

# Management of Epidural Hematoma in the Pediatric Age Group

Mohammed Jaber Al-Mamoori

Department of Neurosurgery, Hilla Teaching Hospital, Babylon, Iraq

## Abstract

**Background:** Epidural hematoma (EDH) is a traumatic accumulation of blood between the inner table of the skull and the stripped-off dural membrane, which has been stripped from the overlying bone both by the direct trauma and by the hydrostatic force of blood. Pediatric EDH presented with both age-specific and/or atypical manifestations when compared with EDH in adults. **Objectives:** The aim of this study is to determine the management protocol of choice for pediatric EDH patients that can lead to an early diagnosis, effective treatment, and a good outcome for both operative and nonoperative groups of patients. Surgical management for EDH is the decision of choice for most cases, but still, there is a role for conservative management in selected cases. **Materials and Methods:** In this prospective study, 210 cases of pediatric EDH under the age of 18 years were studied during a 10-year period in the Neurosurgical Department of Hilla Teaching Hospital in Babylon, Iraq, from 2008 to 2018. This study considered the following parameters: characteristics of patients, clinical manifestations, mechanism of head injury with age-specific distribution, radiological findings, associated pathologies, management, source of bleeding in operated cases, correlated pathologies in dead patients, and outcome. Categorical variables are presented in the form of frequencies and percentages. **Results:** The age group >6-12 years have the highest incidence 33%. Males constituted 75% of the victims whereas females 25%. Accidental fall had the highest incidence of 47.1%. The classic clinical course of lucid interval was present in only 7% of cases. Supratentorial EDHs comprised 91% whereas infratentorial EDHs (posterior fossa) only 9%. Skull fracture was present in 92.2% of cases. Surgical evacuation was done in 110 cases whereas 100 cases were managed conservatively. Middle meningeal artery or vein was identified as a source of bleeding in 50% of the operated cases. Glasgow outcome scale was applied to assess the outcome and to compare the outcome in both operative and conservative groups. The overall mortality was 6.7%. **Conclusion:** EDH is a life-threatening entity in the pediatric age group, so that special attention and a high index of suspicion are required. Clinical and neuroimaging assessments by computed tomography scan have a fundamental role for optimal therapeutic decision. Both surgical and conservative management should have excellent outcome if they accomplished on solid basis. Conservative management should be achieved in specialized neurosurgical centers that can assure rapid conversion to surgical intervention in case of neurological deterioration. In borderline cases, surgical evacuation of EDH may spent less hospital stay time with better cost-benefit ratio than conservative management. Judicious surgical intervention can result in excellent long-term outcome.

**Keywords:** Epidural hematoma, head injury, management, pediatric

## INTRODUCTION

### Definition

Epidural (extradural) hematoma (EDH) is a traumatic accumulation of blood between the inner table of the skull and the stripped-off dural membrane [Figure 1]. In the majority of cases, localized head trauma is the inciting event that causing skull fracture and underlying blood vessel injury, which are the sources of hemorrhage in EDH; however, the underlying brain is minimally injured in most cases.

### History

Many old studies have been shed the light on this clinical entity, of these presented by Hill in 1772, Abernethy in 1811, Bell

in 1816, Cock in 1842, Griesinger in 1862, and Hutchinson in 1867.<sup>[1]</sup> However, it is not until 1886 when Jacobson give a detail review of the presentations and outcomes of (71) cases, it is from that time this clinical entity became firmly established.<sup>[2]</sup>

### Epidemiology

Acute epidural hematoma (EDH) in children is relatively uncommon, accounting for 2%–3% of all traumatic brain

**Address for correspondence:** Dr. Mohammed Jaber Al-Mamoori,  
Department of Neurosurgery, Hilla Teaching Hospital, Babylon, Iraq.  
E-mail: [mohammedjaber76@gmail.com](mailto:mohammedjaber76@gmail.com)

**Submission:** 19-07-2019 **Accepted:** 09-10-2019 **Published Online:** 23-12-2019

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

**For reprints contact:** [reprints@medknow.com](mailto:reprints@medknow.com)

**How to cite this article:** Al-Mamoori MJ. Management of epidural hematoma in the pediatric age group. *Med J Babylon* 2019;16:276-85.

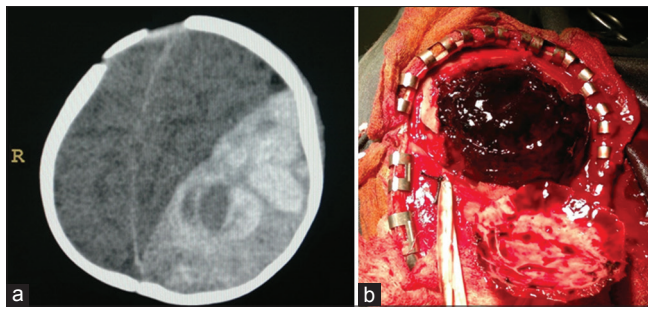
### Access this article online

#### Quick Response Code:



**Website:**  
[www.medjbabylon.org](http://www.medjbabylon.org)

**DOI:**  
10.4103/MJBL.MJBL\_47\_19



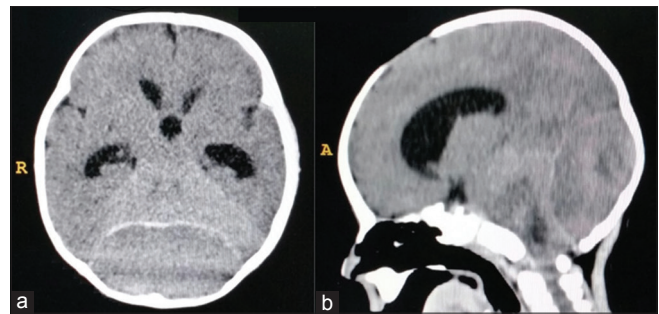
**Figure 1:** Left parietal huge epidural hematoma in a 6-month-old female baby with accidental falling. (a) CT-scan of brain. (b) Operative photograph of left parietal craniotomy

injury cases in this population.<sup>[3]</sup> EDH is more common in young people although it does occur in all age groups, but it is rare before the age of 2 years or after the age of 60 years because of tenacious dural attachment to the inner table of the skull in these age groups.<sup>[4,5]</sup> Male-to-female ratio was 4:1. In adults, EDH is far less common than subdural or intracerebral hemorrhage. In pediatric patients, EDH is comparatively a more common sequel of head injury probably due to the abundant vascularity of the diploe and dura as normal finding in infants and young children.<sup>[6]</sup> Posterior fossa EDH accounts for 5% of EDH, more common in the first two decades of life, especially in infants and children [Figure 2].

### Pathogenesis

Most EDH occurs in the temporoparietal regions (73%) in the territory of the middle meningeal arteries and veins, which is usually torn by squamous temporal bone fracture [Figure 1]. Bruising of the overlying scalp is usually a reliable guide to the site of the hematoma. Skull fracture is associated with EDH in 65%–90% of patients.<sup>[7]</sup> About 10%–40% of EDHs are of venous origin [Figures 2 and 3]. Most traumatic venous EDHs occur in children and mostly not associated with a skull fracture.<sup>[6]</sup> The arterial source of bleeding is reported in only 36% of adults and 18% of children.<sup>[6]</sup> Trauma to potential elastic skulls of adolescents and young children may strip the dura matter from the inner table of the skull causing EDH without fracture, despite the tight adherence of dura to the inner table of the skull, and the meningeal vessels are not embedded in the skull as often seen in later life.<sup>[6]</sup>

Birth injuries fall into two categories: injuries produced by normal force of labor and those produced by obstetrical intervention usually complications of forceps application. EDH is quite uncommon in infants<sup>[3,8]</sup> due to various factors: pliable nature of infant skull resists fracturing due to the presence of unfused suture and fontanels, the middle meningeal artery is less vulnerable to injury because it is not embedded in the skull,<sup>[9-11]</sup> and the dura is tightly adherent to the skull.<sup>[12]</sup> However, infant EDHs are reported probably due to high vascularity of diploe of skull bone and dura.<sup>[3,8,10,12]</sup>



**Figure 2:** Bilateral intratentorial EDH in 6 months age male infant brain CT-scan. (a) Axial view, show isodense large EDH. (b) Sagittal view, shows the mass effect of EDH on brain stem

### Clinical manifestations

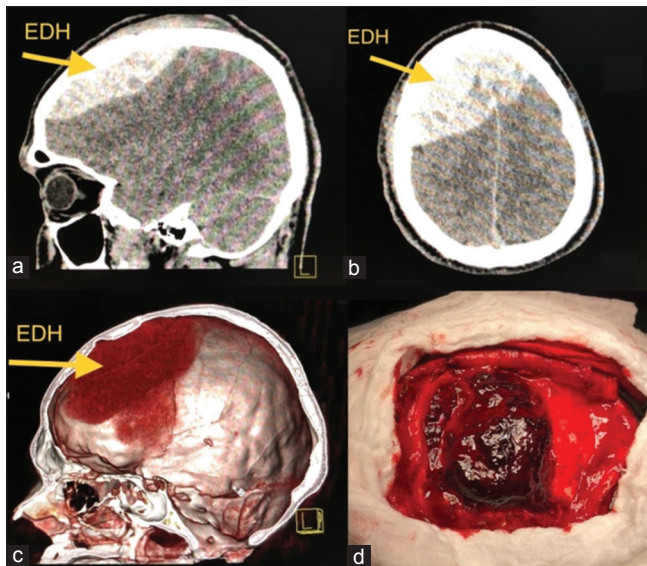
The classic clinical course, first described by Jacobson in 1886,<sup>[2]</sup> is an initial loss of consciousness after trauma, followed by recovery (lucid interval) and then (as the blood clot enlarges) a rapid progression of neurological symptoms: obtundation, contralateral hemiparesis, ipsilateral oculomotor nerve paresis, decerebrate rigidity, arterial hypertension, cardiac arrhythmias, respiratory disturbances, and finally apnea and death. The triad of lucid interval, contralateral hemiparesis, and dilatation of the ipsilateral pupil is usually present in patients with pure large EDHs. This triad not pathognomonic for EDHs, however, EDHs more often present in atypical rather than typical fashion.<sup>[13]</sup>

The “classic” lucid interval occurs in only 14% to 21% of cases.<sup>[14]</sup> The “classic” contralateral hemiparesis caused by the mass effect of EDH on underlying motor cortex occurs in 70% to 80% of cases.<sup>[15,16]</sup> Hemiparesis may be ipsilateral (Kernohan’s phenomenon) because the opposite cerebral peduncle is compressed against the tentorial edge (Kernohan’s notch), a false localizing sign. Temporal EDHs may cause rapid deterioration due to their proximity to the brain stem. EDHs originating from a laceration of the middle meningeal vein or dural sinus have slow clinical course.<sup>[13]</sup> In contrast, frontal or occipital EDH patients present with drowsiness with gradual progression to bilateral decerebrate rigidity, rather than contralateral hemiparesis. Patients with associated intradural lesions will have more severe and rapid neurological deterioration.<sup>[17,18]</sup> The “classic” ipsilateral dilated pupil, the last sign of classic triad, occurs in <50% of cases.<sup>[18]</sup>

Disturbance of the level of consciousness in infants is absent in about 50% of cases, yet at the time of injury.<sup>[19]</sup> Irritability is most commonly seen in infants, which might be due to headache as a result of raised ICP.<sup>[11]</sup> In infant, cephalohematoma is reported in 68% of cases by Ciurea *et al.*<sup>[11]</sup> Convulsion and bulging fontanel may be seen in 34% of cases. Anemia and bradycardia may be suspicious of EDH after exclusion of other sources of bleeding.<sup>[5]</sup> Large subgaleal hematoma is associated with anemia, hypotension, and shock.<sup>[20]</sup>

### Management

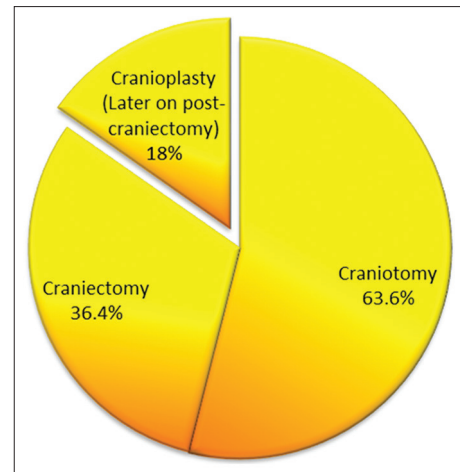
EDH should be suspected when there is a history of head injury even if it is trivial. General and neurological examination of



**Figure 3:** Right sided frontoparietal huge EDH crossing midline in 16 years age male. (a) Sagittal CT-scan view. (b) Axial CT-scan view. (c) three dimensional reconstruction of skull with EDH (d) Operative photograph

the child is mandatory. The diagnosis and management of EDH has been revolutionized by the advent of computed tomography (CT) scan, so that the results of studies are different between pre-CT and post-CT scan era. CT scan should be the first investigation to be performed in suspected cases. “Classic” CT appearance occurs in 84% of cases: high-density biconvex (lenticular) shape adjacent to the skull. In 11% of cases, the side against the skull is convex and that along the brain is straight and in 5% it is crescent shaped (resembling subdural hematoma) [Figures 1 and 3].

EDH is one of the most urgent neurosurgical emergencies that may need prompt evacuation.<sup>[21]</sup> Smaller EDH with exclusion of temporal or posterior fossa may be managed conservatively with careful observation.<sup>[22]</sup> Surgical evacuation by craniotomy or craniectomy indicated if EDH is more than 30 cc, irrespective of the patient’s Glasgow coma scale (GCS) score [Figure 4]. Nonoperative management can be chosen if EDH is <30 cc, <15 mm thick, and with less than a 5-mm midline shift and GCS score lower than 8 without focal deficit, provided that serial CT imaging and careful neurological monitoring facilitate.<sup>[21]</sup> Urgent surgical evacuation is strongly recommended for comatose patients with EDH (GCS score <9) and anisocoria; however, lower surgical threshold is recommended for EDH in the middle cranial fossa/inferotemporal lobe.<sup>[21]</sup> Rapid neurological deterioration or sign of brain herniation permits an initial emergency burr hole to be placed over the maximum thickness of the clot guided by CT image for urgent evacuation of clot to reduce ICP.<sup>[21]</sup> For posterior fossa EDHs causing mass effect by CT scan with neurological deterioration, surgery by suboccipital craniectomy with evacuation of hematoma is the treatment of choice [Figure 2]. Nonsurgical management is recommended when there are no mass effects on CT or sign of neurological dysfunction.<sup>[21]</sup>



**Figure 4:** Types of surgical procedures in the 110 operated cases

### Outcome

The reported mortality rate of EDH is 0% to 17% and even higher in posterior fossa EDH (26%).<sup>[3,8,11,22]</sup> Haselsberger *et al.* revealed that patients with pure EDH have excellent prognosis after surgical evacuation and have significantly better outcome and good recovery than those with associated intradural lesions.<sup>[17]</sup> Outcome of nonsurgical management has affected by many factors, including the size, location, configuration, and the rapidity of accumulation of the clot; the presence of associated intradural lesions; the extracranial decompression of blood through skull diastases; and the age of the patient.<sup>[23]</sup>

### MATERIALS AND METHODS

In this prospective study, 210 cases of pediatric EDH under the age of 18 years were studied during a 10-year period in the Neurosurgical Department in Hilla Teaching Hospital, Babylon, Iraq, from 2008 to 2018. This study considered the following parameters: characteristics of patients, clinical manifestations, mechanism of head injury with age-specific distribution, radiological findings, associated pathologies, management, source of bleeding in operated cases, correlated pathologies in dead patients, and outcome. The clinical data were collected by randomized selection of patients with clinically and radiologically proved EDH.

Patients in question undergo full assessment by history, examination, and investigations. CT scan was the investigation of choice for all cases. Surgery is done for 110 patients by craniotomy or craniectomy and three cases need an emergency burr hole for the decompression of EDH before final evacuation by craniotomy or craniectomy. Conservative management is decided for 100 patients by careful neurological monitoring and serial follow-up CT scans during their hospital stay. The outcome of the patients is assessed during hospital stay and followed from several months to several years later on according to variable patient compliance. Categorical variables are presented in the form of frequencies and percentages.

## Ethical consideration

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patient's verbal and analytical approval before the sample was taken. The study protocol and the patient information and consent form were reviewed and approved by the local ethics committee (Hilla Teaching Hospital and Babylon health directorate).

## RESULTS

In this study, the results of 210 pediatric head-injured patients with EDH are presented in the form of tables involving numbers of patients and their ratios.

### Clinical characteristics of patients

In this study, the highest age group of incidence for EDH is above 6–12 years, which constitutes 33% ( $n = 70$ ). The youngest patient is a female baby of 2 days of age with head trauma due to birth injury. Most of the victims are male (75%). Assessment of level of consciousness for pediatric patients in the emergency room shows that highest percentage of them (40%;  $n = 84$ ) were conscious on arrival. Table 1 shows the clinical characteristics of pediatric patients with EDH.

### Clinical manifestations

More than half of the patients (60%;  $n = 126$ ) have disturbed level of consciousness ranging from drowsiness to deep coma, and 7% ( $n = 14$ ) of the patients have lucid interval in their clinical presentation. Pupillary abnormalities are present in 17% ( $n = 35$ ) of cases and hemiparesis or hemiplegia in 27% ( $n = 57$ ). Most of the patients with EDH have scalp injuries (81%). Table 2 shows the clinical manifestations of pediatric patients with EDH.

### Mechanism of head injury with age-specific distribution

Accidental falling has the highest incidence of causative mechanisms 47.1% ( $n=99$ ) followed by road traffic accident 24.8% ( $n=52$ ), the age group >6-12 year have the higher incidence of accidental falling, road traffic accident and blunt head injury respectively. Table 3 shows the mechanism of head injury in pediatric patients with age-specific distribution.

### Radiological findings

In this study, thickness of EDH is employed to estimate the size in different locations. The highest incidence of EDH thickness is 5–9 mm (27%;  $n = 56$ ) and the least incidence is 55 mm (1%;  $n = 2$ ) [Figure 1]. The majority of EDH is supratentorial (91%;  $n = 192$ ), of which parietal EDH comprises the highest incidence (36%;  $n = 76$ ). Infratentorial (posterior fossa) comprises only 9% ( $n = 18$ ) [Figure 2]. It is found that EDH located more on the right side 53% ( $n=111$ ) than the left side 43% ( $n=90$ ), while 4% ( $n=9$ ) is bilateral. Our 210 studied patients had a total of 233 EDHs: 191 patients had a single EDH and 19 patients had two or more EDHs (ipsilateral in 10 cases and bilateral in 9 cases). Table 4 shows these radiological findings of pediatric patients with EDH.

**Table 1: Clinical characteristics of patients**

| Age          | <i>n</i> (%) | Male     | Female  | GCS in ER | <i>n</i> (%) |
|--------------|--------------|----------|---------|-----------|--------------|
| 0-1 month    | 4 (2)        | 2        | 2       | Conscious | 84 (40)      |
| >1-24 months | 25 (12)      | 14       | 11      | GCS=14-13 | 47 (22)      |
| >2-6 years   | 58 (28)      | 40       | 18      | GCS=12-9  | 58 (28)      |
| >6-12 years  | 70 (33)      | 54       | 16      | GCS=8-3   | 21 (10)      |
| >12-17 years | 53 (25)      | 48       | 5       |           |              |
| Total        | 210 (100)    | 158 (75) | 52 (25) |           |              |

GCS: Glasgow coma scale, ER: Emergency room

**Table 2: Clinical manifestations**

| Symptom and Sign            | <i>n</i> (%) |
|-----------------------------|--------------|
| Intracranial hypertension   | 52 (25)      |
| Psychomotor agitation       | 26 (12)      |
| Drowsiness                  | 105 (50)     |
| Coma (GCS=8-3)              | 21 (10)      |
| Lucid interval              | 14 (7)       |
| Pupillary abnormalities     | 35 (17)      |
| Hemiparesis/hemiplegia      | 57 (27)      |
| Dysphasia/aphasia           | 18 (9)       |
| Dysphagia                   | 1 (0.5)      |
| Paresthesia                 | 5 (2)        |
| Headache                    | 72 (34)      |
| Nausea/vomiting             | 84 (40)      |
| Dizziness/vertigo           | 24 (11)      |
| Ataxia                      | 2 (1)        |
| Seizure                     | 31 (15)      |
| Posttraumatic amnesia       | 8 (4)        |
| Anemia                      | 7 (3)        |
| Scalp injury                | 170 (81)     |
| Raccoon's eyes              | 30 (14)      |
| Subconjunctival hemorrhage  | 13 (6)       |
| Battle sign                 | 14 (7)       |
| Rhinorrhea                  | 2 (1)        |
| Epistaxis                   | 22 (10.5)    |
| Hearing loss                | 6 (3)        |
| Otorrhea                    | 2 (1)        |
| Otorrhagia                  | 26 (12)      |
| 7 <sup>th</sup> nerve palsy | 18 (9)       |
| 6 <sup>th</sup> nerve palsy | 5 (2)        |
| 3 <sup>th</sup> nerve palsy | 11 (5)       |
| Diplopia                    | 16 (8)       |
| Nystagmus                   | 1 (0.5)      |
| Blurring of vision          | 1 (0.5)      |
| Hypertension                | 21 (10)      |
| Hypotension                 | 35 (17)      |

GCS: Glasgow coma scale

### Associated pathologies

In this study, skull fracture is the most common associated pathology in pediatric patients with EDH (92.2%;  $n = 195$ ), of which linear fractures have the highest incidence (55%;  $n = 115$ ) followed by depressed fractures (22%;  $n = 47$ ). Intracranial hemorrhage comprises 33% ( $n = 69$ ) of the associated pathologies, of which intracerebral

**Table 3: Mechanism of head injury with age-specific distribution**

| Mechanism of injury     | 0-1 month | >1-24 months | >2-6 years | >6-12 years | >12-17 years | Total, n (%) |
|-------------------------|-----------|--------------|------------|-------------|--------------|--------------|
| Accidental falling      | 0         | 20           | 35         | 29          | 15           | 99 (47.1)    |
| Road traffic accident   | 0         | 0            | 9          | 23          | 20           | 52 (24.8)    |
| Blunt head injury       | 1         | 4            | 8          | 10          | 7            | 30 (14.3)    |
| Penetrating head injury | 0         | 1            | 6          | 8           | 11           | 26 (12.4)    |
| Birth trauma            | 3         | 0            | 0          | 0           | 0            | 3 (1.4)      |

**Table 4: Radiological findings**

| Thickness of EDH (mm) | n (%)   | Location of EDH                  | n (%)    | Side of EDH                  | n (%)        |
|-----------------------|---------|----------------------------------|----------|------------------------------|--------------|
| 5-9                   | 56 (27) | Supratentorial                   | 192 (91) | Right                        | 111 (53)     |
| 10                    | 46 (22) |                                  |          | Left                         | 90 (43)      |
| 11-15                 | 36 (17) | Frontal                          | 48 (23)  | Bilateral                    | 9 (4)        |
| 16-20                 | 27 (13) | Temporal                         | 25 (12)  | <b>Multi-ipsilateral EDH</b> | <b>n (%)</b> |
| 21-25                 | 17 (8)  | Parietal                         | 76 (36)  | 2 Ipsilateral                | 7 (3)        |
| 26-30                 | 10 (5)  | Temporoparietal                  | 24 (11)  | 3 Ipsilateral                | 2 (1)        |
| 31-40                 | 9 (4)   | Frontoparietal                   | 12 (6)   | 4 Ipsilateral                | 1 (0.5)      |
| 41-50                 | 7 (3)   | Parieto-occipital                | 7 (3)    |                              |              |
| 55                    | 2 (1)   | Infratentorial (posterior fossa) | 18 (9)   |                              |              |

EDH: Epidural hematoma

hematomas have the highest incidence (14%;  $n = 30$ ). Table 5 shows the associated pathologies in pediatric patients with EDH.

### Types of surgical procedures in operated cases

In this study, 52.4% ( $n = 110$ ) of pediatric patients undergo surgical evacuation for EDH, of which craniotomy is the procedure of choice (63.6%;  $n = 70$ ) followed by craniectomy (36.4%;  $n = 40$ ), and half of craniectomy patients undergo cranioplasty for their cranial defect created by craniectomy. Figure 4 shows the types of surgical procedures in the 110 operated cases.

### Source of bleeding in the operated cases

In this study, middle meningeal artery or vein is identified as a source of bleeding in half of the operated cases. Figure 5 shows the source of bleeding in the 110 operated cases.

### Correlated pathologies in dead patients

In this study, correlated pathologies are present in all dead EDH cases. Table 6 shows the correlated pathologies in 14 dead patients.

### Outcome

In this study, Glasgow outcome scale has been applied to assess the outcome of our EDH patients after trauma and to compare the outcome in both operatively and conservatively managed groups. Table 7 shows the outcome.

## DISCUSSION

EDHs in the pediatric age group constitute a different clinical entity from that in adult life due to their nonspecific clinical presentation and the difficulty in obtaining history from the young children.

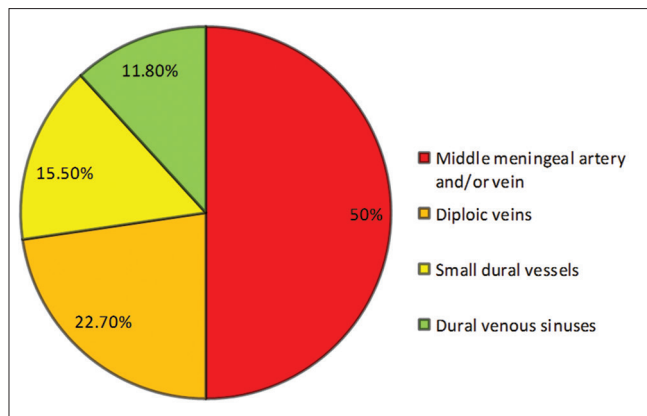
**Table 5: Associated pathologies**

| Type of lesion            | n (%)    |
|---------------------------|----------|
| Linear fracture           | 115 (55) |
| Comminuted fracture       | 9 (4)    |
| Depressed fracture        | 47 (22)  |
| Diastatic fracture        | 10 (5)   |
| Orbital fracture          | 11 (5.2) |
| Facial bones fractures    | 3 (1)    |
| Subdural hematoma         | 10 (5)   |
| Intracerebral hematoma    | 30 (14)  |
| SAH                       | 23 (11)  |
| IVH                       | 6 (3)    |
| Brain contusion           | 31 (15)  |
| Brain edema               | 19 (9)   |
| Intracranial bullet/shell | 10 (5)   |
| Subgaleal hematoma        | 21 (10)  |
| Intracranial aerocoeles   | 21 (10)  |
| Hydrocephalus             | 1 (0.5)  |
| Meningitis                | 1 (0.5)  |
| Fractures of upper limb   | 5 (2)    |
| Fractures of lower limb   | 5 (2)    |
| Pneumothorax              | 2 (1)    |
| Hemothorax                | 3 (1.4)  |
| Lung contusion            | 3 (1.4)  |
| Visceral injury           | 3 (1.4)  |
| Electric shock            | 1 (0.5)  |
| Multiple burns            | 3 (1.4)  |
| Neonatal Jaundice         | 1 (0.5)  |

SAH: Subarachnoid hemorrhage, IVH: Intraventricular hemorrhage

### Clinical characteristics of patients

The age group of above 6 years to 12 years has the highest incidence for EDH (33%) in this study; Lobato *et al.*



**Figure 5:** Source of bleeding in the 110 operated cases

**Table 6: Correlated pathologies in 14 dead patients**

| Correlated pathologies      | n (%)   |
|-----------------------------|---------|
| Brain edema                 | 10 (71) |
| Intracerebral hemorrhage    | 9 (64)  |
| Brain contusion             | 8 (57)  |
| Subarachnoid hemorrhage     | 7 (50)  |
| Subdural hematoma           | 6 (43)  |
| Intraventricular hemorrhage | 4 (29)  |
| Fracture of long bones      | 3 (21)  |
| Postoperative meningitis    | 1 (7)   |
| Hemophilia                  | 1 (7)   |

**Table 7: Outcome**

| GOS   | n (%)     | Operative cases (%) | Conservative cases (%) |
|-------|-----------|---------------------|------------------------|
| D     | 14 (6.7)  | 5 (2.4)             | 9 (4.3)                |
| PVS   | 3 (1.4)   | 0 (0)               | 3 (1.4)                |
| SD    | 10 (4.8)  | 3 (1.4)             | 7 (3.3)                |
| MD    | 38 (18.1) | 17 (8.1)            | 21 (10)                |
| GR    | 145 (69)  | 80 (38)             | 65 (31)                |
| Total | 210 (100) | 110 (52.4)          | 100 (47.6)             |

GOS: Glasgow outcome scale, D: Dead, PVS: Persistent vegetative state, SD: Severe disability, MD: Moderate disability, GR: Good recovery

reported that the age group of 10–19 years has the highest incidence (26%).<sup>[24]</sup> In this study, the incidence of EDH cases increases in a direct proportionate manner with increasing age up to 12 years and decline in older ages and least incidence belongs to the age group of 0–1 month (2%;  $n = 4$ ).

Most of the victims are males probably because they are more vulnerable to trauma than females, which is similar to other studies; this is more apparent in our study in patients above 12–17 years of age for which the majority of victims are male; however, the age group of 0–1 month has equal sex distribution probably because of equivalent risk factors. Noteworthy, there is male predominance in school-age group in this study. In comparison with other studies, a study done by Jamieson and Yelland, male: female ratio has been (4:1) with male predominance.<sup>[22]</sup> A study done in Hong Kong

by Kwan–Hon Chan et al has shown that males constitute 84.62% of the victims while females constitute only 15.38% of EDH victims.<sup>[25]</sup> Browne and Lam also reported overall male preponderance.<sup>[26]</sup>

The highest ratio of victims 40% were conscious on arrival to the hospital, this is probably due to rapid referral to hospital. Jamieson and Yelland mentioned in their study that 30% of their patients do not give a history of loss of consciousness.<sup>[22]</sup> Tomlinson also stated that 25% of his patients do not lose consciousness at the time of impact.<sup>[27]</sup> Lobato *et al.* reported that 35% of their patients do not lose consciousness.<sup>[24]</sup>

### Clinical manifestations

In this study, most of the patients are presented with two or more complaints and 60% of them have disturbed consciousness ranging from drowsiness to deep coma, whereas in Nath *et al.*'s study, up to 70.76% have disturbed consciousness.<sup>[28]</sup> Choux *et al.* reported that 85% of infants with EDH have no loss of consciousness at the time of impact and 7% have no alteration in consciousness at any time following injury.<sup>[3]</sup> Lucid interval is present in 7% of our patients. Lucid interval is reported to be an uncommon manifestation by Jamieson,<sup>[1]</sup> 10% by Lobato *et al.*'s series,<sup>[24]</sup> 18% by Cordobés *et al.*'s study,<sup>[10]</sup> less than one-third by Samudrala and Cooper,<sup>[13]</sup> 10%–27% by McKissock *et al.*,<sup>[29]</sup> and 32% by Paşaoğlu *et al.*<sup>[19]</sup>

In this study, pupillary abnormalities are present in only 17% of cases, in which 13% have unilateral dilated pupil and in 4% bilateral dilated pupils. Cordobés *et al.* reported that 35% have unilateral dilated pupil.<sup>[10]</sup> Most of the patients (73%) have no hemiparesis or hemiplegia on proper neurological examination. Classical contralateral hemiparesis and hemiplegia are present in 27% of cases, 21% have hemiparesis, and 6% have hemiplegia. Cordobés *et al.* found that 12% of cases have full muscle power and no focal sign.<sup>[10]</sup> Jameison and Yelland have found that hemiplegia is present in one-third of cases.<sup>[22]</sup> Reale *et al.* and Hooper have found the classical contralateral hemiparesis in 70%–80% of cases.<sup>[15,16]</sup> Dysphasia and aphasia are present in 9% of cases. No case of dysarthria is found in this study. Cordobés *et al.* have found that dysarthria is present in 7.3% of cases.<sup>[10]</sup>

Nausea and vomiting have a relatively high incidence in the pediatric age group (40%), but it is even higher in Nath *et al.*'s study reaching up to 80% of cases.<sup>[28]</sup> Headache is the presenting complaint in 34% of cases. Miller *et al.* mentioned that acute EDH patients may be present with only minimal complaints such as headache, nausea, and vomiting.<sup>[30]</sup> Zhong *et al.* reported headache and vomiting as one of the common symptoms.<sup>[31]</sup> Posttraumatic seizure is found in 15% of cases in the present study, and the incidence is 7.6% in Nath *et al.*'s study<sup>[28]</sup> and 4.8% in Cordobés *et al.*'s study.<sup>[10]</sup> Posttraumatic amnesia is present in 4% of cases. Pallor due to anemia is present in 3% ( $n = 7$ ) of all cases, in which 85.7% ( $n = 6$ ) of pallor cases are infants. Pallor and anemia are reported in 96% of cases by Ersahin *et al.*<sup>[9]</sup> and 90% of cases by Paşaoğlu *et al.*<sup>[19]</sup> Anemia has been identified as an important laboratory finding in infants with acute EDH.<sup>[3,8,9,29]</sup>

Although symptoms and sign of intracranial hypertension are present in 25% of cases of this study, no case of papilledema is found in this study. Papilledema is present in 4.8% of the cases in Cordobés *et al.*'s study.<sup>[10]</sup> In this study, scalp injuries include abrasions, cutting injuries, penetrating injuries, scalp avulsion, and subgaleal hematoma. Scalp injuries are present in 81% of our cases, which is very close to Cordobés *et al.*'s study (81.7%).<sup>[10]</sup>

Facial nerve palsy is present in 9% of cases: 5% are upper motor neuron and 4% are lower motor neuron. Jamieson mentioned that facial nerve involvement is not rare in EDH.<sup>[11]</sup> Oculomotor nerve palsy is present in 5% of cases and abducens nerve palsy present in 2% of cases. Hypotension is detected in 17% of patients mostly due to blood loss from associated injuries rather than head injury itself and 10% of patients are hypertensive. Only 11 cases are in shock state.

### Mechanism of head injury with age-specific distribution

Accidental falling comprises the highest incidence (47.1%), followed by RTA as the second highest incidence (24.8%) of the causative mechanisms of injury in this study. Penetrating head injuries in this study comprise 12.4%, including bullet injuries, shell injuries, and stab injuries by knives or any sharp tool. Birth trauma is the causative mechanism in three victims out of four newborns. On 1886, Jacobson reported accidental falling in 70% of the cases, blows in 18%, and RTA in 7%.<sup>[12]</sup> Hooper has found accidental falling in 39% of the cases, RTA in 35%, and blows in 16%.<sup>[16]</sup> Jamieson and Yelland have found that 45% are due to RTA.<sup>[22]</sup> Chan *et al.*'s study showed that accidental falling constitutes 45%, quite close to the results of this study, RTA constitutes 37%, and hit by heavy object constitutes 18%, which are almost all sport injuries.<sup>[25]</sup> Beni-Adani *et al.* reported that fall from height was responsible for the development of an EDH in 63.6% of their cases.<sup>[32]</sup> Paşaoğlu *et al.* found that falls were the most common underlying mechanism in 63% of their pediatric cases.<sup>[19]</sup> Ersahin *et al.* identified falls as the most common mechanism of injury in 62% of their pediatric cases.<sup>[9]</sup> On the contrary, Rocchi *et al.* found that RTA is the most common cause of EDH in their series.<sup>[12]</sup> It is interesting that most of accidental falling cases in our study were related to accidents at home or within a familial environment. In this study, accidental falling has higher incidence from 2 to 6 years whereas RTA has higher incidence from 6 to 12 years, and this is consistent with other studies.<sup>[32,33]</sup> Jamous *et al.* reported toddlers and children to be more vulnerable for EDH due to the decrease of fibrovascular attachments between dura and skull as compared with infants where EDH is rare.<sup>[34]</sup>

### Radiological findings

CT scan is the imaging investigation of choice for detailed analysis of EDH location, volume, thickness, and mass effect on the midline structures, ventricular system, and basal cisterns. All the patients in this study undergo CT scan, which has an essential role for diagnosis, monitoring, and choosing treatment modality. 58% of patients have repeated CT scan studies. Close clinical monitoring and possible serial CT studies are mandatory during the first 24–36 h.<sup>[35]</sup> In this study,

the final size of EDH is estimated from the maximum thickness of EDH, which is obtained by one or more CT scan study.

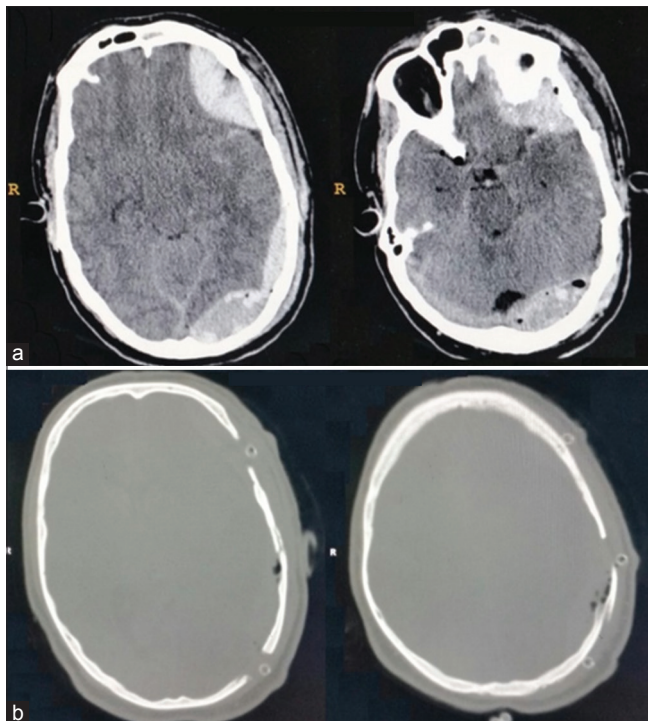
Supratentorial EDH accounts for 91% with higher incidence of parietal EDH in 36% followed by frontal EDH in 23% of all cases of EDH [Figures 1 and 3]. This is consistent with other studies in which regional preponderance toward parietal region is found.<sup>[36,37]</sup> Huisman and F.T.C. Tschirch in their study also found that the majority of the EDH were located in the frontal and parietal region (38% and 43% respectively).<sup>[38]</sup> This difference could be explained by the fact that pediatric skulls are different from those of adults. In children, the frontoparietal-to-temporo-occipital ratio is more than that in adults, which increases the incidence of traumatic frontoparietal impact.<sup>[38]</sup> Rocchi *et al.* reported that temporal EDH is rare in children.<sup>[12]</sup> This may be attributed to the fact that indentation of middle meningeal artery in temporal bone of adults is not seen in children. Jamieson found that 60%–70% of all EDHs are distributed in the lateral walls of the skull.<sup>[11]</sup> Hooper showed that temporoparietal EDH is the most common type and constitutes 65.18% of all EDHs.<sup>[16]</sup> Lobato *et al.* showed that EDH is distributed as follows: 48% parietal, 20% temporoparietal, 12% temporal, 17% frontal, and only 1% in the posterior fossa.<sup>[24]</sup>

Infratentorial (posterior fossa) EDHs account only 9% of all cases of EDH [Figure 2]. In other series, posterior fossa EDH constitutes 4% by McKissock *et al.*,<sup>[29]</sup> 7.5% by Wang *et al.*,<sup>[39]</sup> and 12.9% by Zuccarello *et al.*<sup>[40]</sup> This may be due to the fact that dura attaches more firmly to the posterior cranial fossa as compared to anterior and middle cranial fossae.

More than half of all EDHs present the right side (53%) whereas the left side constitute (43%) and (4%) were bilateral EDHs. Ciurea *et al.* found that overall right side is more frequently involved.<sup>[11]</sup> Gerlach *et al.*<sup>[36]</sup> and Ben Abraham *et al.*<sup>[37]</sup> reported that right parietal region contributes more than half of cases, which is consistent with our study. In contrast, Nath *et al.*<sup>[28]</sup> and Paiva *et al.*<sup>[41]</sup> found that left side is commonly affected than the right side. Multiple EDHs also found in this study, bilateral EDHs is found in 9 patients and multi-Ipsilateral EDHs found in 10 patients. Multiple EDHs characterized by lower GCS at presentation and rapid neurological deterioration than single EDH [Figure 2 and 6]. In comparison with other study, double EDH may be unilateral or bilateral and accounts for 2%–25% of all EDHs.<sup>[42]</sup>

### Associated pathologies

In our study, skull fracture is the most common associated pathology (92.2%) of all cases, of which linear fractures have the highest incidence (55%), followed by depressed fracture (22%). Jennett *et al.* stated that EDH is almost always associated with a skull fracture, which included 91% of adult cases and 75% of pediatric cases.<sup>[43]</sup> Lobato *et al.* found that the skull fracture is present in 80% of EDH cases.<sup>[24]</sup> Chan *et al.* found that the skull fracture is present in 100% of EDH cases.<sup>[25]</sup> Samudrala and Cooper mentioned that, in adults, a skull fracture is present 90%



**Figure 6:** Left sided multi-ipsilateral EDHs in 17 years male brain CT-scan. (a) CT-scan before surgery. left sided multi-ipsilateral EDHs with comminuted fractures in frontal, temporal, parietal and occipital regions. (b) CT-scan after surgery. Surgical evacuation is done by 3 mini craniectomies at frontotemporal, parietal and occipital regions

of the cases, although it is less common in children.<sup>[13]</sup> Teasdale mentioned that tearing of the middle meningeal artery due to a temporoparietal skull fracture results in arterial hemorrhage into the epidural space in more than 50% of the cases.<sup>[44]</sup>

About one-third of cases of EDHs (33%) have another one or more intracranial hemorrhages, of which intracerebral hematoma has the highest incidence (14%), followed by subarachnoid hemorrhage (11%). Jamieson and Yelland. asserted that, in EDH, associated brain lesions are less common than with subdural hematomas.<sup>[22]</sup> Subdural hematoma is present in (5%) of cases of EDH much less than Australian series in infants which comprise (14.3%).<sup>[26]</sup> Associated extracranial injuries are found in 11.6% of cases in our study as compared to 85% of Browne and Lam's study<sup>[26]</sup> and even more rare in Gerlach *et al.*'s study.<sup>[36]</sup> No spinal injury was found in our study.

### Management

In this study, surgical intervention is done for 110 cases of EDH and the rest 100 cases have been managed conservatively by careful neurological monitoring and serial follow-up CT scans during their hospitalization in our neurosurgical department. Minor EDH cases with less than 15 mm thickness, less than 5 mm midline shift, less than 30 ml volume by CT-scan and a GCS score greater than 8 without focal deficit, conservative management by serial CT scanning and close observation in a neurosurgical center can done with successful

outcome.<sup>[45-47]</sup> Pickard *et al.* revealed that EDH evacuation is the most "cost-effective" surgical procedure with excellent outcome.<sup>[48]</sup> In this study, two reliable categories for doing surgery are employed: (1) clinical evaluation – GCS score lower than 8 and presence of neurological deficits, (2) CT-scan findings: EDH more than 15mm thickness, midline shift more than 5mm, which are comparable with surgical indications in other previous studies.<sup>[46]</sup> In posterior fossa EDH; mass effect reveals by obliteration of basal cisterns, displacement of the fourth ventricle and presence of hydrocephalus, which are also comparable with surgical indications in other previous studies.<sup>[46]</sup> Craniotomy is the procedure of choice for 63.6% of cases, followed by craniectomy 36.4% of cases and half of craniectomy patients have large cranial defect that need cranioplasty later on [Figure 4]. Multiple mini-craniectomy is also done for cases of bilateral and multi-ipsilateral EDHs [Figure 6]. All operated infratentorial (Posterior fossa) EDHs have undergone craniectomies, which is proposed as the standard surgical management<sup>[49]</sup> and none of them demand cranioplasty even with large cranial defects. The majority of EDHs associated with depressed fracture and/or comminuted fracture undergo craniectomy.

The youngest patient who undergo surgery is a 2 days age female baby with birth injury during caesarean section presented with left parietal closed Ping-Pong depressed fracture and EDH on the same location, in which craniotomy is done for her with free bone flap including the Ping-Pong depressed fracture with evacuation of EDH then elevation of depressed fracture in the free bone flap before returning back to its bed. In this study, 85% of operative cases undergo surgery within the first 24 h of trauma, in which rapid progressing and life-threatening EDH undergo urgent surgical evacuation and most of them have full recovery. Only 46% of cases operated within 24 h by Jamjoom *et al.*'s study.<sup>[50]</sup> Rapid spontaneous resolution of EDH has been reported in five pediatric cases in our study; all of them have skull fractures. It has been suggested that communication between intracranial and epicranial spaces created by the existence of skull fracture and the pressure gradient provoked by increased intracranial pressure force the hematoma out of epidural space through fracture line.<sup>[51]</sup> Additional cerebral lesions is found in 72.5% of our cases, much less incidence than other series.<sup>[12,34,36]</sup> Active surgical management for associated pathologies is done for all surgical and nonsurgical cases of EDH; one case has intraventricular hemorrhage with hydrocephalus undergo V-P shunt operation, and other cases need extraction of bullets and shells from inside the brain, evacuation of subdural hematomas, fixation of facial bone fractures, fixation of upper and lower limbs fractures, chest tubes, thoracotomies, laparotomies, and tracheostomies.

### Source of bleeding in the operated cases

Middle meningeal artery and/or vein are identified as a source of bleeding in half of operative EDH cases [Figure 5]. The rest of cases constitute 22.7% from diploic veins, 15.5% from small dural vessels, and 11.8% from dural venous sinuses [Figure 5].

Jamieson found that the source of bleeding is mainly middle meningeal artery followed by dural venous sinuses.<sup>[1]</sup> Samudrala and Cooper pointed out that EDH arises from the middle meningeal artery in over one-half of patients, from the middle meningeal vein in one-third, and from diploic veins or a torn dural venous sinus in the remainder.<sup>[13]</sup> In contrast to single EDH, bilateral EDH is most often venous in origin.<sup>[52]</sup>

### Correlated pathologies in dead patients

In this study, correlated pathologies are present in all dead EDH cases. The most common associated pathologies are brain edema (71%), followed by intracerebral hemorrhage (64%), brain contusion (57%), and subarachnoid hemorrhage (50%).

### Outcome

In this study, Glasgow outcome scale has been applied to assess the outcome of our patients after trauma. Good recovery with no disability is reported in 69% of our patients, of which 38% of operative cases and 31% of conservative cases; most of them were conscious at the time of presentation. Moderate disability reported in 18.1% of patients, of which 8.1% in operative cases and 10% of conservative cases. Severe disability is reported in 4.8% patients, of which 1.4% in operative cases and 3.3% of conservative cases. None of the operative cases have persistent vegetative state; only it happened in three conservatively managed cases. In this study, high incidence of death, persistent vegetative state, and moderate and severe disability are reported in conservatively managed cases, this can explain by the fact that all of these cases have associated intracranial and/or extracranial pathologies and were comatose on presentation. Associated intracranial and/or extracranial pathologies have detrimental effects on general neurological status and final recovery, these findings are comparable with other study.<sup>[14]</sup> Cagetti *et al.* stated that patients who are alert or slightly lethargic at the time of operation should have virtually no mortality, while those who are unconscious with brain stem signs will have a mortality of well over 50%.<sup>[53]</sup> Rivas *et al.*<sup>[18]</sup> and Bullock *et al.*<sup>[47]</sup> reported worse outcomes in EDH cases who undergo surgical intervention in association with SDH and/or parenchymal lesions.

Overall mortality in this study is 6.7% ( $n = 14$ ), all of them were unconscious at the time of presentation and had associated intracranial and/or extracranial pathologies. Mortality is less in operative cases (2.4%) as compared to conservatively managed cases (4.3%). Mortality rate varies in different literature from 0% to 12% and even higher in pre-CT era.<sup>[8,36,47,53]</sup> Lobato *et al.* showed that the mortality of GCS = 7 or less is around 75%.<sup>[24]</sup> Servadei cited that the mortality rate varies: 0% for patients who are not in coma, 9% for obtunded patients, and 20% for patients in deep coma.<sup>[54]</sup> Mortality in our study is strongly associated with male gender; only three females died. West China hospital study reported that 66% of dead cases were male and majority of the deaths were due to accidental fall,<sup>[31]</sup> which is nearly comparable with our study. Half of dead cases have GCS = 3–6 at presentation, and the other half have GCS = 9–11. All EDH dead cases presented with 20–55 mm EDH thickness and

temporal epicenter is most common location although extension to adjacent frontal and/or parietal region is also common. Three cases have bilateral EDHs, which include one bifrontal EDH, one bitemporal EDH, and one bilateral posterior fossa EDH. Most of the deaths happened in the first 72 h.

### CONCLUSION

In pediatric head injuries, EDH is a life-threatening entity that may encounter even after minor head trauma and in asymptomatic patients, especially in infants. So that, special attention and a high index of suspicion is required in children presented with irritability, scalp hematoma, pallor, and a history of loss of consciousness. Routine CT examinations for any head injury increase the chance of detection of EDH. Both surgical and conservative management should have excellent outcome if they accomplished on solid basis. Clinical and neuroimaging evaluation have a fundamental role for optimal therapeutic decision. Conservative management need close neurological observation and periodic CT exams with volumetric analysis of EDH, for that reason, it should be achieved in specialized neurosurgical centers. Judicious surgical intervention based on clear clinical and neuroimaging indications can result in excellent long-term outcome. In borderline cases (EDH thickness 10–15 mm, midline shift <5 mm, GCS score 9–12 with mild neurological deficits, and in cases of posterior fossa EDH with mild mass effect and mild dilated ventricles), surgical evacuation of EDH in optimal circumstances may spent less hospital stay time with better cost–benefit ratio than conservative management. Age and sex of the patients, clinical and radiological manifestations, mechanism and severity of head injury, associated pathologies, and mode of management all influence the outcome. Mortality correlated mainly with low GCS and delay neurosurgical management, so that early detection and proper management can reduce the mortality and improve the outcome.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

### REFERENCES

1. Jamieson KG. Epidural hematoma. In: Vin Ken PJ, Bruyn GW, editors. *Injuries of Brain and Skull Part II: Handbook of Clinical Neurology*. Vol. 24. Amsterdam: North Holland; 1976. p. 261-74.
2. Jacobson W. On middle meningeal hemorrhage. *Guys Hosp Rep* 1886;43:147-308.
3. Choux M, Grisoli F, Peragut JC. Extradural hematomas in children 104 cases. *Childs Brain* 1975;1:337-47.
4. Stippier M. Craniocerebral trauma. In: Daroff RB, Jankovic J, Mazziotta JC, Pomeroy SL, editors. *Bradley's Neurology in Clinical Practice*. 7<sup>th</sup> ed., Ch. 62. Philadelphia PA: Elsevier; 2016.
5. Greenberg MS. *Handbook of Neurosurgery*. 7<sup>th</sup> ed. New York: Thieme Medical Publishers; 2010. p. 894-6.
6. Blumbergs PC. Neuropathology of traumatic brain injury. In: Richard Winn H, editor. *Youmans Neurological Surgery*. 6<sup>th</sup> ed.

- Philadelphia: Elsevier Saunders; 2011. p. 3294.
7. McCormick WF. Pathology of closed head injury. In: Wilkins RH, Rengachary SS, editors. *Neurosurgery*. 2<sup>nd</sup> ed. New York: McGraw Hill; 1996. p. 2642-43.
8. Dhellemmes P, Lejeune JP, Christiaens JL, Combelles G. Traumatic extradural hematomas in infancy and childhood. Experience with 144 cases. *J Neurosurg* 1985;62:861-4.
9. Erşahin Y, Mutluer S, Güzelbag E. Extradural hematoma: Analysis of 146 cases. *Childs Nerv Syst* 1993;9:96-9.
10. Cordobés F, Lobato RD, Rivas JJ, Muñoz MJ, Chillón D, Portillo JM, *et al*. Observations on 82 patients with extradural hematoma. Comparison of results before and after the advent of computerized tomography. *J Neurosurg* 1981;54:179-86.
11. Ciurea AV, Kapsalaki EZ, Coman TC, Roberts JL, Robinson JS 3<sup>rd</sup>, Tascu A, *et al*. Supratentorial epidural hematoma of traumatic etiology in infants. *Childs Nerv Syst* 2007;23:335-41.
12. Rocchi G, Caroli E, Raco A, Salvati M, Delfini R. Traumatic epidural hematoma in children. *J Child Neurol* 2005;20:569-72.
13. Samudrala S, Cooper PR. Traumatic intracranial hematoma. In: Wilkins RH, Rengachary SS, editors. *Neurosurgery*. 2<sup>nd</sup> ed. New York: McGraw Hill; 1996. p. 2802.
14. van den Brink WA, Zwienenberg M, Zandee SM, van der Meer L, Maas AI, Avezaat CJ, *et al*. The prognostic importance of the volume of traumatic epidural and subdural haematomas revisited. *Acta Neurochir (Wien)* 1999;141:509-14.
15. Reale F, Delfini R, Mencattini G. Epidural hematomas. *J Neurosurg Sci* 1984;28:9-16.
16. Hooper R. Observations on extradural haemorrhage. *Br J Surg* 1959;47:71-87.
17. Haselsberger K, Pucher R, Auer LM. Prognosis after acute subdural or epidural haemorrhage. *Acta Neurochir (Wien)* 1988;90:111-6.
18. Rivas JJ, Lobato RD, Sarabia R, Cordobés F, Cabrera A, Gomez P, *et al*. Extradural hematoma: Analysis of factors influencing the courses of 161 patients. *Neurosurgery* 1988;23:44-51.
19. Paşaoğlu A, Orhon C, Koç K, Selçuklu A, Akdemir H, Uzunoğlu H. Traumatic extradural haematomas in pediatric age group. *Acta Neurochir (Wien)* 1990;106:136-9.
20. Luerksen TG. Head injuries in children. *Neurosurg Clin N Am* 1991;2:399-410.
21. Chris Zacko J, Harris L, Ross Bullock M. Surgical management of traumatic brain injury. In: Richard Winn H, editor. *Youmans Neurological Surgery*. 6<sup>th</sup> ed. Philadelphia: Elsevier Saunders; 2011. p. 3427-36.
22. Jamieson KG, Yelland JD. Extradural hematoma. Report of 167 cases. *J Neurosurg* 1968;29:13-23.
23. Pang D, Horton JA, Herron JM, Wilberger JE Jr., Vries JK. Nonsurgical management of extradural hematomas in children. *J Neurosurg* 1983;59:958-71.
24. Lobato RD, Rivas JJ, Cordobés F, Altad E, Perez C, Sarabia R, *et al*. Acute epidural hematoma: An analysis of factors influencing the outcome of patients undergoing surgery in coma. *J Neurosurg* 1988;68:48-57.
25. Chan KH, Mann KS, Yue CP, Fan YW, Cheung M. The significance of skull fracture in acute traumatic intracranial hematomas in adolescents: A prospective study. *J Neurosurg* 1990;72:189-94.
26. Browne GJ, Lam LT. Isolated extradural hematoma in children presenting to an emergency department in Australia. *Pediatr Emerg Care* 2002;18:86-90.
27. Tomlinson BE. Brain stem lesions after head injuries. *Journal of Clinical Pathology* 1970;(Supplement)4:154-65.
28. Nath PC, Mishra SS, Das S, Deo RC. Supratentorial extradural hematoma in children: An institutional clinical experience of 65 cases. *J Pediatr Neurosci* 2015;10:114-8.
29. McKissock W, Taylor JC, Bloom WH, Till K. Extradural hematoma observations of 125 cases. *Lancet* 1960;2:167-72.
30. Miller JD, Murray LS, Teasdale GM. Development of a traumatic intracranial hematoma after a "minor" head injury. *Neurosurgery* 1990;27:669-73.
31. Zhong W, Sima X, Huang S, Chen H, Cai B, Sun H, *et al*. Traumatic extradural hematoma in childhood. *Childs Nerv Syst* 2013;29:635-41.
32. Beni-Adani L, Flores I, Spektor S, Umansky F, Constantini S. Epidural hematoma in infants: A different entity? *J Trauma* 1999;46:306-11.
33. Binder H, Majdan M, Tiefenboeck TM, Fochtmann A, Michel M, Hajdu S, *et al*. Management and outcome of traumatic epidural hematoma in 41 infants and children from a single center. *Orthop Traumatol Surg Res* 2016;102:769-74.
34. Jamous MA, Abdel Aziz H, Al Kaisy F, Eloqayli H, Azab M, Al-Jarrah M. Conservative management of acute epidural hematoma in a pediatric age group. *Pediatr Neurosurg* 2009;45:181-4.
35. Durham SR, Liu KC, Selden NR. Utility of serial computed tomography imaging in pediatric patients with head trauma. *J Neurosurg* 2006;105:365-9.
36. Gerlach R, Dittrich S, Schneider W, Ackermann H, Seifert V, Kieslich M. Traumatic epidural hematomas in children and adolescents: Outcome analysis in 39 consecutive unselected cases. *Pediatr Emerg Care* 2009;25:164-9.
37. Ben Abraham R, Lahat E, Sheinman G, Feldman Z, Barzilai A, Harel R, *et al*. Metabolic and clinical markers of prognosis in the era of CT imaging in children with acute epidural hematomas. *Pediatr Neurosurg* 2000;33:70-5.
38. Huisman TA, Tschirch FT. Epidural hematoma in children: Do cranial sutures act as a barrier? *J Neuroradiol* 2009;36:93-7.
39. Wang EM, Lim AT, Yeo TT. Traumatic posterior fossa extradural haematomas: A study of 17 cases. *Singap Med J* 1998;39:107-11.
40. Zuccarello M, Pardatscher K, Andrioli GC, Fiore DL, Iavicoli R, Cervellini P, *et al*. Epidural hematomas of the posterior cranial fossa. *Neurosurgery* 1981;8:434-7.
41. Paiva WS, Andrade AF, Mathias Júnior L, Guirado VM, Amorim RL, Magrini NN, *et al*. Management of supratentorial epidural hematoma in children: Report on 49 patients. *Arq Neuropsiquiatr* 2010;68:888-92.
42. Huda MF, Mohanty S, Sharma V, Tiwari Y, Choudhary A, Singh VP. Double extradural hematoma: An analysis of 46 cases. *Neurol India* 2004;52:450-2.
43. Jennett B, Teasdale G, Galbraith S, Pickard J, Grant H, Braakman R, *et al*. Severe head injuries in three countries. *J Neurol Neurosurg Psychiatry* 1977;40:291-8.
44. Teasdale G. Traumatic acute intracranial hematoma: Comment. In Palmer J editor. *Manual of Neurosurgery*. New York: Churchill Livingstone; 1996. P 544.
45. Chen TY, Wong CW, Chang CN, Lui TN, Cheng WC, Tsai MD, *et al*. The expectant treatment of "asymptomatic" supratentorial epidural hematomas. *Neurosurgery* 1993;32:176-9.
46. Knuckey NW, Gelbard S, Epstein MH. The management of "asymptomatic" epidural hematomas. A prospective study. *J Neurosurg* 1989;70:392-6.
47. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, *et al*. Surgical management of acute epidural hematomas. *Neurosurgery* 2006;58:S7-15.
48. Pickard JD, Bailey S, Sanderson H, Rees M, Garfield JS. Steps towards cost-benefit analysis of regional neurosurgical care. *BMJ* 1990;301:629-35.
49. Malik NK, Makhdoomi R, Indira B, Shankar S, Sastry K. Posterior fossa extradural hematoma: Our experience and review of the literature. *Surg Neurol* 2007;68:155-8.
50. Jamjoom A, Cummins B, Jamjoom ZA. Clinical characteristics of traumatic extradural hematoma: A comparison between children and adults. *Neurosurg Rev* 1994;17:277-81.
51. Tataryn Z, Botsford B, Riesenburger R, Kryzanski J, Hwang S. Spontaneous resolution of an acute epidural hematoma with normal intracranial pressure: Case report and literature review. *Childs Nerv Syst* 2013;29:2127-30.
52. Frank E, Berger TS, Tew JM Jr. Bilateral epidural hematomas. *Surg Neurol* 1982;17:218-22.
53. Cagetti B, Cossu M, Pau A, Rivano C, Viale G. The outcome from acute subdural and epidural intracranial haematomas in very elderly patients. *Br J Neurosurg* 1992;6:227-31.
54. Servadei F. Prognostic factors in severely head injured adult patients with epidural haematoma's. *Acta Neurochir (Wien)* 1997;139:273-8.