REVIEW ARTICLE

PEDIATRIC TRAUMATIC BRAIN INJURY

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Abstract

Traumatic brain injury is a major cause of serious harm and death in children under the age of 15. The injury affects not only the patient, but also impacts heavily on close relatives. Caring for victims with traumatic brain injury is perhaps the most difficult of many professional challenges for nursing staff, requiring both technical and skills and sensitivity to the needs of the relatives. The **aim** of this study was to present a review of recent publications specifically addressing nursing intervention in the care of children with traumatic brain injury. **Methodology :** The approach to this article focus on research and review of studies between 2008–2016, from the online sources of Pubmed/Medline, Elsevier, Saunders Medical Center, Lippincott Williams and Wilkins, New England Journal of Medicine, The Journal of Head Trauma Rehabilitation and the Journal of Neuroscience. The literature featured in this article refers to nursing intervention in cases of children with traumatic brain injury, identified through key words such as: nursing intervention in neurosurgery, nursing intervention in children with cranial trauma, head injuries and nursing care. **Results:** The most recent studies emphasize that nursing interventions in the case of children who have sustained traumatic brain injury should be provided by specially trained persons who have acquired the skills and knowledge within this particular area. Essential to successful outcomes of nursing interventions are frequent training and tutoring sessions where the nurse, in conjunction with the physician, will be able to find, understand and apply scientifically competent solutions to meet the exact needs of the case. The role of the nurse should follow a personalized plan clearly defined as part of the total care and welfare of the patient. **Conclusions :** Nursing interventions for the care of victims with traumatic brain injury include improvement of the neurological status and achieving a better outcome. However, there are few researched facts in the literature that document the detail of the nursing interventions performed. This suggests that further studies of the nature of the nursing interventions are necessary.

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Introduction

Pediatric traumatic brain injury represents more than 80% of all injuries that lead to severe neurological deficits and even death among children younger that the age of fifteen. It occurs when external forces affect the head or the brain resulting in some conditions such as concussions, skull fractures, intracranial hemorrhages (intra-axial and extra-axial hemorrhage), contusions and diffuse axonal injuries. Using the Glasgow Coma Scale (GCS), which is a neurological scale from 3-15 that records the conscious state of patients, a traumatic brain injury can be classified as:

- Mild 13-15/15
- Moderate 9-12/15
- Severe 3-8/15 [(1)]

The pathophysiology of a brain injury is divided into two distinct stages, the primary and secondary injury. The first stage or primary injury occurs at the moment of the traumatic event causing physical trauma to the brain. The second stage or secondary injury is a result of a complex cascade causing hemodynamical changes, excitotoxicity, production of free oxygen radicals, mitochondrial dysfunction and calpain-mediated proteolysis.[(2)] Acknowledging such complex processes, allows us to identify complications and treat such patients more efficiently while improving the outcome and quality of life. The leading causes of traumatic brain injury in children are:

- Falls of any kind, representing a 55%
- Unintentional and blunt trauma, representing a 24%
- Motor vehicle accidents, representing an 18%
- Assaults, representing a 3% [(3)]

The incidence, between males and females in the early stages of life are almost same. After the age of 10, males seem to be more prone to injury than females. Prognosis depends on the severity and location of the injury. Severe brain injuries, tend to have poor prognosis in comparison with mild brain injuries which tend to have a better outcome. Studies suggest that permanent disabilities occur in 10% of mild injuries, 66% in moderate and 100% is severe injuries. [(4)]

Concussion

A concussion or biochemically mediated neuronal dysfunction is the most frequent type of traumatic brain injury. Concussions are closely associated with sports but can be seen in any head

impact or acceleration forces (explosions etc.) resulting in some cases in the loss of consciousness (less than 10%) for a small period. [5] The pathophysiology of the injury is very complicated and involves neuronal injury with the increase of nerve cell depolarization which results in an increased rate of neurotransmitter release from the synaptic vesicle. In a cellular level, the potassium gates open rapidly allowing potassium to leak out of the cell causing the Na-K-ATPase pump to work overtime to overcome the imbalance resulting in anaerobic metabolism due to hyper glycolysis. In addition to the potassium leak, calcium enters the mitochondria causing mitochondrial dysfunction. Excitatory neurotransmitters cause the brain to become hypermetabolic leading the nerve cells to apoptosis (cell death). [2] Concussions have a high incidence in children and statistics suggest that males are more prone to sustain such injury by 67% in comparison to female 33% with a median age of 13 years. [4]

A variety of symptoms might occur after a concussion such as somatic (headaches, vomiting, nausea, vertigo, blurred vision, dizziness, speech dysfunction, and seizures), cognitive (dementia, impaired judgment and lack of coordination) and psychosocial (depression, personality changes, and insomnia). Most findings resolve in a short period after the injury. In some cases, symptoms may maintain after 7 days in 50%, 30 days in 25% and 12-24 weeks in 10%. [5] Most cases arrive at the emergency department with symptoms such as memory loss, blurry vision, confusion and loss of consciousness. A primary survey can identify physical evidence of injuries such as a skull fracture or edema. In addition to those findings a good history of the accident can lead into making a diagnosis. Neuroimaging may be recommended in some patients depending on the severity of the symptoms and physical findings, with a computed tomography being the primary scan of choice. [1] Magnetic resonance imaging is used for a more detailed image of the brain and in later stages of the injury. The management of a concussion is divided into two phases with first combining complete physical and cognitive rest plus medications to relieve symptoms such as pain and nausea and the second phase with the gradual return to normal activities. Resting is crucial and includes limited exercising, playing, scholastic working, reading, watching television and playing video games which

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allow the brain to return to its normal function. The resting period usually lasts 7-10 days or until the patient becomes asymptomatic. The second phase includes the gradual return to normal activities in a particular time frame. [1,5] The two stages are crucial as they minimize sudden death caused by second impact syndrome which occurs when a person sustains a second brain injury causing the patient to slowly sleep in a coma and resulting to death in more than 50% of times. [1] Prognosis is good, and the person has a full recovery. In some cases, patients can suffer from short (postconcussive syndrome, nausea, vomiting, etc.) and long (chronic traumatic encephalopathy, long-term memory loss, psychiatric disorders, etc.) term effects. [6]

Only in severe cases may the patient benefit from an admission for a closer monitor of their injuries. Most often patients with concussion are allowed to go home. Nursing care involves training and informing patients and their relatives about the condition and how to follow the two stages of management mentioned above. A qualified nurse can plan the gradual return to daily activities protocol by taking into account the physician's instructions. During the training session, the patient must understand the importance of the management and information should be provided to identify any complications that may arise. During the second stage of the rehabilitation if the patient becomes symptomatic they should immediately contact their medical team for further investigation. A routine follow-up should always be scheduled to assess the patient's rehabilitation. [1,5,6]

Contusion

Contusion or cerebral contusion is a form of a bruise of the brain tissue that occurs due to acceleration and deceleration forces causing the brain to impact with an area of the skull. The first impact occurs when the brain hits the wall of the skull and is called a coup injury. Coup injuries are generated when the skull is temporarily bent inward and impacts the brain. When the skull bends inwards, it may set the brain into motion, causing it to collide with the opposite side of the skull which results in a secondary injury called a contrecoup. [1] Contrecoup injuries occur in approximately 25% of cases as the external force has to be substantial to set the brain into such motion. Contusions have a high incidence and are related to severe brain injury in 20-30% of times. [6]

Cerebral contusions are divided into non-hemorrhagic and hemorrhagic depending on the force of the impact. Non-hemorrhagic contusions are usually associated with edema of the brain tissue and in some cases causing diffuse axonal injury. Symptoms vary from nausea and vomiting to seizures and coma. Patients may also present blurred vision, faintness and respiratory distress due to herniation. Hemorrhagic contusions or progressive hemorrhagic contusions include a wide variety of hemorrhagic lesions (subdural, epidural, subarachnoid, etc.). The presentation differs depending on the location of the bleed and the severity. Nausea and vomiting, headache, vision abnormalities, loss of consciousness, seizures, and coma are some of the symptoms that may be present. [1,6] Diagnosis is made by obtaining a good history and with the help of radiographic studies. An account of the accident is necessary to understand the exact forces that have caused the injury. The primary scan used for diagnosis is a computed tomography scan. In some cases, magnetic resonance imaging may be used to identify non-hemorrhagic contusions. It is paramount to repeat the scan again in the first 12-24 hours as the injury may have enlarged subacutely. [7] Management of a cerebral contusion involves treating life-threatening symptoms and relieving pressure from the brain. Contusions typically resolve without neurosurgical interventions. In some cases and when the patient deteriorates, surgical intervention is required. [6,8] Prognosis depends on the severity of the injury and varies from full recovery to severe neurological deficits and death. [8]

While caring for patients with contusions like in any other condition, nurses should be aware of the area which has sustained the injury and the severity. Close observations are vital as is examining the level of consciousness with the Glasgow Coma Scale. Patients with such condition are more likely to be transferred to the intensive care unit for a closer monitor of their condition. Early signs of intracranial pressure or seizure activity, are escalated to the attending physician. A scan may be performed to investigate the reason of deterioration. Relatives are informed during meetings with the multidisciplinary team, and early planning should take place, as these patients benefit from rehabilitation. [6,9]

Skull Fractures

Skull fractures are very common during head injuries and occur when external forces affect the skull in such way that it

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cannot withstand the pressure leading to a fracture. A pediatric skull in comparison with an adult is much thinner but more flexible which may result in a fracture with forces equivalent to 2280 N or higher exceeding the skull fracture tolerance. [10] The part of the skull that is most frequently affected is the parietal, then the occipital and then the frontal and the temporal. Linear, depressed, diastatic and basilar are the four major types of fractures seen in pediatric patients. [6]

Linear Skull Fractures

Linear skull fractures are the most often seen fractures in pediatric injury. The definition of a linear fracture is a breakage that runs parallel to the long axis of the bone but does not displace the bone tissue. [11] This kind of fracture is seen in the upper fragment of the frontal, parietal and occipital bones with the parietal being the most common bone affected due to the nature of the bone and the vulnerability of the site of the impact. [6] Frontal and occipital fractures are seen less frequently due to the thickness of the bone. A fracture in the frontal and occipital bone are related to more powerful impacts and therefore more severe injuries. Linear fractures represent approximately 90% of all fractures in children, although some studies suggest that the figure may be incorrect because of the number of fractures that go undiagnosed. [6,10,12]

This type of fracture can be difficult to diagnose as there is no deformation of the bone and there are hardly any symptoms. In some rare cases scalp edema, severe headache, restlessness, irritability, nausea, vomiting, vertigo, short term memory loss and loss of consciousness can present as a result of a linear fracture. A leptomeningeal cyst or growing fracture is a complication that may develop when a laceration of the dura mater causes the arachnoid membrane to get trapped between the fracture edges. This allows the brain to pulse into the fracture preventing it from healing and causing the fracture to grow bigger potentially. This condition develops when the bone is in the healing process and is referred to as a chronic complication. It is more often seen in young children under the age of three causing a variety of symptoms such as headaches, progressive scalp edema, seizures, focal neurological signs and hydrocephalus in rare cases. [13] Linear skull fractures are diagnosed with the help of imaging. A computed tomography is the scan of preference, although a plain X-

ray may also reveal the fracture as long as there are no suspicions of brain injury. Growing fractures can be detected with a computed tomography or a magnetic resonance imaging scan, usually after 14 months of the initial fracture. [14] Treatment of a linear fracture is conservative in neurologically intact patients. Neurosurgical intervention is required in patients that have developed complications such a leptomeningeal cyst which involve the removal of the cyst and repair of the dura mater. [13]

The majority of patients are allowed to go home with pain management and a planned follow-up. Severe cases and early childhood require admission for closer monitoring. Nursing care in those patients involves close observations (vital signs and GCS score) and early detection of any neurological deficits. Physical examination is also necessary to monitor scalp edema and other acute complications. Seizures are a rarity but when detected, require safety precautions and monitoring to manage them effectively. Informing and educating parents is also part of a nurses role. During discharge, it is important to educate parents on performing a physical examination and giving them information on chronic complications that may arise in time so they can seek help without delays. [1,13]

Depressed Skull Fractures

Depressed skull fractures account for 15-25% of children with all skull fractures and are most often the result of a blunt force trauma where the bone breaks and is displaced inwards causing a deformity of the skull. [15] Due to the deformity, there is an increased chance that the brain tissue may get injured as the bone is displaced. As a result, depressed skull fractures are related with moderate to severe traumatic brain injuries. The region most affected is the parietal bone and then the frontal, as it is more prone to injury due to location while the most common cause is falls and motor-vehicle accidents. [13,16]

The bone that extends to the brain tissue can cause direct pressure on the area or even injury, causing a wide variety of symptoms. Depending on the severity of the injury and the area that has been affected, symptoms such as pain, nausea, vertigo, loss of consciousness, edema of the surrounding tissue, seizures, cerebrospinal fluid leakage and focal neurological deficit can be present. Complications such as cerebral contusions and hemorrhages can be associated with depressed skull fractures. [6] A diagnosis

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can be made with a plain radiograph (X-Ray), although a computed tomography is the scan of preference at it can reveal in detail any associated injuries to the cerebral parenchyma. [14] A physical exam can also detect the skull vault that has been depressed. Treatment options vary depending on the nature of the fracture. Simple skull fractures require conservative management. Surgical treatment is recommended in patients with compound skull fractures or when there is dura involvement. Cosmetic appearance may also dictate the path of treatment. [17]

As this type of fracture is associated with moderate to severe brain injury, patients will require admission for further monitoring. Compound skull fractures may even require intensive care. Close neurological observations are paramount to assess the patient for any neurological deterioration and seizure precautions should be taken due to the increased risk of complications secondary to the fracture. Nurses should look for signs of increased intracranial pressure by evaluating the Glasgow Coma Scale, pupil reaction, and search for the Cushing's triad which is a series of symptoms (increased systolic pressure, bradycardia, abnormal respiration pattern and prolonged pulse pressure). Cerebrospinal fluid leakage can be detected if there is dural involvement and early detection is crucial to avoid further complications (infections). In the presence of cerebrospinal fluid, antibiotic prophylaxis should be initiated to prevent infections of the meninges. Due to the severity of the injury nurses should inform parents about the cause, management and outcome of the injury. Early discharge may come in benefit in later stages when the patient is stable and able to be discharged. [6,13]

Diastatic Skull Fractures

Diastatic skull fractures or growing skull fracture are seen in children under the age of three due to the immaturity and flexibility of the skull. A diastatic skull fracture is when two bones separate at a suture line, allowing the brain to herniate outwards beneath the unbroken scalp. The cause of such fracture is usually blunt trauma or deceleration forces with the parietal and temporal-parietal sites being affected the most. [18] It's a condition that can be life-threatening if not treated. The condition can be quite a rarity as it is seen in less than 0,05-1,6% of cases. [19]

A growing skull fracture can be divided into three phases the pre-phase, the early

phase, and the late phase. The pre-phase stage manifests during the time from the initial injury and the time just before there is an enlargement of the fracture. The early stage is seen in a period of two months and after the initial enlargement. The late stage begins after the first two months of the initial enlargement. [20] Depending on the site and the phase of the injury, symptoms vary from nausea, vertigo, headaches, scalp swelling, seizures, hemodynamic instability, impaired vision, contralateral hemiparesis and hydrocephalus. Making an early diagnosis is important as injuries in the pre-phase have a good prognosis. Late stage injuries tend to leave permanent neurological disorders. There is a combination of clinical signs that are seen by a computed tomography scan, which is the most common method used in diagnosing a diastatic skull fracture. [18,20] Unfortunately, a computed tomography may not always differentiate the injury and therefore it is important to proceed with a color Doppler ultrasonographic study or magnetic resonance imaging scan which can disclose more detail of the fracture and underlying injuries. [18] Treatment options vary depending on the phase of the injury. Neurosurgical intervention is almost always required. Craniotomy with the closure of the dura is the first line of treatment. Cranioplasty is needed if the defect is >4 cm in width. A ventriculoperitoneal shunt is required in the presence of hydrocephalus. Prognosis depends on the phase of the injury. Pre-phase and early phase injuries tend to have good outcomes. Even if the patients are symptomatic, they tend to recover after neurosurgical intervention. Late phase injuries tend to have a poor outcome with patient's suffering from long-term neurological deficits (seizures, hydrocephalus, impaired vision, etc.). [20,21,22]

Patients with diastatic skull fractures need close monitoring of their injury. Neurosurgical intervention is almost certain in these cases, and patients will require intensive care for the first 24-48 hours. Some of the nursing intervention involve regular neurological observations and monitor for cerebrospinal fluid leakage. Symptoms that may indicate hydrocephalus such as nausea, vomiting, seizures, drowsiness and confusion should be reported early for a scan to be obtained. The injury site will require attention to avoid any infections. Because seizures may occur it is important to take the necessary precautions to prevent further injury and furthermore

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documentation of the convulsion action is appropriate for diagnostic and therapeutic reasons. Last nurses should provide information to relatives and provide reassurance at all times. [13]

Basilar Skull Fractures

Basilar skull fractures are linear fractures that present in the base of the skull. The temporal and occipital bones the sphenoid wings and sinuses and the foramen magnum form the base of the skull and are involved in this type of fracture. The leading cause of basilar skull fractures is motor-vehicle accidents, falls, and assault, with the temporal bone being affected in almost 18-40% of cases. Incidence range between 3,5-24% of all head trauma and are related to severe traumatic brain injury. [23,24]

Fractures as such may cause a variety of symptoms depending on the region and the extent of the trauma. Meningeal tear is a major concern and can result in cerebrospinal leakage. The leak itself may cause an eardrum perforation due to the excessive pressure in the middle space of the ear, or it may exit into the nasopharynx through the nose or the eustachian tube (anterior skull base fractures). Other symptoms that may present are vomitus, deafness, nystagmus, cranial nerve palsy, blurred vision (1-10% of patients complain of blurred vision) and bleeding. A hemorrhage is a life-threatening complication that should be detected as early as possible through certain signs and presentations. Battle sign occurs when blood accumulates near the mastoid process and is a result of the ruptured posterior auricular artery. Raccoon eyes present when the venous sinuses are ruptured resulting in bleeding into the arachnoid villi and cranial sinuses. Other complications such as cavernous sinus thrombosis, panhypopituitarism, carotid-cavernous fistula, pneumocephalus and carotid artery damage may manifest after a basilar skull fracture. [23] Diagnosis is made with a computed tomography scan as it is considered the gold standard in detecting a fracture of the floor of the skull. Furthermore, by obtaining a history of the traumatic event and with a physical examination of the cranial nerves, one can make a more accurate diagnosis of the condition. [25] Treatment options vary, with most cases (up to 85%) healing without any neurosurgical intervention regardless if there is a cerebrospinal leakage. If conservative management of the leakage does not resolve in a weeks

time, then a lumbar drain should be inserted. Only in complicated cases or when the leakage does not resolve with a lumbar drain, may a patient benefit from neurosurgical intervention. Antibiotic prophylaxis may be indicative to minimize the chance of infection (meningitis). Prognosis depends on the severity of the injury and the presence of underlying complications. Basilar skull fractures usually resolve in a short period with no serious neurological deficits, but the presence of complications can result in poor outcomes. [24,26]

The majority of patients are treated in wards under normal care. In moderate to severe cases, patients may require more aggressive treatment and equal care. During the admission, patients should be monitored for cerebrospinal fluid (CSF) leakage as it is one of the most common symptoms. Nurses should always observe leakage of any kind (serous, clear, blood, etc.) and send the fluid for Beta-2-transferrin analysis. Beta-2-transferrin is a marker found in the CSF and thus making it the best test to detect such leakage. [27] Regular observations and assessment of the conscious level with the Glasgow Coma Scale are necessary to detect any deterioration. As most base fractures resolve spontaneously patients are allowed to go home early provided information, have been given to the relatives. As mentioned, relatives are taught to detect CSF leakage and monitor vital signs. Any significant observation should be looked in detail as complications may occur. [6,13]

Intracranial hemorrhage

Intracranial hemorrhage is defined as a bleed within the skull that can be traumatic in origin. Nontraumatic events or spontaneous intracranial hemorrhages can occur, but traumatic injuries leading to intracranial bleeds have a higher occurrence in childhood. Hemorrhages within the skull can be further divided into intra and extra-axial. Intra-axial hemorrhages, are bleeds that occur within the brain tissue, and can be further divided into intra-parenchymal (intra-cerebral) and intra-ventricular, depending on the region where the blood accumulates. Extra-axial hemorrhages are bleeds that generate outside the brain tissue, between the skull in the different layers of the meninges. Furthermore, they can be divided into three subtypes, epidural, subdural and subarachnoid hemorrhages. Intra-axial bleeds are less common than extra-axial in children. [6] Each hemorrhage is discussed separately to allow readers understand the

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difference, diagnosis, and management of each condition.

Intra-parenchymal hemorrhage

Intra-parenchymal hemorrhage is an acute accumulation of blood within the brain tissue. It is a rare but life-threatening form of hemorrhage in children. [28] The cause can be traumatic or non-traumatic, with trauma being the most common cause resulting in rupture of intracranial vessels. Non-traumatic cases are very rare but are seen in bleeding disorders and arteriovenous malformations. Other non-traumatic causes are coagulopathies, cancer, moyamoya disease, infections and drug abuse. Most bleeds occur in the supratentorial region (80%) and less in the infratentorial (20%). The incidence is unknown due to the rarity of the condition and the lack of studies. [29,30]

Clinical features in children with intra-parenchymal bleeding include a headache, vomiting, seizures, limb weakness, focal neurological deficits, deterioration in sensorium, impaired consciousness and symptoms indicating increased intracranial pressure. Depending on the cause of the hemorrhage, diagnosis is obtained by using different radiographic studies. A computed tomography scan is usually performed on all patients, and after that, a more detailed study can be conducted such as angiography and magnetic resonance imaging. Treatment options vary while taking into consideration the cause. Conservative management is focused in patients with underlying coagulation disorders or when neurosurgical intervention is contradicted. Embolization of an arteriovenous malformation is preferred over craniotomy and clipping of the malformation. Craniotomy for evacuation of the bleed is an approach if other means have failed or when the embolization of the arteriovenous malformation was not a success. Studies suggest that the outcome of a parenchymal hemorrhage is good in 47%, fair in 23%, poor in 23% and death may occur in 7% of cases. [28,29]

Due to the severity of the condition and poor prognosis, patients are initially treated in the intensive care unit. It is crucial that patients remain for the first 24-48 hours under close monitoring and may even require sedation. Early weaning efforts should begin quickly for a more thorough neurological assessment. Patients are usually nursed in an angle of 30^0 with regular neurological observations

in place. In some cases, an intracranial bolt is inserted for measurement of the intracranial pressure. Seizure precautions are necessary due to high risk of convulsion actions. Early rehabilitation and discharge planning should be started as permanent disabilities are common in intra-parenchymal hemorrhages. Part of the nursing role is to engage with relatives and provide answers to their questions as well as scheduling multidisciplinary meetings. Furthermore, in some hospitals, family members under surveillance are allowed to provide some of the daily care to their children. [13,31]

Intra-ventricular hemorrhage

Intra-ventricular hemorrhage is a bleed into the ventricular system of the brain which contains the cerebrospinal fluid. The majority of intra-ventricular hemorrhages (IVH) are secondary either due to an expansion of existing bleeds or due to subarachnoid hemorrhages. [32] Approximately 60% of IVH are secondary and 40% primary. Primary IVH are seen inclosed in the ventricles usually due to the trauma of the ventricular system or arteriovenous malformations, aneurysms and malignancies. These conditions are associated with poor prognosis and have been found to relate to 35% of severe traumatic brain injury. [32,33]

The most common presentation of such condition is a sudden onset of a headache, nausea, vomiting, altered Glasgow Coma Scale, seizures, elevated intracranial pressure, coma, and death. Hydrocephalus is a frequent complication that arises after an IVH. The clot causes obstructions in the aqueducts, and as a result, the CSF is not able to drain causing obstructive hydrocephalus. Also, the breakdown of the clot causes an inflammation process that leads into the arachnoid granulation which does not allow the CSF to be absorbed resulting in communicating hydrocephalus. Diagnosis is made with a computed tomography where the bleed is seen inside the ventricles. Scoring scales such as the Graeb scale can calculate the amount of blood in the ventricles, but the software-dependent volumetric analysis is the gold standard. Management of an IVH includes insertion of an intracranial bolt for accurate intracranial pressure. In some cases, an external ventricular drain is inserted to relieve pressure. Several trials such as the CLEAR trial have been used to treat such condition where recombinant tissue plasminogen activator-mediated has been injected to the ventricles to break down the clot. Craniotomy combined with stereotaxy for clot

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evacuation is used for several cases. [32] IVH has a poor prognostic sign with mortality reaching 80%. [33]

Patients usually benefit from intensive care as they require close monitoring and aggressive treatment. Nursing intervention includes regular neurological observations and assessment of the Glasgow Coma Scale. Measurement of the intracranial pressure (7-15 mm/Hg) and the cerebral profusion pressure (70-90 mm/Hg) is also crucial. The cerebral profusion pressure is calculated by subtracting the mean arterial pressure from the intracranial pressure (CPP=MAP-ICP). [6] Elevated blood pressure should not be treated as it may be a response of the brain to regulate the cerebral profusion pressure (Cushing-Kocher response). Latest guidelines recommend treating systolic blood pressure greater than 180 mm/Hg or diastolic blood pressure greater than 105 mm/Hg. Fluid resuscitation is necessary and the head nursed at 30^0. [34] Coagulopathies should be corrected as soon as possible as they can enlarge the bleed. Complications that may arise such as infections, seizures, etc. should be reported early to be treated. Seizure prophylaxis and anti-epileptic medication should be initiated in all patients with IVH due to the increased risk of seizures. Compression stockings are beneficial to prevent deep vein thrombosis as immobility can cause further complications. Patients are usually sedated for the first 24 hours and after that weaning effort may commence for a more thorough neurological assessment. In the long term patients will require rehabilitation as such conditions cause permanent disabilities. It is, therefore, essential for early discharge planning. Also, nurses have the responsibility to inform relatives on the progress. In some hospital settings, parents are even allowed to be part of the daily nursing care. Regular multidisciplinary meetings should be organized to provide detailed information. [6,13]

Epidural hemorrhage

Epidural hemorrhages are bleeds located between the dura mater and the overlying calvarium and are a result of trauma (impact force). The cause of such hemorrhage is usually a laceration of the dural vessels, including branches of the middle meningeal arteries, veins, dural venous sinuses, and skull vessels. [35] The middle meningeal artery is the most common injured vessel located in the temporal region with an incidence of 70-

75%. Skull fractures are seen in 50% of cases. Epidural hemorrhages are not very frequent in children with an incidence rate of approximately 1-3% of all head injuries and 2-2.5:1 ratio, males being predominant to females. [1,35,36]

Epidural hemorrhages have a unique presentation. Usually, after such injury, patients have altered level of conciseness but tend to recover spontaneously for a short period. As the bleed expands, mass effect occurs which causes elevation of the intracranial pressure and drop of the conciseness level for a second time (herniation syndrome). This phenomenon is called a lucid period and is described by a temporary improvement of a patient's symptoms and then rapid deterioration. In some cases, the injury may lead straight to coma with no lucid period. Cushing triad may kick in which involves hypertension, bradycardia and respiratory depression a result of compromised brainstem perfusion. In addition to that, patients may present with external evidence such as scalp lacerations or contusions and weakness on the extremities of one side (pronator drift). [13] A computed tomography scan is the most sensitive study in diagnosing such hemorrhage. [37] Treatment depends on a variety of factors but is divided into two categories, surgical evacuation of the clot (burr hole, craniotomy, etc.) or conservative management and in a second stage evacuation. Guidelines recommend that patients with less than 30 ml, less than 15 mm thick, and less than 5 mm midline shift, without a focal neurological deficit and a Glasgow Coma Scale greater than 8 can be treated conservatively. [38] Prognosis tends to be good when a lucid period has presented or when the underlying brain structure is intact. Mortality rates average approximately 10%. [39]

Treatment for the first 24 hours is conducted in a monitored setting or intensive care unit. Nursing interventions include regular observations with Glasgow Coma Scale assessment. Early detection of complications (herniation, leptomeningeal cyst, infections, etc.) is important as treatment is crucial. Some patients may even require intracranial pressure monitoring. [6,13] Cushing's triad (hypertension, bradycardia, and respiratory depression) may be present, but hypertension should not be treated as it can compromise the cerebral profusion pressure causing ischemia. Guidelines suggest that systolic blood pressure greater than 180 mm/Hg or diastolic blood pressure greater than 105 mm/Hg

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may be treated. [31] Adequate fluid resuscitation may be needed. Patients are usually stepped down to standard care after 24 hours of close monitoring, where rehabilitation takes place. Discharge planning is important as continuing rehabilitation is necessary as well as regular follow ups with radiographic studies (computed tomography scans). Relatives should always be reassured and informed on the progress of their child. Recovery period in epidural hemorrhages varies, but children tend to have a good outcome. [13]

Subdural Hemorrhage

Subdural hemorrhage occurs due to laceration of vessels (usually veins) on the inner part of the dura matter which connects the cerebral cortex to the venous sinuses. As a result, blood occupies the space between the dura mater and the arachnoid membrane. The hemorrhage is often a result of trauma (acceleration or deceleration forces) and less often due to coagulopathies or infections, with the supratentorial region affected mostly. [1,13] The incidence rate is approximately 5% of all traumatic brain injuries and is accompanied with a fracture in 30% of all cases. [13,40]

Subdural hemorrhages are divided into three categories acute (0-3 days after the traumatic event), subacute (3-42 days after the traumatic event) and chronic (6 weeks and more from the traumatic event). [41] Signs of a subdural hemorrhage are similar to an epidural hemorrhage but come in a slower time frame. In an epidural hemorrhage, there is not enough room for the bleed to expand resulting into a quicker presentation, while in a subdural hemorrhage there is space for the bleed to expand leading to a delayed presentation of symptoms. Even though, the presentation between the two hemorrhages are similar, during the lucid period patients do not always recover completely in a subdural hemorrhage. External evidence of trauma may be present, and other symptoms include a headache, nausea, vomiting, neck stiffness, pupillary dilation, and seizures. [6,13] Diagnosis is made with a computed tomography which also helps identify underlying brain damage and fractures. [42] Treatment option varies depending on the volume of the bleed, symptoms and the time that has elapsed since the traumatic event. Guidelines suggest that surgical decompression (burr hole biopsy, craniotomy) rather than

conservative management should take place in patients:

- with midline shift equal or bigger than 5 mm
- when the clot exceeds 1 cm
- when the Glasgow Coma Scale has decreased by 2 or more points since the time of the injury
- when the pupils are dilated or fixed
- when the intracranial pressure is greater than 20 mm/Hg

Prognosis is poor with mortality rate approximately 60% and may drop to 30% if the clot is evacuated within 4 hours. [43]

Such conditions are treated in a closely monitored setting, usually intensive care unit. Regular observations, assessment of the Glasgow Coma Scale and intracranial pressure monitoring are crucial in the first 24-48 hours. Nurses should be trained to detect and treat elevated intracranial pressure as well as reporting signs of deterioration. Sedation is required for the admission in intensive care. [13] Cushing's triad may also be present in such conditions, but hypertension should only be treated unless systolic blood pressure is greater than 180 mm/Hg or diastolic blood pressure greater than 105 mm/Hg. [31] In herniation syndromes, mannitol should be administered rapidly. Furthermore, seizure precautions and prophylaxis is necessary with anti-epileptic medications. Coagulopathies should be treated as they can enlarge the bleed and cause further complications. Compression stockings are beneficial to prevent deep vein thrombosis due to immobility. Frequent multidisciplinary meetings should be held to provide progress information to relatives. Early rehabilitation is fundamental as is discharge planning. Patients will require intense physiotherapy, regular follow-ups once discharged. [6]

Subarachnoid hemorrhage

Subarachnoid hemorrhage is a term used to describe extravasation of blood between the arachnoid and the pia membrane. The bleed can be arterial or venous in origin. [13,35] Trauma being the most frequent cause, at 80% of cases, causes pre-existing aneurysms or arteriovenous malformations to rupture. Nontraumatic events do exist with cancer, vasculitis, coagulopathies, meningoencephalitis and benign peri mesencephalic subarachnoid hemorrhage with incidence rate at approximately 20%. The condition accounts 18% of all traumatic brain injuries. [6,29]

The signs and symptoms of a subarachnoid hemorrhage vary depending

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on the volume of blood and the affected area. The classic presentation of such hemorrhage consists of a severe headache often called a thunderclap headache, neck pain, and stiffness, nausea, vomiting, symptoms of meningeal irritation (nuchal rigidity and pain and bilateral leg pain), loss of conciseness and seizures. Other nonclassical symptoms may include extremity weakness either on one side or bilaterally, Cushing's triad (hypertension, bradycardia, and respiratory depression) and coma due to elevated intracranial pressure. [44] A computed tomography scan is usually the scan of preference as it can detect the bleed between the arachnoid and the pia membrane. Furthermore, it can reveal underlying trauma of the cerebral cortex and fractures. [45] A lumbar puncture, a procedure that is performed in patients with suspected subarachnoid hemorrhage with negative scans, can determine the presence of blood in the cerebrospinal fluid. [45,46] Multiple tools have been formed to grade a subarachnoid hemorrhage. Two of the most common used grading tools are the World Federation of Neurosurgeons classification, which uses the Glasgow Coma Scale to determine the grade of the hemorrhage, and the Fisher scale, which uses images of a Computed tomography scan to establish the thickness of the bleed and the presence of blood in the ventricles. [47] Early management is crucial to minimize further bleeding. Aneurysms and arteriovenous malformations are treated with coiling or surgery depending on the location and size. Coagulation disorders should also be addressed during the initial management to prevent re-bleeding. [48] Prognosis depends on the grade. Overall 50% of cases result in death, while approximately 60% of patients that survive will have related deficits (a headache, cognitive impairment, etc.). [45,49]

All patients require neuro-critical care, where highly trained nurses provide advance monitoring, assessment, and management. Complications related to subarachnoid hemorrhages may arise such as vasospasm, electrolyte disruption, seizures, hydrocephalus and elevated intracranial pressure. It is, therefore, essential to detect early signs of deteriorations. Hypovolemia should be avoided at all times as it increases the chance of vasospasm. Furthermore, Nimodipine, a calcium channel inhibitor has shown to improve outcome after such

bleed, and also reduces the incident of vasospasm. Correction of electrolyte imbalances is crucial, as is the management of increased intracranial pressure. When norm ventilation and elevation of the head fails, an external ventricular drain may be inserted, to relieve the pressure on the brain by draining cerebrospinal fluid. In this case, nurses must control the rate of drainage and inform of any deterioration. Pharmacological prophylaxis is also paramount with anti-convulsive agents and anesthetics. Hyperosmolar agents are used to reduce the intracranial pressure. In severe cases, a patient may require a decompressive craniectomy, where part of the skull is removed to allow the brain to swell outwards. As the last line of treatment, sedation with barbiturates in combination with therapeutic hypothermia may be used to control the swelling. [50,51] Relatives should always be informed regarding the progress, and regular multidisciplinary meetings are essential to be held for the same reason. Once the child has been stepped down from the intensive care unit, neuro-rehabilitation should take place as early as possible. Discharge planning is also a major part of the nursing role, where rehabilitation, family help, and regular follow-ups must be scheduled to provide a safe and easy discharge. [13,51]

Diffuse axonal injury

Diffuse axonal injury is characterized by axonal tear, at the region where the axon is stretched, resulting in degradation to the part that is furthest to the tear. The axonal tear is not generated during primary injury, but usually during secondary injury, where the various biochemical cascades cause disruption to the region and resulting in damage to the axons. Most axonal injuries occur in regions where white and gray matter converge, the corpus callosum, the basal ganglia, and the brainstem. The cause of such injury, is rotational forces due to high-velocity acceleration or deceleration injuries, resulting into stretching of the neuro-axons. [52,53] A diffuse axonal injury was noticed in 72% of patients with moderate to severe traumatic brain injury, making it a very common. [54]

The classic presentation of a patient that has suffered a diffuse axonal injury is an immediate altered level or loss of consciousness which usually lasts for more than 6 hours. Other symptoms may include pupillary or other cranial nerve dysfunction and abnormal flexion. Furthermore, hypertension, brain stem abnormalities and hyperhidrosis, a typical

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presentation with such injury, may become evident. [6] Diagnosing a diffuse axonal injury is difficult, as computed tomography scans are not able to detect axonal injury. A magnetic resonance imaging scan is preferred, as can detect small artifacts where the white and gray matter meets up and other multifocal areas with abnormal signals. [55] Depending on the anatomical distribution of the injury, a diffuse axonal injury is graded into 3 grades. A grade I injury, involves the white-grey matter, while grade II includes deeper structures of the brain such as the corpus callosum. Grade III, is a combination of grade I and II injuries, plus involvement of the brain stem. [56] Treatment is limited, and efforts to minimizing secondary damage such edema, hypoxia and increased intracranial pressure should take place. [6] The outcome varies depending on the grade of the injury but is usually poor with approximately 90% of patients not being able to regain consciousness (persistent vegetative state) after a severe diffuse axonal injury. [54]

As there is no definitive management, symptomatic treatment and efforts to minimize secondary injury should be initiated without delay. Therefore, intensive care unit is essential for the management of these patients. Close monitoring and early detection of deterioration such as increased intracranial pressure and hypoxia are crucial. Nurses, must also consider instating seizure prophylaxis and be able to detect changes in the neurological status. Early multidisciplinary meetings are important for educating relatives regarding progress. In such conditions, recovery is very slow and in some cases incomplete. Rehabilitation is also paramount, with the patient benefiting from other specialties (physiotherapy, occupational therapy, etc.) in the fight to maximize recovery. Studies suggest that the majority of patients in an unconscious state will remain unconscious; thus long term care in rehabilitation hospitals is necessary. [6,54]Providing holistic care to these patients means that health professionals should take into account patients' needs and provide counseling depending on the age of patients . [57,58]

Conclusions

Traumatic brain injury, leads to serious neurological deficits or even death, and has a high occurrence in childhood. Moreover, a brain injury also impacts the lives of relatives in a financial,

psychological and social prospective. The majority of brain injuries, are a result of motor vehicle accidents and falls. Males past the year of 10 tend to be more prone to injury than females. The initial impact causes the primary injury which is followed by a cascade of biochemical reactions causing further damage to the brain tissue, and is characterized as the secondary injury. Despite our knowledge around the primary injury, the secondary injury needs to be analyzed further, as it causes more damage than the initial impact. Nurses, have a vital role in the diagnosis, management and rehabilitation of patients with such conditions. Furthermore, they are the key professionals in noticing early deterioration signs, a crucial factor in maximizing the patients outcome by allowing early intervention to take place. Finally, victims that have sustained a brain injury require intense rehabilitation, so early discharge planning is crucial.

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