

# PROGNOSTIC FACTORS IN GANGLIONIC AND THALAMIC HAEMORRHAGES: A CLINICAL AND RADIOLOGICAL STUDY

Pages with reference to book, From 62 To 64

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## ABSTRACT

Clinical and radiological features of 43 patients admitted for ganglionic (21) and thalamic (22) haemorrhages were studied to ascertain the factors that would determine prognosis in the acute stage of the illness. Nineteen patients died, 16 of them in the first week. The adverse clinical factors were an altered state of consciousness, decorticate/decerebrate posturing, ataxic respiration, abnormalities of gaze and presence of bilateral Babinski's sign. Adverse radiological factors were the presence of thalamic haemorrhage of 3 cm or more, intraventricular spread of the haemorrhage and midline shift of 0.5 cm or more. The significance of these findings is discussed (JPMA 42: 62, 1992).

## INTRODUCTION

The most common causes of non-traumatic intracerebral haemorrhages - which account for about 10% of the stroke syndromes - are spontaneous hypertensive haemorrhage, ruptured congenital berry aneurysms or arteriovenous malformations<sup>1</sup>. The common locations of the haemorrhage are the thalamus and striatum, the other sites being the hemispheric white matter, pons and cerebellum<sup>2</sup>. Factors that determine the outcome of intracerebral haemorrhages in general, have been fairly well studied<sup>3-5</sup>. Specific behaviour of deep subcortical haemorrhage, in this context, has not been frequently studied as an entity<sup>1,6,7</sup>. We report below our experience with ganglionic and thalamic haemorrhages.

## PATIENTS AND METHODS

Included in this study are 43 patients admitted to the medical service of the Aga Khan University Hospital over a period of 1 year, from November, 1987 to October, 1988, who had deep cerebral haemorrhages diagnosed by a CT scan. The haemorrhage was labelled as thalamic or ganglionic (caudate nucleus and lentiform nucleus) depending upon which site was primarily involved. In each of these, a detailed history, physical examination and neurological assessment was carried out at the time of admission and then on a daily basis for the next 7 days followed by assessment every 3rd day. The state of consciousness was graded as follows: 1) fully conscious - when the patient was fully alert to the surroundings and oriented, ii) stuporose - when the patient was drowsy but arousable by vigorous stimuli and verbal responses were slow or absent, iii) semicomatose - when the patient would respond to deep stimulation by unsustained stirring, moaning or agitation and iv) comatose when there was lack of response to any sensory stimulation<sup>8</sup>. Although increased blood pressure was noted in 42 out of 43 cases at admission (BP  $\geq$  160/90 mmHg) a history and clinical evidence of long standing hypertension was eventually noted in 36 cases. Besides history, other parameters for the diagnosis of hypertension were ECG and/or x-ray changes of left ventricular hypertrophy and fundoscopic examination suggestive of chronic hypertension. Other notable risk factors were diabetes (10), smoking (9), previous history of stroke or transient ischemic attack (6) and history or ECG changes of ischemic heart

disease (27). Besides a CT scan, the routine baseline investigations included a haemogram, liver and renal function tests, blood sugar and electrolyte estimations, x-ray chest and an ECG. Other investigations e.g., for sepsis etc., were done where necessary. Treatment consisted mainly of an appropriate control of blood pressure, treatment of infection where required and other conservative measures. I/V Mannitol and/or steroids - whilst not a routine treatment protocol - was restored to, in cases where there was evidence to suggest a severe increase in intracranial pressure. Neurosurgical intervention was attempted in 5 cases.

## RESULTS

Of the 43 patients, 33 were males and 10 females with mean ages of 59.7 years and 58.6 years respectively. Nineteen (44.2%) patients expired in the acute stage, 5 patients died on day 1, 5 on day 2 and another 6 died within the first week, i.e., 84% of the total deaths occurred within the first week.

### Factors determining prognosis in the acute stage

#### 1. Clinical

The clinical factors that were significant in determining the outcome in the acute stage are shown in Table I,

**TABLE I. Clinical factors determining the prognosis in the acute stage.**

	Died	Survived	P value
<b>1. Level of consciousness</b>			
Conscious/drowsy	4	19	
Semi-coma/coma	15	5	P < 0.01
<b>2. Decorticate/decerebrate posturing</b>			
Present	5	1	P < 0.01
Absent	14	23	
<b>3. Ataxic respiration</b>			
Present	6	2	P < 0.01
Absent	13	22	
<b>4. Abnormalities of gaze</b>			
Present	11	6	
Absent	8	18	P < 0.05
<b>5. Bilateral Babinski</b>			
Present	7	3	P < 0.05
Absent	12	21	

the most significant amongst them being the initial state of consciousness, bilateral increase in tone and ataxic respiration. More significant is the finding noted in

**TABLE II. Prognosis in the acute stage in relation to the state of consciousness at presentations.**

State of consciousness	Survived	Died
Fully conscious	9	0
Stuporose	10	4
Semi-comatose	5	9
Comatose	0	6

Table II which shows that a conscious state at presentation as well as a deeply comatose state, both have a very significant bearing on the prognosis in the acute stage. Seventeen patients had gaze abnormalities, mainly in the form of lateral deviation of gaze (12), limitation of upward gaze (3) and convergence spasm (2). The presence of gaze abnormalities and bilateral Bibinski's sign were also associated with adverse prognosis.

## **2. Radiological (CT scan)**

The significant CT scan factors are listed in Table III.

**TABLE III. Radiological findings determining the prognosis in the acute stage.**

Radiological finding		Died	Survived	P Value
1.	Ganglionic haemorrhages			
a)	Less than 3 cm	2	3	
b)	Greater than or equal to 3 cm	8	8	P > 0.05
2.	Thalamic haemorrhages			
a)	Less than 3 cm	1	8	
b)	More than 3 cm	8	5	P < 0.05
3.	Midline shift			
a)	Less than 0.5 cm	7	10	
b)	More than 0.5 cm	6	1	P < 0.05
4.	Ganglionic and thalamic haemorrhages			
a)	Ventricular spread Not seen	5	16	
b)	Ventricular spread seen	14	8	P < 0.05
5.	Ganglionic haemorrhages			
a)	Ventricular spread not seen	4	10	
b)	Ventricular spread seen	6	1	P < 0.05
6.	Thalamic haemorrhages			
a)	Ventricular spread not seen	1	6	P < 0.05
b)	Ventricular spread seen	8	7	

The risk did not seem to increase with an increased size of 3 cm or beyond in ganglionic haemorrhages, as compared to thalamic haemorrhages. Ganglionic haemorrhages spreading to the ventricles had a more adverse prognosis than thalamic. A midline shift of 0.5 cm or more was also an adverse sign.

## DISCUSSION

Our understanding of deep cerebral haemorrhages has improved considerably since the advent of the CT scan. Clinical factors, however, also remain an important means to determine the eventual outcome. The purpose of the present work was to study both of the above in determining the prognosis in the

acute stage of the haemorrhage episode. The mortality figures in the acute stage are high in all series, though quite variable, ranging from 20% to 58% for ganglionic haemorrhages<sup>9</sup> and 39% to 65% in thalamic haemorrhages<sup>10</sup>. Our mortality figure of 19 out of 43 patients (44.2%) is close to the figure of 46% reported by Portenoy et al<sup>5</sup> in their study comprising all supratentorial (lobar as well as deep) haemorrhages. This would, perhaps suggest that the prognosis in the acute stage in supratentorial haemorrhages does not depend upon the primary site of the bleed. The significance of the state of consciousness as a prognostic factor in our series is consistent with the findings of others<sup>2,5,10,11</sup> and is also true of acute strokes due to all causes. Our finding of zero mortality in patients who were conscious initially and 100% mortality in cases who were initially deeply comatose, is also significant. Kwak et al<sup>11</sup> reported that in their series, the patients who were initially conscious and died, had causes of death that were either secondary or unrelated to the intracranial haemorrhage. Our finding of decorticate/decerebrate posturing being an important factor has not been elaborated by others. We consider this sign significant since it would indicate brainstem compression and only 1 out of 6 patients with this finding survived the acute stage. Ataxic respiration, which would also indicate the eventual involvement of brainstem has a significant adverse prognostic value<sup>5</sup>. In comparison to the above, gaze abnormalities and presence of bilateral Babinski's sign had lesser significance ( $P < 0.05$ ) but important in the overall assessment and have been reported as such by others<sup>10,11</sup>. Thalamic haemorrhages of more than 3 cm size, as measured on the CT scan, are usually associated with a poor outcome. Weisberg<sup>6</sup> and Kwak<sup>11</sup> reported 100% mortality in these patients. Our findings, though not so unequivocal, support the observation. This was, however, not borne out in case of ganglionic haemorrhages, suggesting perhaps, that the proximity to the ventricles and the brainstem could be a significant contributory factor in the adverse prognosis of thalamic haemorrhages. However, in ganglionic haemorrhages, once ventricular spread occurred, mortality figures were significantly high ( $P < 0.05$ ). This has also been the observation of Stein et al<sup>12</sup>. The significance of these findings needs further study. Kwak et al. also studied the effect of a midline shift on CT scan of more than 2 mm and found no difference in prognosis<sup>11</sup>. We attempted to study the significance when the shift was 0.5 cm or more and found a significant difference in the outcome of the 2 groups. We consider this observation as significant, since it would indicate a greater likelihood of herniation/brainstem compression in the tight cranial vault. The management of patients with deep intracerebral haemorrhages is still a daunting task. Despite advances in our understanding of its mechanisms, not much headway has been made in treatment, which mainly remains conservative. Although relief of oedema by steroids or mannitol and neurosurgical intervention in the early stages are logical therapeutic considerations, they have not been found to significantly alter the prognosis<sup>13,14</sup>. An analysis of the prognostic factors in this setting might, in future, determine which of these cases would perhaps benefit from more aggressive treatment.

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