Hyposthetic-Ataxic-Hemiparesis in Thalamic Hemorrhage

ASHOK K. VERMA, D.M. AND MOOL C. MAHESHWARI, M.D.

SUMMARY Acute onset hyposthetic-ataxic-hemiparesis is described in two hypertensive patients. Computed tomography (CT) showed an area of increased attenuation consistent with blood in contralateral thalamus. The pathophysiologic implications of the cerebellar and pyramidal system in thalamic hemorrhage is discussed.

THE SYNDROME OF THALAMIC HEMORRHAGE as described by Fisher' included impairment of vertical gaze and small sluggish or unreactive pupils (Parinaud’s syndrome), in addition to a sensorimotor deficit in which sensory abnormality predominated over motor. Large hemorrhage was usually associated with sudden alteration in sensorium followed by severe and often fatal brainstem dysfunction. This picture has somewhat been modified since the advent of computed tomography (CT). CT Scan has made possible the recognition of clinically inconspicuous small bleed, besides demonstrating the precise site and extent of hemorrhage. We report two cases of hypertensive thalamic hemorrhage, which have presented with hyposthetic hemiataxia with evanescent pyramidal signs.

Report of Cases

Case 1
A 65 year old right handed man, known hypertensive and diabetic for 4 years, suddenly developed slurred speech and unsteadiness. Soon after he noticed weakness and numbness of right arm and leg. There was no headache, altered sensorium, vertigo or diplopia. On admission next day, general examination revealed a BP of 170/105, pulse 92 per minute regular. The heart was normal and no bruit was heard in the neck. He was alert, oriented, and had mild dysarthria. Cranial nerves and fundi were normal. There was mild right hemiparesis with brisk tendon jerks and extensor plantar response. He had marked difficulty in performing finger-nose and heel-knee-shin tests on the right. There was no incoordination on the left side. Intention tremors were noted in right arm. Touch and pain sensations were reduced on the right while normal on the left side. The gait was grossly ataxic, CT scan (fig. 2) revealed a small area of high attenuation consistent with blood in left thalamus. Over the ensuing week his weakness subsided and ataxia improved. However, numbness remained unchanged at discharge one week later.

Discussion
The term 'ataxic-hemiparesis' describes a clinical picture but implies neither localisation nor pathological process. Fisher and Cole4 in 1965 described a clinical stroke syndrome in which the main feature was a combination of pyramidal weakness and ataxia involving the limbs on the same side. Fisher4 coined the term of ataxic hemiparesis after pathological studies of three cases which revealed infarct cavities in the basis pontis on the side contralateral to the pyramidal and cerebellar signs. Ataxic hemiparesis has also been described as a result of lesions in midbrain,6,7 posterior limb of internal capsule8-12 and corona radiata.11-13 The syndrome is now recognised as one of the lacunar syndromes.14 Yet, pontine hemorrhage,7,15 leukemic infiltration,6 tumour and possibly demyelinating disease16 could also be a cause of ataxic hemiparesis. To our knowledge this is first reported account resulting from hypertensive thalamic hemorrhage, thus widening the clinical spectrum of this commonly occurring process.

The symptoms and signs resulting from thalamic disease17 may be categorized into disturbances of sensation, motility, vegetative and mental functions. Speech abnormalities have been described in thalamic lesions of the dominant hemisphere.18,19 Further topo-

From the Department of Neurology, Neurosciences Center, All India Institute of Medical Sciences, New Delhi-110029 India.

Address correspondence to: Dr. M.C. Maheshwari, Professor and Head, Department of Neurology, A.I.I.M S., Ansari Nagar, New Delhi-110029, India.

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FIGURE 1. High attenuation area shown consistent with blood in the left thalamus.

FIGURE 2. Small high attenuation area shown consistent with blood in the left thalamus.

graphic syndromes — such as posterolateral, anterolateral, and medial thalamic syndromes arising from a more discrete lesion have also been mentioned. Posterolateral thalamic lesion, often due to a vascular cause, represents the classical thalamic syndrome of Dejerine and Roussy characterized by the following features: a rapidly regressive fleeting hemiparesis, a persistent hemihypesthesia, intolerable contralateral pains, mild hemiataxic and choreoathetotic movements on the involved side. Significant vegetative and mental dysfunctions may be present in the anterolateral and the medial thalamic lesions respectively. The hemiparesis described in Dejerine and Roussy syndrome, like in our cases, is transient and remains mild to moderate. Cooper concluded that the lesion responsible for ‘thalamic pain’ must include a portion of the internal capsule or part of the parietal lobe along with the thalamic lesion. Our cases did not have a CT demonstrable lesion outside the thalamus; nor did they have ‘thalamic pain’. The characteristic extrinsic and intrinsic oculomotor abnormalities, presumably due to affection of mesencephalic centres, have also been reported to be associated with a sizeable thalamic hemorrhage. However in our cases, clinically normal oculomotor system could possibly be due to a relatively small lesion in the thalamus. On the clinical grounds, although, falling short of so called ‘classical’ thalamic hemorrhage, CT could clinch the accurate diagnosis heretofore unattainable.

In these cases, the site of lesion, CT morphology, accompanying hypertension and conspicuous hemisensory deficit left little doubt that this was indeed a fresh thalamic hemorrhage and coincided with the acute illness. How a thalamic hemorrhage explains the ataxic-hemiparesis is not certain, but the following formulation may apply. The cerebellofugal fibers from dentate nucleus ascend in superior cerebellar peduncle to midbrain and thalamus on its way to the cerebral cortex. At thalamic level, dentato-rubro-thalamo-cortical pathway is not far off from corticospinal tract residing in posterior limb of internal capsule. It seems, therefore, reasonable to postulate that thalamic hemorrhage, by impinging on both the pathways is responsible for the ataxia and pyramidal signs. Mild and evanescent pyramidal deficit favours this speculation. Involvement of corticopontine fibers in posterior limb of internal capsule might also contribute to the ataxia in our patients. However, pyramidal signs resulting from involvement of cerebral peduncle seems unlikely, especially when the intervening mesencephalic structures were not involved.

In our cases, as expected in a thalamic lesion, sensory deficit overshadowed the motor weakness. Such cases of hyposthetic ataxic-hemiparesis may represent a clinicopathological entity different from that resulting from pontine lesions and perhaps from pure capsular lesions.

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References
Lobar Intracerebral Hemorrhage: Etiology and a Long-Term Follow-Up Study of 32 Patients

YASUFUMI TANAKA, M.D.,* MAKOTO FURUSE, M.D.,† HIDEAKI IWASA, M.D.,‡ TOSHIO MASUZAWA, M.D.,‡ KEN SAITO, M.D.,§ FUMIAKI SATO, M.D.,‡ and YOSHIKUNI MIZUNO, M.D.*

SUMMARY Thirty-two patients with lobar hematoma were encountered during a period of six and a half years. Of these patients, 13 had arterial hypertension, 7 had other etiologies, and the remaining 12 were without apparent etiology. In 5 of these patients, cryptic angiomas were suspected from angiograms and CT scans. In one young patient, there was a later recurrence of hemorrhage that resulted in death. Our experience in this series and a review of the literature have led us to conclude that, in young normotensive patients with lobar hematoma, surgical intervention may be a reasonable consideration so that evacuation of the hematoma may be accomplished and a detailed search for small angiomatous malformations may be carried out with a view to preventing recurrences.

Materials and Methods

This study consists of 32 patients admitted to either the neurology or neurosurgery service of Jichi Medical School Hospital during six-and-a-half-year period from September 1976 to March 1983. Hemorrhages in the basal ganglia and thalamic regions were excluded. Also excluded were patients with hematomas due to obvious cerebral trauma, hemorrhagic infarction, ruptured aneurysm, primary and metastatic brain tumors, bleeding diatheses and previously diagnosed arteriovenous malformation (AVM). One patient was initially diagnosed as having a brain tumor by CT scan, but only a hematoma was found at operation (fig. 1). This case was included in the study.

CT scans including intravenous contrast administration were performed in all patients on admission. EMI 1010 head and Toshiba 60 A body scanner were used with one cm interval from Reid’s base line to the parietal level. Cerebral angiography was performed in 28
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A K Verma and M C Maheshwari

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