Extradural Haematoma: Analysis of 190 Cases

Extradural Hematolmalar: 190 Olgunun Analizi

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Abstract: Traumatic extradural haematomas (EDHs) comprise 1 to 3% of all head trauma admissions. The availability of computed tomography has increased the diagnosis of extradural haematomas. From January 1, 1986 to December 31, 1994, 4,553 patients with head injury were admitted to the Department of Neurosurgery, İzmir State Hospital. Of 4,553 patients, 190 patients with surgically treated EDH were included in this study. There were 161 males (85%) and 29 females (15%). The ages of the patients ranged from 4 to 70 years (mean 28 years). Seventy percent of the patients were between 15 and 40 years of age. The overall mortality was 15.7% (30 patients). All deaths occurred in comatose patients, except for one case (p<0.0001). Comatose state, presence of focal motor signs, respiratory irregularities and bradycardia, pupillary changes were determined as the bad prognostic factors. A midline shift greater than 10 mm, haematoma volume greater than 90 ml and haematoma thickness greater than 30 mm significantly increased the mortality. The primary factor on outcome is Glasgow Coma Scale scores of the patients at the time of surgery. Therefore early surgery is crucial in the management of EDH which is a dynamic process.

Key words: Computed tomography, extradural haematoma, head injury.

INTRODUCTION

Traumatic extradural haematomas (EDHs) comprise 1 to 3% of all head trauma admissions [2,7,10,12,16]. Surgical mortality has rapidly decreased since Hutchinson described extradural haemorrhage as an emergency situation in 1867 (8). The availability of computed tomography (CT) has increased the diagnosis of extradural haematomas. We reviewed the records of patients with EDH in order to determine the prognostic factors and the measures that can be taken to reduce the mortality rate.

MATERIALS AND METHODS

From January 1, 1986 to December 31, 1994, 4,553 patients with head injury were admitted to the Department of Neurosurgery, İzmir State Hospital.
Of 4,553 patients, 190 patients with surgically treated EDH were included in this study. Each patient was evaluated in terms of age, type of injury, systemic and neurological findings. The patients with a Glasgow Coma Scale (GCS) score 8 or less were considered comatose and those with a GCS score greater than 8 were considered non-comatose.

Radiological evaluation of the patients such as plain skull X-ray films, angiograms and CT scans were also reviewed. The site, thickness, density pattern and volume of EDHs were noted on the CT scans. The shift of the midline structures was measured at the level of septum pellucidum. The formula used to calculate the volume of the haematomas was as follows:

\[ \text{Haematoma volume (ml)} = 0.5 \times \text{transverse diameter (mm)} \times \text{anteroposterior diameter (mm)} \times \text{craniocaudal diameter (mm)} \] [14].

The origin of bleeding and characteristics of haematoma recorded in the operative notes of the patients were also reviewed. Chi-square test was used in the statistical analyses.

RESULTS

Incidence, Age and Sex

Of 4,553 patients with head injury, 270 (5.9%) had EDH and 190 patients (4.1%) were operated upon for EDH. There were 161 males (85%) and 29 females (15%). The female / male ratio was 1/6. The ages of the patients ranged from 4 to 70 years (mean 28 years). Seventy percent of the patients were between 15 and 40 years of age.

Types of Injury

Motor vehicle accidents were the most common cause of injury in this series. One hundred and three patients (54%) had traffic accidents and fall from a height caused EDH in 58 (30.6%).

Symptoms and Signs

The examination on admission revealed either a scalp laceration or cephalhaematoma in 158 patients (83.2%) and 63 (39.8%) had pyramidal signs such as hemiparesis and extensor plantar response. Sixty-five patients (41.1%) were comatose. A lucid interval was described in 35 patients (18.4%). Pupillary abnormalities were present in 64 (34%). Nineteen (29.2%) of the comatose patients had respiratory irregularities. Bradycardia and arterial hypertension were noticed in 44 patients (23%).

Radiological Evaluation

Of 149 patients who had plain skull X-ray films, 34 had a normal skull X-ray film and a fracture unrelated to EDH was seen on the skull X-ray films of 4 cases. The fractures crossing the middle meningeal artery and a dural sinus were detected in 29 and 9 patients, respectively. EDH was associated with a depressed skull fracture in 14 patients. Angiography was performed in 7 patients. The diagnosis of EDH was based on exploratory burr-holes in 22 patients, and CT in 161. The shape of EDHs on CT scan was biconvex in 133, monoconvex in 17 and bilenticular-biconvex in 11 patients. EDHs appeared hyperdense on CT scans performed within 10 days after trauma. A hypodense EDH was detected in only two patients more than 10 days after trauma.

Site of EDHs

Temporo-parietal region was the most frequent site of EDHs (35.7%). Parietal and frontal regions were the second and third frequent locations, respectively. Three cases had bilateral EDHs and the haematoma was localized in the vertex in one patient. There were 12 patients with posterior fossa EDH (6.3%).

Operative Findings

A clotted EDH was evacuated in the majority of the cases (132 patients). The nature of EDH was both clot and active bleeding in 53 patients. It was clot and partially liquefied in 4 and pure liquefied haematoma in one case. The origin of bleeding was the middle meningeal artery in 68 (35.7%), bone and dural oozing in 70 (36.8%), and dural veins in 15 (7.8%) patients. In 37 (19.4%) patients, the origin of bleeding could not be identified.

Outcome

GOS of the patients is shown in Table I. The overall mortality was 15.7% (30 patients). All deaths occurred in comatose patients, except for one case (p < 0.0001). Patients with bradycardia and hypertension had a higher mortality rate (43%) than those without bradycardia and hypertension (7.5%) (\( \chi^2 = 29.688, df = 1, p < 0.0001 \)). The presence of respiratory irregularities also worsened the outcome.
Table I: Glasgow outcome scale (GOS) scores of 190 patients with EDH.

<table>
<thead>
<tr>
<th>GOS</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>138</td>
</tr>
<tr>
<td>Moderate deficit</td>
<td>16</td>
</tr>
<tr>
<td>Severe deficit</td>
<td>6</td>
</tr>
<tr>
<td>Vegetative state</td>
<td>0</td>
</tr>
<tr>
<td>Death</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>190</td>
</tr>
</tbody>
</table>

The mortality rates were 73% and 9.3% in those with and without respiratory irregularity, respectively ($\chi^2 = 48.490, df = 1, p < 0.0001$). Of 63 patients with pyramidal signs, 16 died and there were 14 deaths in 127 patients without pyramidal signs ($\chi^2 = 4.329, df = 1, p = 0.037$). Mortality rate was 42% in the patients with pupillary changes and 2% in those with a normal pupil size and reaction ($\chi^2 = 47.630, df = 1, p < 0.0001$) (Figure 1).

A midline shift greater than 10 mm and haematoma thickness greater than 30 mm significantly increased the mortality ($\chi^2 = 28.934, df = 1, p < 0.0001$ and $\chi^2 = 7.597, df = 1, p = 0.006$, respectively). Fifty-nine percent of 27 patients having EDH greater than 90 ml in volume died, whereas this rate was only 4% in those with EDH of 90 ml or less ($\chi^2 = 53.696, df = 1, p < 0.0001$) (Figure 2).

The mortality was 38% in patients in which an active bleeding had been detected at surgery and 7.5% in patients with clotted EDH. No death occurred in cases with partially liquefied or liquefied EDHs. This difference was statistically significant ($\chi^2 = 26.834, df = 3, p < 0.0001$). Mortality was highest (35%) in patients in which the origin of bleeding had been the meningeal artery. The mortality rates significantly varied with the origin of bleeding, i.e., 13.3% in venous bleeding, 4.2% in dural and bone oozing and 2.7% in unidentified origin ($\chi^2 = 31.257, df = 3, p < 0.0001$) (Figure 3). The interval from trauma to surgery significantly influenced the outcome. About one-third of the patients operated upon within 6 hours after injury died, whereas fewer patients died as the interval increased, there were no mortalities in the patients undergoing surgery more than 10 days after injury ($\chi^2 = 26.821, df = 4, p < 0.0001$) (Figure 4).

**DISCUSSION**

Almost 6% of 4,553 patients with head injury admitted to our department had EDH. However, 4.1% (190 patients) were operated upon for EDH. Traffic accidents and falls are the first and second predominant types of injury in many series (1,3). In
Twenty-nine (44.6%) of 65 comatose patients died, whereas only one death (0.8%) occurred among non-comatose patients \((p < 0.0001)\). The level of consciousness significantly influenced the outcome.

Pyramidal syndrome and unilateral pupil dilatation are invaluable signs in localizing the site of EDH and are seen in 20% and 45% \((3,9,11,16)\) of patients with EDH, respectively. Hemiparesis was determined in 31.5% of our patients. Hypertension, bradycardia and respiratory irregularities are rarely seen in patients with EDH. The presence of these findings was determined as a significant factor in terms of mortality.

Although the most common site of EDHs has been reported as the temporal region \((1,10,12)\), EDHs were localized in the temporo-parietal or parietal regions in some recent papers \((3,11,16)\), since CT has enabled us to localize the haematoma precisely. Temporo-parietal location was the most common site of EDHs in our patients and was associated with highest mortality. However the difference in mortality rates according to the site of haematoma was not statistically significant. Posterior fossa EDHs comprise 3.4 to 13% of all EDHs and have an 11 to 22% mortality \((4,5,13,18)\). There were 12 patients with posterior fossa EDH \((6.3\%)\). The origin of bleeding was a dural vein in 2, a dural sinus in 6 patients and was undetermined in 4. There was no death in patients with posterior fossa EDH whose GCS scores were 9 or greater.

In some series, surgery was performed within 6 hours, 6 to 24 hours and more than 24 hours after injury in 30-57%, 2-41% and 13-35% of the patients with EDH, respectively \([1,9,11,16]\). Trauma to surgery interval is an important factor in terms of outcome. The mortality rate has been 25-35% in patients operated upon within 6 hours following trauma \((4,11)\). Among 53 patients who underwent surgery within 6 hours after injury, the mortality rate was 37.7%. The mortality rate in patients who underwent surgery between 6 to 24 hours after injury was 7.8% in our series \((p < 0.0001)\). The majority of patients undergoing early surgery were comatose. This may account for the higher mortality rate.

There were 20 deaths in 53 patients \((37.7\%)\) in which an active bleeding and a clot were detected at surgery. However, of 132 patients in which a clot was evacuated, only 10 \((7.5\%)\) died. Active bleeding was significantly associated with the worst outcome.
The number of deaths also varied significantly with the origin of EDH. The mortality rates were 38%, 13.3% and 2.6% in arterial and venous bleedings and in bleedings of unidentified origin, respectively \((p<0.0001)\). It is conceivable that the development of EDH in a short time may be responsible for a greater death rate in patients with an active bleeding.

In general, 73% of patients with EDH have a good outcome. The outcome figure of our patients was consistent with those in the literature \((3,9,11)\). The experimental work of Ford and McLaurin \((6)\) supports the hypothesis that the enlargement of acute EDHs occurs shortly after the trauma. However, the late enlargement of EDH in patients managed conservatively, and the development of delayed EDHs on serial CT scans are contrary to this hypothesis \((15,17)\). The primary factor on outcome is GCS scores of the patients at the time of surgery. Therefore early surgery is crucial in the management of EDHs that are a dynamic process.

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