is no complete consensus on the upper limit for ONSD, upper limits of 5 mm for adults, 4.5 mm for children, and 4 mm for infants are generally accepted.

• Jugular venous oxygen saturation (SvO_2) is a monitoring method used to track changes in CBF and cerebral oxygen consumption.[16] Continuous SvO_2 is measured using a fiber-optic catheter placed in the right jugular vein. The physiological upper limit of jugular SvO_2 is 55%. Values below this threshold indicate reduced CBF secondary to decreased CPP or to vasoconstriction associated with hyperventilation. It indicates that CBF is insufficient to meet the demand. Conversely, elevated SvO_2 indicates mitochondrial dysfunction, reduced demand due to cell death, or increased CBF.[16]

• If brain tissue oxygenation ($PbrO_2$) monitoring is used, maintaining a $PbrO_2$ level above 10 mmHg is recommended. The level of evidence is low. The use of $PbrO_2$ monitoring should be limited to patients without contraindications to invasive neuromonitoring, such as coagulopathy, and patients without a diagnosis of brain death (evidence level III).[6]

• Near-infrared spectroscopy (NIRS) is a technique that measures the transmission and absorption of near-infrared light (700-1000 nm). The different transmission and absorption properties of oxygenated and deoxygenated hemoglobin enable NIRS to monitor brain oxygenation. It operates on the principle of collecting the transmitted infrared light using a receptor and measuring differences in wavelength. It provides information about brain oxygen saturation.

Among the monitoring methods mentioned above, the gold standard is ICP monitoring using a probe placed in the lateral ventricle. However, it requires an invasive procedure and faces challenges regarding applicability across settings. In recent years, measuring ONSD using ultrasound—which does not require an invasive procedure and can be performed at the patient's bedside—has increasingly become the preferred method.[17,18] Although it may require experience, it yields highly consistent results when performed by experienced practitioners.

NIRS is also a non-invasive method, but there may be issues regarding measurement consistency. It may be a more appropriate approach to make decisions based on the trend of increase or decrease rather than the numerical measurement value.[19]

Treatment

The primary goal in the treatment of IIPS is to eliminate the underlying cause. However, this may not always be possible. In such cases, IIPS is treated with a series of palliative measures aimed at reducing or halting the progression of ICP. A significant portion of brain damage resulting from head trauma does not occur at the moment of injury, but rather develops in the hours or days following the trauma. The goal of ICP management is to prevent or minimize secondary damage. In addition to treatment aimed at reducing ICP, maintaining adequate ICP is important for reducing the patient's morbidity. Limited observational evidence suggests that the target ICP in children should be age-specific as follows.[6,7]

CPP 0-5 years: 40-50 mmHg

CPP 6-17 years: 50-60 mmHg

Although the treatment approaches for IIPS may vary depending on the underlying cause, they are generally consistent. Therefore, IIPS treatment will be examined under the subheadings general measures and the approach to traumatic IIPS.

Note: Evidence levels for some treatment recommendations are provided below. The levels of these evidence grades are as follows:

Level I recommendations are based on high-quality evidence.

Level II recommendations are based on moderate-quality evidence.

Level III recommendations are based on low-quality evidence.

General Measures

• Maintaining stable vital signs—particularly avoiding hypotension, ensuring adequate oxygenation, and preventing hypercapnia—is a measure that helps prevent brain tissue damage from secondary causes. To this end, adequate fluid support and, if necessary, the initiation of vasopressors, should be considered.

• Elevating the head helps reduce ICP. Generally, elevating the head at a 30-degree angle is appropriate.[20] Excessive elevation is not recommended, as it may impair cerebral perfusion. When positioning the patient, care must be taken to ensure that not only the head but also the entire body are elevated on a ramp. Maintaining the head in a neutral position is recommended to facilitate CBF. A cervical collar may be applied if necessary. However, caution is required concerning the development of occipital pressure ulcers in patients who remain in this position for extended periods.

• Since glucose is the brain's primary energy source, hypoglycemia must be prevented.

• Anemia should be avoided, as it may impair oxygen delivery to the brain. Discussions regarding the hemoglobin threshold

for determining the ideal level and transfusion requirements are ongoing. In most studies, transfusion is recommended for hemoglobin levels below 7 g/dL, and a restrictive transfusion strategy is advised.[21,22] However, the 7 g/dL threshold does not apply to premature infants, children with severe hypoxia, hemodynamic instability, active blood loss, or cyanotic heart disease.[21] Indeed, a meta-analysis published in 2021 states that "hemoglobin concentration may not be the most informative indicator of transfusion need in patients with different physiological adaptations to anemia".[22] Therefore, making decisions on a case-by-case basis is more appropriate.

• If the patient is at risk of seizures, prophylactic anticonvulsants should be initiated. In cases of sudden-onset seizures, benzodiazepines remain the first choice. However, monitoring the patient's level of consciousness may be challenging. Recent publications offer cautious recommendations regarding seizure prophylaxis.[6,23] While phenytoin was previously the first-line choice, levetiracetam has been used more frequently in recent years. However, there is insufficient evidence to recommend levetiracetam over phenytoin based on efficacy in preventing seizures or toxicit.[6]

• In intubated patients, hyperventilation was previously recommended. However, it is no longer recommended because it reduces CBF and impairs cerebral perfusion. The recommended arterial pCO_2 level in the latest guidelines has been updated to 35-45 mmHg.[6] High positive inspiratory pressure and high positive end-expiratory pressure should be avoided provided that oxygenation remains adequate. There are no studies regarding the temporary application of hyperventilation in children with a risk of herniation or an ICP crisis.[6]

• Sufficient sedation (midazolam) and analgesia (fentanyl) must be provided to allow for controlled ventilation in intubated patients. If ICP remains high despite adequate sedation, neuromuscular blockade may be required. Rocuronium bromide or vecuronium bromide may be used for this purpose. However, monitoring for seizures via bedside EEG may be necessary.

• Since endotracheal aspiration may stimulate the gag reflex and lead to an increase in ICP, administration of lidocaine hydrochloride at 1-2 mg/kg via the endotracheal or intravenous route prior to aspiration is recommended. However, this effect is short-lived (<10 min). Lidocaine hydrochloride suppresses the gag reflex by blocking the superior laryngeal nerve and the glossopharyngeal nerve.

• While prophylactic moderate hypothermia (32-33 °C) is not recommended to improve overall outcomes (mortality, morbidity, functional loss) (evidence level II), moderate hypothermia is recommended for ICP control (evidence level III).[6]

Hyperosmolar Therapies

• Hypertonic saline (3% NaCl solution) is administered by infusion at a dose of 2-5 mL/kg over 10-20 minutes (evidence level II).[6] The goal is to increase serum osmolarity. This may be repeated 4-6 times daily, depending on the patient's serum sodium level. Sodium levels can be monitored via blood gas analysis before hypertonic saline infusions (4-6 times daily). Additionally, serum sodium levels should be checked twice daily in the biochemistry laboratory. A very rapid rise in serum osmolarity and exceeding 360 mmol/L should be avoided (evidence level III).[6,24] Generally, maintaining the patient's serum sodium level between 150-160 mEq/L is sufficient. If the serum sodium level rises above 160 mEq/L, there is a risk of anemia, thrombocytopenia, and deep vein thrombosis. Hypertonic saline is also administered as a continuous infusion at a dose of 0.1-1.0 mL/kg/hour (evidence level III).[6] Neither approach is superior to the other. Unlike mannitol, 3% hypertonic saline does not cause severe osmotic diuresis and carries a low risk of hypovolemia and renal failure. For this reason, it is preferred over mannitol in pediatric applications. However, complications associated with hypertonic saline administration include hypernatremia, hyperosmolality, acute kidney injury, heart failure and/or pulmonary edema due to fluid overload, metabolic acidosis, and osmotic demyelination syndrome (central pontine demyelination). Although rare, a rebound increase in ICP may be observed following hypertonic saline administration.

• Mannitol solution is administered via infusion at a dose of 0.5-1 g/kg over 20-30 minutes. The preparation contains 20% mannitol. The maximum dose for children should not exceed 100 g or 500 mL. It can be administered every four hours, provided that serum osmolality is monitored before each mannitol dose. Rapid increases in serum osmolality and levels exceeding 320 mmol/L should be avoided (evidence level III).[6,24] Additionally, since mannitol causes rapid diuresis, urine output should be monitored, and a urinary catheter should be placed if necessary to prevent bladder distension. Mannitol administration is also associated with potential side effects such as hyperosmolarity, hypovolemia, electrolyte imbalance, and acute kidney injury. Therefore, serum electrolytes and renal function tests should be closely monitored in patients receiving mannitol. It should not be administered via continuous infusion, because when neuronal cells are exposed to a hypertonic environment for prolonged periods, neuronal shrinkage occurs and the cells compensate by increasing intracellular osmolarity to return to their normal size. This situation gradually reduces mannitol's efficacy and may lead to rebound neuronal swelling.[25]

Corticosteroids

Although corticosteroids have been shown to be beneficial in preventing ICP elevation resulting from vasogenic edema associated with brain tumors, they are not recommended in cases of ischemic or hemorrhagic events. For this purpose, dexamethasone is administered at 0.25 to 0.5 mg/kg every six hours, with a maximum daily dose of 16 mg. The single dose should not exceed 8 mg.

Approach to Traumatic IIPS

• New guidelines do not recommend the use of prophylactic phenytoin or valproate to prevent late post-traumatic seizures (PTN). For early PTN prophylaxis, however, phenytoin is recommended to reduce the incidence of early PTN (within 7 days of injury) when the overall benefit is considered to outweigh treatment-related complications (evidence level IIA).[6]

• Two factors determine adequate blood flow to the brain (i.e., cerebral tissue perfusion). The first is CBF, and the second is ICP. Some researchers argue that normalizing CBF is more important, and that ICP should be considered secondary.[26] However, it has been demonstrated that the pathophysiology underlying the development of brain injury cannot be explained by CPP alone; increased ICP is also associated with a poor prognosis. Therefore, while striving to maintain adequate CPP, clinicians must also control ICP. This is only possible through ICP monitoring.

• Providing appropriate sedation to prevent agitation may contribute to controlling ICP. Benzodiazepines are generally preferred for sedation. Considering the use of appropriate analgesia and sedation during routine intensive care monitoring, it is recommended to avoid bolus administration of midazolam and/or fentanyl during ICP crises due to the risks of cerebral hypoperfusion (evidence level III).[6] Alternatively, intervention with pentobarbital may be necessary in such cases.

Traditionally, ketamine has been considered contraindicated in patients with high ICP; there is concern that it may increase CBF and further elevate ICP.[6] The use of etomidate for ICP control is largely of historical significance, as other sedatives and analgesics that do not suppress adrenal function are now available.[6]

• Decompressive craniectomy: The benefit of this procedure for patients with severe traumatic brain injury is controversial.[6] Potential complications of decompressive craniectomy, such as hygroma, hydrocephalus, and aseptic bone resorption, should be considered.

• The use of steroids to reduce trauma-induced ICP elevation is not recommended (evidence level I).[6] In cases of severe head trauma, high-dose methylprednisolone has been associated with increased mortality and is contraindicated.[27]

Refractory IIPS Cases

• A 20% saline bolus may be administered for refractory ICH. The recommended dose is 0.5 mL/kg, with a maximum of 30 mL (evidence level III).[6]

• CSF drainage: In cases of uncontrolled intracranial hypertension, a neurosurgeon may place an intraventricular shunt for CSF drainage and ICP monitoring. Permanent ventricular shunts are another option.

• Decompressive craniectomy: The removal of a portion of the skull to reduce ICP. Decompressive craniectomy is recommended for treatment of medically resistant neurological deterioration, risk of herniation, or uncontrolled intracranial hypertension (evidence level III).[6]

Footnotes

This protocol has been prepared to provide pediatricians with general information on "increased intracranial pressure syndrome" and is not intended to replace the physician's professional judgment. It should not be used alone for patient diagnosis, treatment, or follow-up.

Authorship Contributions

Surgical and Medical Practices: Ç.K., Concept: Ç.K., Design: Ç.K., A.Ç., R.Y., Data Collection or Processing: Ç.K., Analysis or Interpretation: Ç.K., A.Ç., F.İ.G., F.K., M.U.Y., P.Y.Ö., R.Y., U.E.S., Literature Search: Ç.K., A.Ç., F.İ.G., F.K., M.U.Y., P.Y.Ö., R.Y., U.E.S., Writing: Ç.K., A.Ç., F.İ.G., F.K., M.U.Y., P.Y.Ö., R.Y., U.E.S.

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