Surgical Management of Severe Closed Head Trauma

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Abstract: Objective: review the pathophysiology of the severe closed head trauma and to review the recent guidelines in its surgical management and following the prognosis. **Patient and Methods:** This study was an analytical study conducted on 20 patients with traumatic skull bone defects admitted to the neurosurgery department in Al-Azhar Damietta University Hospital undergoing cranioplasty if indicated and clinical & radiological follow up was done after a period of 6 months. **Results:** The age range was 11-55 years, mean age was 30.05 years, male-to-female ratio was 2.75:1. The causative trauma responsible for the head injury in our series were road traffic accident (50%), direct trauma to the head (23.3%) and fall from height (FFH) (26.7%) of all RTA was the most common cause in 50%. Time elapsed to reach hospital within 24 hours from trauma divided either before or after 6 hours which was 24 reach before 6hoursresulting in surgical treatment morbidity and Mortality 29.16% and 6 cases reach after 6 hours with morbidity and mortality 50%. According to Intracranial pathology (EDH, SDH, ICH) the EDH 19case were operated with morbidity and mortality 15.6%, SDH 8 cases were operated with morbidity and mortality 75 %, ICH3 cases were operated with 66.6% morbidity and mortality. **Conclusion:** Head trauma is considered as a major health problem that is a frequent cause of death and disability. Patient's age, conscious level on admission and primary brain pathology markedly determine the outcome of closed head injury patients.
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1. Introduction:

Traumatic brain injury (TBI) is a leading cause of death and disability around the world ***(CDC, 2015).***

Severe traumatic brain injury (TBI), defined as head trauma associated with a Glasgow Coma Scale (GCS) score of 3 to 8 ***(Teasdale et al., 1974).***

TBI results from mechanical forces such as an object striking the head, or from rapid acceleration and deceleration forces that cause vigorous movement, and thereby tissue deformation, of brain tissue within the skull. These forces produce a primary injury that directly affects neurons, blood vessels and glia, and initiates a plethora of secondary processes that result in complex cellular, inflammatory, neurochemical and metabolic alterations ***(McAllister, 2011).***

These secondary changes develop within hours to weeks after the primary injury and lead to a constellation of events that include axonal injury, impaired cerebral blood flow, metabolic changes, edema, raised intracranial pressure (ICP), increased blood-brain barrier (BBB) permeability, calcium influx, elevated oxidative stress, free-radical-mediated damage, excitatory neurotransmitter release, inflammation and cell death ***(Prins et al., 2013).***

2. Patients and Methods

This study was concerned on patients attend to emergency room (ER) and admitted to the neurosurgery department in Al-Azhar damietta universityhospital. Data collection was extracted from hospital records (Admission ICU books, patient admission sheets, operative details and progressive notes).

Inclusion criteria

1- Closed head trauma
2- Severe head trauma with GCS 8 or less
3- Immediate post admission
4- Severe closed head trauma with indication for surgical intervention

Exclusion criteria

1- Severe closed head trauma with no surgical intervention
2- Brain death.
3- Open head trauma including penetrating trauma
4- Associated spinal injuries
5- Severe associated systemic injuries
6- Other old neurological deficits.

Study Design:

Case series with patients follow up and data collection from records.

(Retrospective).

All 30 patients included in this study were subjected to the following methodological scheme and followed up for 6 months:

Management in emergency department:

1. All cases were examined thoroughly on arrival, with detailed history taken from the relatives,

or the ambulance aid according to his states upon presentation about the type, site and time oftrauma.

2. To maintain the air way and exchange of air, the patient's head was dealt with gently after being certain that there is no cervical injury to facilitate suctioning from the tracheobronchial tree and to avoid aspiration. Insure adequate oxygenation and humidification using face masks, airways or endotracheal tube if necessary. Ventilation was used when indicated.

3. Control of bleeding site from any wounds, fluids or blood replacements startedimmediately.

4. Support for disability (e.g long bonefractures).

5. Full exposure for injured patient to examine hidden area to prevent missedinjury.

6. Examination for any associated injuries and prompt management of those needed urgent treatment, mainly chest injuries and stabilization of associated skeletalfractures.

Neurological Examination:

All patients were examined by the neurosurgeons after stabilization of their general condition, with accurate recording of the following:

1. Glasgow coma score (GCS).
   - Mild head injury (MHI) GCS13-15.
   - Moderate head injury GCS9-12.
   - Severe head injury GCS3-8.
2. Vitalsigns.
3. Integrity of the brain stem reflexeslike:
   - Size, equality and reaction of the pupils using strong light.
   - Cornealreflex.
4. Cranial nervesinjury.
5. Head examinationfor:
   - Scalpinjury.
   - Detection of fissure or depressed skullfractures.
   - Detection of fractured base skull (CSF leak and bleeding from externalorifices).
6. Detection of cervical spine injury and applying neck collar immediately when suspected injury waspresent.
7. Motor function test complete the basicexamination.

Investigations:

Patients were subjected to the following investigations:

1) Laboratoryinvestigations:

- Complete blood count (CBC).
- Other laboratory investigations according to patient's condition:
  - Bleedingprofile.
  - Arterial bloodgases.
  - Serumelectrolytes.
  - Renal functions (urea -creatinine).
  - Liver functions (ALT-AST).
- Blood sampling for typing and crossmatching.

2) Imaging:

- Cranial computed tomography (CT):

All patients of head injury were investigated by CT brain without contrast and with bone window after stabilization of the general condition of the patient, e.g correction of any preexisting hemodynamic instability.

- Plain X-rayfilms:

1) Cervicalspine
2) L.S.S X-ray
3) Chest
4) Pelvis
5) Other films according to patient'scondition.

- Focused abdominal sonography for trauma (FAST)

Decision making:

- Conservative ttt with ICU admission
- Surgical management
- Conservative turns into surgical

Surgery:

- Patients were subjected to surgical evacuation of intracranial hematoma or elevation of depressed skull fractures
- Intra – operative difficulties and complications are reported
- Postoperative assessment

1- Clinically focusing on postoperative occurrence of any complications or neurological deficit

2- Radiological: CT Brain

Outcome:

The outcome of patients was categorized according to the Glasgow outcome scale with the sex exclusive categories.

1. Good recovery:

Patients whom returned to their former occupation, though not necessarily their former occupational level.

2. Mild disability:

Patients with mild neurological or mental deficit that not affect their occupational level.

3. Moderate disability:

Implies independence as far as activities of daily life were concerned, but inability to resume former occupational level owing to mental disability, physical disability or both.

4. Severe disability:

Applies to patients dependent on others for the activities of daily life.

5. Persistent vegetative state:

Defined as absence of adaptation to the environment and of any speech or evidence of mental function in a patient apparently awake and at times displaying spontaneous eye opening.

6. Death:

Including within the death category, patients dying directly from brain injury and those dying from secondary systemic complications.

Management:

30 patients with initial GCS 8 or less were operated

Indications

A: Acute subdural hematoma

1- Thickness greater than 10mm

2- Midline shift greater than 5mm on CT

- Patients with less than a 10mm thick lesion or less than 5mm of midline shift

3- A decrease of GCS by 2 or more points,

4- ICP is greater than 20mmHg,

5- Asymmetric or fixed and dilated pupils

B: Acute epidural hematoma

1- Hematoma larger than 30cc, more than 15mm thick ormore than 5mm of midline shift was evacuated regardless of the patient's GCS score.

2- Expeditious hematoma evacuation was done for comatose patients (GCS less than 9) and/or with anisocoria.

C: Focal traumatic parenchymal lesions

1- Progressive neurological deterioration referable to the lesion

2- Medically refractory intracranial hypertension

3- Signs of mass effect on CT

4- Patients with any lesion greater than 50 cm^3 in size

5- Patients with GCS 6-8 with frontal or temporal contusions greater than 20cm^3 in volume with at least 5mm of midline shift and/or cisternal compression on CT scan.

Surgical techniques

A: Acute subdural hematoma

I. Skin Incision.

The hair was generously removed with an electric clipper for a wide exposure of the surgical site. The surgical incision was planned to account for any scalp lacerations to avoid areas of devascularization. If aggressive debridement of complex lacerations was necessary, then a wider incision was made to maintain the option of mobilizing additional tissue for tension-free closure.

Critical structures was marked on the surface anatomy. The superior sagittal sinus is outlined in the midline from the nasion to the external occipital protuberance. The zygomatic arch is palpated and marked. A line connecting the root of zygoma to the inion estimates the level of the transverse sinus. The area between these venous landmarks represents bone that is safe for removal. The standard trauma incision begins at the zygomatic arch, approximately 1 cm anterior to the tragus. The superficial temporal artery crosses the zygomatic arch just in front of the ear was preserved. The incision extends cranially to the level of the top of the pinna thenturns posteriorly and travels medially just above the level of the transverse sinus. At the external occipital protuberance, the incision turns anteriorly, sweeping over the parietal and frontal regions and staying approximately 2 cm lateral to the midline to end at the hairline. To preserve adequate vascular supply, the length of the scalp flap didn't not exceed the width. The temporalis fascia was cut and the temporalis muscle was elevated along with the scalp off calvarium and reflected anteriorly as a single myocutaneous flap.

II. *Bone Flap*

A large 12 × 15-cm frontotemporoparietal bone flap was planned to achieve wide exposure and adequate decompressionshould the bone flap was needed to be left off. Bur holes were placed at the keyhole in the frontal bone behind the zygomatic arch, adjacent to the root of the zygoma and over the parietal bone at the most posterior extent of the planned bone flap. The keyhole approximates the floor of the anterior fossa, and the root of the zygoma approximates the floor of the middle fossa. In elderly patients, the dura may adhere tightly to the endocranial surface of the bone flap. Additional bur holes could have been placed to help with dissection and to avoid violation of the dura during craniotomy. The bur holes are connected epidurally and the bone flap is elevated. The medial cut of the craniotomy, which parallels the superior sagittal sinus, should be 2 to 3 cm lateral to the midline. This provided an adequate view of the bridging veins while avoiding the lacunae contained in the dura adjoining the superior sagittal sinus. Additional bone from the lesser sphenoid wing and the squamosal temporal bone was resected down to the cranial base. Excessive force was avoided during bone removal to avoid extending fractures of the skull base and inciting vascular injuries. The rongeurs was used to cut rather than to break off pieces of bone.

III. Dural Opening.

The dura was opened in a controlled fashion to avoid a sudden plunge in the ICP and to avoid paren-chymal laceration. The dura was opened over the thickest portion of the underlying hematoma, or else over the anterior frontotemporal region. The dura was opened in a stellate fashion with relief cuts to the edge of the skull opening to ensure no restrictions on the underlying swollen brain.

IV. Hematoma Evacuation.

Once the dura was widely open, the intradural space and the parenchymal surface were explored. Subdural hematoma was evacuated with gentle suction and irrigation under direct visualization. Torn bridging veins or pial vessels overlying contused brain were identified as bleeding sources and controlled with electrocautery or hemostatic agents. If a tear in the dural sinus was suspected as the bleeding culprit, the anesthesia team were notified immediately. Vital signs and respiratory parameters were monitored for evidence of air embolism, such as hypotension and decreases in end-tidal carbon dioxide and arterial oxygenation saturation. Additional blood products were made available while the hemorrhage is controlled. An intradural venous sinus tear required either direct repair or patching with a temporalis fascia and muscle graft. If the sinus tear was superficial with epidural bleeding beneath the bone edge, thenthe hemorrhage was controlled by placement of multiple closely spaced tack-up sutures to pull the dura tightly against bone.

V. Closure.

The decision regarding whether to replace the bone flap depended on intraoperative assessment of cerebral swelling, the extent of associated intracranial injuries (both ipsilateral and contralateral to the operative site, as seen on preoperative imaging), and the potential for intracranial hypertension in the postoperative period. If the bone was not replaced, the dural leaflets were loosely reapproximated and covered with an onlay dura substitute, such as DuraGen (Integra LifeSciences, Plainsboro, NJ). Gelfilm (Pharmacia and Upjohn Co., Kalamazoo, MI). The wound is closed in layers over subgaleal drains.

B: Acute epidural hematoma

I. Positioning

Patients undergoing a frontotemporoparietal craniotomy or craniectomy were positioned supine on the operating table. We elevated the head 15 to 20 degrees to promote venous drainage and is turned until the operative side faces upward with superior sagittal sinus 0 to 15 degrees relative to the floor. Spinal precautions was maintained as appropriate, given the co-occurrence of TBI and spinal injury, or were positioned laterally to allow access to the temporal region. Or, positioning was sometimes dictated by the location of the EDH seen on imaging studies.

II. Craniotomy & evacuation of hematoma

A large frontotemporoparietal craniotomy provides the best access for surgical management of EDH, just as was for SDH. But, with improved preoperative localization by CT and earlier detection of smaller EDHs, it was possible to perform a more targeted craniotomy through a limited "slash" incision for evacuation of EDHs. Using the preoperative CT scan as a guide, the incision was planned over the epicenter of the EDH to provide complete exposure of the hematoma. The slash incision, which typically runs vertically, was fashioned so that it can be extended to the larger "trauma flap" if necessary (e.g., if intradural bleeding were to develop with reperfusion of the brain after removal of the EDH). If neurological deterioration was rapid or herniation is present, an initial bur hole was placed over the thickest part of the clot as seen on CT, and the clot is removed to reduce ICP.

After skin incision and muscle-splitting exposure as explained before, the periosteum was stripped to expose the cranium fully in the region of the hematoma. A circular or ellipsoid craniotomy was fashioned using one or multiple bur holes, depending on the size or location of the clot. Correct placement of the craniotomy is crucial to occlude the epidural space optimally and to visualize the bleeding points on the dura, usually the middle meningeal artery. The bleeding source, typically a branch of the middle meningeal artery, was generally controlled with bipolar cautery. Occasionally, the main trunk of the middle meningeal artery was torn because of a fracture involving the petrous bone. In these cases, adequate low temporal exposure was necessary to visualize the foramen spinosum, which was packed with bone wax to control the bleeding. After evacuation of the hematoma, meticulous hemostasis was obtained and bone wax was applied to the bone edges. Dural tack-up sutures were placed at intervals no larger than 2.5 cm in a circumferential fashion with 4-0 silk and the use of bone drill holes. Central dural tack-up sutures were also placed in the center of the bone flap to obliterate the central dead space under the bone flap. The bone flap was then replaced, and the wound is closed in layers over a subgaleal drain.

Rarely, intradural "reperfusion" hematomas developed rapidly after the removal of an extradural hematoma. After evacuation of the extradural hematoma, if the underlying dura becomes tense, a limited opening was made in the dura, and any hematoma was removed with gentle suction and irrigation. A larger dural opening was made rapidly, if there is persistent subdural bleeding, to visualize and to control the bleeding source.

C: Focal traumatic parenchymal lesions

I. Positioning & Skin Incision.

Was dictated by the location of the IPH seen on imaging studies. Because most traumatic IPHs occured in the temporal, subfrontal and frontal regions, most commonly, the patient position was similar to that described previously for craniotomy for frontotemporoparietal procedures. For a bifrontal craniotomy, the patient is placed supine on the operating table with the head resting on a foam pad or horseshoe Mayfield headrest. A bicoronal incision was marked out behind the hairline, starting low at the

level of the zygoma. Following the skin and galeal opening, the superficial temporal fascia and the temporalis muscle are incised, care was taken to avoid possible injury to the facial nerve by remaining above the zygoma at the inferior end of the incision. A myocutaneous flap was reflected as low as possible anterior to the supraorbital margins and the sphenoid wing in the temporal region. The pericranium is preserved and harvested as a separate flap.

II. Bone flap

Multiple bur holes were placed in the temporal and frontal regions, and two were placed parasagittally (approximately 1.5 cm apart) on either side of the sagittal sinus near the plane of the coronal suture. A Kerrison punch and a curved curet were then used to enlarge the bur holes and to undermine the inferior aspect to allow a Penfield No. 3 dissector to separate the dura from the inner aspect of the skull. The dura was gently and carefully stripped over the midline sagittal sinus. A cutting craniotome blade was used to perform the craniotomy cuts, keeping as low as possible in the frontal and temporal areas. The last cut of the craniotomy was the one carried over the midline sagittal sinus region to have immediate access to the sinus should bleeding be encountered. Alternatively, the craniotomy was elevated in two pieces to avoid injury to the sagittal sinus. The first piece of the craniotomy extends just lateral to the sagittal sinus and does not cross midline. The dura over the sagittal sinus is dissected away from the inner table along the bony edge under direct visualization. After the midline dura has been freed along the entire length of the craniotomy, the second piece could be elevated safely. The frontal air sinus would be exenterated, and its ostia was plugged afterward. Bone wax was applied to the bone margins, and strips of Surgicel (Ethicon, Somerville, NJ) or Gelfoam (Pfizer, New York, NY) were placed around the craniotomy margins and over any small bleeding points from the sagittal sinus. Dural tack-up, 4-0 silk sutures were placed around the margins of the craniotomy site.

III. Dural opening

Bilateral dural openings were created with the flaps based toward the sagittal sinus. Care was taken during the dural opening to protect the underlying brain tissue and parasagittal draining veins with cottonoid pads. If bifrontal contusions or SDH were present or if frontobasal access is required, the most proximal superior sagittal sinus was double-ligated and cut as close to the crista galli as possible, where it is usually small.

IV. Hematoma evacuation

Using bipolar cautery, the pia mater and superficial vessels were cauterized over the cortical area with the most damage, swelling, and contusion. The pia mater was opened with a No. 11 blade, and a subpial plane was developed, if possible, to minimize bleeding. With gentle suction and bipolar cautery, the contused and hemorrhagic areas were removed. Gentle retraction of the surrounding brain tissue adjacent to the cavity was performed with a handheld malleable retractor. If significant brain swelling was present, partial resection of the right frontal or temporal lobe was performed to minimize the pressure effects of postoperative brain swelling. But, no more than 3 to 4 cm of the frontal lobe and 5 to 6 cm of the right temporal lobe was removed. On the left side, only contused, amorphous tissue was removed. The areas of the frontal lobe most prone to hemorrhagic contusions were the inferior orbital aspects of the gyrus rectus and inferior frontal gyrus. In the temporal regions, the anterior tips of the temporal lobes were most susceptible to injury. After adequate decompression, hemostasis was achieved in the surgical bed with Surgicel or gentle tamponade can be maintained with cotton balls soaked in thrombin or half-strength hydrogen peroxide, which is an effective hemostatic adjunct.

V. Closure

The dura was then closed in watertight fashion with 4-0 silk sutures, especially over the subfrontal region behind the frontal air sinus. The previously harvested pericranium flap was placed over the frontal sinus and beneath the bone flap. If there was significant brain swelling, the dura was left open and the bone flap wasalso left out.

Pre and Post-operative **Intensive Care Unit (ICU)** admissionwhere a standard protocol for treatment were applied as follows while the patient in a 30 degree head up position.

1. **Intubation** and **hyperventilation** (if the patient was not intubatedin emergencydepartment.
2. **Bronchial suction** was performed as necessary, once anhour.
3. **Mechanical ventilation** was used to keep PCO2 between 27 and 35 mm Hg using assisted control mode, ABG was done half an hour after starting ventilation was readjusted accordingly and the samples were withdrawn every 12hours.
4. **Reassessment** was done as to the followingguides:

- Glasgow ComaScale. - Pupillary sizes andreactivity. Neurologicaldeficit. - Eyemovement. - Vitalsigns.

5. Insertion of a **central venous line** under strict aseptic technique for infusion of fluids and medications which was done during the first 6 hours of admission, then after stabilization of the patient, C.V.P. was measured every 6 hours for adjustment of the I.V fluidsbalance.
6. **Foley's catheter** was inserted and urine output was recorded hourly and every 24 hours. A

Foley catheter isplaced for urinary bladder drainage, for evaluation of adequate urine output, and to accommodateincreasedurinary volume should *mannitol* be required. Urine issampled for 24 urine anlysis and drug screening.

7. Insertion of **nasogastric tube** (**NG**) for passive drainage of the stomach contents in the early days of management. nasogasteric intubation done to decompress the stomach, prevent aspiration, and detect any gastrointestinal hemorrhage and later on forfeeding.

8. Continuous **monitoring** of blood pressure, pulse, respiration and temperature were done. The results were recorded every hour to detect any suddenchanges.

9. **Venous blood samples** were taken for CBC and repeated on daily basis. Serum electrolytes, blood sugar, urea and creatinine were also checked.

10. Plain **chest x-ray** for assessment of the endotracheal tube position and the central venous line. It also helps to follow the chest conditions, early detection and treatment of any complications such as chest infection.

11. **Osmotic agents and diuretics** were used in those cases with persistent intracranial hypertension in presence of hyperventilation. *"Mannitol"* 20% was administered ina dose of 0.5-1gm/kg/day which would be given every 8-12 hours Loop diuretics especially "*furosemide"* was employed in a dose 0.3-0.5 mg/kg to potentiate the effect of"mannitol".

12. **Antiepileptic** treatment in the patients with history of post-traumatic fits or for prophylaxis patients more susceptible to develop fits in patients (e.g cortical lesions and operatedpatient).

a."*Phenytoin"*, as a loading dose (15-20 mg/kg) and a maintenance dose (5-10 mg/kg) was givenslowlyI.V (1 ml/minute) to avoid cardiovascular depressant effect.

b. "*Diazepam*", continuous I.V infusion at a rate (0.1-0.2 mg/kg/hour or as needed) in cases of *"status epilepticus"* In combination with "*phenytoin"*.

c. *Other" antiepileptic"* drugs were given ifneeded.

13. **Serial follow-up CT brain** were done starting from 24 hours following admission if not earlier, in patients who do not show improvement or deteriorate in neurological status, follow up CT in patient with hematoma was done after 6, 12, 24 and 48 hours to detect any increase in the hematoma size or developing of masseffect.

14. **Weaning ventilator** in patients was the transition from artificial to spontaneous ventilationthatwould be decided by both the neurosurgeon and anesthetist. Generally it started from the fifth day of admission unless the patient's clinical condition didn't allowit.

15. According to the degree of recovery, patients were scheduled to attend the neurosurgery outpatient clinic for the followup.

3. Results:

The study included 30 patients (8 females and 22 males) diagnosed as severe closed TBI.

Preoperatively all the patients were evaluated using a standardized sheet and the findings were tabulated as preoperative clinical (subjective and objective) and radiological data.

1. Frequency of causes of head injury (table 1):

Causes	No. of cases	%
Road traffic accident	15	50.0%
Direct Trauma	7	23.3%
Falling from high	8	26.7%
Total	30	100.0%

The causative trauma responsible for the head injury in our serious were road traffic accident (50%), direct trauma to the head (23.3%) and fall from height (FFH) (27.7%) of all Causes of head injuries.

2. Age and sex distribution of the head injury (table 2):

The youngest patient included in this serious was 4 years, the oldest patient was 57 years with mean age 30.97 and standard deviation ± 24.04, and males were more commonly involved, male to female ratio 2.75:1

Age (years)	Total		Male		Female	
	No.	%	No.	%	No.	%
Up to 10	2	6.67%	1	3.33%	1	3.3%
<10-20	5	16.67%	4	13.33%	1	3.33%
<20-30	8	26.67%	6	20%	2	6.67%
<30-40	8	26.67%	6	20%	2	6.67%
<40-50	4	13.33%	3	10%	1	3.33%
<50-60	3	10%	2	6.67%	1	3.33%
Total	30	100%	22	73.33%	8	26.67%

Morbidity and mortality according to time to reach hospital (table 3)

	Time to reach hospital		Morbidity and mortality	
	No.	%	No.	%
First 6 hours	24	80%	7	29.16%
6-12 hours	4	13.4%	2	50%
12-24 hours	2	6.6%	2	100%

The time elapsed between trauma and hospital admission ranged from 1 to 48 hours, 24 patients were admitted in the first 6 hours after trauma, 4 patients were primarily admitted to our hospital within 6 to 12 hours after trauma while 2 patients were admitted to general hospital then transferred to our hospital from 12 to 24 hours after trauma.

Prehospital care (table 4):

For evaluation of prehospital care patients were divided into 2 groups (both received medical care at hospital) but one did not provide emergency medical services at the field by paramedics and was delivered to the hospital by their relatives.

	Total no.	survival	dead	Percentage of dead pt.
With prehospital Care	11	*10*	*1*	9.2%
Without prehospitalcare	19	15	4	21%

Morbidity and Mortality outcome of surgical management according to intracranial pathology (table 5):

The outcome of patients was categorized according to the Glasgow outcome scale with the six exclusive categories.

Outcome	Number of cases total and according to pathology							
	Total		AEDH		ASDH		IPH	
	No.	%	No	%	No.	%	N	%
Good recovery	19	63.7%	16	84.2%	2	25%	1	33.33%
Mild disability	1	3.33%	1	5.3%	0	0.0%	0	0.0%
Moderate disability	1	3.33%	0	0.0%	0	0.0%	1	33.33%
Severe disability	2	6.67%	0	0.0%	2	25%	0	0%
Persistent vegetative state	2	6.67%	0	0.0%	1	12.5%	1	33.33%
Death	5	16.7%	2	10.5%	3	37.5%	0	0.0%
Total	30	100%	19	100%	8	100%	3	100%

Morbidity and Mortality in operated cases related to age and sex (table 6):

		Total		Outcome			
				Good recovery		Morbidity & mortality	
		No.	%	No.	%	No.	%
Age	**Up to 10**	2	6.6%	2	100%	0	0.0%
	> 10-20	5	15.5%	4	80%	1	20%
	> 20-30	8	26.4%	4	50%	4	50%
	> 30-40	8	26.4%	5	62.5%	3	37.5%
	> 40-50	4	13.2%	2	50%	2	50%
	> 50-60	3	9.9%	2	66.6%	1	33.4%
Sex	**Male**	22	73.6%	14	63.6%	8	36.4%
	Female	8	26.4%	5	62.5%	3	37.5%
Total		30	100%	19	63.33%	11	36.67%

Morbidity and Mortality in operated AEDH cases related to age and sex (table 7):

		Total		Outcome			
				Good recovery		Morbidity & mortality	
		No.	%	No.	%	No.	%
Age	**Up to 10**	2	10.5%	2	12.5%	0	0.0%
	> 10-20	2	10.5%	2	12.5%	0	0.0%
	> 20-30	6	31.6%	5	31.25%	1	33.33%
	> 30-40	4	21%	4	25%	0	0.0.%
	> 40-50	2	10.5%	1	6.25%	1	33.33%
	> 50-60	3	15.9%	2	12.5%	1	33.33%
Sex	**Male**	14	73.6%	11	78.6%	3	21.4%
	Female	5	26.4%	5	100%	0	0.0%
Total		19	100%	16	84.21%	3	15.79%

Morbidity and Mortality in operated IPH cases related to age and sex (table 8):

		Total		Outcome			
				Good recovery		Morbidity & mortality	
		No.	%	No.	%	No.	%
Age	Up to 10	0	0.0%	0	0.0%	0	0.0%
	> 10-20	1	33.33%	0	0.0%	1	33.33%
	> 20-30	0	0.0%	0	0.0%	0	0.0%
	> 30-40	1	33.33%	1	33.33%	0	0.0%
	> 40-50	1	33.33%	0	0.0%	1	33.33%
	> 50-60	0	0.0%	0	0.0%	0	0.0%
Sex	Male	2	66.67%	1	50%	1	50%
	Female	1	33.33%	0	0.0%	1	100%
Total		3	100%	1	33.33%	2	66.67%

Morbidity and Mortality in operated ASDH cases related to age and sex (table 9):

		Total		Outcome			
				Good recovery		Morbidity & mortality	
		No.	%	No.	%	No.	%
Age	Up to 10	0	0.0%	0	0.0%	0	0.0%
	> 10-20	2	25%	2	100%	0	0.0%
	> 20-30	2	25%	0	0.0%	2	100%
	> 30-40	3	37,5%	0	0.0%	3	100%
	> 40-50	1	12.5%	0	0.0%	1	100%
	> 50-60	0	0.0%	0	0.0%	0	0.0%
Sex	Male	6	75%	2	33.33%	4	66.67%
	Female	2	25%	0	0.0%	2	100%
Total		8	100%	2	25%	6	75%

4. Discussion:

Our study of patients attend casualty unit and admitted to neurosurgery department. The 30patients with different types of closed head injury that received surgical management.

Head trauma represents one of the causes of significant morbidity and permanent disability in the adult population. Emergency room traumatic brain injury (TBI) admissions include a spectrumthat goes from concussions to significant intra-axial and extra-axial cerebral hematomas ***(Bullock et al.,2006).***

In our study number of patient treated **surgically** are 30and the morbidity and mortality of surgical treatment is36.3%. this study in accordance with **(Darryl Lau et al., 2012).** He take 103 patient operated and overall morbidity and mortality35.2%**.**

In our study number of **EDH** cases 19cases treated surgically resulting in morbidity and mortality 15.8%. This is not significantly differ from **(Kuday et al., 2004)**. that he take115 cases of extradural hematoma **(EDH)** 17cases of them operated results in morbidity and mortality 23%.

In our study the number of **SDH,** 8 patients were treated surgically and resulting in morbidity and mortality is 75% with mortality up to 37.5%. This study not significantly differ from ***(Bernard et al., 2004).*** The subdural hematoma **(SDH)** of a patient with brain injury, and its poor prognosis without or with treatment, reflects that association. The overall mortality rate of patients with surgical treated SDH is roughly 50%.

The mortality rate of acute subdural hematoma (ASDH) remains high despite application of advanced surgical methods, medical treatments, and technologies. Recent studies have reported that the overall rate of favorable outcomes (good recovery and moderate disability) only reaches 19–27 %. Decompressive craniectomy has been recommended as an effective strategy to manage ASDH, but its associated risk of secondary complications adversely affect outcome. ***(Shen J et al., 2013).***

Studies from United States and Europe demonstrated that traumatic ASDH has an important role on the mortality under the age of 45. **Ryan CG et al., 2012.** reported that 63% of traumatic ASDH patients were male and the mean age of the study population was 58 years. **Yanagawa Y et al., 2012**. reported that 67% of the patients of traumatic ASDHs were male and the mean age was 43 years. **Li LM et al., 2012.** reported a male percentage of 60% with a

mean age of 51 years. **Shen J et al., 2013** reported that the majority of the affected patients were male and the patient population had a mean age of 36 years. **Okten AI et al., 2006.** studied a traumatic ASDH patient population with a mean age of 39 years and they observed that 76% of the patients were male. ***(Fatih A et al., 2017).*** In our study, 75% of our patients with ASDH were male and their meanage was 30.8 years.

Outcome of Our study of patients with (brain **contusions** and **ICH**) encountered in this work, 3patients who were treated surgically and morbidity and mortality outcome of surgically treated patients are 66.6%. This study significantly similar to ***(McClellan DR et al., 2009).*** however mortality rates have been greater than 50% for the patients with head injury who develop posttraumatic intracerebral hematomas. Likewise other result significantly differ from outcome of patient with post traumatic intracerebral hematoma **(ICH)** depends upon multiple factors: size and location of the hematoma, presence of other intracranial lesion, and neurological condition of the patient upon admission. Overall, mortality rates have been in the 25% to 30% range. ***(McClellan DR et al., 2009).***

Outcome from cerebral **contusions** varies widely, large and multiple contusions are associated with a significance different prognosis than small isolated ones. In addition, the presence of other intracranial mass lesions, as well as the severity of the primary injury itself, greatly affects the prognosis. Therefore, it's impossible to give a specific morbidity and Mortality figure for cerebral contusions ***(Steinbok et al., 2007).***

A significance intracerebral hematoma **(ICH)** resulting from direct non penetrating head trauma is unusual. However, cerebral **contusions** commonly occur, such contusions will coalesce into a hematoma that reaches a significance size ***(Soloniuk D et al., 2006).***

A cerebral **contusion** consists of small perivascular hemorrhages surrounded by necrotic brain tissue. Typically they involve crests of gyri and can assume wedge-like shape extending through the cortex toward the white matter ***(Rockswold GLet al., 2003).***

In several series of closed head injuries patients studied by CT scan, the occurrence of cerebral **contusions** has varied from 30 to 40% ***(Macpherson et al., 2000).***

Pure cerebral contusions are fairly common. The majority of contusions occur in the frontal and temporal lobes although they can occur at almost any site including the cerebellum and brain stem ***(Sheinberg MA et al., 2004).***

In our finding the time elapsed between trauma and hospital admission **(time to reach hospital)** ranged from 1 hourto 24 hours, patients who were primarily admitted in the first 6hours after trauma are 24, patients who were admitted (primarily or referred cases) to our hospital after 6hours are6. Morbidity and Mortality of patient reach hospital with first6hours29.16 % and after 6hours50 % respectively so the early treatment reduce secondary brain injury. this study in accordance to ***(Luerssen TG et al., 2008).*** The time elapsed between trauma and hospital admission **(time toreach hospital**) is of crucial value as early resuscitation and assessment of vital signs as well as neurological condition in advanced multi trauma center will reduce the secondary brain damage and improve the outcome**.**

In our study number of patient presented with **severe TBI**are 30 cases all of them were operated and the morbidity and mortality of **severe TBI**with surgical treatment is 36.34%. so it highly differ from **(Sandeep Jain et al., 2008)**. That he take 102patient with **severe** TBI68 case operated and mortality was 68.4%.

Inourstudy show Morbidity and Mortality of surgically treated cases of age group less than 20 year 14.2% and 21-40 year 43.75% and for >40 year was 42.8% so the increase age lead to increase morbidity and mortality outcome this is in accordance with **(Wittstatt AW et al., 2017).** the increase mortality in elderly attributed to systemic complications such as pulmonary infection. Also similar to **(Shimoda K et al., 2014).** That he say there is strong correlation exists between age and outcome. In general, increasing age is associated with poorer outcomes in adults although for children the opposite may be true.

In our study the causative trauma responsible for the head injury in our serious were **road traffic accident (RTA)** (50%), **direct trauma** to the head (23.3%) and **fall from height (FFH)** (26.7%) of all RTA was the most common cause in50%. The highest percentage ofallcauses is Road traffic accidents were the most cause of closed head injury this is in accordance with **(Sarani B et al., 2009).** That the RTA most common cause of morbidity and mortality but in contrast by percentage of incidence in United States of America (USA) show the road traffic accident (RTA) (62%), while fall from height (FFH), direct trauma to the head and sports injuries represent (38%). (**Sarani B et al., 2009).**

In our study the Incidence of TBI in **male**73.3% Incidence of TBIin female 26.7% There was a male predominance noted among the studied cases,22 of total number of cases were males, while females were only 8 cases This can be explained as out-door male activities in developing countries more than females. This is In accordance with study of **(Zhang S et al., 2009).** He found incidence rates of brain injury by gender and age shows that males have higher rates

than females at all ages except the very young, where the rates are about the same or only slightly higher formales.

Conclusion:

Traumatic brain injury (TBI), also known as intracranial injury, occurs when an external force traumatically injures the brain. TBI can be classified based on severity, mechanism (closed or penetrating head injury), or other features (e.g., occurring in a specific location or over a widespread area). Head injury usually refers to TBI, but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull.

In our study the morbidity and mortality outcome with respectsurgical management is 36.3 %.

Time elapsed to reach hospital within 24 hours from trauma divided either before or after 6 hours which was 24 reach before 6 hours resulting in surgical treatment morbidity and Mortality 29.16% and 6 cases reach after 6 hour with morbidity and mortality 50%.

Morbidity and Mortality of age group less than 20 years was 14.2% and >20-40 years was 43.75 % and for >40 years was 42.8% so Morbidity and Mortality increased with increased age.

The causative trauma responsible for the head injury in our series were road traffic accident (50%), direct trauma to the head (23.3%) and fall from height (FFH) (26.7%) of all RTA was the most common cause in 50%.

Incidence of TBI in male 73.3% Incidence of TBI in female 26.7% so male predominance is clear.

According to Intracranial pathology (EDH, SDH, ICH) the EDH 19 case were operated with morbidity and mortality 15.6 %, SDH 8 cases wereoperated with morbidity and mortality 75 %, ICH 3 cases were operated with 66.6 % morbidity and mortality.

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