Delayed onset of traumatic extradural hematoma

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During a 4½-year period, seven patients with delayed onset of an extradural hematoma were seen among 80 consecutively treated cases of extradural hematoma for a frequency of 8.75%. The hematomas were insignificant or not present on initial computerized tomography (CT) scanning. Repeat CT scans within 24 hours of admission showed sizeable hemorrhages. Six hematomas were evacuated, and one was reabsorbed spontaneously. In only one patient did neurological deterioration herald the onset of the extradural hematoma, four patients remained unchanged, and two improved before diagnosis. Intracranial pressure (ICP) was monitored in five patients, four of whom showed intermittent rise in pressure despite preventive treatment. Intracranial hypotension and rapid recovery from peripheral vascular collapse seemed to be contributory factors in the delayed onset of an extradural hematoma. Awareness of this entity, a high degree of vigilance, ICP monitoring, and repeat CT scanning within 24 hours of injury are strongly recommended in these cases, especially after decompression by either surgical or medical means, recovery from shock, or whenever there is evidence of even minimal bleeding under a skull fracture on the initial CT scan.

KEY WORDS □9 brain injury □9 extradural hematoma □9 delayed onset

EXTRADURAL hematoma may be classified into two clinical groups, acute and subacute, based on the time of onset of symptoms and signs. Acute cases include those patients with neurological compromise that requires surgery within 3 days of trauma; the vast majority of these undergo surgery within the first 24 hours. Subacute cases are less well defined and include patients who present 4 days or longer after injury.3,6,8,31

Computerized tomography (CT) scanning has revolutionized the evaluation of the head-injured patient, and has made it easier to perform initial and follow-up examinations for intracranial hemorrhage. As a direct consequence, a new pathological dimension has been added to the clinical classification mentioned above, reviving the concept of delayed traumatic intracerebral hematoma and bringing to light the problem of delayed traumatic extradural hematoma. Both of these conditions are increasingly recognized as CT scanners become more widely available and as the indications of repeat CT scanning increase. Extradural hematomas of delayed onset are rare and, in large series of head injuries evaluated by CT scanning, are often not discussed.4,13,22,23,27,30

In this report, we present our experience with seven cases of delayed traumatic extradural hematoma. We describe the features of this dangerous condition, which may be difficult to diagnose. The cases are summarized in Table 1 and two cases are described in detail.

Illustrative Case Reports

Case 1

This 39-year-old man fell from a tanker truck, striking his head against the pavement. He was unconscious at the scene of the accident but was alert on arrival at a local hospital. His pupils were normal and he could move his limbs freely, but within half an hour he became comatose. He was admitted to the Rambam Medical Center in a coma. Both pupils were dilated and nonreactive, and he manifested bilateral decerebrate posturing. He was noted to have a right temporal hematoma under the scalp and brisk bleeding from the right ear. Blood pressure was 150/90 mm Hg, pulse was 64 beats/minute, and breathing was deep and rapid. He was immediately hyperventilated. Skull x-ray films revealed a right temporal skull fracture and a CT scan showed a left subdural hematoma, which was removed.
Delayed traumatic extradural hematoma

**Case 1**

Initial computerized tomography scans showing a left acute subdural hematoma with ventricular compression and midline shift to the right (Fig. 1).

Repeat computerized tomography scans 24 hours postinjury and 18 hours after left parietal craniectomy. A contralateral delayed traumatic extradural hematoma is revealed with slight ventricular compression and midline shift to the left (Fig. 2).

Intraoperative swelling was only partially controlled with mannitol and Pentothal (thiopental) and a temporal lobectomy was required. Intracranial pressure (ICP) was not monitored, but was controlled by medication throughout the postoperative period.

The following morning the patient was unchanged and a repeat CT scan revealed a contralateral extradural hematoma associated with the fracture (Fig. 2). The patient failed to improve and died 8 days after trauma.

**Case 4**

This 33-year-old man was rendered unconscious after sustaining a head injury in a motor-vehicle accident. On arrival at our institution he was comatose, his right pupil was dilated and nonreactive (he had a prosthetic left eye), and he exhibited a right hemiparesis. He had bilateral periorbital ecchymosis and brisk bleeding from the left ear. Breathing was slow and superficial, blood pressure was 165/90 mm Hg, and pulse was 64 beats/minute. He was immediately intubated and hyperventilated. Skull x-ray films revealed bilateral fractures in the right parietal and left frontal areas. ACT scan showed a right parietal extradural hematoma and a left parietal contusion with a thin subdural hematoma (Fig. 3). The extradural hematoma was removed and a feeding tube was left in the subdural space to monitor ICP.

Some 8 hours later, the patient's neurological status deteriorated, bilateral decerebrate posturing appeared, and ICP rose sharply. A repeat CT scan revealed that a large extradural hematoma had collected over the brain contusion (Fig. 4). The hematoma was immediately evacuated. The patient gradually improved and was discharged to a rehabilitation hospital 1 month after surgery. When last seen 18 months later, he had returned to his previous work as a mechanical engineer and was neurologically normal except for difficulty in speaking foreign languages.

**Discussion**

During a 4½-year period, we have treated a total of 105 acute traumatic extracerebral hematomas; 80 of these were extradural hematomas, seven of which had a delayed onset. Delayed traumatic extradural hematoma is considered to be uncommon, and in some large series is not mentioned at all. Nevertheless, the incidence in our total series is high: 6.6% of the 105 acute extracerebral hematomas and 8.75% of...
FIG. 3. Case 4. Initial computerized tomography scans showing a right parietal extradural hematoma. A left parietal brain contusion and a thin subdural hematoma are also visible.

FIG. 4. Case 4. Repeat computerized tomography scans 8 hours postinjury and 3 hours after right parietal craniotomy with cerebral contusion underneath. A left contralateral delayed traumatic extradural hematoma and hemorrhagic contusion of the brain can be seen, with ventricular compression and midline shift to the right.

the extradural hematomas. This compares with the experience of other recent large series where extradural hematomas of delayed onset reached 10%. Six of our seven patients underwent surgery and in the seventh the hematoma reabsorbed spontaneously. A summary of findings and results is displayed in Table 1.

The term "delayed traumatic intracranial hematoma" describes the dynamics of the postinjury bleeding. Patients with this disorder have no or only insignificant hemorrhage at first evaluation (usually on CT scanning). The hematoma is found only on a routine follow-up CT scan or a repeat scan to assess clinical or ICP deterioration. Only by scanning the patient more than once can this condition be diagnosed. Otherwise it would be impossible to distinguish between delayed bleeding and late presentation of symptoms as in subacute and chronic subdural hematomas.

The ever-increasing indications for early CT scanning in head-injured patients have disproved, at least in part, the statement that extradural hematoma formation achieves near-maximum size minutes after trauma. The fundamental causes of delayed traumatic extradural hematoma are not only the source of bleeding or the extension of the separation of the dura mater from the inner surface of the calvaria, as was shown in acute experimental models by Ford and McLaurin, but also include the disturbance of the equilibrium in the brain and the hemostatic effect of the ICP. Low ICP, high blood pressure, or rapid correction of hypotension, acting concurrently or in isolation, favor the production of an extradural hematoma, especially at areas already injured such as under a skull fracture.

Six of the seven patients in this series developed an extradural hematoma in an area underlying a previously known skull fracture; four of them (Cases 2, 3, 4, and 7) showed minimal bleeding at that site on the initial CT scan. In Cases 2 and 7, the findings were correctly interpreted as nonsurgical extradural hematomas; in the other two, the tentative diagnosis was that of brain contusion and subdural hematoma. All the patients in this series were treated soon after trauma by a regimen that reduced the ICP rather drastically, with intubation and hyperventilation; mannitol was given in Cases 2, 5, and 7, and Pentothal in Case 1. Five patients (Cases 1, 3, 4, 5, and 6) developed an extradural hematoma within 24 hours of removal of a previous acute extracerebral hematoma that left them with lowered ICP. Four patients (Cases 1, 4, 6, and 7) had brisk otorrhea, equivalent of continuous cerebrospinal fluid drainage.
Delayed traumatic extradural hematoma

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Injury Site</th>
<th>Clinical Status</th>
<th>Initial CT Scan</th>
<th>Therapy</th>
<th>Interval (hrs)</th>
<th>Findings</th>
<th>Repeat Scan</th>
<th>Therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39, M</td>
<td>rt temporal</td>
<td>coma, bilat midriasis</td>
<td>rt parietal SDH</td>
<td>evacuation of SDH</td>
<td>24</td>
<td>condition unchanged, ICP the same</td>
<td>rt EDH</td>
<td>evacuation of EDH</td>
<td>died</td>
<td></td>
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<tr>
<td>2</td>
<td>30, M</td>
<td>bilat parietal</td>
<td>agitation, no focal signs</td>
<td>small bifrontal EDH</td>
<td>conservative</td>
<td>16</td>
<td>regained consciousness, ICP unchanged</td>
<td>large bifrontal EDH</td>
<td>evacuation of EDH</td>
<td>excellent</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>53, M</td>
<td>It parietal</td>
<td>coma, rt hemiplegia</td>
<td>rt parietal EDH, rt temporal contusion</td>
<td>evacuation of EDH</td>
<td>24</td>
<td>improved, alert; ICP normal</td>
<td>rt parietal EDH</td>
<td>conservative</td>
<td>fair</td>
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<tr>
<td>4</td>
<td>33, M</td>
<td>bifrontal</td>
<td>coma, rt hemiplegia, rt midriasis</td>
<td>rt parietal EDH, lt parietal SDH, &amp; contusion</td>
<td>evacuation of EDH</td>
<td>8</td>
<td>deterioration, decerebrate, ICP elevated</td>
<td>lt parietal EDH</td>
<td>evacuation of EDH</td>
<td>excellent</td>
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<tr>
<td>5</td>
<td>25, M</td>
<td>It temporal</td>
<td>coma, rt midriasis, decerebration</td>
<td>lt parietal EDH</td>
<td>evacuation of EDH</td>
<td>24</td>
<td>condition unchanged, ICP elevated</td>
<td>lt frontal EDH</td>
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<td>6</td>
<td>10, F</td>
<td>rt frontal</td>
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<td>rt temporal EDH, rt parietal SDH</td>
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<td>condition unchanged, ICP elevated</td>
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<td>7</td>
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*Abbreviations: CT = computerized tomography; EDH = epidural hematoma; SDH = subdural hematoma; ICP = intracranial pressure.

Low ICP can bring about intracranial bleeding by itself, without trauma, as in cases of extracerebral hemorrhage complicating shunt surgery, ventriculography, ventricular and subarachnoid drainage, spinal anesthesia, and posterior fossa and spinal intradural operations. One patient (Case 3) was hypotensive at the time of emergency evacuation of an extradural hematoma. Blood pressure recovered soon after, and was combined with postoperative intracranial hypotension. Within 24 hours a contralateral delayed traumatic extradural hematoma was demonstrated (Table 1).

Neurological deterioration is not a reliable indication in the diagnosis of delayed traumatic extradural hematoma, and was a precursor to that disorder in only one case. Four patients remained neurologically unchanged throughout their course, and two even improved during the interval preceding discovery of extradural hematoma. Intracranial pressure is a more useful prognosticator, and ICP monitoring is advised since it preceded delayed extradural hematoma in four of the five patients in whom it was recorded.

A high clinical suspicion or awareness of this entity is necessary to diagnose this disorder. Complete reliance on neurological and ICP monitoring, trust in an early scan, and a relative complacency after an apparently successful initial decompressive operation were the principal obstacles to a diagnosis of delayed extradural hematoma. Head-injured patients who undergo measures to control their ICP, patients with decompression of their injury, thus releasing a certain tamponade effect, and patients who arrive at the hospital in a state of shock are all at high risk for delayed extradural hematoma. We strongly recommend that this group of patients undergo routine repeat CT scanning within 24 hours of admission or of emergency decompressive surgery, regardless of neurological status or ICP. This is especially true in cases with a skull fracture contralateral to the original hemorrhage or signs of bleeding underneath any previously detected skull fracture.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Hyper-ventilation</th>
<th>Mannitol</th>
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<th>Low Blood Pressure</th>
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The concept was the moving force that made early diagnosis possible in Cases 2 and 7.

Finally, the increasingly liberal early use of CT scanning for neurologically normal head-injured patients will probably identify some patients with subacute or chronic presentation as having delayed traumatic intracranial hematomas.

References


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