



Traumatic Extradural Hematoma Overview, Clinical Study and Management

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Abstract

The study was conducted at neurosurgical hospital in Baghdad depending on patient's case sheet data. The study was carried retrospectively. The patients from 1.1.2004 till 1.5.2005 were taken in consideration and we found 50 cases treated by surgical intervention and 12 cases treated conservatively. Patients with EDHs less than 0.5 cm were treated conservatively and those with postoperative EDHs of elective neurosurgical procedures in addition to incompletely filled patients' files were excluded. All patients data were recorded including the demographic characteristics and clinical parameters: side, site and size of haematoma (maximum thickness). The study concluded that conservative treatment had a role in management of EDHs.

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Introduction

Extradural haematoma (EDH) is a collection of blood between the inner table of the skull and the dura. It is an infrequent sequel of head injury, occurring in less than 2 percent of patients admitted with cranio-cerebral trauma. Ratio of male: female is 4:1. It usually occurs in young adults and it is rare in those over age of 60 years and children in the first 2 years of life (Robert *et al.*; Brambilla *et al.*, 1986; Gutman *et al.*, 1992; O'sullivan *et al.*, 1990; Servadei *et al.*, 1980).

Pathogenesis

Impact to the skull can cause in bending and stripping of the dura mater from the inner table. The skull is usually fractured, and meningeal vessels are torn. Bleeding occurs into the region where the dura has been separated

from the inner table, and the dura is further stripped by a confluent hematoma (FITZGERALD *et al.*; Bruce, *et al.*, 1978; Carlsson *et al.*, 1968). EDH results from injury to the middle meningeal artery or vein, the diploic veins, or the dural venous sinuses. Hematoma is almost always unilateral, and most are found in the temporal region. Associated intracranial injury occurs in a minority of patients (Hendricks *et al.*, 1964; Zimmerman *et al.*, 1981; Adams *et al.*, 1984).

Another conceptualization emphasizes the existence of primary and secondary injury. These classification systems are not incompatible, since the concept of focal and diffuse injury may be subsumed under the categories of primary and secondary injury, which are considered as stages in the evolution of head trauma (Graham *et al.*, 1987; Graham *et al.*, 1993; Gennarelli, 1993).

Pre-injury status

Many premorbid factors can modify post-traumatic events. Variations in the thickness of the scalp and skull and in the shape of the head influence the forces transmitted to the contents from an external impact (Doberstein, 1993; Gurdjian, 1972). The thinness and degree of adhesion of the dura to the skull, which are greater in the elderly, decreases the ease with which the dura tears in relation to an overlying skull fracture. This factor also influences the incidence and extent of epidural hematoma formation after head injury. Studies of patients older than 70 years of age have shown a low incidence of epidural hematomas (4 to 7 per cent) (Cagetti *et al.*, 1992; Kotwica *et al.*, 1992). In children, excluding infants, show a considerably higher incidence of epidural hematomas (35 per cent) (Hahn *et al.*, 1993). The type and incidence of transtentorial herniation depends on the configuration of the tentorium cerebelli and the size of the tentorial notch, both of which have considerable variation (Cohen *et al.*, 1990; Corsellis *et al.*, 1958; Ropper *et al.*, 1986). Also, the relationship between the size and rate of expansion of an intracranial mass lesion and intracranial pressure is modified by pre-existing brain atrophy. Thus, a patient with advanced brain atrophy may harbor a large epidural or subdural hematoma and demonstrate few or no neurological manifestations. The level of function before injury is a strong predictor of the potential for recovery of neurological function. Recently, the concept of "individual vulnerability" has been advanced, which takes into consideration host factors including preinjury conditions (e.g., medical diseases, psychiatric disorders, substance abuse, previous injury, learning disability), sociodemographic factors (e.g., age, gender, economic status), and pending litigation or other compensation issues (Kay, 1992). All these pre-injury factors may influence post-traumatic morbidity and mortality (Middelboe *et al.*, 1992; Mittenberg *et al.*, 1992).

Mechanism of injury

Epidural hematomas are contact injuries resulting from blunt trauma to the skull and meninges. Fractures, most often linear, are present in 30 to 91 per cent of patients with epidural hematomas (Baykaner *et al.*, 1988; Gallagher *et al.*, 1968; Rivas *et al.*, 1988). It is thought that the initial impact, with deformation or fracturing of the cranium, produces detachment of the dura directly beneath the site of the blow and injures blood vessels (most commonly branches of the middle meningeal artery). Once bleeding has begun, the blood fills the

extradural pocket. Experimental evidence indicates that arterial bleeding into the resulting pocket creates a hydraulic "water press" effect, progressively stripping away the dura from the skull and widening the perimeter of the hematoma (Crooks *et al.*, 1991; Weinman *et al.*, 1969).

The pathological effects of epidural hematomas are primarily due to compression of the underlying brain and later to swelling of this compressed area together with brain distortion and increased intracranial pressure. Clinical signs of these effects vary according to the location of the hematoma. Most traumatic epidural hematomas become rapidly symptomatic, but cases of delayed radiographical and clinical appearance of these lesions have also been documented (Phomprasat *et al.*, 1980; Cucciniello *et al.*, 1993; Lobato *et al.*, 1991; Chandrasekaran *et al.*, 1993; Gelabert, 1993).

Epidural hematoma in infant and children

In infants, an epidural hematoma frequently arises from arterial or venous bleeding that is remote from the location that is seen in older children and adults. Epidural hemorrhage is rare in the neonate (2 per cent of intracranial hemorrhages) (Takagi, 1978). As in other age groups, bleeding may result from traumatic tearing of branches of the middle meningeal artery, major venous sinuses, or diploic vessels, and there is often an associated linear skull fracture. An instrument or a difficult delivery in a neonate with signs of raised intracranial pressure could raise the possibility of an epidural hematoma (Faix, 1983). Epidural hematoma is less common in children than in adults and is even rarer in infants. With an incidence of 1.5 to 3.5 per cent in the pediatric head injury population (Choux, 1986). The decreased incidence in children, particularly in infants and newborns, is because of the increased adherence of the dura to the calvarium, which reduces the potential of epidural bleeding. The source of epidural bleeding differs in children than in adults. Hematoma caused by venous bleeding from a dural sinus. Because the production of this epidural hematoma is slower, anemia may be present before any neurological abnormalities appear. Epidural hematomas in children can decompress through the fracture line under the scalp. The classic presentation of the child with epidural hematoma often includes a relatively minor injury. Before a definitive computed tomography study is made, measures should be instituted to reduce intracranial pressure (Zimmerman *et al.*, 1983). Epidural hematomas of the posterior fossa are more likely to be seen in children than in adults.

These lesions require emergency evacuation. The mortality rate for epidural hematoma in children ranges between 7 and 15 per cent, the highest rate being for hematomas in the temporal region (Pagni *et al.*, 1975).

Clinical manifestation

Patients with EDH follow one of the following five clinical courses:

Conscious throughout, Unconscious throughout, Initially conscious and subsequently unconscious, Initially unconscious and subsequently lucid, Initially unconscious followed by a lucid interval and then unconscious again. Patients with acute EDH may present with only minimal complaints such as headache, nausea, and vomiting (Miller *et al.*, 1990). The clinical signs associated with EDH depend on the location and speed of growth of the hematoma and on the presence of associated intradural lesions (Poon *et al.*, 1992; Smith *et al.*, 1991; Marshall *et al.*, 1983). Some patients had a transient loss of consciousness immediately after impact followed by a lucid interval, followed by a second deterioration in consciousness as the hematoma expands. Pupillary changes, hemiparesis (Cooper *et al.*, 1993; Bricolo, 1984). Other presenting findings: seizure, bradycardia, a drop in hematocrit after admission, ipsilateral hemiparesis (Kernohan *et al.*, 2001; Jamison *et al.*, 1976). The diagnosis of EDH depends on the History of head injury and clinical examination; Searching for scalp wounds evidence of external trauma and examination of papillary abnormalities and focal neurological deficit (Aldrich, 1993); Skull radiography, angiography, and ventriculography, CT scan and magnetic resonance imaging,

Indications for surgery

Surgical intervention depends on patient's neurological status, the imaging findings, and the extent of extracranial injury, however, surgery indicated in any symptomatic EDH, an acute asymptomatic EDH > 1cm in its thickest measurement Threshold for surgery in pediatrics should be very low (Cooper *et al.*, 1993; Bricolo *et al.*, 1984).

Outcome

Mortality following treatment of EDH varies from 5 to 43 percent (Cook *et al.*, 1988; Jamieson, 1968; Jamjoom *et al.*, 1992; Phonprsert, 1980; Cagetti *et al.*, 1992). Mortality is low (5 to 10 percent) in children and

increases sharply in those over age 40 (35 to 50 percent). Associated intracranial lesions such as SDH intracerebral hematoma, cerebral contusion have a detrimental effect on outcome and result in a mortality rate four times greater than in patients without such lesions. Increased age, poor neurological condition, large hematoma volume, delay in operative evacuation, large midline shift, and postoperative elevation in ICP are all associated with poor outcome (Phonprsert *et al.*, 1980; Lobato *et al.*, 1988; Ali *et al.*, 1984).

Materials and Methods

A retrospective study was conducted at the Neurosurgical hospital in Baghdad depending the patient's case sheet data.

The patients' files for the period 1st of January 2004 to the 1st of May 2005 were included and analyzed, a total of 50 cases treated by surgical intervention and 12 cases treated conservatively, were reported.

Inclusion criteria

All EDH patients were included regardless their age or gender

Exclusion criteria

1. EDHs less than 0.5 cm and treated conservatively.
2. Postoperative EDHs of elective neurosurgical procedures.
3. Incomplete files or uncertain diagnosis were also excluded

Patient data were reported carefully including the demographic and clinical data in addition, specific data regarding the EDH were collected, neurological examination at admission and later follow up.

Statistical analysis

Through the statistical package for Social Sciences (SPSS) software for windows, version 16 and appropriate statistical tests were used accordingly.

Results and Discussion

The current study included 62 patients with EDH, of them 50 patients (80.65%) were surgically managed and the remaining 12 patients (19.35%) treated conservatively and this means that there is some role of conservative treatment in EDH cases.

In a study conducted by of Dr. Ali Kamil in 1994, 94% of cases treated surgically and 6% treated conservatively. We have some of conservatively treated patients presented with large size till 2cm maximum thickness and get good improvement that followed carefully by serial CT scans. All of our patients that treated conservatively had normal neurological examination and no any neurological deficit; this is very important factor influences decision of management. Tsaiy in 1984 reported 2 cases treated conservatively for EDH followed up by serial CT scans. Conversely, Jamieson mentioned that any EDH of more than 1 cm thickness or more than 25 ml volume should be dealt with by surgery. Our study proved the role of conservative treatment, where conservative treatment was successful with good outcome in 11 out of 12 patients, hence, we disagreed the opinion of Jamieson who mentioned that all EDHs patients should be surgically treated.

In the present study, the highest age incidence of EDHs in age group 1-10 years (28%), followed by age group 11-20 years (22%) and 12-30 years (22%), then age group 31-40 years (18%), age group >40 years (8%), lastly <1 year (2%). However Hooper R.S at 1959 found that EDH is more common in third decade of life and it is rare in elderly age group. Romario and Javan (1988) show that the highest age group incidence is 10-19 years and 20-29 years by 26% and 20% respectively (Table 1). An Iraqi study in 1993 by Dr. Zaid D. Sarsam mentioned that higher incidence of head injury in Iraq in children aged below 12 years. In 1994 Dr. Ali Kamil showed that highest age incidence is 30-39 years followed by 0-9 years 30% and 28% respectively. However, in our study, it is clear that pediatric age group take the highest incidence and this is may be due to new circumstances in our country for the last two years. In our study we didn't find any case above 55 years and this is due to adherent dura to the inner table of the skull.

Regarding surgically treated patients, 90% of them are males and 10% females, while those of conservative treatment 83.3% are males and 16.7% are females. A study done in Hong Kong at 1990 by Kwan Hon Chan *et al.*, show those males 84.62% and 15.38 are females of EDH victims. Jamieson and Yelland at 1968 showed a male to female ratio of 2.7:1. This predominance is due to environmental factors that males more exposed to injuries than females. Previous studies also showed predominance of males among EDH patient.

The present study found that the highest incidence in those of private work 48%, followed by children

(preschool age) 26% then students 16% and officers 10%, this is for surgically treated patients. For conservative one the students constitute highest incidence 83.3%, followed by private work 33.3% then children (preschool age) 16.6% (Table 1).

The current study found that majority of the patients were residents in Baghdad (Figure 1), and this is due to the availability of neurosurgeons in almost all cities of our country so the referral from other provinces much decreased.

In the present study there was sharing of the bulk of incidence between RTA and FFH 44% to each of them, followed by hit by heavy object 10% and only 2% for bullet injury. This is for surgical management. For conservatively treated patients, RTA group show highest incidence 50%, followed by FFH 33.3% and lastly hit by heavy object 16.6% (Table 1). Kwan-Hon Chan *et al.*, (1990) showed that FFH constitute 45%, RTA 37%, and hit by heavy object 18%. Of course it was found that the type of injury changes with time and country according to traffic and civilization of country.

In the current study, 54% of surgically treated patients are right sided, 42% are left sided and 4% bilateral EDH. While in conservative group, the ratio is 1:1 (50% right and 50% left (Table 1). Anyhow this ratio is changeable according side of trauma.

Among the surgically treated patients 54% arrived within first 2 hours, 54% during 3-10 hours, 18% during 10-24 hours, 12% from 1 day to 1 week and only 2% >1week.

Conservatively treated patients: 58.33% within 2hours, 16.66 from 3-10 hours and 25% from 1 day to 1week (Table 2).

The time spent between the injury and receiving the patients in neurosurgical emergency unit is very important in management and all studies concentrate on this point and recommend to be as less as it possible and to decrease the delay in transferring the victim to neurosurgical hospital in order to get a chance of early interference and better outcome. But unfortunately, some times the patients referred from general hospital without full first aid management that worsen and complicate the condition of the patients. Those presented after one week are due to either missing in general hospital or slowly developed EDH. In the literature series this interval is variable. Lokkeberge *et al.*, (1984) U.S.A., 1984

reported that (42.2%) of head injured patients admitted within 1-2hours and 9.2% of them admitted half hour, while 70%admitted within the first 4 hours of injury. Jannet *et al.*, (1977) showed a delay in admitting head injured patient in Scotland where less than 6 hours 32%, 7-24 hours 38%, and more than 24 hours 30%. This shows that there is a case delay in our country and it is due to the system used which is the same system used in

Scotland where the patient is first transferred to general hospital and then to neurosurgical unit in another hospital after assessment that it might be wrong, while in U.S.A., the patient is transferred to neurosurgical unit from emergency room at the same general hospital without delay. All our patients have good quality x-rays with multiple views, (Table 3) summarizes the associated fracture showed by X-Ray.

Table.1 Age and sex distribution of the studied group according to the type of management

Variable		Type of management	
		Surgical	Conservative
Age (year)	Below 1 year	1 (2%)	0 (0%)
	1 – 10	14 (28%)	2(16.66%)
	11 – 20	11 (22%)	6(50%)
	21 – 30	11 (22%)	4(33.33%)
	31 – 40	9 (18%)	0 (0%)
	> 40	4 (8%)	0 (0%)
	Gender	Male	45 (90%)
Female		5 (10%)	2(16.6%)
Occupation	Child (preschool)	13 (26%)	2 (16.6%)
	student	8 (16%)	6(50%)
	Military	5 (10%)	0 (0%)
	Civil work	24 (48%)	4(33.33%)
Type of injury	RTA	22 (44%)	6 (50%)
	Fall from height	22 (44%)	4 (33.3%)
	Hit by heavy object	5 (10%)	2 (13.7%)
	Bullet injury	1 (2%)	0 (%)
Side of Hematoma	Right	27 (54%)	6 (50%)
	Left	21 (42%)	6 (50%)
	Bilateral	2 (4%)	0 (0%)
Total		50 (100%)	12 (100%)

Table.2 Time between injury and admission to emergency room the patient

Time	Type of management	
	Surgical	Conservative
First 2 hours	17 (34%)	7(58.3%)
3 – 10 hours	17 (34%)	2(16.7%)
11 – 24 hours	9 (18%)	0(0%)
1 day-1 week	6 (12%)	3(25%)
>week	1 (2%)	0(0%)
Total	50 (100%)	12 (100%)

Table.3 Associated fracture showed by X-Ray

Associated fracture	Type of management	
	Surgical	Conservative
Depressed fracture	6 (12%)	0(0%)
Linear parietal	9 (18%)	2 (16.66%)
Linear temporal	9 (18%)	2 (16.66%)
Linear frontal	4 (8%)	0 (0%)
Linear occipital	1 (2%)	1 (8.33%)
Linear Parieto-temporal	6 (12%)	3 (25%)
Linear Parieto-occipital	5 (10%)	0 (0%)
Linear Fronto -parietal	3 (6%)	1 (8.33%)
No fracture	7 (14%)	3 (25%)
Total	50 (100%)	12 (100%)

Table.4 Site of haematoma by CT-scan

Site of hematoma	Type of management	
	Surgical (n = 50)	Conservative (n = 12)
Parietal	12 (24%)	2 (16.66%)
Frontal	9 (18%)	1 (8.33%)
Parieto-temporal	9 (18%)	3 (25%)
Temporal	7 (14%)	1 (8.33%)
Fronto -parietal	7 (14%)	3 (25%)
Parieto-occipital	6 (12%)	1 (8.33%)
Occipital	0 (0%)	1 (8.33%)
Total	50 (100%)	12 (100%)

Table.5 Maximum thickness of haematoma by CT-scan

Thickness of haematoma (cm)	Type of management	
	Surgical	Conservative
< 0.5	0 (0%)	0 (0%)
0.5 – 1.0	12 (24%)	4 (33.3%)
1.1 – 1.5	9 (18%)	4 (33.3%)
1.6 – 2	7 (14 %)	4 (33.3%)
2.1 – 2.5	5 (10 %)	0 (0%)
2.6 – 3	6 (12 %)	0 (0%)
> 3	2 (4 %)	0 (0%)
Midline shift	10 (20%)	0 (0%)
Total	50 (100%)	12 (100%)

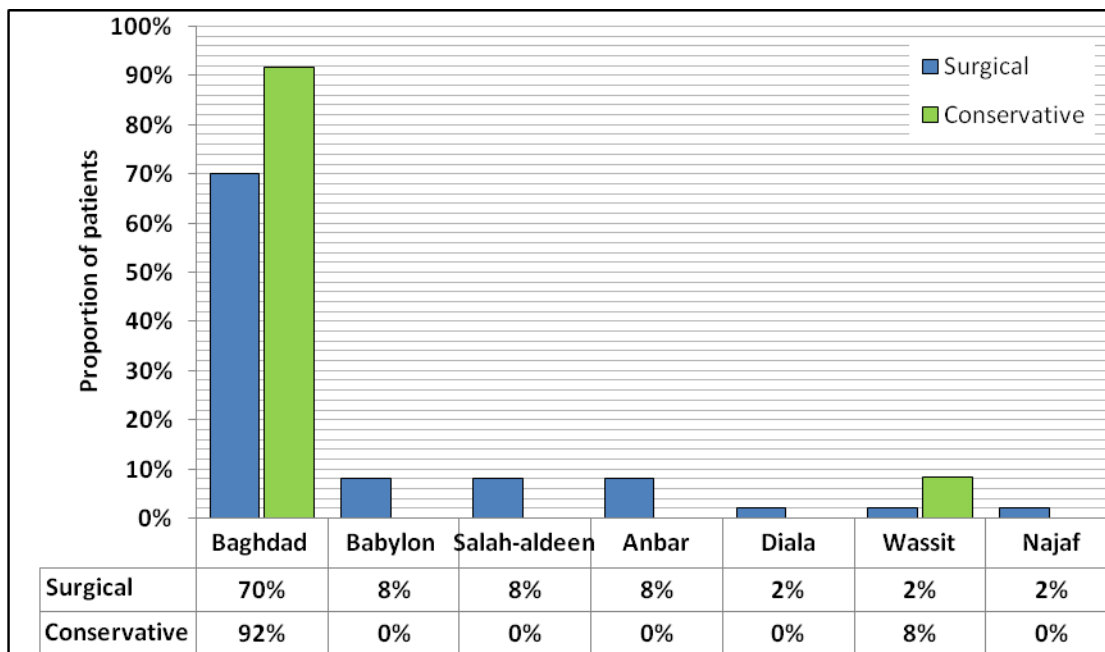
Table.6 Patients' Glasgow Coma Scale (GCS) at arrival

GCS	Type of management	
	Surgical	Conservative
Mild (13-15)	28 (56%)	9 (75%)
Moderate (9-12)	15 (30%)	2 (16.66%)
Severe (3-8)	7 (14%)	2 (16.66%)
Total	50 (100%)	12 (100%)

Table.7 Outcomes of the studied group

	Type of management	
	Surgical	Conservative
Full recovery	40 (80%)	11 (91.67%)
Neurological deficit	4 (8 %)	0 (0%)
Death	6 (12%)	1 (8.33%)
Total	50 (100%)	12 (100%)

Fig.1 Distribution of the studied group by residence



However, vast majority of the patients had linear fractures and only 6 patients had depressed fractures. Our

findings agreed that reported in previous studies (Kwan *et al.*, 1990; Lokkeberge *et al.*, 1984; rutage, 1978).

Fortunately, all our patients have a chance for good quality CT-scan in our hospital that can be done immediately at arrival; this helps the neurosurgeon to make a precise decision to manage the patient as fast as possible. The CT-scan in surgically treated patients, revealed that 24% of EDHs at parietal region, 18% at frontal, 18% at parieto-temporal, 14% at temporal, 14% at fronto-parietal, and 12% at parieto-occipital region. In conservatively treated patients, 25% at parieto-temporal, 25% at fronto-parietal, 16.6% at parietal, 8.3% at each of frontal, temporal, occipital and parieto-occipital regions (Table 4). These findings were close to that reported in Iraq by Dr. Ali Kamil.

Hooper in 1960 showed that temporo-parietal area is the commonest area of EDH that constitutes 65.18%. Ramiro and Javan in 1988 show parietal 48%, parieto-temporal 20%, temporal 12%, frontal 17% and posterior fossa 1%.⁵⁵ Mckissockw *et al.*, (1960) found that posterior fossa EDH is 4%, while Zucarko *et al.*, (1981) found that posterior fossa EDH is 12.9%⁵⁶. On the other hand, in our study 56% of all EDHs are distributed in the lateral wall of the skull, Jamieson in 1976 found that 60-70% of all EDH distributed in the lateral walls of the skull⁶⁰. From other point of view, in our study none of the cases of posterior fossa EDH was found.

Regarding, the maximum thickness of haematoma by CT-scan among surgically treated patients it had been found that 24% of these patients had an EDHs thickness of 0.5-1cm, 18% with thickness of 1.1-1.5cm, and the maximum thickness was 1.6-2cm in 14%, 2.1-2.5 in 10%, 2.6-3cm in 12% and >3cm in only 4% of those patients. These associated with midline shift in 20% of cases. Among the conservatively treated patients the maximum thickness was 0.5-1cm in 33.33%, 1.1-1.5cm in 33.33% and 1.6-2cm 33.33% . while none of them had a thickness of more than 2 cm (Table 5) The 24% of surgically treated patients with a maximum EDH thickness of 0.5-1cm, treated surgically because they present with decreased level of consciousness and/or neurological deficit, but we don't know whether these signs and symptoms are due to EDH itself or to associated edema or concussion. So we must examine the patient carefully and assess his condition many times before we decide to interfere with these small size hematomas. In our study, we have 56% of surgically treated patients presented with GCS 13-15 (mild), 30% with 9-12 (moderate) and 14% with 3-8 (severe), while in conservatively treated patients 75% with 13-15, 16.66% with 9-12 and 8.33% with 3-8, (Table 6). Four

out of 6 death of surgically treated patients (66.66%) presented with GCS of <9 ranging from 4-8.

The outcomes of the patients were full recovery in 80% of the surgically treated patients and discharged with good condition and normal physical exam. Unfortunately, 4 patients (8%) developed neurological deficit and we don't know whether they improve or not later on, because our study is retrospective and no follow up information available in their case sheet, in addition 6 patients (12%) died. All the conservatively treated patients except one (8.33%) had full recovery and only one patient unfortunately died (Table 7). The mortalities were mainly in those who were presented with severe head trauma and low GCS, death occur either due to large sized EDH that not received by neurosurgical unit immediately after injury or due to associated brain pathology. Ramiro in 1988⁷⁶ showed that mortality of GCS<8 are around 75%. Many studies are done about EDH and mortality was ranging from 10% to 30% (Bruce *et al.*, 1978). Romario in 1988 showed that EDH operated upon coma, the mortality range from 25-71%.

The present was not free of limitations; the retrospective design lead to loss of information about the patients post management particularly those with neurological deficit, additionally, data were not complete in some patients case-sheets particularly among died patients. However, further prospective studies with larger sample size and longer duration could reveal additional information about the traumatic EDH.

Conclusion and recommendations

Patient with EDH without midline shift and had high GCS even with mild neurological manifestations could be managed conservatively with close follow up and careful serial CT scans, which can decrease the need to surgical intervention and its complications. Craniotomy is the main type of surgical treatment, and with providence of CT scans, it became the treatment of choice instead of burr hole or craniectomy.

Patients with EDH should be examined carefully, CT scan should be performed immediately and early surgical intervention in indicated cases, and these could improve the outcome. CT scan must be available in every hospital with providing well equipped ambulances with well trained staff and good emergency room and neurosurgical unit.

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References

- Adams, J.H. 1984. Head injury. In Adams, J., Corsellis, J., and Duchen, L., eds.: Greenfield's Neuropathology, 4th ed. London, Arnold, 1984, pp. 85-124 trauma.
- Aldrich, E.F., and Eisenberg, H.M. 1993. Acute subdural hematoma. In Apuzzo, M. L. J., ed. Brain Surgery Complication Avoidance and Management. New York, Churchill Livingstone, pp. 1283– 1298
- Ali Kamil. 1994. Study of 50 cases of EDH.
- Baykaner, K., Alp, H., Ceviker, N., *et al.* 1988. Observation of 95 patients with extradural haematoma and review of literature. *Surg. Neurol.*, 30: 339.
- Bigler, E.D., Kurth, S., Blatter, D., *et al.* 1993. Day-of-injury CT as an index to pre-injury brain morphology: Degree of post-injury degenerative changes identified by CT and MR Neuroimaging. *Brain Injury*, 7: 125.
- Brambilla, G., Rainoldi, F., Gipponi, D., Paolatte, P. 1986. Extradural haematoma of the posterior fossa report of eight cases and a review of the literature. *Acta neurochir (Wien)*, 80: 24-29.
- Bricoli, A.P., Pasut, L.M. 1984. EDH, toward zero mortality a prospective study, *Neurosurg.*, 14: 8-12.
- Bricolo, A.P., and Pasut, L.M. 1984. Extradural hematoma: Toward zero mortality: A prospective study. *Neurosurgery*, 14: 8–12.
- Bricolo, A.P., Pasut, L.M. 1984. Extradural hematoma: toward zero mortality, a prospective study, *Neurosurgery*, 14: 8-12.
- Bruce, D.A., Schut, L., Bruno, L.A., *et al.* 1978. Outcome following severe head injuries in children. *J. Neurosurg.*, 48: 679–688.
- Bruce, D.A., Schut, L., *et al.* 1978. outcome following severe head injuries in children, *J. Neurosurg.*, 48: 679-688.
- Cagetti, B., Massimo, C., Pau, A., *et al.* 1992. The outcome from acute subdural and epidural intracranial haematomas in very elderly patients. *Br. J. Neurosurg.*, 6: 227.
- Cagetti, B., Pau, A., *et al.* 1992. the outcome from acute subdural and epidural intracranial haematomas in very elderly patients. *Br. J. Neurosurg.*, 6: 227-231.
- Carlsson, C.A., Von Essen, C., and Lofgren, J. 1968. Factors affecting the clinical course of patients with severe head injury: Parts 1 and 2. *J. Neurosurg.*, 29: 242–251.
- Chandrasekaran, S., and Zainal, J. 1993. Delayed traumatic extradural haematomas. *Aust. N.Z. J. Surg.*, 63: 780.
- Choux, M. 1986. Extracerebral hematomas in children. In Vigoroux, R. P., and McLaurin, R. L., eds. *Adv. Neurotraumatol.*, Vol. 1. New York, Springer-Verlag.
- Clinical neuroanatomy and related neuroscience, fourth edition, M.J.T. Fitzgerald, Jean folan-currant.
- Cohen, A.R., and Wilson, J. 1990. Magnetic resonance imaging of Kernohan's notch. *Neurosurgery*, 27: 205.
- Cook, R.J., Dorsch, N.W.C., Fearnside, M.R., Chaseling, R. 1988. Outcome prediction in extradural hematomas'. *Acta neurochir (wein)*, 95: 90-94.
- Cooper, P.R. 1993. Post-traumatic intracranial mass lesions. In Cooper, P. R., ed. *Head Injury*, 3rd ed. Baltimore, Williams & Wilkins, pp. 275-329
- Cooper, P.R. 1993. Post-traumatic intracranial mass lesions. In Cooper, P. R., ed. *Head Injury*. 3rd ed. Baltimore, Williams & Wilkins, pp. 275-329.
- Corsellis, J.A.N. 1958. Individual variation in the size of the tentorial opening. *J. Neurol. Neurosurg. Psychiatry*, 21: 279.
- Crooks, D.A. 1991. Pathogenesis and biomechanics of traumatic intracranial hemorrhages. *Virchows Arch. [A]*, 418: 479.
- Cucciniello, B., Martellotta, N., Nigro, D., *et al.* 1993. Conservative management of extradural haematomas. *Acta Neurochir*, 120: 47.
- Dharker, R., and Bhargava, N. 1991. Bilateral epidural haematoma. *Acta Neurochir*, 110: 29.
- Doberstein, C.E., Hovda, D.A., and Becker, D.P. 1993. Clinical considerations in the reduction of secondary brain injury. *Ann. Emerg. Med.*, 22: 993.
- Faix, R.G., and Donn, S.M. 1983. Immediate management of the traumatized infant. *Clin. Perinatol.*, 10: 487– 505.
- Frutage, *et al.* 1978. x-ray in head injury, *J. Radiol.*, 130-132.
- Gallagher, J.P., and Browder, J. 1968. Extradural haematoma: Experience with 167 cases. *J. Neurosurg.*, 29: 1.

- Gelabert, M., Prieto, A., Rumbo, R.M., *et al.* 1993. Simultaneous bilateral extradural haematoma. *Br. J. Neurosurg.*, 7: 95.
- Gennarelli, T.A. 1993. Mechanisms of brain injury. *J. Emerg. Med.*, 11: 5.
- Graham, D.I., Adams, J.H., and Gennarelli, T.A. 1987. Pathology of brain damage in head injury. In Cooper, P., ed.: *Head Injury*. Baltimore, Williams & Wilkins, 1987, pp. 72-88
- Graham, D.I., Adams, J.H., Doyle, D., *et al.* 1993. Quantification of primary and secondary lesions in severe head injury. *Acta Neurochir*, 57(Suppl.): 41.
- Guillermain, P. Traumatic extradural haematomas. In Vigouroux, R.P. 1986. ed. *Extracerebral Collections: Advances in Neurotraumatology*. Wien, Springer-Verlag, pp. 1-50.
- Gurdjian, E.S. 1972. Recent advances in the study of the mechanism of impact injury of the head. *Clin. Neurosurg*, 19: 1.
- Gutman, M.B., Maulton, R.J., Sullivan, I., *et al.* 1992. risk factors predicting operable intracranial haematomas in head injury, *J. Neurosurg.*, 77: 9-14.
- Hahn, Y.S., and McLane, D.G. 1993. Risk factors in the outcome of children with minor head injury. *Pediatr. Neurosurg.*, 19: 135.
- Hendricks, E.G., Harwood-Nash, D.C.F., and Hudson, A.R. 1964. Head injuries in children: A survey of 4465 consecutive cases of the Hospital for Sick Children, Toronto, Canada. *Clin. Neurosurg.*, 11: 46-65.
- Hooper, R.S. 1959. Observations on EDH, *brit. J. Surg.*, 47: 71-87.
- Jamieson, K.G., and Yelland, J.D.N. 1968. Extradural hematoma: Report of 167 cases. *J. Neurosurg.*, 29: 13.
- Jamieson, K.G., Yelland, J.D.N. 1968. Extradural hematoma: report of 167 cases, *J. Neurosurg.*, 29: 13-23.
- Jamison, K.G. 1976. Epidural hematoma in vin ken pj, bruyn GW. (eds), *injuries of brain and skull part II, hand book of clinical neurology vol.24* Amsterdam north Holland, 261-74.
- Jamjoom, A. 1992. The influence of concomitant intradural pathology on the presentation and outcome of patients with acute traumatic extradural haematoma. *Acta Neurochir (wein)*, 15: 86-89.
- Jannet, Teosdale. 1977. aspects of coma after severe head injury, *Lancet*, 1: 878-881.
- Jennett, B. 1975. *Epilepsy after Nonmissile Injuries*. 2nd ed. Chicago, Year Book Medical Publishers.
- Kay, T. 192. Neuropsychological diagnosis. *State Art Rev. Phys. Med. Rehabil.*, 6: 109.
- Kernohan, J.W., Woltman, H.W. 1929. Incisura of the Crus due to Contralateral Brain Tumor. *Arch. Neurol. Psychiatr.*, 21: 274. *Handbook of neurosurgery, fifth edition, by Mark S. Greenberg* 2001.
- Kotwica, Z., and Jakubowski, J.K. 1992. Acute head injuries in the elderly: An analysis of 136 consecutive patients. *Acta Neurochir*, 118: 98.
- Kwan-Hon Chan, kirbal, S., *et al.* 1990. The significance of skull fracture in acute traumatic intracranial haematomas in adolescents, a prospective study, *J. Neurosurg.*, 72: 189-194.
- Lobato, R.D., Rivas, J.J., Cordobes, F., *et al.* 1988. Acute epidural hematoma; an analysis of factors influencing the outcome of patients undergoing surgery in coma, 68: 48-57.
- Lobato, R.D., Rivas, J.J., Gomez, P. A., *et al.* 1991. Head-injured patients who talk and deteriorate into coma. *J. Neurosurg.*, 75: 256.
- Lokkeberge, Gr. 1984. Assessing the influence of non-treatment variables in a study of outcome from severe head injuries, *J. Neurosurg.*, 61: 254-262.
- Marshall, L.F., Gautille, T., Klauber, M.R., *et al.* The outcome of severe closed head injury. *J. Neurosurg.*, 75: S28.
- Marshall, L.F., Toole, B.M., and Bowers, S.A. 1983. The National Coma Data Bank: Patients who talk and deteriorate: Implications for treatment. *J. Neurosurg.*, 59: 285-288.
- Middelboe, T., Anderson, H.S., Birket-Smith, M., *et al.* 1992. Minor head injury: Impact on general health after 1 year: A prospective follow-up study. *Acta Neurol. Scand.*, 85: 5.
- Miller, J.D. 1992. Evaluation and treatment of head injury in adults. *Neurosurg. Q.*, 2: 28-43. *Handbook of neurosurgery, fifth edition, by Mark S. Greenberg, 2001.*
- Miller, J.D., murry, L.S., Teasdale, G.M. 1990. Development of a traumatic intracranial hematoma after a "minor" head injury. *Neurosurgery*, 27: 669-673.
- Mittenberg, W., DiGiulio, D.V., Perrin, S., *et al.* 1992. Symptoms following mild head injury: Expectation as etiology. *J. Neurol. Neurosurg. Psychiatry*, 55: 200.
- Morris, J.A., MacKenzie, E.J., Damiano, A.M., *et al.* 1990. Mortality in trauma patients: The

- interaction between host factors and severity. *J. Trauma*, 30: 1476.
- O'sullivan, M.G.J., Gray, .W.P, Buckley, T.F. 1990. Extradural haematoma in the Irish Republic:an analysis of 82 cases with emphases on 'delay'. *Br. J. Surg.*,77:1391-1394.
- Pagni, C.A., Horton, J.A., and Herron, J.M. 1983. Nonsurgical management of extradural hematomas in children. *J. Neurosurg.*, 59: 958– 971.
- Pagni, C.A., Signovone, G., Crotti, F., *et al.* 1975. Severe traumatic coma in infancy and childhood: Results after surgery and resuscitation. *J. Neurosurg. Sci.*, 19: 120– 128.
- Peter, J.C., and Domingo, Z. 1990. Subacute traumatic extradural hematomas of the posterior fossa: A clinicopathological entity of the 5- to 10-year old child. *Child's Nerv. Syst.*, 6: 135– 138.
- Phomprasat, C., Suwanwela, C., Hongsaprabhas C., *et al.* 1980. Extradural hematoma: Analysis of 138 cases. *J. Trauma*, 20: 679.
- Phonprsert, C., suwanwela, C., Hongsaprabhas, C., *et al.* 1980. Extradural hematoma: analysis of 138 cases, *J. Trauma*, 20: 679-683.
- Poon, W.S., Rehamn, S.U., poon, C.Y.F, Li, A.K.C. 1992. Traumatic extradural hematoma of delayed onset is not a rarity. *Neurosurgery*, 30: 681-686.
- Reider-Groswasser, I., Frishman, E., and Razon, N. 1991. Epidural haematoma: Computerized tomography parameters in 19 patients. *Brain Injury*, 5: 17.
- Rivas, J.J., Lobata, R.D., Sarabia, R., *et al.* 1988. Extradural haematoma: Analysis of factors influencing course of 161 patients. *Neurosurgery*, 23: 44.
- Robert, H., Wilkins, M.D. and Setti, S., Rengachary, M.D. *Neurosurgery*, second edition, vol 4 page 2801.
- Romario, D., Javan, R. 1988. acute epidural haematoma: an analysis of factors influencing the outcome of patients undergoing surgery in coma, *J. Neurosurg.*, 48-57.
- Ropper, A.H. 1986. Acute increased intracranial pressure. In Asbury, A. K., McKhann, G. M., and McDonald, W. I., eds. *Diseases of the Nervous System*. Philadelphia, W. B. Saunders, pp. 1064– 1073.
- Rosenthal, M. 1993. Mild traumatic brain injury syndrome. *Ann. Emerg. Med.*, 22: 1048.
- Servadei, F., Piazza, G., Seracchioli, A., *et al.* 1988. Extradural haematomas: an analysis of the changing characteristics of patients admitted from 1980 to 1988; 2: 87-100.
- Servadei, F., Piazza, G., Seracchioli, A., *et al.* Extradural haematomas: an analysis of the changing characteristics of patients admitted from 1980 to 1988, 2: 87-100.
- Smith, H.K., Miller, J.D. 1991. The danger of an ultra-early computed tomographic scan in a patient with an evolving acute epidural hematoma. *Neurosurgery*, 29: 258-260.
- Takagi, T., Nagai, R., Wakabayashi, S., *et al.* 1978. Extradural hemorrhage in the newborn as a result of birth trauma. *Child's Brain*, 4: 306– 318.
- Tsaify, F.Y. 1984. Air in epidural haematomas, *J. Neurosurg.*, 61: 417
- Weinman, D., and Muttucumar, B. Extradural hematoma. *Ceylon Med. J.*, 14: 60.
- Zaid, D., Sarsam. 1993. Craniocerebral injuries, thesis.
- Zimmerman, R.A., and Bilaniuk, L.T. 1981. Computed tomography in pediatric head trauma. *J. Neuroradiol.*, 8: 257–271.
- Zimmerman, R.A., and Bilaniuk, L.T. 1983. Radiology of pediatric craniocerebral trauma. In Shapiro, K., ed. *Pediatric Head Trauma*. New York, Futura, pp. 69-142
- Zucarko, M. 1981. Pardatcsches *et al*, epidural haematomas of the post cranial fossa, *Neurosurg*, 8: 434-437.

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