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مقایسه GCS و APACHE Score در پیش بینی پروگنوز بیماران بستری در بخش مراقبتهای ویژه (ICU)

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Comparison of GCS and APACHE Score in prognosis of patients admitted in intensive care unit (ICU)

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LIST

Comparison of GCS and APACHE Score in prognosis of patients admitted in intensive care unit (ICU)

It was a comparative cross-sectional survey among patients admitted in intensive care unit of Sina Hospital of Tehran. Mean age of patients was 55.64 years. 64% were male and 36% were female. Mean initial **Glasgow Coma Scale** 9.48 with standard deviation of 3.75 and mean APACHE Score was 16.12 with standard deviation of 6.096. Mean GCS of expired subjects was 9.15 and alive patients was 9.89 which there was no statistically significant association between GCS and prognosis of patients (P=0.339). Mean APACHE Score of expired subjects was 17.67 and alive patients was 14.22 which there was statistically significant association between APACHE Score and prognosis of patients (P=0.004).

Keywords: APACHE Score, Glasgow Coma Scale, Prognosis, ICU

Introduction

Trauma is a leading cause of death in children older than 1 year in the United States, with head trauma representing 80% or more of the injuries. In approximately 5% of head trauma cases, patients die at the site of the accident. Head trauma has a high emotional, psychosocial, and economic impact because these patients often have comparatively long hospital stays, and 5-10% require discharge to a long-term care facility.

One of the most presumed factors contributing to patients' status and also prognosis may be their consciousness level. This factor is usually measured by Glasgow Coma Scale (GCS) which is a conventional method of scoring. Current study has performed to evaluate the association of GCS with prognosis and clinical course of head trauma patients admitted in intensive care units because of a low consciousness level.

Review of Literatures

Pathophysiology: Primary and secondary injuries are described with head trauma, and the presence of these injuries affects the outcome of these patients.

The primary injury occurs at the time of impact, either by a direct injury to the brain parenchyma or by an injury to the long white matter tracts through acceleration-deceleration forces. Direct injury to the brain parenchyma occurs as the brain is impacted on the bony protuberances of the calvaria or by penetration of the brain by bony fragments or a foreign body. In children, the compliant skull is easily deformed, and impacts on the brain at the time of the insult result in a coup injury, as opposed to adults, in whom the brain is forced against the bony protuberances opposite the point of the impact, resulting in a countercoup injury. Intracranial hemorrhage may also result from shearing or laceration of vascular structures. Acceleration-deceleration forces cause shearing of the long white matter tracts, leading to axonal disruption and secondary cell death.

The secondary injury is represented by systemic and intracranial events that occur in response to the primary injury and further contribute to neuronal damage and cell death.

The systemic events are hypotension, hypoxia, and hypercapnia and may occur as a direct result of primary injury to the CNS or can result from associated injuries in a person with multiple traumas.

The intracranial events are a series of inflammatory changes and pathophysiologic perturbations that occur immediately after the primary injury and continue over time. Their presence adds to the adverse outcome of the head trauma patient. The inflammatory events are the result of a cascade of biomolecular changes triggered by the initial insult, leading to microcirculatory disruption and neuronal disintegration. A series of factors such as free radicals, free iron, and excitatory neurotransmitters (glutamate, aspartate) are the result of these inflammatory events, and their presence contributes to the negative outcome. The pathophysiologic events are cerebral edema, increased ICP, hyperemia, and ischemia.

The brain has minimal ability to store energy; thus, it is dependent on aerobic metabolism. The delivery of oxygen and metabolic substrate to the brain is maintained by a constant supply of blood known as cerebral blood flow (CBF). CBF, defined as the amount of blood in transit through the brain at one given point in time, is estimated to be 50mL/100g/min in a healthy adult, and it is known to be much higher in children. However, the minimum amount necessary to prevent ischemia remains unknown. CBF is influenced by mean arterial blood pressure (MAP), ICP, viscosity

of the blood, metabolic products, and the diameter of brain vessels. CBF should not be confused with cerebral blood volume (CBV), which represents the amount of blood present in the brain vasculature. CBV is the major contributor to the ICP and is dependent on the diameter of intracranial vessels. When CBV is increased, the pressure gradient across the compartment is decreased, and the CBF is decreased.

The brain has the capacity of maintaining constant blood flow through a mechanism known as autoregulation. This occurs over a wide range of blood pressures through changes in cerebral resistance in response to fluctuations in MAP pressure. The CBF is maintained at a MAP of 60-150 mm Hg. At 60 mm Hg, the cerebral vasculature is maximally dilated, and at 150 mm Hg, it is maximally constricted. Fluctuations past this range lead to alterations in CBF and contribute to ischemia or disruption of the blood-brain barrier. Several mechanisms are known to affect autoregulation of CBF, and they can be divided into metabolic products and arterial blood gas content and myogenic, neurogenic, and endothelium-dependent factors. Their effect is not fully known, and their mechanism of action is still under experimental investigation.

CBF is closely linked to cerebral metabolism. Although the mechanism of coupling is not clearly defined, it is suspected to involve vasodilators released from neurons. Several factors have been implicated such as adenosine and free radicals. Pathophysiologic states, such as fever and

seizure activity, that are known to increase the metabolic activity lead to an increase in CBF.

CBF can be altered by changes in the partial pressure of oxygen or carbon dioxide. Alteration in the partial pressure of oxygen acts on the vascular smooth muscle through mechanisms that remain unclear. Hypoxia causes vasodilatation with significant increase in CBF. Increases in oxygen pressure cause vasoconstriction but to a lesser degree than hypoxia. Hypercarbia increases CBF up to 350% of normal; hypocapnia produces a decrease in blood flow. The mechanism appears to involve alteration in tissue pH that leads to changes in arteriolar diameter. This mechanism is preserved even when autoregulation is lost.

The myogenic mechanism was considered for a long time to be the most important in the autoregulation process. The changes in the actin-myosin complex were thought to lead to rapid changes in the vasculature diameter, thus affecting the CBF. Currently, changes in the actin-myosin complex have been shown to mostly cause dampening of arterial pulsation and to have little direct effect on cerebral autoregulation.

The neurogenic mechanism is represented by the effect of the sympathetic system on the cerebral vasculature. The sympathetic nervous system shifts autoregulation towards higher pressures, whereas sympathetic blockade shifts it downwards.

Recent studies identified nitric oxide as one of the factors affecting cerebral autoregulation by producing relaxation of cerebral vessels. It is present in several conditions such as ischemia, hypoxia, and stroke. Nitric oxide has been shown to be generated by different cells at rest but also under direct stimulation by factors such as cytokines.

Traumatic brain injury may lead to loss of autoregulation through alterations of the described mechanisms. These mechanisms represent the foundation upon which the medical management of increased ICP and cerebral perfusion pressure (CPP) is based in patients with traumatic brain injury.

Frequency:

- **In the US:** Head injury is estimated to occur in approximately 200 per 100,000 population per year. The number includes all head injuries that resulted in hospitalization, death, or both in persons aged 0-19 years.

Mortality/Morbidity: The overall outcome for children with head injuries is better than that of adults with the same injury score. Recovery in children takes longer, from months to sometimes years, whereas adults reach maximum recovery by about 6 months following the injury. Outcome assessment based on the Glasgow Coma Scale (GCS) could be used as an early predictor, but it has limitations regarding long-term

outcome. Patients with multiple organ injuries, including head trauma, generally have a far worse outcome than those with head injury alone.

- Mortality: According to the National Center for Health Statistics, the mortality rate from head trauma is 29% in the pediatric population. These data are based on death certificate information, and 29% could be an underestimation of the actual rate. Data reported by studies in trauma centers show that head injury represents 75-97% of pediatric trauma deaths.
- Neurologic deficits: Ten to 20% of children with moderate-to-severe head injury (GCS of 6-8) have short-term memory problems and delayed response times, especially if the coma lasts longer than 3 weeks. More than half of children with GCS of 3-5 have permanent neurologic deficits.

Race: Black adolescent boys account for most of the firearms-related CNS injuries in the pediatric population.

Sex: Males are twice as likely to sustain head injuries as females, and they have 4 times the risk of fatal trauma.

Age: The distribution of head trauma is relatively stable throughout childhood. An increase in the incidence of head trauma was identified in 2 age groups.

- At approximately age 15 years, a dramatic increase occurs, mainly in males, related to their involvement in sports and driving activities.
- Infants younger than 1 year have also been identified by several studies as having an elevated incidence of head trauma, which is attributed to falls and child abuse.

CLINICAL

History: Head trauma patients may experience one or a combination of primary injuries, depending on the degree and mechanism of trauma. Specific types of primary injury include scalp injury, skull fracture, basilar skull fracture, concussion, contusion, intracranial hemorrhage, subarachnoid hemorrhage, epidural hematoma, subdural hematoma, intraventricular hemorrhage, subarachnoid hemorrhage, penetrating injuries, and diffuse axonal injury.

- Scalp injury
    - Often observed with traumatic brain injuries, scalp injury can overlie other intracranial pathology; therefore, it requires careful exploration for foreign bodies or underlying skull fractures.
    - Bleeding associated with scalp lacerations could be significant enough to cause hypotension and shock in a small infant.

- Caput succedaneum and cephalohematoma are observed with birth-related head trauma. Caput succedaneum involves molding of the neonatal head and crosses the suture lines, whereas cephalohematoma involves subperiosteal bleeding and is limited by the suture lines.

- Skull fracture
  - Skull fractures are linear, comminuted, depressed, and diastatic. In children, 90% of the fractures are linear and tend to be more diastatic; thus, the radiographic appearance is more impressive. An open fracture is a fracture overlaid by a laceration. The presence of cerebrospinal fluid (CSF) in the wound indicates a violation of the dura and warrants further exploration.
  - Location of the fracture is important because it may cross the path of a major vessel and be associated with an intracranial bleed.
  - Depressed skull fracture is defined as displacement of the inner table of the skull by more than one thickness of the bone. One third of depressed fractures are simple, one third are associated with dural laceration, and one fourth have cortical lacerations.

- Basilar skull fracture
    - This is present in 6-14% of pediatric patients with head trauma and is suggested by a history of a blow to the back of the head.
    - Loss of consciousness, seizures, and neurologic deficits may or may not be present. Children with basilar skull fracture usually have prolonged nausea, vomiting, and general malaise, most likely because of the vicinity of the fracture to the emesis and vestibular brainstem centers.
    - Physical findings such as Battle sign, raccoon eyes, and CSF otorrhea and rhinorrhea are pathognomonic; ocular nerve entrapment may occur in 1-10% of patients.
- Concussion
    - A transient loss of consciousness, concussion occurs as the result of head trauma. Patients often have normal findings on neurologic examination; the diagnosis is usually a retrospective one.
    - Infants and young children have a higher incidence of posttraumatic seizures and most often increased delayed somnolence and vomiting; older children have a history of posttraumatic amnesia.

- Waxing and waning of mental status in the absence of any morphologic changes is also characteristic of concussion and is more often observed in older children.

- Contusion
  - Caused by a direct injury to the head, a contusion is an area of bruising or tearing of the brain tissue. The temporal and frontal lobes are the most vulnerable areas because of their anatomical relationship with the bony protuberances of calvaria.
  - The typical presentation is of progressive neurologic deterioration secondary to local cerebral edema, infarcts, and/or late-developing hematomas.
- Epidural hematoma
  - Developing between the skull and the dura and secondary to the laceration of an artery or vein, epidural hematomas of arterial origin peak in size by 6-8 hours after the injury. Epidural hematomas of venous origin may grow over 24 or more hours. Common locations are the temporal, frontal, and occipital lobes. An overlying skull fracture may be present.
  - Patients may present with the classic lucid interval between the initial loss of consciousness and subsequent neurologic

deterioration, but this is less frequent in the pediatric population.

- When neurologic deterioration with hemiparesis, unconsciousness, posturing, and pupillary changes develops, it is due to the expansion of hematoma and exhaustion of compensatory mechanisms, with subsequent compression of the temporal lobe and/or brain stem.

- Subdural hematoma
  - Located between the dura and the cortex, subdural hematoma results from tearing of the bridging veins across the dura or laceration of the cortical arteries during acceleration-deceleration forces; it is usually associated with severe parenchymal injury, and the presentation is that of profound and progressive neurologic deterioration.
  - Subdural hematoma may develop secondary to birth trauma, in which case the presentation is within 12 hours of life and includes seizures, full fontanel, anisocoria, and respiratory distress.
  - Subdural hematoma is also a feature of shaken baby syndrome; the usual presentation is of new-onset seizures, increased head circumference, poorly thriving infant, and tense fontanel. Focal neurologic deficits are usually absent.

- Penetrating injuries: Resulting from various sources, penetrating injuries should be considered neurosurgical emergencies because rapid deterioration and fatal hemorrhages may ensue.
- Intraventricular hemorrhage: This type of hemorrhage is usually the result of minor trauma and resolves spontaneously. Large hemorrhages could lead to obstructive hydrocephalus, especially when they are located at the level of the foramen of Monroe and the aqueduct of Sylvius, in which case surgical intervention is required.
- Subarachnoid hemorrhage
    - The most common form of hemorrhage associated with head trauma, subarachnoid hemorrhage, results from disruption of the small vessels on the cerebral cortex. The usual location is along the falx cerebri or tentorium and the outer cortical surface.
    - Common symptoms include nausea, vomiting, headache, restlessness, fever, and nuchal rigidity caused by blood in the subarachnoid space.
- Diffuse axonal injury
    - A result of rapid acceleration-deceleration forces, this type of injury causes disruption of the small axonal pathways.

- The most commonly affected areas are the basal ganglia, thalamus, deep hemispheric nuclei, and corpus callosum. Their increased vulnerability to shear injuries is attributed to a different momentum of these structures from the rest of the brain at the time of the injury.
- Patients usually present with various states of altered mentation and often remain in a vegetative state for long periods. A marked discrepancy exists between the highly abnormal neurologic examination findings and the lack of findings on CT scanning. Occasionally, small petechial hemorrhages may be present.
- Prognosis for full recovery often is poor.

Physical: Head trauma patients often have multiple organ injuries. Assessment of patients with severe head injuries involves a primary and a secondary survey. The primary survey is a focused physical examination directed at identifying and treating life-threatening conditions present in a trauma patient and by this, preventing secondary brain injury. The secondary survey of patients with head trauma is a detailed examination and assessment of the system with the goal of identifying all traumatic injuries and directing further treatment.

- Airway (primary survey)

- Airway inspection should be directed at identifying the presence of foreign bodies, loose teeth, facial lacerations and bone instability, deviation of trachea, and circumoral cyanosis indicative of hypoxia.
- Auscultation of airway may suggest the presence of upper airway obstruction, especially when a turbulent flow pattern is noted.

- Breathing (primary survey): Apnea and hypoventilation secondary to pulmonary or neurologic causes are common findings in patients with head trauma. When present, they require immediate intervention and endotracheal intubation.
- Circulation (primary survey)
  - Cushing triad, bradycardia, hypertension, or alteration of respiration, if present, is a late manifestation indicative of herniation.
  - When present, hypotension should not solely be attributed to intracranial hemorrhage. Several other causes may lead to this finding, such as internal hemorrhages, spinal cord injury, cardiac contusion, and dysrhythmias with secondary impaired cardiac output. Hypotension associated with bradycardia in a trauma patient should be highly suspicious of spinal cord injury.