INTRODUCTION

Traumatic brain injury:

Traumatic brain injury (TBI) occurs when there is a blow or jolt to the head due to rapid acceleration or deceleration or a direct impact. It can also be caused by direct penetrating injury of the brain. Brain function is temporarily or permanently impaired and structural damage may or may not be detectable.[1]

Traumatic brain injury, according to the World Health Organization, will impass many diseases as the major cause of death and disability by the year 2020.With an estimated 10 million people affected annually by TBI, the burden of mortality and morbidity that this condition imposes on society, makes TBI a pressing public health and medical problem.[2]

Falls are the leading cause of TBI among all age groups (35.2%), followed by motor vehicle collisions or traffic accidents (17.3%), being struck by/against an object (16.5%), and assaults (10%). However, causes of TBI fatalities are slightly different. As another example, the lethality of gunshots wounds to the head is approximately 90%. Because of this, gunshot wounds are a much higher percentage of TBI fatalities than the overall incidence would suggest. [3]

Clinically, TBI has been traditionally divided into mild, moderate, and severe injury, as judged by the widely used Glasgow Coma Scale(GCS) score which is a simple type of assessment scale used as an aid in the clinical assessment of unconsciousness. It is based on eye opening, verbal response, and motor response. The GCS consists of 15 points, four for eye opening, five for best verbal response, and six for best motor response. A score of 13-15 indicates mild injury, a score of 9-12 indicates moderate injury, and a score of 8 or less indicates severe injury.[4,5] (Table I) For infants and young children, a modified Glasgow Coma Scale for infants and children are used, pediatric GCS.[6] (Table II)

Acute extradural hematoma is one of the known secondary complications of head injuries. It accounts for 2-3% of all head injuries.[7,8]

Extradural hematoma:

Extradural (or epidural) hematoma (EDH): Is a collection of blood between the inner table and the stripped off dural membrane.[9] The incidence of EDH is highest among adolescents and young adults.

Table (I): Glasgow Coma Scale[5]

Area assessed	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Best verbal response	Oriented	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best motor response	Obey commands	6
	Localizes pain	5
	Withdrawal (normal flexion)	4
	Abnormal flexion (decorticate)	3
	Extension (decerebrate)	2
	None	1

Table (II): Comparison of pediatric GCS with GCS[6]

Area Assessed	Pediatric-GCS	GCS	Score
Eye opening	Open spontaneously	Open spontaneously	4
	Open in response to verbal stimuli.	Open in response to verbal stimuli.	3
	Open in response to pain only.	Open in response to pain only.	2
	No response	No response	1
Verbal response	Coos and babbles.	Oriented, appropriate	5
	Irritable cries.	Confused	4
	Cries in response to pain.	Inappropriate words	3
	Moans in response to pain .	Incomprehensible words or nonspecific sounds	2
	No response.	No response.	1
Motor response	Moves spontaneously.	Obeys commands	6
	Withdraws to touch	Localizes painful stimulus	5
	Withdraws in response to pain	Withdraws in response to pain	4
	Responds to pain with decorticate posturing(abnormal flexion)	Responds to pain with decorticate posturing(abnormal flexion)	3
	Responds to pain with decerebrate posturing(abnormal extension)	Responds to pain with decerebrate posturing(abnormal extension)	2
	No response	No response	1

In observational studies, the mean age of patients with EDH is between 20 and 30 years of age. EDH is rare in patients older than 50 to 60 years of age.[10,11]

Extension of the hematoma is usually limited by suture lines owing to the tight attachment of the dura at these locations. Recent analyses have revealed that epidural hematomas may actually traverse suture lines in a minority of cases.[12] Laceration of the middle meningeal artery and its accompanying dural sinuses is the most common etiology.[13] EDH could also occur because of venous bleeding.[14]The temporoparietal region is the most likely site for an EDH.[15]

An EDH is usually unilateral, and 20% of patients have other intracranial lesions, usually subdural hematomas or contusions, They rarely show bilateral localization and consist of 2–10% of all acute epidural hematoma in adults and in the pediatric age group, it is exceedingly rare. The deterioration of a patient who has an EDH from arterial bleeding can be rapid and dramatic. Because of their rapid formation, EDHs from arterial bleeding

are usually detected within hours after injury and often earlier in children. EDHs that develop from a dural sinus tear develop more slowly, and clinical manifestations may be delayed, with resultant delays in detection.[16]

Delayed extradural hemorrhage(DEH) comprised about 10% of traumatic extradural hematomas in several reported series.[17] There are several contributory factors in the development of DEH that have been described in previous reports. Many of these risk factors may occur after hospital admission. Some authors believe that rapid correction of hypotension and recovery from peripheral vascular collapse in multiple trauma victims plays a role by elevating cerebral perfusion pressure, which may cause an extradural hematoma to develop. Bleeding from venules as a result of venous congestion has also been proposed. Venous bleeding would enhanced by mechanical ventilation, as elevated intrathoracic pressure results in elevated intracranial venous pressure. Loss of a tamponading effect of an evacuated blood collection or the use of mannitol to reduce intracranial pressure can also enhance the appearance of subsequent DEH, disseminated intravascular clotting and fibrinolysis after head injury and Traumatic cerebrospinal fluid leak are also recognized as contributory factors for the development of DEH.[18]

Posterior fossa extradural hematomas(PFEDHs) represent 5% of all cases of epidural hematomas. They are usually associated with linear fracture of the occipital bone, diastases fracture of lamboid suture, or both. Diagnostic difficulties are caused by atypical clinical pictures and rare occurrence of these hematomas. Symptoms can develop slowly, but the condition may deteriorate suddenly with fatal outcome unless evacuated in a timely manner.[19,20]

Computed tomography (CT) scan remains the imaging modality of choice for diagnosis of this life threatening condition. The decision and timing for treatment of EDH has to be made individually in each case depending on patient's age, hematoma size, location, patient's neurological status and course.[21,22]

Early diagnosis and treatment reduces mortality and improves outcome. Mortality was 20-55% prior to the CT era but this has improved to about 12-20%. The standard recommendation for symptomatic patients is surgical intervention within the golden hours.[23,24]

A lot of factors acting independently affect outcome in patients with acute traumatic EDH. Admission (GCS) and the presence of associated intracranial lesions appear to be the most important predictors of outcome. Other important factors that determine outcome includes the age of the patient, time from injury to treatment, immediate coma or lucid interval, presence of pupillary abnormalities, CT findings (hematoma volume, degree of midline shift, presence of signs of active hematoma bleeding, associated intradural lesion) and post-operative intracranial pressure.[25,26]

Unlike most forms of TBI, people with EDH usually have good prognosis if they can receive surgery quickly.[27]

Extradural hematoma is widely recognized as a time-critical surgical emergency. Guidance from the Royal College of Surgeons of England recommends that surgical decompression should be carried out within 240 minutes (four hours) of the onset of significant symptoms.[28]

Zero mortality from EDH is a realistic goal for a modern, well run care system for head injured patients that include prompt referral and suitable hospital facilities for constant access emergency neurosurgery.[29]

Clinical presentation:

Classical presentation:

EDH is primarily a disease of the young and accounts for 0.5 to 1% of all patients who have experienced TBI. EDH are rare in elders and children younger than 2 years of age because of the close attachment of the dura to the skull in both of them. The development of symptoms and signs of EDH is entirely dependent on how quickly the EDH is developing within the cranial vault. Classically, the epidural hematoma is associated with a lucid interval between the initial loss of consciousness at the time of impact and a delayed decline in mental status(10-33%), alterations in the level of consciousness may have a variable presentation. Patients with an EDH often complain of a severe headache, sleepiness, dizziness, nausea, and vomiting. A small EDH may remain asymptomatic, but this is rare. In some cases brain herniation may develop.[30,31]

Signs of brain herniation:

Signs of brain herniation include lethargy, coma, loss of all brain reflexes(blinking, gagging, pupils reacting to light), loss of consciousness, respiratory and cardiac arrest.[32]

Signs of delayed extradural hematoma(DEH):

Otorrhea or rhinorrhea can cause intracranial hypotension and enhance the separation of the dura matter from the inner surface of the clavaria.[33] Extradural hematoma underlying a skull fracture is a common feature and predisposing factor for this complication. Skull fracture sites can be the source of hemorrhage. Sharp bone edges along the fracture site can lacerate dural vessels and cause extradural bleeding. However, the presence of skull fracture does not explain the delay in the evolution of an extradural hematoma.[34] All patients, Who sustained mild head injury had neurological impairment before detection of DEH.[35,36]

Signs of extradural hematomas of the posterior fossa:

Posterior fossa epidural hematoma may exhibit a rapid and delayed progression from minimal symptoms to even death within minutes. It may be classified as acute (with signs of brainstem compression within 24 hours of injury). or as delayed (with brain stem compression appearing after 24 hours of injury). Acute PFEDH frequently gives signs of medullary failure within hours of injury. Typically, occipital trauma is associated with severe nuchal occipital pain, marked alteration in the level of consciousness, and occasionally, a lucid interval. This is followed by rapid brainstem compression, with respiratory depression and death if not treated promptly.[32]

Delayed PFEDH often gives rise to headache and neck pain as well as symptoms of a PF lesion, including lower cranial nerve dysfunction and cerebellar signs. concurrent systemic traumatic lesions leading to a hypotensive state and intracranial traumatic lesions with associated increased ICP have been classically identified as mechanisms responsible

for the development of delayed PFEDH.[37] The presence of skull fracture has been identified by several authors as a common feature of reported cases of delayed EDHs and should be considered as a predisposing factor for the development of this complication. Some authors tried to explain the development of delayed EDH as result of the venous origin of bleeding. Among the misleading signs seen in PFEDH, unilateral mydriasis is frequent and possibly explained by the upward herniation of the cerebellum through the tentorial hiatus deforming and compressing the oculomotor nerve, with associated lesions being other possible factors. Numerous examples in which a relatively insignificant supratentorial hematoma was evaluated overlooking a life-threatening PFEDH has been described.[32]

Signs of bilateral EDH:

Among the EDH cases the incidence of bilateral EDH has been reported in various studies ranging from 2-25%.[38] Its presence at more than two sites is extremely rare. It has been suggested that the force of impact to the head could produce bilateral hematomas, which is more predominant in the anteroposterior direction than from the lateral direction.[39]

This is probably the reason for a higher frequency of EDH in the frontal region.[40] Patients with double EDH less frequently have a lucid interval, have a lower GCS, lateralization is frequently absent and deteriorate more often than individuals with unilateral hematomas.[41] There are two types of bilateral EDH. In the commoner first type, the bleeding is venous in nature and occurs as a delayed phenomenon, while in the second type the bleeding is arterial in nature.[18]

Clinical evaluation and management in emergency medicine department:

Overview:

A clear, simple, and organized approach is needed when managing a severely injured patient. The primary survey promulgated in Advanced Trauma Life Support (ATLS) provides such an approach.[42] The primary survey is organized according to the injuries that pose the most immediate threats to life and is performed in the order described immediately below. Patients with TBI should be transferred to a hospital with neurosurgical services as soon as they are hemodynamically stable.[43-47]

1. Primary trauma survey and Resuscitation.

The primary survey and Resuscitation occurs simultaneously and consists of the following steps:[48]

- Airway assessment and protection, while maintaining cervical spine stabilization.
- Breathing and ventilation assessment (maintain adequate oxygenation).
- Circulation assessment (control hemorrhage and maintain adequate end-organ perfusion).
- Disability assessment (perform basic neurologic evaluation).
- Exposure, with environmental control (undress patient and search everywhere for possible injury, while preventing hypothermia).

2. Secondary trauma survey.

A secondary survey is completed once the patient is relatively stable and includes history, clinical neurological examination, laboratory investigation and radiological investigations.[49,50]

I. History

The relevant facts to be ascertained from the history are the time and mechanism of injury, whether there was any initial loss of consciousness, and whether the level of consciousness has improved or deteriorated since the injury.[51]

II. Clinical neurological examination

Neurological assessment requires serial documentation of conscious level, pupillary signs, fundus examination, signs of basal skull fractures, associated injuries and motor power and response.[52]

1. Conscious level

The Glasgow coma scale(GCS) score is universally used to describe conscious level and has prognostic value. It should be performed as soon as the patient is stabilized. It is repeated throughout the resuscitation process to identify any deterioration in patient condition, which may suggest expanding extradural hematoma or brain swelling.[52,53]

2. Pupils

Its size and response to light should be documented regularly. Any asymmetry greater than 1 mm or a change in response to light must be assumed to be due to the effects of an intracranial space-occupying lesion causing compression of ipsilateral third nerve. Urgent CT scan of the brain is required. Bilateral, dilated and unresponsive pupils in the context of a brain injury and in the absence of mydriatic agents is a grave sign.[51]

3. Fundus examination

To detect vitreous or retinal hemorrhages and papilledema that can affect the papillary light reflex.[51]

4. Signs of basal skull fractures

Including otorrhea, rhinorrhea, periobital ecchymosis, retrooccular ecchymosis.[52, 54]

5. Associated injuries

Other signs may accompany head injury that should be clinically evaluated:

- Spinal injuries occurs in about 10% of severely head injured patients. In these patients the cervical spine is most frequently involved.[52]
- Thoracic injuries: such as rib fractures, pneumo- or hemothorax, cardiac tamponade, aspiration or acute respiratory syndrome(ARDS).[52]
- Abdominal injuries: especially liver, spleen or kidney laceration. Substantial hemorrhage usually results in abdominal distension. However these signs may not manifest early and may be obscured in a comatosed patient.[52]
- Pelvic injuries: that are often associated with significant occult blood loss.[52]

6. Motor power and response

The basic examination is completed by testing the motor strength. Each extremity is examined and graded on the internationally used scale as follow normal power is 5; moderate weakness is 4; severe weakness (antigravity) is 3; severe weakness (with gravity) is 2; (tracer movements) is 1; and no movement is 0.[55]

III. Investigations

Laboratory investigations:

These include blood grouping, CBC, coagulation profile, blood gases, renal and liver profile.[56]

Imaging studies:

Because neurological examination is frequently unreliable predicting intracranial pathological processes, thus radiological evaluation is always indicated in TBI patients.[57] In the setting of acute head trauma, imaging serves a key role in both diagnosis and appropriate initial treatment.[58] In addition to EDH, head trauma is a major cause of a variety of other central nervous system lesions including subdural hematoma, subarachnoid hemorrhage, cerebral contusion, diffuse brain swelling, and laceration.[59,60]

1. Plain x-ray skull:

Plain x-ray may reveal a skull fracture whether linear or depressed.[61]

2. Head CT:

Computed tomography (CT) of the head is the most widely used imaging study for acute head trauma owing to its speed, relative simplicity, and widespread availability.[58]On CT scan with bone window, an EDH appears hyperdense, biconvex, ovoid, and lenticular. The EDH does not usually extend beyond the dural attachments at the suture lines. The margins are sharply defined, and the hematoma usually bulges inward toward the brain. EDHs of mixed density on CT may be actively bleeding. Acute EDH may not be apparent on initial head CT in up to 8 percent of cases.[62]

Progressive hematoma enlargement of small EDHs over several weeks has also been reported in patients followed by serial CT.[60] The first follow-up CT scan should be obtained six to eight hours after head injury, A repeat head CT scan is recommended sooner upon neurological deterioration.[63]

A posterior fossa EDH is the most common traumatic mass lesion of the posterior fossa and accounts for 5% of EDHs.[16] An occipital fracture is present in approximately 85% of PFEDH cases. The most significant fracture is linear and vertical, extending across the lateral sinus toward the foramen magnum.[64] A Towne projection will usually best demonstrate a fracture or sutural diastases in the occipital region. The absence of fracture does not rule out traumatic expanding lesions in the PF.[31] Posterior fossa EDH is less dense in more than 50% of cases.[35]

Hematoma volume estimation:

Hematoma volume influences management decisions in adult patients with acute EDH because it correlates with outcome. The hematoma volume can be estimated quickly from the head CT scan by using the formula ABC/2, which approximates the volume of an ellipsoid. This formula was originally used to estimate intracerebral hemorrhage volume, but can be applied to EDH as well.[60]

The formula is calculated using the centimeter scale on the CT images as follows:[65]

- A is the greatest hemorrhage diameter on the CT slice with the largest area of hemorrhage.
- B is the largest diameter 90 degrees to A on the same CT slice.
- C is the approximate number of CT slices with hemorrhage.

3. Portable x-rays:

Plain radiographs play an important role in the primary evaluation of the unstable trauma patient. For hemodynamically compromised patients proceeding directly to the operating room after the primary survey, plain x-rays of the lateral cervical spine, chest, and pelvis, obtained in the ED or immediately upon arrival to the OR, can detect life threatening injuries that might otherwise be missed.[66-68]

4. Ultrasound (FAST exam):

Focused Abdominal Sonography for Trauma (FAST) is an integral part of the primary circulation survey for unstable patients, in whom it often determines management.[69-73]

5. Spine evaluation:

The cervical spine should be evaluated during the initial evaluation by plain radiographs and if any abnormalities are noted, this area should be further evaluated with a CT scan. Magnetic resonance imaging (MRI) may be necessary to image a spinal cord injury. A rigid cervical collar should remain on at all times while the patient is being evaluated.[74]

6. Others:

ECG: patients with head trauma are prone to developing dysrhythmias through a re-entry mechanism as ST segment changes, T wave abnormalities and prolonged QT interval.[52]

Management of Traumatic brain injury and Extradural hematoma:

The goal of medical care of patients of TBI is to recognize and treat life threatening conditions and to eliminate or minimize the role of secondary injury. Patients with severe head trauma are at increased risk of developing cerebral edema, respiratory failure, and herniation secondary to the increased ICP, therefore, frequent serial assessments of the neurologic status must be performed.[75]

1- Prehospital phase

The prehospital phase is the most important interval in determining the ultimate outcome after TBI. The initial goals are to maintain a patent airway, begin fluid resuscitation, immobilize the cervical and thoracolumbar spine, and assess the level of consciousness, followed by the immediate transport to a trauma center with neurologic services. Patients with systolic B.P less than 110 mmHg require fluid resuscitation. Lactated Ringer's solution is generally recommended, another recent studies suggest the use of hypertonic saline.[76,77,78]

2. Management in the Emergency Room as early hospital management:

i-Establishment of an adequate airway

A stable airway should be obtained to provide adequate ventilation and oxygenation. If endotracheal intubation is required, adequate sedation and paralysis must be assured to avoid further increase in ICP. Rapid sequence induction and endotracheal intubation are generally recommended in case of GCS<8 or falling rapidly, hypoxia, hypercarbia ($PaCO_2$>65mmHg) or hypocarbia ($PaCO_2$<30 mmHg), inability to protect airway, significant facial injuries and bleeding, seizures, major injuries elsewhere especially chest injuries and evidence of shock state.[52]

Patient with TBI and altered mentation should be supplemented with 100% oxygen and should be supported with positive pressure ventilation.[52,79]

Premedication for rapid sequence induction (RSI) includes atropine (0.02 mg/kg) to blunt the effect of vagal stimulation and decrease secretions. Lidocaine (1-2 mg/kg) may be used to decrease airway stimulation during intubation and prevent an increase in ICP. Thiopental (75-250mg/kg) etomidate (0.3 mg/kg) or midazolam (0.02-0.1 mg/kg) have successfully been used to sedate the patient for intubation.[52,79]

ii- Immobilization of cervical spines

Proper immobilization techniques at the accident site and during patient transport must be maintained. All patients with a suspected spinal cord injury are immobilized in a rigid cervical collar and placed on spine board for transport.[52]

iii- Cardiovascular management

Achieving normotension and euvolemia is the goal in cardiovascular management. Cerebral perfusion pressure (CPP) is defined as the mean arterial pressure (MAP) minus the ICP (CPP=MAP-ICP).

It is the physiologic variable that defines the pressure gradient driving the cerebral blood flow (CBF) and metabolic delivery, it is therefore closely related to ischemia. Several clinical studies suggest that maintaining CPP at 70-80 mmHg may be the critical threshold. Adequate volume resuscitation with isotonic saline or lactated Ringer's solution should be conducted to maintain adequate filling pressure, normal cardiac output, and ultimately normotension (MAP>90mmHg). More recent adult and pediatric studies have shown that the use of hypertonic saline in the resuscitation process is superior to that of lactated Ringer's solution or isotonic chloride solution. Patients who have received hypertonic saline solution have improved blood pressure response, overall decreased fluid requirements, fewer interventions in controlling the ICP, fewer complications, and improved survival.[77,78,80] Hypertension if present, could represent a compensatory mechanisms in response to increased ICP, thus reflex treatment of it may significantly comprise the cerebral perfusion. When normotension is desired in the presence of intracranial or intracerebral hemorrhage following surgical evacuation, calcium channel blockers are the drugs of choice instead of direct vasodilators to avoid hypotension.[80]

Continuous cardiac monitoring should be performed because of the high incidence of ventricular dysrhythmias present in patients with TBI and in those in whom cardiac contusion is suspected.[52,78]

iv- Prophylactic hypothermia:

Hyperthermia has long been correlated with poor outcome in patient with TBI, and control of fever is an important initial intervention to limit secondary brain injury.[81,82,83]

v- Management of elevated intracranial pressure:

Efforts to evaluate and manage increased intracranial pressure (ICP) should begin in the emergency department. Patients with severe TBI (GCS $\leq$8) and clinical symptoms suggesting possible impending herniation from elevated ICP (unilaterally or bilaterally fixed and dilated pupil(s), decorticate or decerebrate posturing, bradycardia, hypertension, and/or respiratory depression) should be treated urgently.[84,85]

Intracranial hypertension is variably defined as an intracranial pressure of over 20-25 mmHg. Treatment of hypertension should proceed in a stepwise manner:[80]

a- **Head position:** elevation of the head of the bed to 30-45 degree has been shown to optimize arterial in flow and venous drainage, also keeping the neck in the midline prevent kinking of jugular veins.[80]

b- **Hyperventilation:** should be used carefully for treating acute ICP elevation. studies have shown that prolonged prophylactic use of hyperventilation in head trauma patients is associated with negative outcome. CBF is known to diminish in the first 24 hours in patients with severe traumatic brain injury, with absolute values close to those of ischemia. hyperventilation decrease CBF. it also potentially leads to loss of autoregulation. this may cause further ischemic injury and does not produce consistent reduction in ICP. therefore, mild hyperventilation with $PaCo_2$ level of 30-35 mmHg is tolerated better over a long period with less deleterious effects.[86] it has been suggested that a high PaO_2 may improve brain tissue oxygenation.[87] continuous pulse oxymetery is recommended, with the arterial saturation maintained more than 94%.[88]

c- **Analgesia and sedation:** they may be needed to facilitate ventilation in intubated patients as well as imaging in restless patients and whom elevated ICP is difficult to control if patient is in pain. traditionally, narcotics in combination with benzodiazepines have been commonly used.[89]

Table (III): Use of midazolam in TBI[90]

Drug name	Midazolam
Description	Short acting benzodiazepine with rapid onset of action. Used in treating increased I.C.P.
Dose	0.02-0.1 mg/Kg/Dose I.V.
Contraindication	Documented hypersensitivity, uncontrolled pain, pre-existing hypotension, narrow angle glaucoma.
Interaction	Sedative effects of midazolam may be antagonized by theophylline, narcotics. Erythromycin may accentuate sedative effect of midazolam because of decreased clearance.
Pregnancy	D-unsafe in pregnancy
Precautions	Careful monitoring of cardio respiratory state during administration, caution in congestive heart failure, pulmonary disease, renal impairment and hepatic failure.

d- Osmotic diuretics: Mannitol is very effective and can be life saving.[80]

Table (IV): Use of mannitol in TBI.[80]

Drug Name	Mannitol (Osmitrol)
Description	Osmotic diuretic, which lowers the blood viscosity and produces cerebral vasoconstriction with normal CB. ICP decrease occurs subsequent to a decrease CBV.
Adult Dose	1.5-2 g/kg IV as 20% solution (7.5-10 mL/Kg) or as 15% solution (10-13 mL/Kg) over a period as short as 30 min.
Pediatric Dose	0.5-1 g/kg/dose IV initial dose 0.25-0.5 g/kg/dose IV q4-6h
Contraindications	Documented hypersensitivity; anuria; severe pulmonary congestion; progressive renal damage; severe dehydration; active intracranial bleeding; progressive heart failure.
Interactions	May decrease serum lithium levels.
Pregnancy	C - Safety for use during pregnancy has not been established.
Precautions	Carefully evaluate cardiovascular status before rapid administration of mannitol because a sudden increase in extracellular fluid may lead to fuliminating CHF. if used every 4-6h, serum osmolarity should be monitored and dose held if osmolarity exceeds 320 mOsm/kg.

e. Loop diuretics: Furosemide is the most commonly used loop diuretics in TBI. It can be administered every 6 hours at the midway point between mannitol doses.[80]

Table (V): Use of loop diuretic in TBI.[80]

Drug Name	Furosemide (lasix)
Description	A loop diuretic helpful in decreasing the ICP via 2 mechanisms. One influences CSF formation by affecting the sodium- water movement across the blood-brain barrier; the other mechanism is the preferential excretion of water over solute in the distal tubule.
Adult Dose	20-80 mg/d IV /IM; may increase dose; not to exceed 600 mg/d
Pediatric Dose	1-2 mg/kg/dose IV q6-12h
Contraindications	Documented hypersensitivity; hepatic coma, anuria and sever electrolyte depletion
Interactions	Metformin decreases furosemide concentrations; furosemide interferes with hypoglycemic effect of antidiabetic agents and antagonizes muscle-relaxing effect of tubocurarine; auditory toxicity appears to be increased with coadministration of aminoglycosides and flurosemide; hearing loss of varying degrees may occur; anticoagulant activity of warfarin may be enhanced when taken concurrently with this medication; increased plasma lithium levels and toxicity are possible when taken concurrently with this medication.
Pregnancy	C - Safety for use during pregnancy has not been established.

f. Steroids: No role for steroid therapy in TBI or for post traumatic brain edema.[91]

vi-Management of early post-traumatic seizures:

Post-traumatic seizures are present in 10% of TBI mainly in children . These may affect the outcome adversely by increasing the ICP, increasing the metabolic demands of the brain, and causing hypoxia and/or hypoventilation in a spontaneously breathing patient. Short acting benzodiazepines may be used to control seizures, and phenytoin or phenobarbital should be used for maintenance as an anticonvulsant.[92,93]

Table (VI): Use of phenytoin for seizure prophylaxis in TBI [52,80]

Drug Name	Phenytoin (dilantin)
Description	May act in motor cortex where may inhibit spread of seizures activity.
Adult Dose	Loading dose for status epilepticus : 15-20 mg/kg PO/IV once or in divided doses, followed by 100-150 mg/dose at 30 min intervals. Initial maintenance dose (administered 12 h after loading dose):100mg (if administering oral susp, use dose of 125mg) PO/IV tid Maintenance: 300-400 mg/d PO/IV divided tid or qd/bid if using ER; increase to 600 mg/d (625 mg/d for PO susp) may be necessary; not to exceed 1500 mg/24h Rate of IV infusion must not exceed 50 mg/min to avoid hypotension and arrhythmias.
Pediatric Dose	Loading dose : 15-20 mg/kg PO/IV once or in divided doses Initial maintenance dose (administered 12 h after loading dose):5 mg/kg/d PO/IV divided bid/ tid Maintenance:4-8 mg/kg PO/IV divided bid/ tid; children > 6y may require minimum adult dose (300 mg/d); not to exceed 300 mg/d
Contraindications	Documented hypersensitivity; sinoartrial block; second-and third-degree AV block; sinus bradycardia; Adams-Stokes syndrome
Interactions	Amiodarone, phenylebutazone, omeprazole, ethanol(acute ingestion), and valproic acid may increase phenytoin toxcicity; phenytoin effects may decrease when taken concurrently with barbiturates, diazoxide, ethanol (long term ingestion), antacids, charcoal, carbamazepine and theophylline.
Pregnancy	D-unsafe in Pregnancy.
Precautions	Rapid IV infusion may result in death from cardiac arrest, marked by QRS widening; discontinue use if hepatic dysfunction occurs.

3. Neurosurgical consultation

Once the patient's condition is stabilized neurosurgical consultation is required. The critical factors in deciding to proceed directly with surgical evacuation of an intra-cranial hematoma are the patient's neurological status and the CT-scan findings.[52]

4. Definitive management of EDH:

The decision to perform surgery in patients with acute EDH is based primarily upon the patient's neurologic status, as assessed by the (GCS) score, neurologic examination and pupillary signs, and brain imaging findings.(56)

Retrospective studies suggest that stable patients with EDH who have small hematomas and mild symptoms can be managed nonoperatively.(94-96)

- An epidural hematoma (EDH) greater than 30 cm^3 should be surgically evacuated regardless of the patient's (GCS) score. Evacuation should be done as soon as possible especially in comatose patients (GCS <9) with anisocoria by craniotomy.(97)
- An EDH less than 30 cm^3 and with less than a 10-mm thickness and with less than a 5-mm midline shift (MLS) in patients without focal deficit can be managed conservatively with serial computed tomographic (CT) scanning and close neurological observation in a neurosurgical center.(94-97)

Surgical treatment of epidural hematomas (figure 1) involves opening the calvaria over the site of the hemorrhage. The EDH is readily apparent after elevating the bone flap, and it is removed. Coagulation of bleeding dural vessels is usually performed. Epidural tack-up sutures are placed from the dura to the craniotomy bone edge and to the center of the craniotomy flap to tamponade epidural bleeding from area beyond the craniotomy edges and to prevent recurrence. Dural venous sinus bleeding is controlled with tamponade by gelatin sponges and cotton strips and head of the bed elevation, taking care to avoid venous air embolism. The utmost care should be taken when elevating depressed bone fragments on or near the dural venous sinuses. If present, the cushing response remains untreated until it resolves spontaneously as the mass effect is relieved.(98)

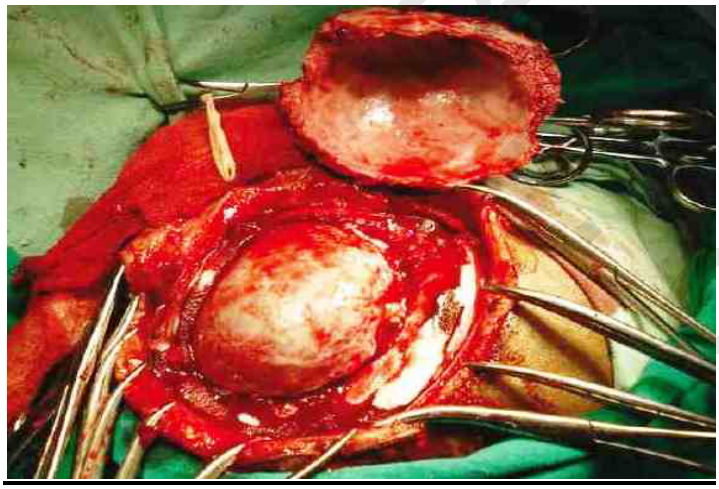


Figure (1): Evacuation of an extradural hematoma.
From http://www.cybermedicine2000.com Chapter 47: Brain Injury. Available at:http://www.cypermedicine2000.com/pharmacology2000/822_1/page1.htm

Complications:

Many of the complications from EDH occur when the pressure they exert results in significant brain shifting. When the brain is subject to subfalcine herniation, the anterior and posterior cerebral arteries may be occluded, resulting in cerebral infarction. Downward herniation of the brain stem can result in Duret hemorrhages within the brainstem, mostly in the pons. Transtentorial herniation may result in an ipsilateral cranial nerve III palsy manifests as ptosis, pupillary dilation, and the inability to move the eye in medial, upward, and downward directions.[98]

Prognosis:

Data regarding outcome after EDH are mainly from observational studies. In surgical series, mortality after EDH in adults and children is approximately 10 and 5 percent, respectively.[59] In general, the preoperative motor examination, the Glasgow Coma Scale score, and pupillary reactivity are significantly correlated to the functional outcome of patients with acute epidural hematomas when they survive. Because many isolated epidural hematomas do not involve underlying structural brain damage, the overall outcome is excellent if prompt surgical evacuation is undertaken.[98]

a. Prognostic indicators:

In observational studies, the following variables have been associated with unfavorable outcome from EDH:[59]

- Low (GCS) score on admission or before surgery.[29,99-101] The GCS grades coma severity according to three categories of responsiveness: best eye opening, best verbal, and best motor response. The GCS is scored between 3 and 15, with higher scores indicating better performance.
- Presence of pupillary abnormalities, particularly contralateral or bilateral unreactive pupils[100-104]
- Older age.[101]
- Longer time interval between neurologic deterioration and surgery[102,105]
- Postoperative elevated intracranial pressure.[104,106]

In addition, several studies have identified head CT findings that correlate with poor outcome:

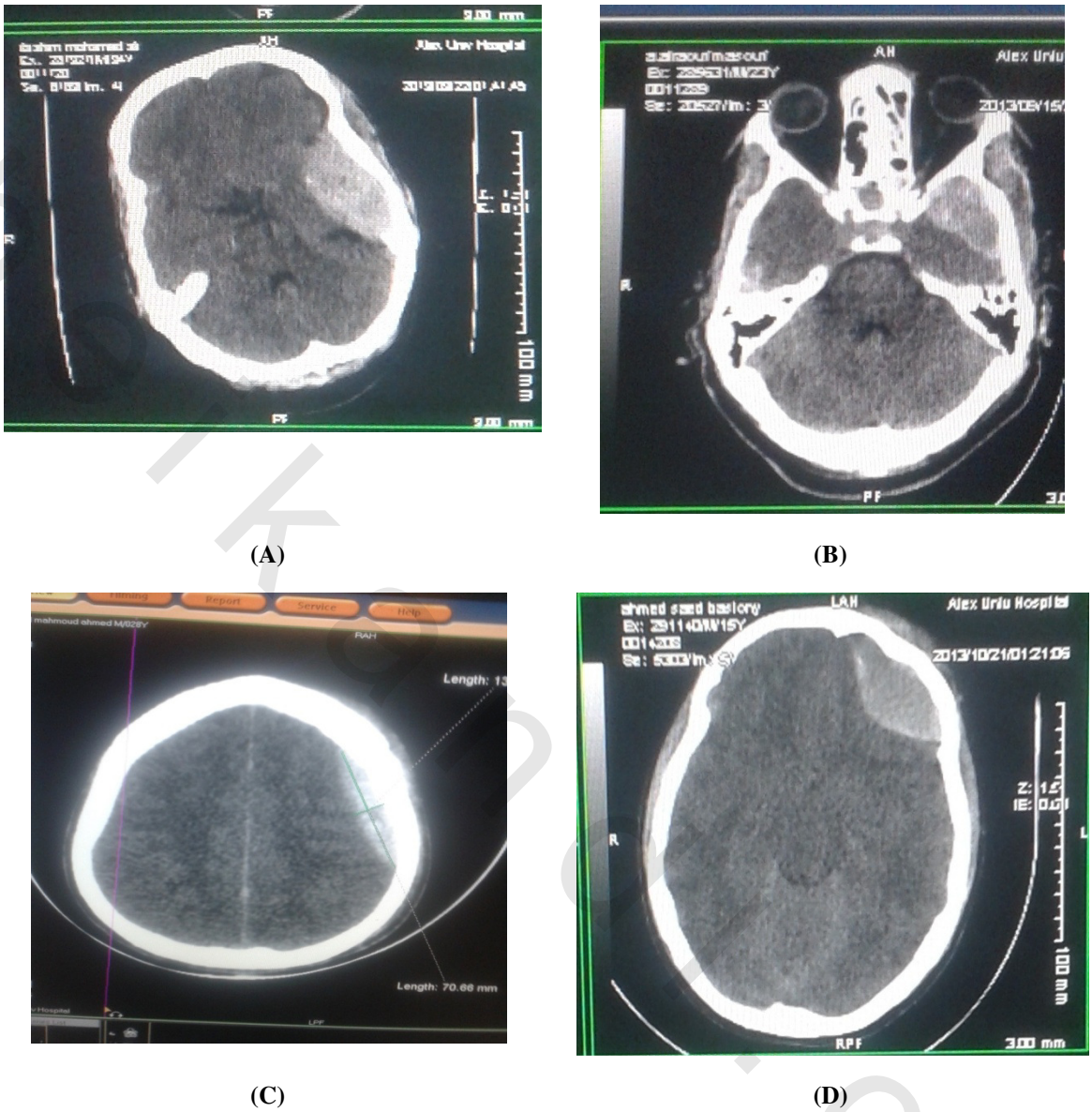
- Hematoma volume >30 to 150 cm^3 [100,103]
- Presence of midline shift >10 to 12 mm[100]
- Mixed density blood clot, indicating acute bleeding[103]
- Presence of associated intracranial lesions such as contusions, intracerebral hemorrhage, subarachnoid hemorrhage, and diffuse brain swelling.[100,101,103,105]

b. Glasgow Outcome Scale(GOS):[107]

By far, the most common method chosen to assess outcome is the Glasgow Outcome Scale. It was introduced by Jannett and Bond in 1975. It measures the ability following severe head injury, it takes into account both physical and mental impairment. It includes (1) death, (2) persistent vegetative state, (3) severe disability (conscious but disabled), (4) moderate disability (disabled but independent) and (5) good recovery.

Table (VII): Glasgow Outcome Scale (GOS)[107]

Outcome	Definition
Good recovery	Patients able to return to former occupation, though not necessarily at the same level. may have minor neurological or psychological impairment.
Moderate disability	Patients unable to return to work, but otherwise able to perform the activities of daily living independently.
Severe disability	Patients require assistance to perform daily activities and cannot live independently.
Persistent vegetative state	Absence of speech or evidence of mental function in a patient who appear awake with spontaneous eyes opening.
Death	



(A) **(B)**

(C) **(D)**

Figure (2): CT images. A: left temporal EDH measuring 30 ml ,note the brain edema. B: left temporal EDH measuring 30 ml, managed surgically. C: left parietal EDH measuring 15 ml managed conservatively. D: left frontal EDH measuring 30, managed surgically.