Basal ganglia hematomas in severely head injured patients: clinicoradiological analysis of 37 cases

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Object. The authors analyzed the clinicoradiological presentation of traumatic basal ganglia hematomas (TBGHs) in severely head injured (SHI) patients.

Methods. The records of 37 patients (28 male and nine female patients with a mean age of 28 years) in whom computerized tomography (CT) scans revealed TBGHs 2 ml or more in volume were retrospectively reviewed. These cases represented 2.4% of the total series of 1526 SHI patients admitted to the authors’ institution between 1979 and 1998. Thirty-five patients (94%) were involved in traffic accidents and only two exhibited a period of lucidity. Associated extracranial injuries were seen in 21 patients (57%) and coagulation disorders in 32 (86%). Skull fracture was present in 10 (43%) of the 23 patients in whom skull x-ray films were obtained. Computerized tomography findings indicated diffuse axonal injury in 27 patients (73%), intraventricular hemorrhage in 22 patients (59%), and subarachnoid hemorrhage in 16 patients (43%). In all but two patients, the TBGHs were visible on the initial CT scan, and in 28 cases (76%) these hematomas were contralateral to the side of impact. Hematoma enlargement over the first few posttraumatic days was noted in 65% of the patients in whom control CT scans had been obtained (22 of 34 patients). Four patients (11%) underwent surgery to remove their TBGHs. Final outcomes were poor: 22 patients (59%) died, two (5%) became vegetative, seven (19%) experienced severe disabilities, and only six patients (16%) made a favorable recovery.

Conclusions. Traumatic basal ganglia hematomas are dynamic lesions that tend to enlarge during the acute posttraumatic period. The overall prognosis in this series was poor. Patients in whom the volume of the hematoma was larger than 25 ml and those in whom hematoma volume enlargement or raised intracranial pressure occurred had the worst outcomes, perhaps indicating the need for a more aggressive surgical treatment.

KEY WORDS • basal ganglion hematoma • severe head injury • computerized tomography

Traumatic basal ganglia hematomas occur mainly in young persons; however, the clinicopathological profile of these lesions is still poorly defined. The purpose of this work was to analyze retrospectively the clinical characteristics of 37 SHI patients with TBGHs, while paying special attention to sequential CT-visualized and ICP changes that occur during the acute posttraumatic period and to the final prognosis.

Clinical Material and Methods

Inclusion Criteria and Patient Population

For the purposes of this study, TBGH is defined as an intracerebral hemorrhagic lesion located in the basal ganglia (caudate nucleus, putamen, and globus pallidus) or neighboring structures such as the internal capsule and the thalamus, which acutely develops after head injury. Two clinicoradiological criteria were necessary to include patients in this study: 1) a GCS score of 8 or less following nonsurgical resuscitation determined at the outset or following the patient’s subsequent deterioration into coma.
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within 48 hours postinjury; and 2) a presenting TBGH with a total volume of 2 ml or more observed on either the initial CT scan or a control CT scan obtained during the acute posttraumatic period. Hemorrhagic lesions measuring less than 2 ml were considered to be petechiae and not hematomas.

From November 1979 to April 1998, 37 consecutive patients with TBGHs were admitted. They represented 2.4% of the total series of 1526 SHI patients who were treated at our institution during the same period. There were 28 male and nine female patients, ranging in age from 14 to 81 years (average 28 years). The causes of injury included a car accident in 28 cases (76%), motorcycle accident in four (11%), pedestrian accident in two (5%), fall in two (5%), and bicycle accident in one patient (3%). Patients arrived at our hospital either directly from the scene of the accident or from regional hospitals, and were transported by either a well-equipped ambulance or a helicopter. Twenty-nine patients (78%) were admitted to a hospital within 1 hour after injury (range 20–135 minutes), and two patients (5%) were conscious on arrival.

Radiological Studies

The site of impact on the head was suspected by the presence of a skull fracture and the location of the scalp wound. Skull x-ray films were obtained in 23 patients (62%). All patients underwent CT scanning as soon as possible after admission (admission or initial CT scan); 29 (78%) of these patients underwent the initial scanning study within the first 4 hours posttrauma (range 40 minutes–18 hours posttrauma). Delays in obtaining the initial CT scan occurred in patients referred from regional hospitals that did not have CT scanners. All but two patients, who died within the first 24 hours of injury, subsequently underwent CT scanning sessions. The CT scan that revealed the most significant change in the volume of the TBGH was selected as the control CT scan. The mean interval between initial and control CT scans was 42 hours (range 2–146 hours). In 27 patients (77%) the control CT scan was obtained within 48 hours of the initial examination. Intracranial pathological conditions were classified according to the Traumatic Coma Data Bank classification.39

The number and side of hemorrhagic lesions in the region of the basal ganglia, as well as the presence of low attenuation areas and the blood level associated with the hematoma were noted. Hematoma volume was calculated using the following formula: length × width × height / 0.5.40 The length and width were obtained from the CT slice demonstrating the largest hyperdense area of the TBGH; the height of the hematoma was determined by the number of slices in which it appeared and, thus, a TBGH that appeared in only one slice was assumed to be 1 cm high. In cases in which a patient harbored several hemorrhagic lesions located in the basal ganglia region, the volumes of the separate lesions were summed. Sequential changes in volumes of the TBGHs, as gleaned from the initial and the control CT scans, could be recorded in 34 patients.

Case Management

Patients were treated according to a standardized protocol that included aggressive treatment of raised ICP, which was initiated at an upper threshold of 20 mm Hg. The ICP was measured in 30 patients (81%) with the aid of an intraventricular catheter, an epidural fiberoptic sensor, or a parenchymal fiberoptic device. The duration of the ICP recording ranged from 1 to 14 days (average 5 days). Patients were separated into six categories, depending on the ICP course: low, low-high-low, high–low, low–high, high-low-high, and high. We considered the last three categories to be elevated ICP. Almost every patient in whom raised ICP developed experienced it within the first 3 days postinjury. Only four patients underwent evacuation of their TBGHs. Although the majority of the patients had associated intracranial pathological conditions apart from the TBGH visible on CT scans, only two required surgical intervention for focal lesions.

Final Outcomes

Final outcomes in patients were graded at 6 months postinjury was classified according to GOS categories: good recovery, moderate disability, severe disability, vegetative state, and death. The categories of good recovery and moderate disability were considered to represent a favorable outcome. Motor and language impairments, other focal deficits, and psychological sequelae were assessed in patients whose conditions evolved into the categories of good recovery, moderate disability, and severe disability. In most of the patients, the neurological injury was the major source of mortality and morbidity.

Results

Clinical Findings

In 29 patients (78%) the GOS score was 6 or less following resuscitation. The average GCS score in the whole group was 6, and the motor scores were as follows: localizing response in eight patients; withdrawal in 12; abnormal flexion in 11; decerebration in five; and flaccidity in one. The mean motor score was 4. Eight patients (22%) exhibited pupillary changes (unilateral mydriasis) on arrival at the hospital. In 21 patients (57%) there were relevant associated extracranial lesions. Early after injury, arterial hypotension (systolic blood pressure ≤ 90 mm Hg) was present in nine patients (24%), hypoxia (SaO2 ≤ 90%) in 13 (35%), and hypotension associated with hypoxia in five (14%). Prothrombin time (normal > 75%) was altered in 24 patients (65%) and activated partial thromboplastin time (normal < 34 seconds) in 19 patients (51%). The plasma level of fibrinogen (normal > 2 g/L) was abnormal in 19 patients (51%) and the platelet count (normal > 150 × 10⁹/L) was altered in six (16%). As a whole, coagulation disorders occurred in 32 patients (86%) early after injury, and all of these patients received aggressive treatment (fresh frozen plasma and platelets) as soon as possible.

Radiological Findings

Skull fractures were present in 10 (43%) of the 23 patients in whom skull x-ray films were obtained. Apart from the TBGHs, the following lesions were observed on the CT scans: signs of DAI in 27 patients (73%), intraven-
tricular hemorrhage in 22 (59%), subarachnoid hemorrhage in 16 (43%), focal brain contusion in six (16%), and acute subdural hematoma in five (14%). The TBGHs were located on the right side in 20 patients (54%) and on the left side in 17 (46%); they were located contralateral to the side of impact in 28 patients (76%). In three patients basal ganglia petechiae were present on the side opposite the TBGH. Two patients in whom the results of the initial CT scans, obtained 90 or 180 minutes after injury, were normal later sustained TBGHs (3.2 and 43.3 ml in volume, respectively) that were visible on control CT scans obtained 40 hours and 11 hours later, respectively. Volumes of the TBGHs, associated midline shift, and the presence of surrounding low-density areas or blood levels are reflected in Table 1. In 10 patients (27%) hematomas larger than 25 ml were visible on either admission or control CT scans. Of the 34 patients in whom control CT scans were obtained to investigate sequential changes in the volume of the TBGH, enlargement was noted in 22 (65%), a decrease in two (6%), and no change in 10 (29%). The increase in volume ranged from 0.6 to 47.6 ml (average 12.9 ml, median 6.5 ml; Fig. 1), and it occurred as early as within 2 hours postinjury (Fig. 2). In one of the two patients who were conscious on arrival at the hospital, hematoma enlargement was revealed on the control CT scan, which was considered responsible for the patient’s deterioration. The time intervals between initial and control CT scans in the two patients in whom a decrease in hematoma volume was apparent were 36 hours and 38 hours.

**Table 1**

<table>
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<tr>
<th>CT Findings</th>
<th>Initial CT Scan (37 patients)</th>
<th>Control CT Scan (34 patients)</th>
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<td>TBGH volume (ml)</td>
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<td>midline shift &gt;5 mm (%)</td>
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* Initial CT scans were available in all patients. Control CT scans were not obtained in two patients and one was not obtained in another until after surgery to remove the TBGH had been performed.

Fig. 1. Computerized tomography scans obtained in an 81-year-old man who was admitted to the hospital 40 minutes after he had been struck by a car. At the time of admission this patient’s GCS score was 8 (motor score 5); there were no pupillary changes, arterial hypotension, hypoxia, or coagulation disorders, and findings on a skull x-ray film were normal. **Left:** The initial CT scan obtained 2 hours after injury revealing a left-sided TBGH (volume 39.5 ml) with a midline shift less than 5 mm. Because of the patient’s advanced age, his ICP was not recorded and the hematoma was not evacuated. **Right:** Control CT scan obtained 44 hours after the initial scan, revealing massive enlargement of the hematoma (volume 87.1 ml), intraventricular hemorrhage, and a marked midline shift. The patient died 4 days posttrauma. This was the largest enlarged TBGH observed in this series.

Fig. 2. Computerized tomography scans obtained in a 26-year-old man who was unconscious after being involved in a traffic accident. His GCS score, which was determined on his arrival at the hospital 1 hour after injury, was 7. There were no pupillary changes, arterial hypotension, hypoxia, or skull fracture. Prothrombin time was altered. **Left:** Admission CT scan obtained 115 minutes postinjury revealing two hemorrhagic lesions (total volume 4.5 ml) in the left basal ganglia, contralateral to the side of impact. Hemorrhagic lesions scattered throughout the deep white matter were also observed (not shown). A parenchymal fiberoptic ICP sensor was placed. Initially the patient’s ICP was normal but, suddenly, bilateral mydriasis developed and his ICP became uncontrollable, rising to over 40 mm Hg. **Right:** The patient’s control CT scan obtained 2 hours after the admission scan, demonstrating enlargement of the TBGH (46 ml) accompanied by a low-density area and a midline shift greater than 5 mm. The patient died 23 hours after admission. This was the most rapidly enlarging TBGH in this series.

**Course of ICP**

Intracranial pressure measured in 30 patients was low throughout the course of the injury in eight patients, low-high-low in four, high–low in one, low–high in seven, high–low-high in seven, and high throughout the course of the injury in three patients. As a whole, 17 patients (57%) exhibited elevated ICP and all of them died. Of the remaining 13 patients (43%) who had normal ICPs, only two died of associated extracranial lesions. In the three patients who died following surgery for their TBGHs raised ICP was demonstrated after surgery, and the only survivor had normal postoperative ICP. Final outcome was more closely related to ICP levels than to hematoma size.
Surgical Treatment

One patient underwent surgery for a focal brain contusion and another for an acute subdural hematoma on arrival. Overall, only four patients (11%) underwent surgical evacuation of TBGHs larger than 25 ml between 3 and 59 hours after injury (average interval 21 hours; Figs. 3 and 4). In the remaining patients, medical treatment was selected, even in the presence of raised ICP. Of the surgically treated patients, three died and one became severely disabled.

Final Outcomes

The final outcomes of patients are shown in Table 2. Overall, final outcomes were unfavorable in 31 patients (84%) and favorable in only six (16%). In the 22 patients who died, death was directly attributable to the TBGH in 10 (45%; seven patients with TBGHs larger than 25 ml and three with smaller clots surrounded by a large low-density area); to uncontrollable ICP related to diffuse brain swelling in six patients (27%), including one patient who required surgery for an acute subdural hematoma; and to associated extracranial lesions in six patients (27%). Prognoses as related to hematoma volumes observed on initial and control CT scans are reflected in Figs. 5 and 6, respectively. The mortality rate was not significantly different in patients with hematomas smaller than 10 ml, compared with those with hematoma volumes of 10 to 25 ml or larger than 25 ml. Of the 10 patients in whom TBGHs measured more than 25 ml at the time of either the initial or the control CT scanning session, only two who had normal ICPs survived (Figs. 4 and 6). Figure 7 depicts final outcomes as related to changes in hematoma volume. The mortality rate was 73% (16 of 22) in patients in whom there was hematoma enlargement compared with 33% (four of 12) in those without clot enlargement. However, because of the small size of the samples, this difference did not reach statistical significance. Moreover, causes of death and overall outcomes in the subgroup of patients in whom hematoma enlargement was identified were not different from outcomes observed in the global series.

In all 13 patients who comprised the GOS categories of good recovery, moderate disability, and severe disability, persistent morbidity developed: motor impairments were observed in 12 patients (92%), language disturbances in seven (54%), other focal deficits in eight (62%), and psychological sequelae in 10 patients (77%). Only one patient with a small TBGH measuring 2.6 ml, which did not exhibit enlargement on the control CT scan, displayed normal motor function. The patient who underwent surgery for a focal brain contusion experienced severe disability related to the basal ganglia lesion.

Discussion

Definition and Mechanism of Formation of TBGHs

Courville and Blomquist believed that TBGHs were small, usually multiple, and located in the zone of the lentiform nucleus and external capsule, on either one or both sides. In contrast, spontaneous hemorrhages in this area were large, solitary, and located in the region of the thalamus and internal capsule. Therefore, solitary deep-seated intracerebral hematomas with mass effect were always deemed spontaneous, and, thus, when a patient suffered a head injury it was considered to be the consequence rather than the cause of the hematoma.

However, in 1959, Mosberg and Lindenberg reported on one patient whose clinical condition progressively de-
teriorated and who died after suffering a head injury. In their patient a massive hematoma in the pallidum and a ruptured arterial twig of the anterior choroidal artery within the clot were found at autopsy. After histological examination of the vessel, these authors judged the arterial tear to be traumatic in origin, demonstrating that massive hematomas in the basal ganglia might be produced by head injury. Furthermore, they reviewed 19 more cases of traumatic lenticular hemorrhage. To these authors, traumatic hemorrhage in the region of the basal ganglia should be the diagnosis in cases in which trauma occurs while the head is in motion (coup and contrecoup) and the impact, sufficient to deform the skull, is applied to the vertex, forehead, or occipital area and directed toward the tentorium. Under these circumstances, there would be a shift of the brain through the tentorial notch,24,32 producing shearing forces among different tissues, which in turn would produce stretching and tearing of the pallidum branches of the anterior choroidal artery, and occasionally, of the striate body branches of the MCA, resulting in hemorrhages in the basal ganglia region. Because other brain structures might also be displaced through the tentorial notch, contusions and/or small hemorrhages in areas such as the corpus callosum, thalamus, hypothalamus, hippocampus, and midbrain could be associated with the TBGH. Thus, previous occurrence of a head injury and the evidence of other typically traumatic lesions in the same patient would suggest a traumatic origin of the hemorrhage in the basal ganglia. It should also be noted that TBGHs may occasionally be accompanied by ventricular hemorrhage, simulating hypertensive hematoma.15

Mosberg and Lindenberg32 also suggested that the compression of vessels irrigating the basal ganglia region might cause not only hemorrhage but also ischemia, and considered the traumatic hemorrhages of the basal ganglia to be hemorrhagic contusions, in contrast to Courville and Blomquist,8 who labeled them hematomas. Whether TBGHs should be considered contusions or, by contrast, intracerebral hematomas remains a secondary question, and the ultimate mechanisms of these lesions still remain unclear.

In 1980, Maki and colleagues28 reported on a 6-year-old girl who after suffering severe head injury presented with a massive unilateral hemorrhage measuring 20 ml and located contralateral to the side of impact; the lesion required surgical aspiration through a craniotomy. The authors postulated that the anterior stretch of the lateral branch of the perforating vessel of the MCA, secondary to the opposite directions of rotation of the skull and the brain after injury, plays a major role in the pathogenesis of these lesions. This mechanism could also explain the occurrence of TBGHs located contralateral to the side of im-

TABLE 2
Final outcomes in 37 patients with TBGHs

<table>
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<tr>
<th>Outcome</th>
<th>No. of Patients (%)</th>
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<tbody>
<tr>
<td>good recovery</td>
<td>2 (5)</td>
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<tr>
<td>moderate disability</td>
<td>4 (11)</td>
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<tr>
<td>severe disability</td>
<td>7 (19)</td>
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<tr>
<td>vegetative state</td>
<td>2 (5)</td>
</tr>
<tr>
<td>death</td>
<td>22 (59)</td>
</tr>
<tr>
<td>total</td>
<td>37 (100)</td>
</tr>
</tbody>
</table>

FIG. 4. Sequential CT scans obtained in a 17-year-old man admitted 2 hours after being injured in a bicycle accident. The patient’s GCS score at admission was 4 and his pupils were isocoric. Prothrombin time, activated partial thromboplastin time, and the plasma level of fibrinogen were abnormal. There was no arterial hypotension, hypoxia, or skull fracture. Left: Admission CT scan obtained 150 minutes after trauma revealing a large hematoma (volume approximately 52 ml) with a blood–fluid interface (high attenuation area in the lower portion of the lesion and a low-density zone in the upper portion) in the left basal ganglia, contralateral to the side of impact; petechiae in the deep white matter and the contralateral basal ganglia, as well as intraventricular and subarachnoid hemorrhage and a midline shift greater than 5 mm were also noted. The TBGH was evacuated via a craniectomy 3 hours postinjury, and the patient’s ICP was recorded by means of a parenchymal fiberoptic device. Center: Postoperative CT scan obtained 13 hours after the admission scan, demonstrating a small residual clot in the left basal ganglia region, a midline shift less than 5 mm, and enlargement of the hemorrhagic lesion in the right extreme capsule, which is associated with the blood–fluid interface and a low-attenuation area. The ICP course during the following 8 days was low-high-low. Right: A later CT scan obtained 40 days posttrauma demonstrating residual hypodense lesions and ventricular enlargement. The patient was severely disabled 6 months after injury. This is the largest TBGH observed on an initial CT scan in this series.
Basal ganglia hematomas in severe head injury

Impact, which were observed in 76% of the patients in our series. Recently, Fujioka, et al., have demonstrated that traumatic dissection of the MCA produces infarction and hemorrhage in the basal ganglia. On the other hand, the basal ganglia and the thalamus are the most frequent places in which hypoperfusion is detected using single-photon emission CT brain perfusion scanning after mild and moderate head injury.1

In an autopsy series of 635 fatal nonmissile head injuries, Adams, et al., found 63 patients with basal ganglia hematomas; the lesion was defined as an intracerebral hemorrhage involving the striatum, pallidum, or thalamus. Most patients were involved in traffic accidents. They rarely experienced a period of lucidity and they exhibited a high incidence of DAI, gliding contusions, and large contusions, suggesting that these deep-brain hematomas were a primary event that occurred at the moment of injury and arose from the shear strains elicited by acceleration/deceleration forces.2,9,10,20,27 Patients with this type of injury have a relatively low incidence of skull fractures, in contrast with those suffering falls, in whom TBGHs rarely develop.22,27 However, it should be noted that TBGHs can occur in the absence of DAI, and not all patients with DAI have TBGHs.

The majority of patients in our series were involved in traffic accidents, suggesting that inertial phenomena were responsible for most TBGHs. Thus, in 73% of our patients there were CT findings suggestive of DAI, and skull fracture was present in 43% of those in whom skull x-ray films were obtained. Although the occurrence of a period of lucidity is not consistent with DAI, two of our patients were conscious after injury.

Enlargement and De Novo Formation of TBGHs

It is well known that contusion hemorrhages occur at the moment of the impact and may evolve into large hematomas as early as 30 to 60 minutes after injury. The rate and extent of volume increase has been related to factors such as the caliber of the injured vessel, the reduction in cerebral blood flow causing secondary ischemia and necrosis in the surrounding white matter leading to hematoma enlargement by coalescence, the occurrence of blood dissection, the presence of hypoxia or systemic arterial hypertension, and a bleeding tendency, which occurs in persons suffering from alcohol abuse.25 In the present series, 65% of TBGHs enlarged during the acute posttraumatic period and it should be remarked that 86% of the pa-

![Figure 5](image5.png)

**Fig. 5.** Bar graph depicting the correlation between hematoma volumes seen on the admission CT scans and final outcomes in 37 patients with TBGHs. GR = good recovery; MD = moderate disability; SD = severe disability; V = vegetative state; D = death.

![Figure 6](image6.png)

**Fig. 6.** Bar graph demonstrating the correlation between hematoma volumes observed on control CT scans and final outcomes in 35 patients with TBGHs. Only one patient in whom the TBGH was larger than 25 ml on both initial and control CT scans survived without surgery; that patient had moderate disability.

![Figure 7](image7.png)

**Fig. 7.** Bar graph demonstrating the correlation between hematoma volumes throughout the course of the injury and final outcomes in 34 patients with TBGHs. Two patients were excluded because no control CT scan was available and another because the control CT scan had been obtained after surgery for TBGH. The mortality rate was particularly high in the subgroup of patients in whom there was hematoma enlargement.
patients exhibited some type of coagulation disorder that might have contributed to the development of delayed hematomas or the enlargement of a preexisting one.

It is noteworthy that on the initial CT scan low attenuation areas were associated with a TBGH in 54% of our patients, a percentage that increased to 91% on the control CT scans. In keeping with the findings of Wong,40 we believe that the degree of midline shift may be more reliable than the volume of the clot in evaluating the mass effect of a TBGH, because the former relates to both the hyperdense and the hypodense components of the lesion. In the present series, a midline shift greater than 5 mm was observed in 22% and 46% of the patients on initial and control CT scans, respectively (Table 1).

Okada41 reported on 83 cases of traumatic intracerebral hematomas, 49 of which were delayed; 10 hematomas (four of them delayed) were located in the basal ganglia. According to this author, completion of hematoma formation displays two incidence peaks, the first within 6 hours postinjury and the second between 12 and 24 hours postinjury. In our series two patients had delayed TBGHs, one of which reached a volume of 43.3 ml in a patient with hypoxia, arterial hypotension, and coagulopathy.

Another interesting aspect is the occurrence of bilateral TBGHs. Yanaka, et al.,43 reported on two cases of bilateral TBGHs. Ozgun and Castille45 described a case of bilateral TBGH occurring after a lightning strike to the head. In our series, studies revealed petechiae in the basal ganglia contralateral to the TBGH in three patients.

Management of TBGHs

As has occurred in previously reported series, the majority of our patients with TBGHs were treated nonoperatively. Mosberg and Lindenberg46 first published the case of an SHI patient in whom a hematoma located in the region of the pallidum was successfully aspirated through a brain cannula. Traumatic basal ganglia hematomas may be evacuated by means of open surgery,4,5,28,33,38 CT-guided stereotactic aspiration surgery,41,42 or ultrasound-guided aspiration surgery.42 Because only one patient with a TBGH larger than 25 ml survived without surgery in our series, and all patients with raised ICP as well as 73% of the cases showing hematoma volume enlargement died, we believe that a more aggressive surgical management should have been conducted, at least in patients in whom ICP was difficult to control. It is noteworthy that the only surgically treated patient in our series who survived underwent complete hematoma evacuation via a craniectomy. Intracranial pressure monitoring is sometimes tapered before increased intracranial volume normalizes and ICP measurement is not always able to predict failure of intracranial volume compensatory mechanisms and sudden deterioration,40 as occurred in one of our patients with a disastrous result (Fig. 2). It has been recently published that early treatment with citalopram, a selective serotonin reuptake inhibitor, may have a beneficial effect on the extent of morbidity associated with TBGHs.3

Outcomes of TBGHs

Intermediary contusions of the basal ganglia may cause extrapyramidal signs secondary to changes in the substantia nigra,6,11,12,14,23,28,31,37 motor paralysis due to involvement of the pyramidal tracts in the internal capsule,12,28,30 visual deficits as well as language and sensory impairments due to injury to the lateral geniculate bodies and the thalamus, respectively, and prolonged unconsciousness due to involvement of the reticular activating system.13,20

Differences in the incidence of TBGHs in clinical and autopsy series suggest that TBGHs may be an important cause of death after severe closed head injury and are associated with a worse prognosis than other types of posttraumatic intracerebral hematomas.13,27 However, these lesions are compatible with a good recovery when they occur in isolation, and their final prognosis is strongly related to the existence of associated DAI.19,22,23 Data from different series of TBGHs are summarized in Table 3. Munemoto and coworkers33 related the final outcome of five children with TBGHs to the size of the lesion, demonstrating that prognosis is poor in patients with massive hematomas. In 1986, Macpherson and colleagues27 reviewed a series of 2000 patients with confusion, focal deficits, or coma after head injury; in 61 (3%) of these patients TBGHs were observed on CT scans. Sixty-six percent of the latter patients had unfavorable outcomes but the authors did not provide the volumes of the hematomas. Jayakumar, et al.,16 reported on 22 patients with TBGHs

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<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>SHI (Patients (%)</th>
<th>Age (yrs)</th>
<th>Road Traffic Accident (%)</th>
<th>Interval of Lucidity (%)</th>
<th>Skull Fracture (%)</th>
<th>TBGH Contralateral to the Impact (%)</th>
<th>Presence of DAI (%)</th>
<th>ICP Recordings (%)</th>
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<td>43</td>
<td>76</td>
<td>73</td>
<td>81</td>
</tr>
</tbody>
</table>

* Unfavorable outcome includes severe disability, vegetative state, and death. Abbreviations: % = percentage of cases; — = not stated.
‡ Autopsy series.
§ Data inferred from study.
¶ Patients with white matter hemorrhage.

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**TABLE 3**

Summary of TBGH series in the literature*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Patients</th>
<th>SHI (Patients (%)</th>
<th>Age (yrs)</th>
<th>Road Traffic Accident (%)</th>
<th>Interval of Lucidity (%)</th>
<th>Skull Fracture (%)</th>
<th>TBGH Contralateral to the Impact (%)</th>
<th>Presence of DAI (%)</th>
<th>ICP Recordings (%)</th>
<th>Unfavorable Outcome (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mosberg &amp; Lindenberg, 1959 ‡</td>
<td>20</td>
<td>55 ‡</td>
<td>40</td>
<td>5–68</td>
<td>—</td>
<td>—</td>
<td>35</td>
<td>71</td>
<td>55 ‡</td>
<td>—</td>
</tr>
<tr>
<td>Munemoto, et al., 1985</td>
<td>5</td>
<td>60</td>
<td>—</td>
<td>3–13</td>
<td>100</td>
<td>0</td>
<td>40</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Adams, et al., 1986 †</td>
<td>63</td>
<td>—</td>
<td>2–82</td>
<td>68</td>
<td>9</td>
<td>71</td>
<td>—</td>
<td>36</td>
<td>—</td>
<td>100</td>
</tr>
<tr>
<td>Macpherson, et al., 1986</td>
<td>61</td>
<td>84</td>
<td>33</td>
<td>—</td>
<td>69</td>
<td>2</td>
<td>46</td>
<td>71</td>
<td>28</td>
<td>—</td>
</tr>
<tr>
<td>Jayakumar, et al., 1989</td>
<td>22</td>
<td>63</td>
<td>21</td>
<td>0–60</td>
<td>71</td>
<td>0</td>
<td>33</td>
<td>—</td>
<td>14</td>
<td>—</td>
</tr>
<tr>
<td>Katz, et al., 1989</td>
<td>6</td>
<td>60</td>
<td>28</td>
<td>13–71</td>
<td>100</td>
<td>—</td>
<td>—</td>
<td>60</td>
<td>—</td>
<td>0</td>
</tr>
<tr>
<td>Lee &amp; Wang, 1991</td>
<td>52</td>
<td>87</td>
<td>36</td>
<td>3–73</td>
<td>98</td>
<td>0</td>
<td>11.5</td>
<td>—</td>
<td>26.98</td>
<td>6</td>
</tr>
<tr>
<td>present study</td>
<td>37</td>
<td>100</td>
<td>28</td>
<td>14–81</td>
<td>95</td>
<td>5</td>
<td>43</td>
<td>76</td>
<td>73</td>
<td>81</td>
</tr>
</tbody>
</table>
Basal ganglia hematomas in severe head injury

and concluded that the location and size of the hematoma did not correlate with prognosis; however, once more the volume of the hematomas was not defined. Lee and Wang reported on 52 patients suffering from TBGHs, the volume of the hematomas was not defined. Lee and Ma did not correlate with prognosis; however, once more and concluded that the location and size of the hematoma was unfavorable in 46% of their cases, but the volume of the hematomas was again not provided. Advanced patient age (>60 years), the presence of abnormal pupillary changes, impaired oculocephalic and motor responses, and intraventricular and brainstem hemorrhage correlated with poor prognosis.

In our series, nearly 60% of the patients died and 84% had an unfavorable outcome. Likewise, 73% of patients in whom there was hematoma volume enlargement died, but the overall outcome in this subgroup was not different from that seen in the global series. The poorer outcomes in our series compared with other series may be related to our inclusion of SHI patients and our exclusion of those with hemorrhagic lesions measuring less than 2 ml.

Conclusions

Traumatic basal ganglia hematomas are dynamic lesions that tend to increase in volume during the acute post-traumatic period. Occasionally, they are not observed on the initial CT scan and, thus, occur as delayed hematomas. The overall mortality rate in our series was high, particularly in patients with large hematomas and in those in whom there was hematoma volume enlargement or elevated ICP. According to our results it seems that surgical evacuation should be considered in patients with TBGHs larger than 25 ml in volume at any time through the acute posttraumatic period. On the other hand, morbidity was considerably high in the survivors. The pathogenesis and biomechanics of these lesions are still unclear and further studies are necessary.

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