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Traumatic Brain Injury, Current Clinical Management Strategies in ICU: Hands on Manual

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ABSTRACT: Traumatic brain injury (TBI) is a leading cause of morbidity and mortality, frequently requiring intensive care unit (ICU) management. Current ICU strategies aim to prevent secondary brain injury through early stabilization and continuous neurological monitoring. Initial care focuses on airway protection, adequate ventilation, and hemodynamic stability to ensure sufficient cerebral oxygenation. Intracranial pressure (ICP) monitoring guides targeted interventions to control intracranial hypertension. Maintenance of optimal cerebral perfusion pressure is achieved through fluid management and vasopressor support when necessary. Due to the severity of the social economic health related issue is important to carefully provide veritable guide to doctors especially intensivist on the current protocol from years of experience of different scholars, local and internationally accepted guidelines to serve as a hand on local protocol of management of TBI in the intensive care unit. This manual is based on existing regulatory documents and international accepted method of managing TBI, special and detailed emphases is placed on careful calculation of fluid balance and maximising fluid administration through the enteral route. Overall, ICU management of TBI emphasizes

multidisciplinary, protocol-driven care to improve neurological outcomes.

Keywords: Traumatic Brain Injury, Intensive Care, Hands on Manual.

INTRODUCTION

Traumatic brain injury is one the commonest reason for admission into the intensive care units or the emergency room worldwide, with advent of industrialisation, sporting and other activities, the burden of TBI is very high. In Nigeria the incidence is over 2,710 per 100,000, this much higher than in develop countries. The population mostly involve are young adults between 20 -30 years, often traders or motorcyclists.

The major cause is road traffic accidents (RTA), this takes over 70-80% of the cases in Nigeria, over 50% are moderate and severe TBI. This creates high and costly impact in the society and in the health care system especially in developing countries. The health care burden ranges from resource strains, financial needs, poor emergency transport system, poor or lack of trained personnel and surgical logistics needs.

The social economic impact is heavy as most people affected are bread winner of their families and most times don't recover or recover with disability, left to their families to carter for them all their life, with frequent readmission into the hospital without universal health coverage, (UHC), this leaves the relatives to bear huge financial burden. There is total economic loss to the families who was ones productive.

Due to the severity of the social economic health related issue is important to carefully provide veritable guide to doctors especially intensivist on the current protocol from years of experience of different scholars, local and internationally accepted guidelines to serve as a hand on local protocol of management of TBI in the intensive care unit.

This manual is based on existing regulatory documents and international accepted method of managing TBI, special and detailed emphases is placed on careful calculation of fluid balance and maximising fluid administration through the enteral route. Specific indications for a balanced sedation and management involving hyperosmolar therapy for cerebral oedema.

For easier understanding and management of TBI this simple classification is adopted as it closely related to the management strategy.

Classification of Traumatic Brain Injury.

A]. Closed, B]. Open.

II. Closed TBI: Mild, Moderate and Severe.

III. Mild TBI: Concussion and Mild Cerebral Contusion

IV. Moderate TBI: Moderate cerebral contusion

V. Severe TBI: Severe cerebral contusion,

Hematomas: Epidural, Subdural, Intracerebral, Subdural Hygromas.

Depression fracture of the cranial Vault.

When a case of suspected TBI is suspected, most times from RTA, patient presented with a confused, altered state consciousness or once unconscious but recovered consciousness, it is necessary to care take this step. Note proper history must be taken.

Primary Examination:

Visual inspection, determine the level of consciousness using the Glasgow Coma scale (GCS.) as

it is in Table 1below. There are other scale and other modification of GCS, this is proven to serve the purpose.

Table 1:

Response	Score
Eye opening	
Opens eyes spontaneously	4
Opens eyes in response to speech	3
Open eyes in response to painful stimulation (eg, endotracheal suctioning)	2
Does not open eyes in response to any stimulation	1
Motor response	
Follows commands	6
Makes localized movement in response to painful stimulation	5
Makes nonpurposeful movement in response to noxious stimulation	4
Flexes upper extremities/extends lower extremities in response to pain	3
Extends all extremities in response to pain	2
Makes no response to noxious stimuli	1
Verbal response	
Is oriented to person, place, and time	5
Converses, may be confused	4
Replies with inappropriate words	3
Makes incomprehensible sounds	2
Makes no response	1

1. Examination of the pupil, noting the following, size, symmetry, reaction to light.
2. Evaluate the patient's general condition.
3. Elicit focal neurological symptoms.
4. Look out for Meningeal signs
5. Examine for reflexes, ability to swallow etc.
6. Mandatory measurement of vital signs, Non-invasive blood pressure (NIBP), heart rate, respiratory rate, and capillary refill time.

   Check for urine output rate.

As a guide: clinically severe TBI in acute period will present as follows:

GCS of 3-8 points this is a lesion that has affected the upper and lower part of the brain, medulla oblongata, clinically they are comatose, stupor but less common, normothermia or could also be hyperthermia, their blood pressure could be increased or decreased. There could be change in heart rate and respiration rate rhythms, breathing pattern could change to abnormal breathing. The internal organ and skin could have some changes; asymmetrical reflexes can be elicited.

Moderate TBI on other hand has a higher score of GCS 9-12 this is mostly lesions affecting large hemispheres and extrapyramidal system. Clinically patient presents with stupor, slow reduced movement, movement with decreased amplitude, masked face, loss or reduced facial expressiveness. Patient often present also with increased muscle tone in the limbs, muscular rigidity. Temperature will show hyperthermia from 37.0 to 38.5 degree centigrade. Cardiovascular examination will show normal or slightly elevated blood pressure and heart rate; reflexes could be asymmetry.

Consultation of a Neurologist / Neurosurgeon is necessary, close monitory of patients is needed and mandatory this will include but not limited assessment of neurological status this mandatory on admission and after three hours' reassessment I s done then daily if symptoms do not progress and patient did not deteriorate. In case of deterioration repeated neurological examination is needed and actions taken. In the consultation of the Neurologist there are nonspecific sign of brain oedema and increase intracranial pressure ICP, headache nausea, vomiting, elevated blood pressure, bradycardia, papilledema, paresis of the VI cranial nerve, transient visual disturbances,

cerebrospinal fluid pressure changes and loss of consciousness. The progression of symptoms and further decrease of GCS by 1-2 indicates that there is an increase in ICP.

In event of increased ICP and possible herniation that will require anti oedema therapy:

1. Diencephalic herniation will occur with supratentorial lesions, with displacement of diencephalon through the tentorial notch. Clinically respiratory pattern will change from normal to Cheyne-Strokes respiration (cycles of deep and shallow breathing), miosis (small or constricted pupils) with preserved reaction to light, there could be the presence of upward gaze palsy and changes in mental health.

2. Medial temporal lobe herniation may occur with lateral supratentorial lesions, with the medial temporal lobe displaced through the tentorial notch, creating pressure on the midbrain structures. Clinically patient presents with depressed consciousness, mydriasis (dilated pupils) without light reaction on the side of herniation, this caused by the compression of the III cranial nerve, hemiparesis on the opposite side, the eye movement are always not affected.

3. Cerebellar tonsillar herniation this is as a result of pressure compressing the lower part of the cerebellum through the foramen magnum, this in turn compresses the medulla oblongata, clinical presentation is impaired consciousness and irregular breathing or apnoea.

Patient with TBI moderate or severe will require laboratory work up, all necessary laboratory and radiological investigation must be done on admission.

Complete full blood counts and differentials, blood sugar and urine test must be done, platelet count – caogulogram, biochemistry – creatinine levels, urea, total protein bilirubin, AST, ALT, amylase. Blood gases, electrolyte, acid and bases balance and lactate are all compulsory. Plasma osmolality. Patient blood group and Rh factor, toxicology investigations of blood and urine for alcohol, drugs and toxic substances.

Radiological investigation is important as lesion could be seen and informed management strategy is determined. This includes but not limited Computer tomography (CT), of the skull, in poor centre that do not have CT, head sonography could be done, but the gold standard is CT. X- rays of the chest and skull in two projections or views, also the X-ray cervical spine and echoencephalos copy.

Other investigations are ultrasonography of internal organs, ECG, lumber puncture, monitoring of intracranial pressure, brain oxygenation, SpO2, and cerebral blood flow. It is important to note that central line is important to set.

Anaesthesiological support/ considerations includes method of analgesia, general anaesthesia with mechanical ventilation, preferably using a semi open circuit especially if there is no capnography. Mechanical ventilation is set at normal mode, or hyperventilation keeping PaCO2 at 28-35mmHg. Blood gases checked and cross checked.

Selection of anaesthetic drugs is very important, recommended are as follows sodium thiopental, propofol, narcotic analgesics, benzodiazepines, isoflurane, sevoflurane, and non-depolarising muscle relaxants.

Selection of infusion should have to be done carefully, 0.9% sodium chloride, 3-7,5 % sodium chloride solutions for hypotension and sign of intracranial hypertension. In management of severe TBI there are the dos and the don’ts, the don’ts are do not use nitrous oxide, halothane, ketamine, glucose solutions. Dextran and albumin except in cases of severe hypovolemic shock. The use of frozen plasma except in cases of clinically significant coagulopathy. Hypotension and hypovolemia must be prevented and corrected quickly if it occurs. Arterial blood pressure must be maintained above 90mmHg. After surgical procedure patient must be transferred to ICU managed on mechanical ventilation.

Treatment Protocol:

For proper understanding of accurate management of severe TBI, the protocols will be simplified into various pillars in the treatment. Each of them will be discussed for proper understanding.

1. Patient positioning: Patient should be nurse in head elevated at 30 degrees, the use of bed anti bed sore foam and regular antiseptic skin care products are mandatory and patients body position should change every 3hrs if no contraindication. Manual physical therapy should be done, especially massage and exercise by physiotherapist and patient relatives. For proper approach of physiotherapyis advisable that patient relative is trained to assist as this will reduce workload and burnout from staff, this will also create a good relationship and trust in the medical professional.

2. Most often in the ICU patients die due to lack of proper nutrition protocol, hence it is very pertinent that feeding should be a major treatment pillar. In feeding look out for patient ability to swallow, if the swallowing reflex is active and preserved, to ascertain patient can swallow give a drink of normal saline solution (0.9%). If there is no risk of aspiration, immediately initiate oral intake of fluids and food as soon as possible. If swallow is impaired, then pass a gastric NG tube for feeding and feed, this will check not only energy needs but will enhance gut mobility and reduce risk of multiorgan disorder. Patient can also benefit jejunostomy tube, placed under the guidance of endoscopic or radiological manipulations. This has great advantages of gastric tube as it reduces risk of aspiration. For prolonged comatose condition, the best method to provide nutrition is gastrostomy or jejunostomy. For enteral feeding special balanced mixtures are necessary, that allows patient to receive adequate fluids and nutritional ingredients, proteins, carbohydrates, lipids, vitamins minerals and electrolytes.

3. Parenteral feeding should be used when enteral feeding is not possible, the energy requirement within the first 24hrs should be up to 10kcal/kg, the goal is to achieve within the first week 30kcal/kg.

4. Management of airway is very crucial, once the GCS if 8 or below patient must be intubated, if the coma will prolong above five-day tracheostomy should be inserted, preferably a tube that will allow subglottic suctioning

5. In a severe TBI patient proper breathing mechanism is impaired, hence there is reduced oxygen supplies to the brain. So ventilation and oxygenation is an integral part of the management of patients with TBI. Oxygenation should be maintained such that SpO2 should stand above 95%. However, unnecessary hyperoxia should be avoided. Capnography is necessary for patients on mechanical ventilation. Normocapnia should be maintained at etCO2 should be within 31-35mmHg, PaCO2 at 35-40 mmHg. Peep should be at 3-6 cmH2O, as it raises ICP, this level helps to reduce lung collapse /atelectasis in the lungs without increasing ICP, Pmax = up to 30cm H2O, set tidal volume at 6-8 mI/kg. FiO2should be the minimum required to maintain SpO2 at 95% and above. PaO2 is preferably maintained at 150-200mmkg.

6. The patient should be well synchronized with the ventilator, this may not be achieved unless sedation is applied, sedation is very important to maintain patient in the ventilator, for relaxants, a non-depolarizing muscle relaxant should be used. We must avoid prophylactic hyperventilation of patients (PaCo2 $\leq$ 25mmHg), other methods of measuring cerebral blood flow, oxygenation and metabolism can be useful. Monitoring of blood in the jugular vein and partial oxygen pressure in the brain tissue are important, their values should for jugular vein < 50% and in brain tissue < 15mmHg.

7. Sedative drug serve the purpose of stabilizing patients in the ventilator, hence is needed mostly in cases of patient agitation and adaptation to the mechanical ventilation. There are many sedative drugs in use today, diazepam, thiopental, propofol in their smallest doses that can achieved the desired goal should be used. It is important to understand that over suppression of patients is not recommended, the prophylactic use of barbiturates up to the EEG isoelectric level what is termed burst suppression is not recommended. High doses of barbiturates are only

recommended when controlling increased ICP, that is refractory to medical / surgical treatment. Barbiturates can only be administered only be administered when the hemodynamic parameter is stable. In prolonged mechanical ventilation propofol is highly beneficial in controlling ICP in high doses given through an infusion pump. While using propofol attention should be given to the symptoms of propofol infusion syndrome. Drugs that increase cerebral seizure activity and ICP should avoided - ketamine and GABA drugs.

8. In cases of cerebral oedema that does not respond to drugs then in that circumstance the use of hyperventilation is permitted. The sedatives and analgesics recommended for optimal result are midazolam given at the doses of 2mg as a test dose then 2-4mg/hour in a continuous IV infusion. Fentanyl 2mcg/kg test dose, then 2-5mcg/kg/hour in continuous IV infusion, sufentanil 10-30mcg test dose, 0.05-2 mcg/kg continuous infusion. Propofol is a good potent sedative drugs that can be used for management of TBI, 0.5mg/kg test bolus then 20-75mcg/kg/min continuous infusion, do not exceed 5mg/kg/hour. Pentobarbital loading dose of 10mg/kg over 30min, then 5mg/kg every hour for 3 doses then maintenance dose 1mcg/kg/hr. sodium thiopental 2-4mg/kg IV bolus the continuous infusion or titrated to 0.5-3mg/kg/hr.

9. Seizures might occur in TBI cases, anticonvulsants recommended for usage to stop seizures are, if they are available lorazepam, phenytoin, valproate. Approximately 50% of patients with TBI experience early seizures within the first week. Preventive use of anticonvulsant drugs is reasonable if the following risk factors of seizures are present in the patients. GCS < 10, cortisol contusion, depressed skull fracture, subdural haematoma, epidural haematoma, intracerebral haematoma. Penetrating injury, seizure within the first 24 hrs after admission.

10. Fluid therapy is an integral part of the management of patient with TBI so proper assessment of patient s fluid status should be conducted to note increase in BP, cardiac output, reduce HR. on daily basis careful calculation of fluid requirement should be done and cumulative fluid balance throughout the period of treatment in ICU should monitored. When calculating fluid needs should be considered all intake, infusion solutions, drugs, blood components, parenteral nutrition, enteral intake in form of food, food mixtures, water, solution, endogenous water (4mI/kg/day approximately 300ml for an adult). Fluid loses must be calculated from urine, insensible losses through skin approximately 400mI/day in adults, through the lungs about 400ml/day in adults, if patient is being ventilated with humidified oxygen or through a viral – bacterial filter that returns moisture and heat, no water is loss through the lungs however hyperventilation or mechanical ventilation without humidifier losses 2/3of body weight per hour in ml. in hyperthermia , skin losses is 1/3 of body weight multiply by each degree above 37 degree centigrade multiply by hours in ml.in hypothermia when temperature is below 37 degree centigrade, skin losses accordingly decreases. In terms of pathological diarrhoea, vomiting bleeding drains, cerebrospinal fluid leakage and others.

To determine the required volume, it is necessary to consider the following, physiological needs-25mI/kg in case of excess body weight the ideal body weight should be used (height in cm – 100) in kg, existing deficit, and expected pathological loses. The administration of fluid via routes, the best and safest route is the enteral, which should this should be used if gastrointestinal tract is intact .Fluid therapies in brain pathology, glucose solution are contradicted, they source of free water and can cause brain oedema. The main solution is saline solution, normal saline, Ringers lactate, Hartman's solution, Darrow's solution, sterofudin and others. Electrolyte deficiency must be corrected. In deficiency of major electrolytes (kn-kp) *0.2* body weight = deficiency in mmol. Kn – normal concentration, kp is patient's concentration in mmol/l.1mL of 7.5% of KCl is equivalent to 1Lof 0.9% of NaCl = 154 mmol of sodium and chloride each. Hypernatremia is permissible at the beginning of the treatment. Correction should be done slowly. Hyperkalaemia and hypokalaemia should be corrected immediately, to correct hypokalaemia 7,5% KCl can be administered using a syringe pump. Darrow solution which contains 36 mmol of potassium per litre also can be used. Physiological needs for potassium and sodium is 1 mmol/kg each. Is important to note that in patients with cerebral pathology colloid solution should not be used,

especially albumin and hydro ethyl starch (HES) solution.in cases of extreme necessity for fluid resuscitation, gelatine preparations, FFP, or as required it can be used. Any disorder of theantidiuretic hormone (ADH) secretion will affect the fluid status of the patient both decreased and increased secretion as a result of cerebral pathology. The determination of urine specific gravity and urine sodium level is very important, increase or decrease of urine output maybe associated with the of ADH in the blood.

11. Intracranial pressure (ICP) is very important as it concern management TBI, there is a specific device for measuring ICP, in the measuring there is a measurement method, invasiveness, attract cost, and possibility of correction. On the other hand, ICP can be monitored clinically, with the following signs, level of consciousness, neurological symptoms, fundus examination, also using the CT data. In other to get accurate result, a ventricular catheter Is connected to the external device, it is less expensive and assessable method of ICP monitoring. It has a great advantage that it can be recalibrated while in use. The other method available is using a fibre optic device or instruments with a micro deformation sensor installed in a ventricular catheter offers the same advantages at a higher cost. A parenchyma sensor cannot be re calibrated while monitoring, and with the use of a micro deformation sensor will have a minimal data drift. It is worth to note that data drift does not depend on the duration of monitoring. Other methods are less accurate, the subdural, epidural or subarachnoid sensors with either hydraulic or pneumatic mechanism.

In measuring ICP our goal is ICP should be <20mmHg.

12. Another important aspect in management of severe TBI is the hyperosmolar therapy in correcting increased ICP, mannitol is effective for this control at a dose of 0.25g/kg to 1g/kg. Precautions should be taken while using mannitol, when systolic BP is less than 90 mmHg or plasma osmolality is above 320mmol/l. the use of mannitol is better in severe cases of trans tentorial herniation or progressive worsening of neurological condition that is not limited to extracranial causes. To reduce ICP a hypertonic solution of NaCl 7,5%- 3ml/kg over 20-30 minutes' daily will be useful. This have an advantage over mannitol, its effect is longer up to a day while the mannitol last 4-6 hours, it can also be used in hypovolemia while mannitol cannot. Other advantages are the rebound phenomenon that occurs with mannitol is not seen, also in term of plasma osmolality the range of hypertonic solution is wider up to 360mmol/L as against mannitol of 320mmol/L. One caution in managing ICP is without the monitoring the use of hyperosmolar therapy should be limited to 1 to 2 days, and plasma concentration of NaCl should be maintained at 144-155mmol/L.

13. Antibiotic stewardship is another aspect that require proper management, antibacterial prophylaxis should be conducted according to the microbial profile of the department. The most common sources of infection for these patients are catheter associated blood stream infections, urinary tract infections, ventilator associated pneumonia, pressure sores and bacterial complications from ICP monitoring devices.

14. Venous thrombosis: To prevent deep venous thrombosis is done using compression stocking routinely and administration of anticoagulant, heparin prophylaxis or clexane, if there is no contraindications 5000 unit of heparin is given 6hly, on the first day and subsequently transition to a low molecular weight heparin under INR control.

15. Management and prevention of stress ulcerin the gastrointestinal tract is best done using enteral feeding, if feeding is delayed then drugs should be prescribed H2 histamine blockers and others. Any patient that has ulcer as a comorbidity then this drug must be given with feeding.

16. Cerebral protectors have a lot of controversy surrounding it so the use is limited.

17. Blood pressure control: lowering blood pressure below 90mmHg is not acceptable, autoregulation of blood flowing to the brain is impaired, cerebral perfusion depends solely on mean arterial pressure: CPP= MAP-ICP. The MAP level should be maintained at 100mmHG. Arterial hypertension with MAP at 160mmHg, even up to 200mmHg in the first days of treatment,

may not need to be correction, as this may be a compensatory mechanism to increased ICP. Note that if ICP is monitored it should not exceed 20mmHg, and target CPP at 50-70mmHg. ICP may be difficult to manage with IV infusion so use vasopressors, noradrenaline, adrenaline, avoid dopamine as it can cause oedema. In controlling BP magnesium sulphate, beta blockers are used. Hypomagnesemia occurs in 40-60%of patients, the normal blood magnesium level is 0.75-1.0 mmol/L magnesium is widely used to control BP, is natural NMDA receptor antagonist, helps to eliminate vasospasm, contributes to reduction of secondary brain injuries, and has sedative and anticonvulsant effects. Is given in a high doses of 2-4 mmol/L due its poor penetration through the brain blood barrier. This can cause intermittent hypotension. There are contraindication to magnesium, hypovolemia, blood pressure < 100mmHg, renal impairment urine output. <40mi/hr. the dose of magnesium sulphate is 20ml and given 25%(5g) intravenously over 20 to 30 minutes, then intravenously given at the rate of 1-2,5 grams per hour. The rate is then adjusted to suit hemodynamic parameters, if the decrease in BP is not more than 10-15%of base line value, then increase rate to 2-2.5g/hr. if the BP decreased by >15%, then reduce the infusion rate. The infusion duration is 3 days. It is advisable to maintain plasma magnesium concentration at 2-4 mmol/L. if there is a sign of over dose (suppression of knee reflex, bradycardia, decreased blood pressure), stop the infusion, in severe cases the antidote is 10% calcium chloride administered slowly IV 10,0 ml.

18. Antioxidants can be used tocopherol acetate Vitamin E, given 30% -2.0-4.0 mi IM daily for 7 -10 days.

19. Steroids, corticosteroids are not recommended for treatment of TB.

20. Hyperthermia is another aspect that is important to manage, if temperature is greater than 37.5-degree cent, give drugs to control it. Paracetamol IV or IM. Indomethacin slightly reduces ICP, it has advantages over other NSAID, is given 50mgthree times daily. Note if this drugs help it means is caused by infection but if does not or just slightly it means is of central origin, use cooling methods to control the hyperthermia. Wet wipes, air stream blowing, applying ice packs, to extremities. During physical cooling deepsedation with diazepam or other drugs should be applied.

21. Blood sugar / Glycaemia control: blood sugar should be maintained at 4.0- 10.0mmol/l. in case of hyperglycaemia administer insulin using a pump, insulin infusion pump at 2unit/hr. blood glucose should be monitored 2hrly in the first day, if there is any tendency of <4mmol/L hypoglycaemia then administer glucose IV. After stabilizing the patient blood should be monitored every 4 to 6hrs. Glycaemia control should continue until patient is out of critical period.

22. Complication and possible side effects: there could be deterioration of neurological conditions resulting from intracranial complications. Secondary neuron damage, haemorrhagic complications, infection complications, and intracranial hypertension. Most organs ranging from kidney, lungs, liver, catheter-associated bloodstream infections, urinary tract infections VAP, pressure sores/ulcers, microbial infections resulting from measuring ICP, and stress induced ulcers and gastrointestinal bleeding.

23. Characteristics of the expected final treatment outcome, is expected that there will be total regression of all neurological symptoms, restoration of consciousness and vital organ functions.

24. Recommendations: prevent infections and its complications, prevent deep vein thrombosis, rehabilitation.

25. Criteria to discharge: Complete restoration of tissue perfusion, function of vital organs, consciousness, hemodynamic stable., adequate urine output, adequate independent breathing, gas exchange in lungs, blood clothing system.

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