ORIGINAL ARTICLE

Extradural Hematoma - An Experience of 300 Cases

M. L. Babu, Sanjay Kumar Bhasin, Adarsh Kumar

Abstract

Three hundred cases of extradural haematoma seen over a period of nine years from June 1995 to May 2004 in Neurosurgery unit of Postgraduate Department of Surgery, Government Medical College, Jammu were analysed retrospectively for their clinical profile and operative measures. Their clinical presentation varied from intense headache to deep coma and fixed pupil. Two hundred forty five of them were operated and rest managed conservatively. Overall mortality in operated cases was 14%. Cases managed conservatively were monitored closely and serial CT Scans were done to assess progress. In two cases we had to operate as haematoma increased in size.

Key Words

EDH, Middle meningeal artery, RTA.

Introduction

Extradural haemtoma constitutes a major source of preventable mortality. It occurs in 1-2% of head injury cases (1). Extradural haematoma is very rare in extremes of ages. Mortality rate vary from 10-40% (2) and is an index of alertness and efficiency of health care and hospital set up. Blood collects between duramatar and bone, generally brain underneath is not injured. In most of the cases X-Ray's reveal fracture.

Material and Methods

Three hundred cases of extradural haematoma treated over a period of nine years at Govt. Medical College Hospital, Jammu formed base of the study. The mode of injury, clinical presentation, age, CT findings and operative measures were studied.(Table 1 to 5).

Results

Commonest of injury was RTA (n= 156) followed by fall (n=75). Commonest clinical presentation was altered sensorium (n=200) followed by headache / vomiting. Commonest age group involved was 21 to 30 years (n= 102). Males were more affected than females. 18 cases were deeply comatosed at the time of admission and 40 cases had pupillary changes.

In our series temporal site was involved in 43% (n=128) followed by frontal in 30% (n=90). Posterior fossa was involved in just 2% cases (n=06). Associated injuries were seen in14.3% cases (n=43).

Table 1. Cases of injury in 300 cases.

Cause of injury	Number of cases	Percentage
Fall	75	25
Assault	54	18
RTA	156	52
Ballistic injury	15	5

Table 2. Clinical Features in Patients.

Signs/Symptoms*	Number of Patients
Lucid interval	60
Headache/Vomiting	170
Altered sensorium	200
Neurodeficit	109
Bradycardia	70
Pupillary changes (single)	35
Papillary changes (bilateral)	05

* More than one sign/symptom observed in single patient.

Table 3. Age Distribution in 300 cases.

Age in years	Number of cases
1-10	30
11-20	78
21-30	102
31-40	60
41-50	30

From The Department of Neurosurgery, Unit of P.G Department of Surgery, Government Medical College, Jammu, J&K-180 001.
Correspondence to: Prof. M. L. Babu (Neurosurgeon), 258/C, Ramvihar, Old Janipur, Jammu.

Table 4: Distribution of cases as per site of Haematoma.

Site of haematoma	Number of Patients
Temporal region	128
Frontal region (unilateral)	90
Frontal region (Bilateral)	11
Temproparietal	61
Occipital region	04
Posterior fossa	06

Table 5. Associated injuries in 300 cases of EDH.

Associated injury	Number of cases (n=43)
Bilateral	03
Counter-coup contusion	17
Acute SDH/ICH/Contusion	23

Out of 300 cases, 245 cases were operated on emergency basis while rest were treated conservatively. Cases treated conservatively included small haematoma with no midline shift on CT Scan and general condition of the patients was grade I. Theses cases were closely monitored and serial CT Scans done to assess clot size. In 02 cases only, we had to operate as repeat scan showed increase in size of clot. We lost 42 patients, none in conservative management group. 14 of these cases had associated brain injuries, 15 cases were deeply comatose at admission, 10 had fixed pupil/pupils at admission and 03 case developed malignant brain oedema after evacuation of EDH. Brain oedema was confirmed at autopsy in this patient.

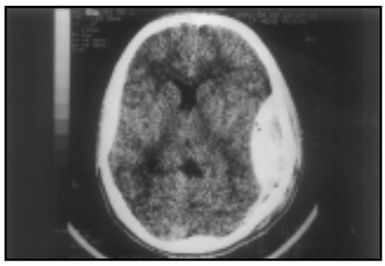

Fig. 1. CT scan showing Parietal EDH extending into occipital region.

Discussion

Extradural haematomas are contact injuries resulting from blunt trauma to the skull and meninges. Fracture, most often linear is present in 30-40% of patients with EDH (3). It is thought that initial impact with deformation or fracturing of cranium produces detachment of the dura directly beneath the site of the blow and injures blood vessels. Once bleeding begins the extradural space is

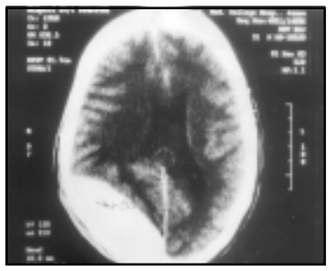

Fig. 2. CT scan showing rare Occipital EDH.

filled with blood. Experimental evidence indicates that arterial bleeding into the resulting pockets creates a hydraulic "water pressure" effect, progressively stripping away the dura from the skull and widening the perimeter of the haematoma (4). This is because of the thinness of the temporal squamous and the close approximation of the middle meningeal artery and vein to the inner table in this region, 70% of EDH are located in the temporal region (5). In rest of the cases EDH occurs in the frontal, occipital and posterior fossa region. EDH contra lateral to impact site is extremely rare. One such case was reported by Mishra *et al* (6). There is a lower incidence of EDH in elderly due to adherent dura with the cranium. Very young infants have a lower incidence presumably due to the pliable nature of the skull that resists fracturing. The pathological effects of epidural haematoma are primarily due to compression of the underlying brain and later due to distortion and increased ICP. Clinical signs of these effects vary according to the location of the haematoma.(7)

The classical clinical picture of EDH with a lucid interval is present in only 20-50% of cases (8). We observed it only in 20% cases. Most EDH became rapidly symptomatic, but cases of delayed radiographic and clinical appearance of these lesions have also been documented (9). One delayed manifestation EDH was noticed by us on serial CT Scan, this patient was operated subsequently. With the development of Neuroimaging facilities, the mortality rate caused by EDH has steadily decreased from 90% at the turn of 20th century to 30% in the 1950 and to 0-12% now (10, 11). Even minor injury can cause EDH, if the dura at particular site strips easily. Commonest cause of bleeding is middle meningeal artery rupture but source can be bleeding from superior saggital sinus/lateral sinus and Diploic vein. We had 02 cases with saggital sinus tear. Bilateral EDH are rare, we just had 03 case. Spontaneous Vertex EDH was reported by Metellus *et al* (12). EDH following Temporomandibular

joint arthrocec tesis and lavage was reported by Carroll *et al* (13). Bradycardia is a reliable sign of increasing ICP but is seen in small number of cases. It may also occur in absence of haematoma. Posterior fossa haematoma should be suspected if there is change in pulse rate, respiration rate or mild neck stiffness.

In cases treated conservatively, haematoma is seen to have resolved in three weeks time. Kolodziej *et al* reported just 04 cases of EDH treated non-surgically (14). Complications after treatment of EDH like hydrocephalous was not seen in our series but complications like malignant brain oedema (n=03), secondary haemorrhage in primary contusion (n=01) were encountered in our series. Outcome depends on the presence of associated injuries, primary neuronal damage, nature of first aid given, age of the patient, speed with which symptoms develop and condition at admission. A slower evolution of symptoms means better prognosis. Level of consciousness just before surgery largely influence prognosis. Mortality is higher in comatose patients. Massive cerebral oedema following evacuation of EDH is probably due to loss of cerebral auto regulation. Incidence of this has been reported to be 8% by Lobato (15). In our series we had three cases, all three died. In theses cases autopsy conformed malignant brain oedema. Mortality in our series was 15%. We lost 45 patients in our study, none in cases treated with conservative line of management. Of 45 cases who died, 03 developed malignant brain oedema after evacuation. 15 had associated brain injury and rest came to hospital in moribund condition (fixed pupil/pupils). In one series, it was observed that in younger patients in whom surgery is done earlier and have no or minimal associated brain injury recover better than the patients in whom surgical intervention is delayed.(16)

Step hanov (17) analysed and showed that pre CT era mortality ranged from 16-52% and in post CT era from 8-41%. He concluded that faster transportation of patients to neurosurgery centre remained most important factor for further reduction in mortality. Jones (10) reported fall of mortality from 29% to 8% in last 35 years.

Conclusion

From our experience of 300 cases of EDH we conclude that early diagnosis, improved neurosurgical services and availability of CT have been the main reason for decreasing mortality trends in EDH. Added to it is the fact that early transportation or early report by the patient to the centre where neurosurgical services are available also have bearing on the final outcome in the management of EDH. Conservative treatment should be tried only if patients general condition is good, haematoma is small and at non-dangerous zone and when serial CT facilities are readily available.

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