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Managing Epidural Hematoma: A Case Study of Perioperative and Postoperative Strategies after a Traumatic Injury

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Abstract: ***An epidural hematoma (EDH) is an extra-axial collection of blood within the potential space between the outer layer of the dura mater and the inner table of the skull. It is confined by the lateral sutures (especially the coronal sutures) where the dura inserts. It is a life-threatening condition, which may require immediate intervention and can be associated with significant morbidity and mortality if left untreated. Rapid diagnosis and evacuation are important for a good outcome.The following case illustrates our own experience in the peri operative and post-operative management of a patient who had fall from 12 feet height , following which she developed EDH . The Patient was Operated and shifted to ICU for further management.***

Keywords: EDH, GCS, Aspiration, Intubation, ICU, Tracheostomy, Bronchoscopy

1. Introduction

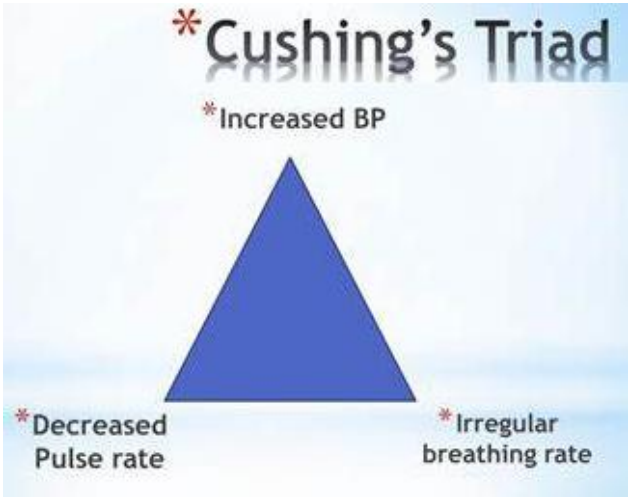
Epidural hemorrhage (EDH) is an event characterized by bleeding into the epidural space between the dural layers of the meninges and the skull. The primary mechanism triggering bleeding is trauma (i.e., closed head injury), which causes arterial injury, most commonly middle meningeal artery injury. Epidural hemorrhage presents acutely, usually immediately (seconds to hours) following head trauma, with an altered level of consciousness that may span from a momentary loss of consciousness to coma.

Typical symptoms of EDH include:

- Headache
- Nausea and vomiting
- Confusion
- Loss of consciousness (typically immediately after a head injury) followed by a period of lucidity
- Progressively decreasing level of consciousness (typically developing several hours after the initial injury)

Typical clinical findings in EDH may include:

- Tenderness of the skull (in the context of injury)
- Confusion
- Low Glasgow Coma Score {GCS}
- Cranial nerve deficits (e.g. occulomotor nerve palsy causing fixed dilation of the ipsilateral pupil)
- Motor or sensory deficits of the upper and/or lower limbs (e.g. hemiparesis, paraesthesia)
- Hyperreflexia and spasticity
- Upgoing plantar reflex (Babinski's sign)
- Cushing's triad: a physiological response to raised intracranial pressure including bradycardia, hypertension and deep/irregular breathing.



Aetiology

An extradural haematoma is commonly caused by skull trauma in the temporoparietal region, typically following a fall, assault or sporting injury. An EDH is associated with a skull fracture in 75% of cases. The pterion is an anatomical landmark where the parietal, frontal, sphenoid and temporal bones fuse.

The pterion is particularly vulnerable to fracture as the bone at this location is relatively thin. The middle meningeal artery (MMA) also lies underneath the pterion and therefore fracture at this location can result in rupture of the MMA. As a result, the middle meningeal artery is involved in 75% of extradural haematomas.

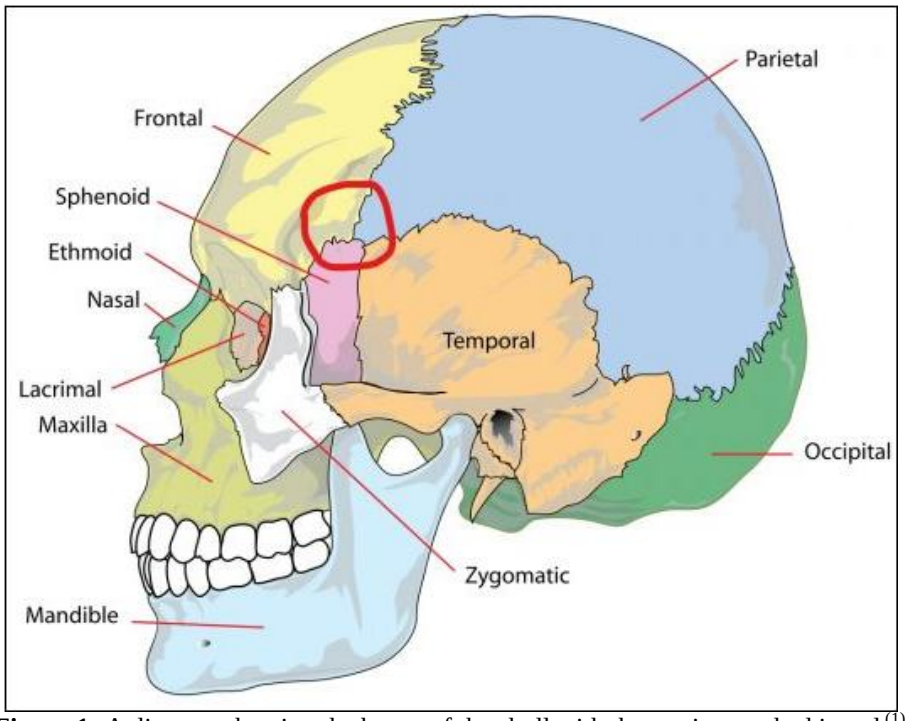


Figure 1: A diagram showing the bones of the skull with the pterion marked in red.[1]

EDH can also occur secondary to the rupture of a vein, particularly if the middle meningeal vein or dural sinuses are involved. Rarely, EDH can occur secondary to arteriovenous abnormalities or bleeding disorders. [2]

An epidural hematoma (EDH) can be a life-threatening condition. It usually requires immediate treatment or can cause brain damage or possibly death if left untreated. An EDH results in death in up to 15% of cases. [3]

Diagnosis is based on clinical suspicion following head trauma and is confirmed with neuroimaging (i.e., noncontrast head CT).

Management includes stabilization , monitoring in a neurologic ICU, and neurosurgical intervention.

Pathophysiology

Arterial Injury:
Most epidural hematomas result from arterial bleeding from a branch of the middle meningeal artery. The anterior meningeal artery or dural arteriovenous (AV) fistula at the vertex may be involved. [4][5]

Venous Injury:
Up to 10% of EDHs are due to venous bleeding following the laceration of a dural venous sinus.

In adults, up to 75% of EDHs occur in the temporal region. However, in children, they occur with similar frequency in the temporal, occipital, frontal, and posterior fossa regions.

A skull fracture is present in the majority of patients with EDH. These hematomas often present beneath a fracture of the squamous part of the temporal bone.

If this condition occurs within the spine, this entity is described as a spinal epidural hematoma.

Based on radiographic progression, it can be classified into one of the following:

- Type I: Acute; occurs on day 1 and associated with a "swirl" of un-clotted blood
- Type II: Subacute occurring between days 2 to 4 and usually solid
- Type III: Chronic occurring between days 7 to 20; mixed or lucent appearance with contrast enhancement

Study
55 year old patient with underlying Hypertension (on Tablet Telmisartan + Chlorthiazide 40 mg 24 hourly) got admitted in Neurosurgical emergency of SMHS Hospital on 22nd June 2022 as a case of fall from 12 feet height.

The time of injury was 8:30 am and the time of arrival to the hospital was 10:00 am. On the way to the hospital patient had no episode of vomiting, bleed from ear or nose, no episode of seizures.

As the patient reached to the neurosurgical emergency, the patient was examined:

GCS: $E_1V_2M_4$ (7/15)
Pupils: Normal size bilaterally
Reaction to light: normal
CVS: Heart sounds normally heard
RS: Bilaterally air entry present

Aspiration +
P/A: Soft , non distended , non tender
ENT: No bleed present
Vitals:
HR: 68b/m
BP: 100/64 mmhg

Spo2: 83% on room air
RR - 22/min
Temp: Afebrile

Immediately Anaesthesiologist and radiologist were called.

After securing 2 IV lines with 18G and 16G IV cannula, the patient was resusitated with normal saline and simultaneously intubated. Rapid sequence Induction (RSI) along with Manual in-line neck stabilization (MILS) was done. E-fast was done which came negative.

The base line Investigations were sent and the patient was shifted to CT-Scan unit for evaluation on transport ventilator accompanied by us.

CT chest and abdomen were normal . The CT of spine was normal.

CT head revealed 40 mm EDH in the temporal region with 8 mm mid-line shift. Small contusions were present over the frontal region.

CT scan is the most common imaging modality to assess for intracranial bleeding. Its popularity is related to its widespread availability in emergency departments. The majority of EDHs are identifiable on a CT scan. The classic presentation is a biconvex or lens-shaped mass on brain CT scan, due to the limited ability of blood to expand within the fixed attachment of the dura to the cranial sutures. EDHs does not cross suture lines.

The blood **investigations** revealed:
Hb - 9g/dl
WBC - 2300/mm^3
Plt - 180000
LFT - Normal
Kft - Normal
ECG - Normal
Triple Serology - Negative
Coagulogram - Normal

The Patient was immediately shifted to the emergency theatre at 11:30 am for evacuation of EDH.

The patient was coupled to mechanical ventilation with Vt - 450 ml and RR - 14/min. Standard ASA monitors were connected.

Arterial line was secured in right Radial artery and tranducer was connected for Invasive BP monitoring. Central venous line of 7 Fr size was secured in right Internal jugular vein under USG guidance using Seldingers technique.

Craniotomy with evacuation of EDH was done. Intra operatively the patient got hemodynamically unstable and infusion Noradrenaline was started @ 3 ml/hr and titrated according to the BP. She received 2.5 L of crystalloids and 1 unit of PRBC. The urine output was 1.8 L. The ABGs done intra operatively were normal. The surgery took 2.5 hours and the patient was shifted to Surgical-ICU on transport ventilator (intubated and sedated) with infusion of Noradrenaline going on at 2:15 pm.

On arrival to SICU:
Patient was put on PSIMV mode of mechanical ventilation, monitors were connected. Arterial line was transduced for IBP.
GCS - Sedated
Pupils - B/l normal size and reacting to light
CVS : Heart sounds normally heard
RS : Bilaterally air entry present

Aspiration +
P/A : Soft , non distended , non tender
Renal : urinary catheter in situ

Vitals:
HR: 68b/m
BP: 100/64 mmhg on inotropic support
Spo2: 95 % on $Fi0_2$ of 100% , Vt - 450 ml
RR - 12 / min
Infusion Fentanyl was started @ 5 ml / hour.
$Fi0_2$ was reduced according to the ABG values of the patient .

Treatment
Inj Clindamycin 600 mg IV 12 hourly was started for aspiration.
Inj Meropenem 1g IV 8 hourly was started.
Inj Pantoprazole 40 mg IV 24 hourly was started.
Inj Paracetamol 1g IV 8 hourly was started.
Hypertonic Saline 100 ml was given over 6 hours IV 12 hourly.
Inj Levetiracetam 500 mg IV 24 hourly was started.
Inj Phenytoin 200 mg IV 24 hourly was started.

Methyl cellulose eye drops were added to the treatment, along with chlorhexidine mouth wash.

Regular oral and endotracheal suctioning was done .The head end of the patient was kept at 30 degrees, ABG, input/output, blood glucose and temperature monitoring was done 4 hourly.

The $EtCo_2$ of the patient was kept between 30-35 mmhg , normo-thermia was maintained. Base line investigations were sent everyday. The patient was put on air mattress and position of the patient was regularly changed. Central Venous pressure (CVP) was checked regularly.

On 24th June 2022, the inotropic support to the patient was stopped and patient was hemodynamically stable . Ryles tube was inserted and nasogastric feeding was started @ 100 ml/hour.

The patient was taken for a check NCCT Scan of head on 25th June 2022 at 10:00 am. There was no fresh bleed. Sedation was stopped and GCS of the patient was assessed.

GCS - $E_3V_TM_4$ (8/15)

Deep vein thrombosis (DVT) prophylaxis was started (Inj Enoxaparin 0.4 ml S/C 24 hourly).

Sedation was started again , patient was put on infusion Fentanyl @ 5 ml/hr.

Following this the GCS of the patient was assessed everyday.

The best GCS score was: $E_4V_TM_4$ (9/15) on 29th June 2022.

On 1st July 2022, we did percutaneous tracheostomy as a part of weaning the patient off from the ventilator and to limit the need for sedation and make oral toileting easy.

Following trachestomy the oxygen requirements of the patient got decreased .The patient was regularly checked for presence of any bedsores.

On 5th July 2022, the patient was put on T piece with 6 L/min of oxygen flow and maintained a SpO_2 of 95% with GCS - $E_4V_2M_6$ (12/15)

On 9th July there was sudden drop in $Sp0_2$ with decreased chest rise on the left side . Portable Chest Xray was done which revealed collapse of left lung . The patient was coupled back to ventilator which revealed high peak and plateau airway pressures.

Physiotherapist was called and chest physiotheraphy was done. Suctioning was regularly done along with nebulization with Normal Saline 6 hourly and Salbutamol and Budesonide 24 hourly. ABG sampling was done 4 hourly.

On 11th July 2022, we did bed side bronchoscopy of the patient that revealed a mucus plug present in left upper main stem bronchus . The mucus plug was removed immediately. The lung protective measures were followed.

On 13th July 2022, the patient was put back on spontaneous mode of mechanical ventilation and maintained a $Sp0_2$ of 98% of $Fi0_2$ 40%.

On 15th July 2022, T piece trail was again given on and patient could breathe comfortably on 8-10 L oxygen and .

On 18th July 2022, chest xray was repeated which showed improvement . The oxygen requirement of the patient also came down to 5 L/min.

GCS - $E_4V_4M_6$ (14/15)

From 20th July 2022, decannulation of the tracheostomy tube was started ($Sp0_2$ - 97% on 3 L/min Oxygen)

On 29th July 2022, patient was completely de-cannulated and trachestomy tube was removed. Sutures were applied and patient was spontaneously breathing on room air and maintaining a $Sp0_2$ of 97-98 %.

Deep breathing exercises and incentive spirometry was encouraged for the patient.

The patient was ambulated.

The patient was fully conscious and oriented to time , place and person. The patient had no neurological deficit .

The Power in all the four limbs was normal (5/5).

The Cranial nerves examination was done and were found to be bilaterally intact.
CVS : Heart sounds normally heard
RS : Bilaterally air entry present
P/A : Soft , non distended , non tender

Vitals:
HR : 78b/m
BP : 133/74 mmhg
Temp : Normal
On 30th July 2022 , patient was shifted to ward .and on 31st July 2022 , the patient was discharged .

2. Summary

The Patient got admitted in our hospital as a case of traumatic brain injury. She was immediately operated and shifted to SICU where she stayed for 38 days . During this course she was mechanically ventilated and trachestomized. Neuro and lung protective measures were followed and supportive treatment was given simultaneosuly.

3. Conclusion

The management of the patient with severe TBI is often complex and requires a multidisciplinary approach.Anesthesiologists are involved in the care of patients with TBI in various situations, including (but not limited to) resuscitation and stabilization in the emergency department (ED), sedation and anesthesia for diagnostic imaging, craniotomy or decompressive craniectomy, extracranial surgery, and intensive care management.

References

[1] Rengachary SS (ed.), Ellenbogen RG (ed.). Principles of Neurosurgery, 2nd Edition. Elsevier; 2005
[2] https://geekymedics.com/extradural-haematoma-overview/
[3] https://my.clevelandclinic.org/health/diseases/22034-epidural-hematoma
[4] Burjorjee JE, Rooney R, Jaeger M. Epidural Hematoma Following Cessation of a Direct Oral Anticoagulant: A Case Report. Reg Anesth Pain Med. 2018 Apr;43(3):313-316.
[5] Bonow RH, Barber J, Temkin NR, Videtta W, Rondina C, Petroni G, Lujan S, Alanis V, La Fuente G, Lavadenz A, Merida R, Jibaja M, Gonzáles L, Falcao A, Romero R, Dikmen S, Pridgeon J, Chesnut RM., Global Neurotrauma Research Group. The Outcome of Severe Traumatic Brain Injury in Latin America. World Neurosurg. 2018 Mar;111:e82-e90