Thalamic haemorrhage

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Summary
Thalamic haemorrhage is usually considered a single entity although the thalamus is composed of anatomically as well as functionally discrete subregions receiving blood from different arteries. The clinical features vary according to the intrathalamic location of the haematoma and the bleeding artery. We investigated the impact of haematoma location and vascular territory on the clinical symptoms and signs, neuro-imaging findings and clinical courses of patients with thalamic haemorrhages by a retrospective analysis of 175 consecutive patients with thalamic haemorrhage. Based on the neuro-imaging findings we classified thalamic haematomas into four regional types and one global type according to the primary bleeding sites: (i) anterior type occurring in the territory of the tuberothalamic arteries, (ii) posteromedial type occurring in the territory of the thalamic-subthalamic paramedian arteries, (iii) posterolateral type occurring in the territory of the thalamogeniculate arteries, (iv) dorsal type occurring in the territory of the posterior choroidal arteries and (v) global type occupying the entire area of the thalamus. We studied the clinical and neuro-imaging characteristics of each type. Eleven patients (7%) had the anterior type; these were the smallest haematomas and often ruptured into the anterior horn of the lateral ventricle. The major clinical signs were acute behavioural abnormalities; the clinical course was usually benign. Twenty-four patients (14%) had the posteromedial type in which haematomas often ruptured into the third ventricle, causing marked hydrocephalus, and often extended medio-caudally, involving the mesencephalon. The prognoses of this type depended on the presence of mesencephalic involvement which was associated with the worst outcome among the types even if the size of the haematoma itself was not large. The posterolateral type was most frequent (77 patients, 44%) and was characterized by large haematomas, rupture into the posterior horn of the lateral ventricle and frequent extension into the posterior limb of the internal capsule. Clinical signs included marked sensory and motor signs, hemimeglect in right-side haematomas and language abnormalities with left-side haematomas. The case fatality with this type was relatively high (35%) and permanent neurologic sequelae frequently resulted. In the dorsal type (32 patients, 18%) haematomas were best visualized at the level of the body of the lateral ventricle on CT scans. The size was moderate and haematomas often extended posterolaterally into the adjacent subcortical white matter. Sensory and motor signs were common and about one third of the patients were first misdiagnosed as having lacunar infarcts. The prognoses were excellent. The global type (31 patients, 18%) of thalamic haemorrhage was clinically and radiologically very similar to the posterolateral type except that the haematomas were too large to define the bleeding focus. Severe sensory and motor signs were almost always present. In this type 25 patients died (the case fatality was 81%).

Keywords: thalamic haemorrhage; classification; vascular territories; clinical courses

Introduction
In 1959 C. Miller Fisher described the clinical features of thalamic haemorrhage, based on his experiences with necropsy-proven haematomas that he examined clinically. He emphasized three clinical signs which have long been considered as the classical findings of thalamic haemorrhage: (i) predominance of sensory deficits over motor; (ii) oculomotor signs including abnormalities of vertical gaze; and (iii) language disturbances in dominant-hemisphere thalamic haematomas. Because his descriptions preceded the CT era, they mostly concerned large, fatal thalamic haematomas. Smaller haematomas were not recognized. Later, with the introduction of CT, small thalamic
haematomas began to be diagnosed in living patients (Walshe et al., 1977; Weisberg, 1979; Barraquer-Bordas et al., 1981; Choi et al., 1983; Kawahara et al., 1986). More diverse clinical presentations of thalamic haematomas were reported and various classifications have been proposed according to the intrathalamic location of haematoma and their size, shape and spread outside of the thalamus (Ikeda et al., 1985; Kawahara et al., 1986; Weisberg, 1986; Wie, 1987; Caplan, 1994; Kumral et al., 1995).

The clinical features and courses of thalamic haematomas have not been well studied in relation to regional anatomy, particularly in association with vascular territories. Four different arteries supply functionally discrete subregions of the thalamus (Bogousslavsky et al., 1988, Barth et al., 1995). In a simplistic view, the anterior thalamic nuclear group subserves mostly memory and emotional behavioural functions. They connect mostly with the mamillary bodies, fornix and cinguli. The polar (tuberothalamic or pre-mamillary) arteries which usually arise from the posterior communicating arteries supply this part of the thalamus. The functions of the posteromedial or medial group of thalamus are mostly related to memory and behavioural functions and so are often classified as 'frontal.' The dorsomedial nucleus projects mostly to the prefrontal cortex. The intralaminar nuclei are functionally related to the brainstem reticular formation and influence the state of alertness and consciousness. The thalamoperforant (thalamo-subthalamic paramedian) arteries are the major supply to this region. The ventral posteromedial and ventral posterolateral nuclei are the principal somatosensory relay nuclei. The ventral lateral and, to a lesser extent, ventral anterior nuclei receive inputs from cerebellar and basal ganglionic structures and project to the motor cortex. The thalamogeniculate arteries arising from the posterior cerebral arteries supply the posterolateral part of the thalamus. The dorsal and far posterior groups probably have the most poorly characterized functions. This group projects mostly to the association cortices in the posterior parietal, temporal and occipital lobes. They probably relay complex multimodal sensory information (auditory, visual and somatosensory) to the posterior hemispheres and subserve some visual-spatial and language functions. This area is supplied by the posterior choroidal arteries arising from the posterior cerebral arteries.

In this study, our working hypothesis is that haematomas occurring in the different thalamic regions supplied by the different vessels should cause different clinical and neuro-imaging features. Until recently, very few studies have subdivided thalamic haematomas into different types and they involved only a small number of cases (Kawahara et al., 1986; Kumral et al., 1995). We herein define the types of thalamic haemorrhages according to the vascular territories of the thalamus. We characterized the clinical and radiological features of the individual types and correlated the neuro-imaging findings with the clinical courses and outcomes.

Patients and methods

Patients, demographic characteristics and clinical analyses

We reviewed the CT and/or MR images of all patients with thalamic haemorrhage at three hospitals: the Chungnam National University Hospital, Taejon, Korea; the Samsung Medical Center, Seoul, Korea and the Tufts New England Medical Center, Boston, USA. Thalamic haemorrhage was defined as a haematoma primarily located in the thalamus. We did not include ganglio-thalamic haematomas in which it was impossible to tell whether the source of bleeding was thalamic or basal ganglionic, but we included those that extended partly into adjacent structures.

A total of 175 patients met our inclusion criteria (140 from the Chungnam National University Hospital, 21 from the Samsung Medical Center; and 14 from the Tufts New England Medical Center). They included 161 Koreans, nine Caucasians and five Chinese. Seventy-five patients (43%) were men. Their ages ranged from 11 to 93 years, with a mean of 61.2 (±11.7, SD) years. Hypertension was responsible for thalamic haemorrhage in 144 patients (82%), chronic alcoholism in 14 (8%), moyamoya disease in two (1%) and cocaine use in two (1%). Thirteen patients (7%) had no identifiable underlying diseases or risk factors on laboratory and angiographic investigations.

We obtained the clinical data from medical records, including the initial level of consciousness, sensory and motor signs, neuroophthalmologic signs, and cognitive and behavioural abnormalities. The level of consciousness was categorized as alert, drowsy, stupor or coma. With regards to sensory manifestations, we evaluated the presence of paraesthesiae at the time of onset, sensory loss found on neurological examination and later development of a thalamic pain syndrome at follow-up visit. Motor weakness and the occurrence of decerebrate posturing were also noted. Neuroophthalmologic signs that we recorded include pupil size, pupillary light reflex, primary eye position (adversion and 'wrong-way' deviation), nystagmus, ocular movement abnormalities and Horner's syndrome. Cognitive and behavioural dysfunctions included confusion or disorientation, memory disturbances, neglect and language abnormalities. Clinical outcome was assessed at the time of discharge from the neurology department and the mean duration of the hospital stay was 14.5 (±8.77, SD) days (range 1–46). We classified the outcome into three categories: Class 1 when the patient recovered, completely or nearly completely, enough to resume independent living activities; Class 2 when the patient survived but required continuous assistance from others; and Class 3 if the patient died in the hospital or was discharged moribund.
Classification of thalamic haemorrhages and CT and MR imaging analyses

Classification and typing of thalamic haematomas was performed independently by two of the authors (C.-S.C. and L.R.C.). For identification of the vascular territories of haematomas, the focus of primary bleeding was considered as the centre of the largest circle that best fit the contour of the haematoma on the CT or MR images.

The haematomas were classified into four regional types and one global type. The regional types were: anterior, posteromedial, posterolateral and dorsal. The anterior type was defined as a haematoma mainly located in the anterior thalamic region supplied by the polar or tuberithalamic artery (Fig. 1). The posteromedial type was defined as a haematoma located in the medial thalamic region supplied by the thalamogeniculate arteries (Fig. 3). The posterolateral type was defined as a haematoma located in the territory of the thalamogeniculate arteries (Fig. 3). The dorsal type was defined as a haematoma located in the dorsal aspect of the thalamus with the main haematoma best seen at a higher level of the CT scans than other types (i.e. the cella media) (Fig. 4). The global type was defined as a large haematoma that occupied the whole thalamus and its bleeding focus was difficult to assess on the brain images (Fig. 5).

We analysed the CT features with respect to haematoma size (as the longest diameter), patterns of extrathalamic extension of haematomas and the presence and severity of hydrocephalus. Two kinds of extrathalamic extension were evaluated. One was the entry site of haematoma into the ventricle causing intraventricular haemorrhage and the other was the direction of parenchymal extension and involvement of specific adjacent structures such as the limb of the internal capsule, the lentiform nuclei, the head of the caudate nucleus and the adjacent subcortical white matter. The direction of
haematoma extension was determined as the vector line that was drawn from the centre of the haematoma to the farthest point of the haematoma. For each type of thalamic haemorrhage, we correlated the clinical data with the neuro-imaging features to characterize the individual types clinically and radiologically. The neurological findings in patients with the five types of thalamic haematomas were analysed and compared.

**Statistical methods**

For comparison between the types, we carried out one-way analysis of variance using multiple range tests for the numerical data and the $\chi^2$ test for the categorical data with Yates correction for continuity; $P < 0.05$ was considered significant.

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**Results**

**Clinical and neuro-imaging characteristics of individual types**

**Anterior type of haematoma**

The anterior type was the least common of the haematomas (11 of 175 cases, 6%) in this series. Haematoma size was the smallest among all types (Fig. 6). They usually ruptured into the anterior horn of the lateral ventricle (Fig. 7C). Occasionally the haematomas extended anterolaterally (Fig. 7A and B), involving the anterior limb of the internal capsule and, in some, even the caudate nucleus (Fig. 1B). Ventricular enlargement was usually slight (Fig. 7D).

The patients were usually alert (Fig. 8A), but often had acute confusion and behavioural abnormalities such as memory impairment and apathetic and indifferent affect (7 of 11). Sensory and motor dysfunctions were rarely observed.
Thalamic haemorrhage

Fig. 3 The posterolateral type of thalamic haemorrhage as seen in CT pictures. Haematomas can be limited to the posterolateral portion of the thalamus (A) or may extend caudally into the mesencephalon (B). More frequently they compress the posterior limb of the internal capsule (C) or extend more laterally, involving the internal capsule, the posterior portion of the corpus striatum, or both (D). They usually rupture into the posterior horn of the lateral ventricle (C and D). Each picture is from a different patient.

and, when present, were very slight and transient (Fig. 8B and C). The patients usually remained alert. Eyeball movement and pupillary abnormalities were absent. Clinical outcome was so excellent that all patients survived and no important neurological deficits remained after recovery (Fig. 8D).

Posteromedial type of haematoma

Twenty-four haematomas (14%) belonged to the posteromedial type. The haematomas of this type were small in size if they are limited to the medial thalamus (Fig. 6). They frequently ruptured into the third ventricle, resulting in marked obstructive hydrocephalus (Fig. 7C and D), often exerting severe mass effect. The haematomas often extended mediocaudally into the midbrain (Figs 2C and D, 7A and 7B).

Clinical presentations and courses were quite different between those patients with small localized haematomas and those with larger, extensive haematomas that involved the midbrain.

Five patients (21%) with small localized haematomas (Fig. 2A) were alert but confused and initially had acute memory, cognitive and behavioural changes. Sensory and motor dysfunctions were not observed. All patients survived but behavioural abnormalities persisted for months after recovery of other neurological signs. Oculomotor and pupillary abnormalities were absent (Fig. 8D).

Nineteen patients (79%) with midbrain involvement ("thalamomesencephalic haemorrhage") were stuporous or comatose at onset. They showed marked motor deficits due to involvement of the cerebral peduncles (Fig. 8B and C). Ten of these patients showed decerebrate posturing and soon
expired. Oculomotor abnormalities were quite frequent and six patients showed 'wrong-way eyes'. Case fatality of mesencephalic involvement was high (13 of 19 cases, 68%).

**Posterolateral type of haematoma**
Seventy-seven patients had the posterolateral type (44%). The haematomas were usually large (Fig. 6). In 58 patients (75%) the haematomas ruptured into the ventricles, mostly into the posterior horns of the lateral ventricles (29 patients) (Fig. 7C and D). In the brain parenchyma, they often extended posterolaterally, involving the posterior limb of the internal capsule (in 61 patients) and the lentiform nuclei (in 30 patients) (Fig. 7A and D). They extended deep into the adjacent white matter in 10 patients and rarely involved the midbrain (six patients).

The clinical features and courses of this type correlated well with the size. Initial consciousness level was related to the haematoma size. Sensory symptoms and signs were frequent (Fig. 8B). Thirty-one patients had paraesthesiae at onset and had decreased touch and pain sensation on examination. About one third of them developed Dejerine-Roussy thalamic syndrome (see Dejerine and Roussy, 1906) between 3 and 15 days after the onset (Fig. 8B). Contralateral hemiparesis was usually moderate or marked and often persisted when the lesions involved the internal capsule (Fig. 8C). Ipsilateral Horner’s syndrome was found in 10 patients with large haematomas (13%). Hemineglect was observed in 20 of 35 patients with right-sided thalamic haematomas and language abnormalities in 18 of 42 patients with left-sided thalamic haematomas. Consciousness, memory and vertical gaze were preserved unless the haematomas were large enough to extend into the upper brainstem (Fig. 8A). The case mortality rate was 30% (23 of 77 patients) and neurologic sequelae persisted in many survivors (Fig. 8D).
Fig. 5 The global type of thalamic haemorrhage as seen in CT pictures. Haematomas are very large and occupy the entire area of the thalamus (A–D). They rupture into the ventricles and destroy the neighboring structures (A–D). Each picture is from a different patient.

**Dorsal type of haematoma**

There were 32 patients with this type (18%). The main portions of these haematomas were best seen at the level of the body of the lateral ventricle on CT scans. The size was moderate (23.5±1.16 mm, Fig. 6). The haematomas usually ruptured into the body of the lateral ventricle (26 cases, 81%, Fig. 7C) while in six other patients the haematomas remained localized. They frequently extended posterolaterally into the corona radiata or centrum semiovale, thereby causing motor and sensory deficits (in 14 patients) (Fig. 6D, 7A and 7B).

The patients were usually alert on admission and only three patients were stuporous initially (Fig. 8A). None was comatose. Sensory signs were relatively frequent and eleven patients had paraesthesia at onset (Fig. 8B). Motor weakness was often present but was slight or moderate (Fig. 8C). The clinical features of 11 patients (34%) were quite similar to lacunar infarction, consisting of sensorimotor and pure motor strokes (eight and three patients, respectively). Dysphasia was observed in three patients with a haematoma in the left

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**Fig. 6** Sizes of individual types of thalamic haematomas.

* Denotes the mean length of the largest diameter; the range represents the 95% confidence interval. PM = posteromedial type; PL = posterolateral type.
hemisphere. Three patients with haematomas localized to the far posterior area of the thalamus (mostly pulvinar) had slight confusion and memory dysfunction with transient uncharacterizable diplopia at onset. The prognosis was very good and all patients survived (Fig. 8D). Sensory and motor recovery was also better than in the other types of thalamic haemorrhages.

**Global type or very large thalamic haemorrhages**

There were 31 patients (18%) with this type. These haematomas were large enough to occupy nearly the entire area of the thalamus (Figs 5 and 6). The mean length was 37.6 (±1.59) mm. The blood entry site into the ventricles was usually the posterior horns of the lateral ventricles (Fig. 7C and D). These haematomas often extended into adjacent structures such as the internal capsule and putamen (Fig. 7A and B). Hydrocephalus and mass effect were common (Fig. 7D).

More than three quarters of the patients were stuporous or comatose on admission (Fig. 8A). They had the classical features of thalamic haemorrhages as described by Fisher (1959). The patients often had abnormal eye movements, predominantly affecting vertical gaze and convergence. The commonest abnormality was paralysis of upward gaze (15 of 31, 48%). However, sometimes both up and down gaze were affected or down-gaze was selectively involved. One or both eyes were often deviated downward and inward, giving the appearance of peering down at the nose. Pupillary abnormalities were also common. Usually both pupils were small and poorly reactive. The ipsilateral pupil was often smaller and occasionally only the ipsilateral pupil became miotic. Severe sensory and motor deficits were always present. Many patients had decerebrate posturing in the acute phase. In most cases the clinical and radiological characteristics were very similar to those of the posterolateral type. Prognosis was very poor and 24 patients (77%) died.
Thalamic haemorrhage

Fig. 8 Clinical characteristics of individual types of thalamic haemorrhage. (A) Initial level of consciousness; (B) sensory features; (C) motor dysfunctions; and (D) clinical outcomes. See the text for clinical outcome grades (Classes 1, 2 and 3). PM = posteromedial type; PL = posterolateral type.

Discussion

Brain haemorrhage and vascular territory

The history of the development of modern knowledge about haematomas at other common intracranial sites parallels that of thalamic haemorrhage. Initially only the largest haematomas were diagnosed clinically and at necropsy (Fisher, 1959; Fisher, 1961a, b). Basal ganglionic ('putaminal') haematomas were initially described as involving the striatum and internal capsule near the capsular genu and extending into the posterior limb of the internal capsule and often into the adjacent lateral ventricle. The clinical findings described included: contralateral hemiplegia, hemsensory loss and hemianopia, ipsilateral conjugate gaze deviation, and language abnormalities in dominant hemisphere lesions and contralateral neglect in nondominant hemisphere lesions. These large haematomas result from rupture of the largest lateral lenticulostriate arteries. With the advent of CT and MR technology, it has become apparent that smaller haematomas at different locations with different clinical findings resulted from rupture of different arteries that supply the anterior and posterior and the medial and lateral basal ganglionic regions. Caudate haemorrhages (Stein et al., 1984; Weisberg, 1984) result from rupture of the Heubner's arteries or branches of the medial lenticulostriate arteries. They rupture generally into the lateral ventricles anteriorly and cause mostly behavioural abnormalities and meningeal irritation (Stein et al., 1984). Haematomas within the anterior limb of the internal capsule and anterior putamen cause a contralateral hemiparesis usually with good recovery. Far posterior putamo-capsular haematomas involve the retroretricular portion of the internal capsule and often branch into the temporal isthmus white matter. These lesions involve mostly the geniculo-calcarine fibres and cause no, or only minimal or transient, contralateral hemiparesis and aphasia, agitation and behavioural changes. These different syndromes depend on haematoma location, size, drainage pattern and site of arterial rupture.

Similarly pontine haemorrhages were also first thought to
cause a uniform clinical picture (Fisher, 1961a) but recently a new classification has been proposed based on the haematoma location and extension and clinical courses: massive, basis-tegmentum, bilateral tegmental and unilateral tegmental (Chung and Park, 1992). Massive haematomas cause a syndrome of coma, quadriplegia, absent horizontal gaze, small reactive pupils and sometimes oculomotor bobbing. They are usually located centrally, occupy a large area at the tegmento-basal junction of the pons and often rupture into the fourth ventricle. The bleeding vessel in this type is the paramedian penetrator of the basilar artery in its distal portion and forms a haematoma starting at the junction of the tegmentum and basis pontis; these haematomas grow into their final round or oval shape and replace most of the pons (Kase and Caplan, 1986). The bleeding vessels of the bilateral tegmental and basal-tegmental types are the same but the degree and direction of the extensions of the haematomas differ. The prognoses of these three types are uniformly poor. In contrast, the haematomas of the unilateral tegmental type are small and present an excellent prognosis with a case survival rate over 90% (Chung and Park, 1992). This type of haematoma is caused by rupture of the penetrators arising from the long circumpedal arteries which enter the tegmentum laterally and course medially (Caplan and Goodwin, 1982; Kase and Caplan, 1986). Occasionally, bleeding may result from rupture of a vessel penetrating from the dorsal region or of the distal portion of a paramedian vessel penetrating from the base (Kase and Caplan, 1986).

Haematomas are often classified as putaminal, thalamic, caudate, lobar, pontine or cerebellar but lesions at any of these sites differ in haematoma size, location, extensions, clinical presentations and prognosis according to the arteries that have bled. We analysed our clinical and radiological material to determine if thalamic haemorrhages had the same variability depending on the arteries involved.

Anatomical substrates for clinical presentations of thalamic haemorrhages

The CT and MR technology has allowed detection of small haematomas and several classifications of thalamic haematomas have been proposed on the basis of clinical and CT criteria. The criteria of classification and designations of each type vary among authors, including the neuroanatomical location of haematomas (Hirose et al., 1985; Kawahara et al., 1986; Kumral et al., 1995) size, shape, extension (Weisberg, 1986; Mori et al., 1995) and vascular territories (Steinke et al., 1992; Caplan, 1994). Recently Mori et al. (1995) reported 104 patients with thalamic haematomas and proposed extension and volume of haematomas as the major determinants of mortality, motor functional outcome and disabilities in performing activities of daily living after 6 months, while disturbances of consciousness and ventricular extension of the haematomas were the major predictors of cognitive function. The authors did not consider the intrathalamic location of haematomas as a major factor affecting the functional outcome.

Using our criteria, the anterior type of haematoma affects the nuclear group that lies within the most rostral portion of the thalamus where it projects forward as the anterior tubercle. The major nucleus is the anteroventral nucleus while the anterodorsal and anteromedial nuclei are smaller components. These nuclei receive projections from the mamillary bodies through the mamillothalamic tract and also receive projections from the fornix. These anterior nuclei project to the cingulum through the posterior portion of the anterior limb of the internal capsule and are functionally related to the frontal lobe. Haematomas often spill out of the confines of the thalamus anterolaterally to involve the medial parts of the anterior limb of the internal capsule and the head of the caudate nucleus. The haematomas also often reach the ventral lateral nucleus, an important motor relay structure. They usually ruptured into the frontal horn of the lateral ventricle.

Clinically, behavioural and cognitive abnormalities predominate and sensory-motor deficits are usually slight and reversible (Kawahara et al., 1986). Memory impairment and abulia with decreased spontaneity, apathy and indifference to the environment were often noted (Ikeda et al., 1985; Kawahara et al., 1986). Memory dysfunction often persisted. The behavioural abnormalities were quite similar to those found in patients with occlusion of the tuberothalamic artery (Graff-Radford et al., 1985; Bogousslavsky et al., 1986; Caplan, 1988; Bogousslavsky et al., 1988). Usually patients remained alert and oculomotor abnormalities were absent. Although this type of thalamic haematoma is not commonly encountered, clinical and radiological abnormalities are quite uniform among the reports. The radiological and clinical findings are summarized in Tables 1 and 2.

The posteromedial type of haematoma involves mainly the dorsomedial and centromedian nuclei in the distribution of the thalamic-subthalamic paramedian (thalamoperforant) arteries. This nuclear group lies posteromedially, extending from just behind the anterior group to the pulvinar. The dorsomedial nucleus is the largest structure within this nuclear group and has extensive connections with other thalamic nuclei, especially the intralaminar and posterior nuclei. It receives input from the amygdala, temporal lobe neocortex and orbital frontal cortex. A large projection goes to the prefrontal cortex. The midline nuclei (paratenial, paraventricular and reuniens nuclei) lie medial to parts of the dorsomedial nucleus and connect with the hypothalamus. The intralaminar nuclei lie within the internal medullary lamina which divides the medial and lateral thalami. The largest intralaminar nucleus is the centromedian nucleus, which is located between the dorsomedial and ventral posterior nuclei. The dorsomedial and centromedian nuclei are important relay stations for subcortical behavioural processing through cortico-striato-thalamo-cortical circuits (Alexander et al., 1986). The other prominent intralaminar nucleus is the parafascicular nucleus. The intralaminar nuclei...
Table 1 Summary table of CT/MRI characteristics of individual types of thalamic haemorrhage

<table>
<thead>
<tr>
<th>Haematoma size: long diameter (mean±SD)</th>
<th>Anterior (n=11)</th>
<th>Posteromedial (n=24)</th>
<th>Posterolateral (n=77)</th>
<th>Dorsal (n=32)</th>
<th>Global (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smallest (17±6.0 mm)</td>
<td>Occasionally antero-laterally into the anterior limb of the internal capsule and the medial portion of the head of caudate nucleus</td>
<td>Occasionally antero-laterally into the midbrain: occasionally rostro-laterally into the lateral thalamus</td>
<td>Usually posterolaterally into the posterior limb of the internal capsule and lentiform nucleus, infrequently into deep subcortical white matter</td>
<td>Usually posterolaterally into the white matter (corona radiata/cerebral semiovale)</td>
<td>Widespread extension into neighbouring structures</td>
</tr>
<tr>
<td>Intraparenchymal extension</td>
<td>Usually into the third ventricle</td>
<td>Usually into the posterior horn of the lateral ventricle</td>
<td>Infrequently into the body of the lateral ventricle at the level of cella media</td>
<td>Almost always into the posterior horn of the lateral ventricle</td>
<td>Very large (37±8.4 mm)</td>
</tr>
<tr>
<td>Ventricle drainage</td>
<td>Occasionally into the frontal horn of the lateral ventricle</td>
<td>Usually into the third ventricle</td>
<td>Usually into the posterior horn of the lateral ventricle</td>
<td>Infrequent</td>
<td>Rare</td>
</tr>
<tr>
<td>Associated ventricular enlargement</td>
<td>Rare</td>
<td>Usually severe hydrocephalus, often requiring extraventricular drainage</td>
<td>Infrequent</td>
<td>Rare</td>
<td>Frequent and marked enlargement, often requiring extraventricular drainage</td>
</tr>
<tr>
<td>Mass effect</td>
<td>Usually absent and only slight if any</td>
<td>Moderate to marked, compressing the cerebral peduncle</td>
<td>Mild to moderate, exerting pressure on the posterior limb of the internal capsule and neighboring structures</td>
<td>Mild</td>
<td>Very severe</td>
</tr>
</tbody>
</table>

Table 2 Summary table of clinical characteristics of individual types of thalamic haemorrhage

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Anterior (n=11)</th>
<th>Posteromedial (n=24)</th>
<th>Posterolateral (n=77)</th>
<th>Dorsal (n=32)</th>
<th>Global (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>6%</td>
<td>Alert</td>
<td>Usually acute stupor or coma</td>
<td>Alert to drowsy, depending on the haematoma size</td>
<td>Usually alert</td>
<td>Stupor or coma in 3/4 of patients</td>
</tr>
<tr>
<td>Consciousness level</td>
<td>Alert</td>
<td>Usually acute stupor or coma</td>
<td>Alert to drowsy, depending on the haematoma size</td>
<td>Usually alert</td>
<td>Stupor or coma in 3/4 of patients</td>
</tr>
<tr>
<td>Behavioural and language dysfunctions</td>
<td>Acute confusion and behavioural changes</td>
<td>Prominent memory dysfunction in case of haematoma limited to the medial thalamus; deconic posture at early stage with concomitant mesencephalic involvement</td>
<td>Hemineglect in right-sided lesion; dysphasia as left-sided lesion</td>
<td>None and simultating lacunar syndrome (coronaradialis/cerebral semiovale)</td>
<td>Frequent decerebrate postures at early stage, very similar to the posterolateral type in less severe cases</td>
</tr>
<tr>
<td>Sensory manifestation</td>
<td>Rare</td>
<td>Uncommon</td>
<td>Frequent; preceding paraesthesia in one third; frequent sensory dysfunction</td>
<td>Preceding paraesthesia in one third; frequent sensory dysfunction</td>
<td>Almost always, severe</td>
</tr>
<tr>
<td>Motor manifestation</td>
<td>Usually absent and only slight if present</td>
<td>Absent when localized to the medial thalamus, moderate to marked contralateral hemiparesis in case of involvement of the cerebral peduncle</td>
<td>Frequent moderate to marked contralateral hemiparesis due to compression of the posterior limb of the internal capsule</td>
<td>Mild to moderate contralateral hemiparesis due to involvement of the corona radiata</td>
<td>Severe contralateral hemiparesis</td>
</tr>
<tr>
<td>Ocular findings</td>
<td>None</td>
<td>Very frequent</td>
<td>Infrequent EOM dysfunctions, occasional Horner's syndrome</td>
<td>None</td>
<td>Frequently presents the classical ocular features</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Excellent (Survival = 100%)</td>
<td>High fatality (Survival = 46%)</td>
<td>High fatality and morbidity (Survival = 70%)</td>
<td>Very good (Survival = 84%)</td>
<td>Very high mortality (Survival = 23%)</td>
</tr>
</tbody>
</table>

EOM = extracocular movement.

receive input mostly from the brainstem reticular formation and project to the putamen and caudate nuclei.

Most reports on small medial thalamic haemorrhages characterized the haematomas as being localized to the medial thalamus and the related clinical signs mostly were memory dysfunctions. The outcome was usually good (Choi et al., 1983; Kawahara et al., 1986; Hankey and Stewart-Wynne, 1988). When the haematomas are localized, sensory-motor findings are usually absent or minor and, if present, they are always transient. The major findings
are reduced alertness and cognitive and behavioural abnormalities.

Early in the course, these patients are often described as confused. Later they are often abulic and have difficulty with memory acquisition. Amnesia may be severe and persist after recovery from the stroke (Choi et al., 1983; Kawahara et al., 1986; Hankey and Stewart-Wynne, 1988). Oculomotor abnormalities are not present in the small localized medial haematomas.

The large haematomas of the posterolateral type extend into the diencephalic-mesencephalic junction very frequently, rupture into and obstruct the third ventricle, and often cause marked hydrocephalus (Waga et al., 1979; Caplan, 1992). Lethargy and even stupor are common early, especially if the lesion spreads to the ventricle and involves the reticular activating system near the third ventricle and in the midbrain. Severe contralateral hemiplegia is related to involvement of the cerebral peduncle and persists in survivors. The majority of our patients had altered consciousness, pupillary dysfunction, vertical gaze abnormalities and fatal outcome. Thus, extension into the midbrain predicts a poor outcome.

The posterolateral region is the most common location of thalamic haematomas as reported by many authors (Walshe et al., 1977; Kawahara et al., 1986; Weisberg, 1986). In the series of Kawahara et al. (1986), for example, about three quarters of haematomas (29 of 39 patients with small thalamic haematoma) were posterolateral in location while the other sites, anterolateral, medial and posterior (dorsal) probably each accounted for 8%.

In our series of 144 regional types, the posterolateral type accounted for 53% (77 patients), the dorsal type 22% (32 patients), the postero medial type 17% (24 patients) and the anterior type 8% (11 patients). The 31 patients with the global type of haematoma were quite similar to the posterolateral type in clinical presentations and the majority of them were probably caused by rupture of thalamogeniculate arteries. So if we add these patients classified as global type, the posterolateral type accounts for >60% of thalamic haematomas.

The thalamogeniculate arteries are the largest and most consistent of the penetrating branches to the thalamus. With rupture of these arteries haematomas often spill out of the thalamus posterolaterally, involving the posterior limb of the internal capsule and a part of the lentiform nuclei, and rupture into the posterior horn of the lateral ventricle.

Sensory signs usually predominate in this type (Fisher, 1959). Dense hypaesthesia was often preceded by paraesthesia at onset and was then followed by the so-called thalamic syndrome as in some patients with lateral thalamic infarcts (Caplan et al., 1988). Sensory signs are due to involvement of the thalamic somatosensory relay nuclei (ventral posteromedial and ventral posterolateral) (Walshe et al., 1977; Hirose et al., 1985; Kawahara et al., 1986). However, pure sensory stroke is an infrequent manifestation of thalamic haemorrhage (Abe et al., 1992; Kim, 1992) but often concomitant involvement of the internal capsule causes a mixed sensorimotor syndrome and leaves a painful hypaesthetic hemiparesis as a late sequela. Recovery of motor function is good when the surrounding brain oedema resolves. Rarely sensory ataxic hemiparesis can occur in this type of thalamic haemorrhage (Dobato et al., 1990; Solomon et al., 1994; Tatu et al., 1996) with or without accompanying dystonic, choreic movements. These cerebellar and extrapyramidal motor abnormalities relate to interruption of cerebellofugal fibres from the red nucleus synapsing at the ventral anterior and ventral lateral nuclei and interruption of the relay from the striatum to these nuclei through the ansa lenticularis (Caplan et al., 1988; Solomon et al., 1994).

Pupillary and eye-movement abnormalities are slight or absent unless the haematoma is quite large and spreads to or compresses the posteromedial thalamus. Alertness is usually maintained in patients with a smaller posterolateral thalamic haematomata. An ipsilateral small pupil and ptosis are commonly noted. These patients usually have abnormalities of conjugate lateral gaze consisting of: hypometric saccades to the contralateral side, abnormal pursuit movements to the side of the lesion with 'catch up' saccades and reduced optokinetic nystagmus when the (visual stimulus) drum is rotated toward the damaged side (Brigell et al., 1984; Hirose et al., 1985). Occasionally hemianopia is found.

Behavioural abnormalities include aphasia in patients with dominant hemisphere lesions and left-sided neglect, poor drawing and copying in right-sided haematomas (Hirose et al., 1985). Altered consciousness, memory disturbances and vertical gaze abnormalities can develop if the posterolateral type haematoma spreads medially and exerts pressure on the diencephalo-mesencephalic junction or involves the midbrain directly.

Haematomas due to rupture of the medial and lateral posterior choroidal arteries have not been well studied and there has been some confusion in the literature because the haematomas cause two separable clinical syndromes. One syndrome develops in haematomas which are localized in the dorsal part of the thalamus but do not involve the pulvinar, resulting from disruption of the distal branches of the lateral posterior choroidal artery. Such haematomas are best seen on the sagittal MR images (Fig. 4C) or on the CT scan slice that shows the body of the lateral ventricle (Fig. 4A and B). Thus only these haematomas can be named as the dorsal type. They usually present focal sensorimotor symptoms and signs only, simulating lacunar syndromes such as sensorimotor or pure motor stroke. They are usually mild and reversible. Language or behavioural abnormalities are not observed.

Another distinct syndrome is rare but presents a variety of behavioural dysfunctions. This results from damage to the pulvinar caused by the haematomas that develop after rupture of the proximal portion of the lateral posterior choroidal artery. On CT or MR images these haematomas are seen in the far posterior part of the thalamus (Fig. 4D). Language abnormalities are sometimes the most important feature of a left-sided pulvinar haematoma. The first few sentences may
be normal except for minor paraphasic errors but as the patients continue to talk, they lapse into jargon aphasia, closely resembling the speech of patients with Wernicke-type aphasia (Ciemens, 1970; Mohr et al., 1975; Samarel et al., 1976). Repetition of spoken language is good but writing is poor (Cappa and Vignolo, 1979). Patients with right-sided pulvinar haematomas may show constructional apraxia and abnormal topographic memory and disorientation to place (Hirose et al., 1985; Kawahara et al., 1986). Other clinical features of this type of thalamic haemorrhage include transient vertical diplopia with grossly normal ocular motility, memory disturbances and confusion. These findings are usually transient and subside over several weeks.

The global type represents large thalamic haematomas originating at any of the anatomical sites but the clinical features are quite similar to those of the posterolateral type except for their fulminant clinical courses. Global haematomas were often associated with abnormal eye movements predominantly affecting vertical gaze and convergence. The reversibility of the vertical gaze palsy with ventricular drainage in some patients suggests that this finding is often due to spread or pressure of the lesion on the pretectal region or pressure related to obstructive hydrocephalus and aqueductal dilatation (Gilner and Avin, 1977; Waga et al., 1979). Vertical gaze paresis is common in patients with large lesions. The commonest defect is paralysis of upward gaze. However, sometimes both upwards and downwards gaze are affected or downward gaze may be selectively involved. One or both eyes are often deviated downward and inward giving the appearance of peering down at the nose (Fisher, 1959; Fisher, 1961b; Fisher, 1967; Caplan, 1993).

**Prognosis of thalamic haemorrhage**

The outcome grade at the time of discharge in our series was Class 1 in 58 patients (33%), Class 2 in 52 patients (30%) and Class 3 in 65 patients (37%). Thus, the case fatality of thalamic haemorrhage was 37% in this series. The prognosis was clearly related to the anatomical type of thalamic haematoma (Fig. 8D and Table 3). Decreased level of consciousness at the time of drainage. Only two of these patients survived. None of the anterior and dorsal types required ventricular drainage. Ventricular drainage did not affect the natural course of severe thalamic haemorrhage in this series. In no patients of this series the haematoma was surgically aspirated. The clinical and neuro-imaging features that suggested a poor outcome in this study are listed in Table 3.

In this study we classified thalamic haemorrhages according to their vascular territories and presented the neuro-imaging characteristics and clinical presentations of the individual types. Thus, we propose that the concept of vascular territory, as in ischaemic strokes, should be introduced to help in the understanding of the clinical and radiological features of thalamic haemorrhages. This might also be applicable to haemorrhages in other brain regions as a testable hypothesis. If the concept is proved valid, it will allow more precise prediction of natural, at least medical, courses of spontaneous intracerebral haemorrhages. And this will also provide a basis for decision of the feasibility and effectiveness of various therapeutic options, which should be investigated with prospective, case-controlled, medical and surgical trials.

<table>
<thead>
<tr>
<th>Table 3 Early clinical and CT scan features indicating a poor outcome in thalamic haemorrhage</th>
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<tr>
<td><strong>Clinical features</strong></td>
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<tr>
<td>Low level of consciousness at onset</td>
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<td>Severe motor weakness and appearance of decerebrate posturing</td>
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<td>Ocular movement abnormalities, particularly fixed-gaze; vertical gaze limitation and wrong-way eyes</td>
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<td><strong>Initial CT scan features</strong></td>
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<td>Large haematoma</td>
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<td>Global and posteromedial types</td>
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<td>Mediocaudal extension of the haematoma, involving the midbrain</td>
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<td>Posterolateral extension into the basal ganglia</td>
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<td>Presence of dense blood clot in the third ventricle</td>
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<td>Markedly enlarged ventricles and severe mass effect, causing a midline shift</td>
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</table>

**References**


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