Thalamic Hemorrhage. A Study of 23 Patients With Diagnosis by Computed Tomography

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SUMMARY A series of 23 patients with thalamic hemorrhage with computed tomography confirmation is reported. Nine of these died, all had hematomas larger than 3.3 cm. The value of the syndrome of downward and convergent ocular deviation is stressed, and its possible mechanisms are analyzed. The characteristics and mechanisms of the pupillary abnormalities are reported, as well as the speech abnormalities observed in patients with lesions of the dominant hemisphere. Prognostic conclusions are drawn.

The clinical syndrome of thalamic hemorrhage was not delineated until recently. Prior to the introduction of computed tomography (CT) scan, only a few observations provided useful clinical-pathological correlations, pointing to characteristic disturbances of extrinsic and intrinsic ocular motility in the diagnosis of thalamic hemorrhage.

Walshe, Davis and Fisher reported the most extensive study of the clinical features of thalamic hemorrhage diagnosed by CT scan. Their observations were made on a total of 18 patients, and the salient diagnostic features were limitation of vertical gaze, downward eye deviation, and small but reactive or sluggish pupils. A sensory-motor deficit contralaterally was present in all. They reported 10–15% of hypertensive intracerebral hemorrhages were thalamic in location.

Clinical Series

We have studied 23 patients (14 female, 10 male; 38–86 years of age — average 68) with thalamic hemorrhage. All were diagnosed by CT scan, and also had autopsy verification. In several of the patients, the lesion was not limited to the thalamus, but was thalamo-capsular. In 12, there was ventricular extension of blood, and in 5 there was distortion and/or dilatation of the ventricular system. In 17 patients there was a history of arterial hypertension; 6 were diabetic.

One patient had a peculiar anatomical distribution with bilateral symmetric involvement of the thalamus and upper brain stem, probably due to hemorrhagic infarction in the distribution of the great vein of Galen rather than a true thalamic hemorrhage. However, due to the clinical course and interest, we have included this patient as an "atypical" one in the series.

The distribution of hemorrhages showed a curious preponderance for the right side, where 17 of the 23 hemorrhages occurred.

The level of consciousness of the patients at the time of the first neurological examination was: alert and awake, 9; drowsy, 9; stuporous, 4; comatose, 5.

The symptoms of presentation to the observer are detailed in table 1, and the ocular abnormalities appear in table 2.

A bilateral Babinski sign was found in only 6 of the 23 patients (26%), a finding which is in contrast with previous reports. Dysphasic speech abnormalities were found in the majority of our patients with left thalamic hemorrhage, with absolute mutism in one. Recovery of normal speech function followed after approximately 5 days.

The "spasmodic" convergence of the eyes was sometimes difficult to differentiate from a 6th nerve palsy, in particular if the inward deviation was unilateral, as in at least one patient.

Discussion

The diagnosis of thalamic hemorrhage has mainly been made by analysis of ocular motility disorders, in addition to speech abnormalities characteristic of dominant hemisphere lesions. The studies of Bottinelli et al., Fisher et al., and our own observations serve as the basis for a discussion of the extrinsic and intrinsic ocular abnormalities. Patients with left thalamic hemorrhage allow a detailed analysis of the characteristic speech disorders.

Extrinsic Ocular Motility Disorders

The most frequent abnormality is gaze depression with convergence, but variants occur. In 1966, Bottinelli et al. reported the characteristic ocular abnormalities in 40 patients with thalamic hemorrhage. In some of these patients oblique deviation was found in addition to the horizontal deviation, with the ipsilateral eye at the lowest or highest level of abduction. This was interpreted to be due to a partial lesion of the third or fourth cranial nerves respectively. In one of our patients, the eye contralateral to the lesion was lower than the ipsilateral eye.

In the 18 patients reported by Walshe and colleagues, a vertical gaze palsy was found in 94% and a downward eye deviation at rest in 55%. In one of our patients, ice water stimulation on the left ear provoked a depression of the ipsilateral eye, with abduction of the contralateral eye, whereas stimulation of the right ear produced a tonic deviation to the right, although with incomplete abduction of the right eye. That hemorrhage was on the left side, and ventricular hemorrhage with hydrocephalus were present.

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TABLE 1 Initial Symptoms in 23 Cases of Thalamic Hemorrhage

<table>
<thead>
<tr>
<th>Symptom/Medical Condition</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemiplegia/hemiparesis and contralateral sensory deficits</td>
<td>23 cases</td>
</tr>
<tr>
<td>Vomiting</td>
<td>11 cases</td>
</tr>
<tr>
<td>Headache</td>
<td>7 cases</td>
</tr>
<tr>
<td>Bilateral Babinski sign</td>
<td>6 cases</td>
</tr>
<tr>
<td>Homonymous hemianopia</td>
<td>4 cases</td>
</tr>
<tr>
<td>Aphasic disorders</td>
<td>4 cases</td>
</tr>
<tr>
<td>Mutism</td>
<td>1 case</td>
</tr>
<tr>
<td>Anosognosia</td>
<td>2 cases</td>
</tr>
<tr>
<td>Hemisomatognosia and spatial unilateral agnosia</td>
<td>1 case</td>
</tr>
</tbody>
</table>

TABLE 2 Oculomotor Disorders in 23 Cases of Thalamic Hemorrhage

<table>
<thead>
<tr>
<th>Condition</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>EXTRINSIC MOTILITY:</td>
<td></td>
</tr>
<tr>
<td>Limitation of upward gaze</td>
<td>8 cases</td>
</tr>
<tr>
<td>&quot;Convergence spasm&quot;</td>
<td>7 cases</td>
</tr>
<tr>
<td>Lateral deviation of gaze:</td>
<td></td>
</tr>
<tr>
<td>—towards opposite side</td>
<td>6 cases</td>
</tr>
<tr>
<td>—towards side of lesion</td>
<td>3 cases</td>
</tr>
<tr>
<td>PUPILLARY ABNORMALITIES:</td>
<td></td>
</tr>
<tr>
<td>Miosis</td>
<td>16 cases</td>
</tr>
<tr>
<td>Loss of light reflex</td>
<td>3 cases</td>
</tr>
<tr>
<td>Anisocoria:</td>
<td></td>
</tr>
<tr>
<td>—early ipsilateral miosis</td>
<td>3 cases</td>
</tr>
<tr>
<td>—delayed ipsilateral miosis</td>
<td>1 case</td>
</tr>
</tbody>
</table>

In order to make a just evaluation of the number of cases with the typical syndrome of extrinsic motility, we must state that the first patients were not searched with this peculiar disorder in the examiner's expectation.

Vertical gaze abnormalities and convergence spasm were attributed by Bottinelli and colleagues to involvement of nuclei in the vicinity of the posterior commissure and the periaqueductal region. Walshe et al. commented that such disturbances are probably due to a pressure effect from the hemorrhage on the vertical gaze apparatus in the area of the nucleus of Darkschewitsch.

Experimental studies have facilitated the understanding of these extracranial abnormalities. In studies performed in monkeys by Pasik and colleagues, the pretectal region was found to contain structures that appear to be essential for vertical ocular movements. This area includes the posterior commissure and the interstitial nucleus on both sides. These authors dismiss a role for the nuclei of Cajal and Darkschewitsch, the pretectal area, the pretectal nucleus, and the olivary or posterior nucleus in this function.

Christoff et al. pointed to the fact that detailed anatomical studies of patients with vertical gaze palsy always show that lesions are either bilateral or unilateral with extension to the midline in the region of the posterior commissure. Vertical gaze paralysis mainly occurs with bilateral lesions of the pretectal region, posterior commissure, or dorsal mesencephalic tegmentum.

According to Denny-Brown and Fisher, a bilateral lesion of the interstitial tegmental nucleus in the macaque monkey produces a total loss of the conjugate elevation and depression of the eyes. The downward gaze mechanism has its control in areas located caudal to those responsible for upward movements. This has been shown by the recent experiments of Kompf et al., who carried out in monkeys, which demonstrate the crucial importance of the prerubric areas for downward ocular movements. The dissociation between upward and downward eye deviation is accounted for by these anatomical differences, and they help explain the "Bottinelli-Fisher syndrome."

Lateral conjugate eye deviation, if this appears, is often found toward the side of the lesion.

Intrinsic Ocular Motility Disorders

The pupils were miotic in 16 of our 23 patients (69.5%). Miotic pupils have been one of the main diagnostic features of thalamic hemorrhage, as pointed out by Walshe et al. In their patients, the pupillary diameter was below 3 mm. Anisocoria was only observed in 2 patients, the smaller pupil being ipsilateral to the side of the hemorrhage in one and contralateral in the other.

In 4 of our patients, we found anisocoria with the smaller pupil always ipsilateral to the lesion. In one where there was no anisocoria, a slight ptosis was present ipsilaterally. Pupillary dilatation was occasionally present, and it was bilateral in one of our patients, due to transtentorial herniation. This latter patient appeared to have a hemorrhagic infarction in the distribution of the vein of Galen, rather than a true thalamic hemorrhage.

The pathophysiology of the pupillary changes in thalamic hemorrhage deserves further comment. Garcin and Kipfer performed studies in dogs, and suggested that a lesion of the upper thalamus, particularly rostrally, can produce an ipsilateral miosis which is sometimes accompanied by ptosis. In this ocular-sympathetic syndrome sometimes occurred as a delayed phenomenon and its duration was frequently transient.

Karplus and Kreidl described a "center" for the ocular sympathetic system, which they called "area S" in the floor of the hypothalamus. This "center" is a small gray nucleus located in the region immediately adjacent to the subthalamic body of Luys, which is crossed by fibers of pallidal origin. In the monkey, the effects of stimulation of this "center" are bilateral, but predominantly contralateral, appearing in the form of anhydrosis. A lesion of this structure causes miosis, predominantly contralaterally.

Garcin et al. described a patient with an acute onset of hemiballismus. Miosis and ptosis appeared ipsilaterally 2 weeks from the onset. At autopsy, a hemorrhage restricted to the subthalamic nucleus was found, as well as a slight paleness of the internal part of the internal capsule.

The presence of ipsilateral miosis is commonly seen...
with lesions affecting the subthalamic region, as was the case, for example, in infarctions of the area supplied by the thalamo-perforating area branches of the posterior cerebral artery.

Houdart and colleagues described the occurrence of Horner's syndrome ipsilaterally to the lesion in patients after small stereotactic operations in areas immediately below the thalamus. In 38 patients subjected to stereotactic thalamic or paratalamal surgery, Carmel found 16 patients with Horner's syndrome ipsilaterally, with ipsilateral anhidrosis in 12. This author concluded that the area responsible for these sympathetic effects is in the prerubric area, at a higher level and slightly in front of the red nucleus, reaching the capsule of this nucleus below the ventrolateral nucleus of the thalamus. Sympathetic nerve projections, among which are those of the ocular sympathetic system, pass through this area on their way down from their origin in the hypothalamus. These ocular sympathetic fibers continue their downward course, which is at all times ipsilateral, with a definite lateral position in the pons and medulla.

Compression of the mesencephalic tegmentum at the level of the third nerve nucleus, according to Kerr and Hallowell, would cause miosis rather than midriasis. Plum and Posner point out that in mesencephalic lesions, the pupils can be either midposition due to a nuclear lesion, or moderately midriatic due to lesions of the third nerve fibers before their exit from the midbrain, the light reflex being lost in both instances. Ipsilateral miosis found with thalamic hemorrhage cannot be explained by a lesion of the "S area" described by Karpus and Kreidl. It can be attributed to either a lesion of the prerubric area described by Carmel, or by involvement of the thalamus itself, in the areas outlined in the experiments of Garcia and Kipfer.

The light reflex was lost in 3 of our patients, a lower frequency than the 62% found in the series of Walshe et al. In our series the pupillary diameter was beyond the 3mm limit found by Walshe. In fact, one patient had equal reactive pupils of 4mm diameter.

**Speech Disorders**

The dysphasic disorders due to hemorrhages in the left thalamus have been the subject of numerous reports. These have been summarized by Luria, Elghozi and colleagues, Dimond, and by Peña et al. A detailed description of the characteristics is not possible here. In Walshe’s series, speech could be tested in 7 of 10 patients with hemorrhages on the left side. Four patients had dysphasia, ranging in severity from a few paraphasic errors to a complete receptive and expressive disorder. In 1974, Mohr et al. published one such observation. Two patients of Walshe et al., including one with a small hemorrhage, had dysarthria without dysphasia. One patient was mute for 3 days and then spoke fluently without aphasic abnormalities. This clinical observation is similar to one of our patients who was mute for 5 days, thereafter rapidly returning to normal speech.

**Agnosic Disorders**

Walshe and colleagues studied this in 3 of the 6 patients with a right-sided hemorrhage, 2 were found to have anosognosia and one had motor impersistence. Our 17 patients with right-sided hemorrhage were not studied in detail, but the presence of anosognosia was detected in 2 and in another one hemisomatoagnosia with left unilateral spatial agnosia was present.

Watson and Heilman studied 3 patients with a right thalamic hemorrhage diagnosed by CT scan, who had left unilateral neglect-accompanied akinesis of the left limbs, in addition to anosognosia and visual-spatial disorders. They interpreted the presence of this syndrome as a consequence of interruption of a reticular-cortical-limbic activator circuit which also includes the thalamic intralaminary nuclei.

**Prognosis**

In the large series of Bottinelli et al., 22 of 40 patients died (65%). In the series of Walshe et al. the mortality was 50%, mostly during the acute phase (8 of the 9 deaths). In our series, 9 of 23 patients died (39%). On analyzing the deaths in our series, it is surprising to find that 4 of the 5 patients who were described as alert on admission, later died. In one, death was due to an unrelated neoplasia, in another to pulmonary embolism, and in still another, to bronchopneumonia. Of the other 5 patients who died, 4 were admitted in coma, and one was described as drowsy. The 4 patients who were stuporous on admission survived. Eight of the 9 patients who were drowsy on admission survived and only one of those admitted in coma survived. Overall the level of consciousness constitutes an important prognostic index. According to Walshe et al., the patients who survived usually became more alert within 3 days from the onset, and recovery was gradual, usually taking 4 to 6 months to reach a plateau. Although the extension of the bleeding to the ventricular system is a sign of a poor prognosis, it is not necessarily fatal. In our series, there were 12 patients with intraventricular hemorrhage, not all fatal. In the series of Walshe, 9 of the 12 patients with ventricular hemorrhage died.

Wiggins and colleagues have commented on the fact that the improvement in diagnosis by the CT scan has revealed that mortality from hypertensive intracerebral hemorrhages is lower than was originally believed. The present series and that of Walshe, demonstrate the important prognostic value of the size of the hemorrhage as shown by CT scan. Our experience confirms the conclusion of these authors of a 100% mortality in patients with thalamic hematomas measuring more than 3.3 cm on the CT scan.

Gilner and Avin reported a 53-year-old patient who had stupor, left sensory-motor deficit, sluggishly reactive pupils (3 mm), and downward and inward deviation of the eyes. She was treated with hyperventilation. A catheter was inserted into her left lateral ventricle for drainage of 15 cc of CSF. This was followed by a drop in the intraventricular pressure from 45 to 20 mm Hg. With this, her Cheyne-Stokes
respiration and tonic spasms of decerebration disappeared. In addition, the extrinsic ocular abnormalities disappeared, and were replaced by conjugate tonic deviation of the eyes toward the side of the lesion. During continuous intracranial pressure monitoring, when pressure was elevated to twice or three times normal, breathing became irregular, and the inward and downward deviation of the eyes returned. The authors suggested that the increasing intracranial pressure was a contributing factor to the characteristic extraocular abnormalities.

Waga and colleagues reported a patient with a left thalamic hemorrhage who had typical extraocular abnormalities, with reactive miotic pupils, although with anisocoria (the smaller pupil being contralateral to the lesion). On CT and angiography a left thalamic hemorrhage with hydrocephalus was confirmed. By the 10th day, although the patient was improved, the paralysis of gaze elevation and hydrocephalus (with papilledema) persisted. This was accompanied by signs of brain herniation through the tentorial incisura. A ventriculo-peritoneal shunt was emplaced, and the following day the paralysis of gaze had ceased. The authors attributed the extraocular abnormalities to periaqueductal abnormality, pointing to the possibility of reversing it by therapeutic intervention.

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