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Hemorrhagic Sensorimotor Stroke: Spectrum of Disease

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Abstract

Background: This study is aimed to describe the clinical characteristics of hemorrhagic sensorimotor stroke based on data collected from a prospective hospital-based acute stroke registry.

Methods: Twelve patients with hemorrhagic sensorimotor stroke were identified, which accounted for 9% of all cases of pure sensorimotor stroke (n = 133) and 2.9% of intracerebral hemorrhage (n = 408) entered in the database.

Results: Patients with hemorrhagic sensorimotor were more likely to have hypertension, sudden onset, headache, altered consciousness, and internal capsule and basal ganglia involvement than patients with sensorimotor stroke of ischemic origin. When compared with patients with hemorrhagic stroke, hypertension, presence of previous TIA, obesity, heavy smoking, and involvement of the internal capsule were significantly more frequent in patients with hemorrhagic sensorimotor stroke, whereas altered consciousness, basal ganglia, parietal topography and ventricular involvement were less frequent. In the multivariate analysis, altered consciousness (odds ratio 17.2) and basal ganglia involvement (odds ratio 10.3) were independent predictors of hemorrhagic sensorimotor stroke.

Conclusions: Hemorrhagic sensorimotor stroke is a very infrequent stroke syndrome. Altered consciousness at stroke onset may be a useful sign for distinguishing hemorrhagic sensorimotor stroke from other causes of lacunar stroke. There are important differences between hemorrhagic sensorimotor stroke and the remaining intracerebral hemorrhages.

Keywords: Hemorrhagic sensorimotor stroke; Lacunar stroke; Intracerebral hemorrhage; Stroke

Introduction

Sensorimotor stroke is a distinctive lacunar syndrome [1, 2]. Lacunar syndromes are usually caused by symptomatic lacunar infarctions [1-4]. In 5-20% of cases, however, sensorimotor stroke and other lacunar syndromes not due to lacunar infarctions also occur, mainly due to small intracerebral hemorrhages or large non-lacunar ischemic brain infarctions [5-8]. Despite the recognition of the hemorrhagic cause of lacunar syndromes, little is known about the frequency of sensorimotor stroke due to intracerebral hemorrhage or hemorrhagic sensorimotor stroke in stroke registries, as well as their clinical characteristics in relation to the remaining cases of intracerebral hemorrhages and sensorimotor stroke of ischemic origin.

Our aim was to examine clinical features of hemorrhagic sensorimotor stroke in patients included in a prospective hospital-based stroke registry, as well as to compare demographic characteristics, clinical manifestations, neuroimaging data and outcome of patients with hemorrhagic sensorimotor stroke with those of ischemic sensorimotor stroke and the remaining patients with intracerebral hemorrhage.

Materials and Methods

Between January 1986 and December 2004, data of 3808 stroke patients admitted consecutively to the Department of Neurology of the Sagrat Cor Hospital (an acute-care 350-bed teaching hospital in the city of Barcelona) were collected prospectively in a stroke registry [9, 10].

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Table 1. Clinical Data, Topography of Lesion, Neurological Findings and Outcome in 12 Patients With Hemorrhagic Sensorimotor Stroke

Sex/Age, Year	Risk Factors	Altered Consciousness	Topography	Outcome
Male/67	Hypertension	No	Internal capsule/ basal ganglia	Moderate hemiparesis
Male/67	Hypertension, heavy smoking, obesity, peripheral vascular disease	No	Internal capsule/ basal ganglia	Moderate hemiparesis
Female/89	Hypertension	No	Internal capsule/ basal ganglia	Mild hemiparesis
Female/77	Hypertension, heavy smoking	No	Internal capsule	Mild hemiparesis
Male/80	Hypertension, congestive heart failure	No	Internal capsule	Mild hemiparesis
Female/73	Hypertension, atrial fibrillation	Yes	Internal capsule	Moderate hemiparesis
Male/78	Hypertension	Yes	Internal caosule/ basal ganglia	Mild hemiparesis
Male/78	Hypertension, alcohol abuse	No	Internal capsule/ basal ganglia	Mild hemiparesis
Male/57	Hypertension, TIA, hyperlipdemia	No	Internal capsule/ basal ganglia	Mild hemiparesis
Female/73	Hypertension, hyperlipidemia	No	Thalamus	Moderate hemiparesis
Female/81	Hypertension, diabetes, TIA, obesity	No	Thalamus	Symptom free
Female/87	Hypertension, TIA	No	Thalamus	Mild hemiparesis

Subtypes of stroke were classified according to the Cerebrovascular Study Group of the Spanish Neurological Society [11], which is similar to the National Institute of Neurological Disorders and Stroke Classification [12] and has been used in previous studies [3, 5, 9, 10]. Definitions of cerebrovascular risk factors and lacunar syndromes were previously reported [3, 5, 13]. Sensorimotor stroke was defined as a unilateral partial or complete paresis and/or sensory deficit involving at least two of the three areas (face, upper and/or lower limb) with no evidence of aphasia, apraxia and agnosia, or visual field deficit, eye movement disturbance, ataxia, or bilateral weakness.

For the purpose of this study, patients with transient ischemic attack (TIA), subarachnoid hemorrhage, spontaneous subdural hematoma and spontaneous epidural hematoma were excluded. There were 2703 patients with brain infarction, 121 of whom had ischemic sensorimotor stroke. The remaining 407 patients in the stroke registry had intracerebral hemorrhage, 12 of whom had hemorrhagic sensorimotor stroke. Ischemic sensorimotor stroke occurred as a result of a lacunar infarction (n = 83), an atherothrombotic infarction (n = 17), a cardio-embolic infarction (n = 15), a brain infarction of unusual etiology (n = 1) or an infarction of unknown cause (n = 5). Sensorimotor stroke was attributed to a lacunar infarction when the lacunar syndrome correlated with a small size infarction (deep hypodense areas with a maximum diameter of 15 mm on no more than two adjacent 10-mm tomographic cuts) in the vascular territory of the perforating arteries or when the two computerized tomographic (CT) scans obtained during hospitalization were negative and in the absence of cortical ischemia, carotid stenosis or major source for cardio-embolic stroke. Prior to conducting the study, approval was obtained from the Ethical Committee of Clinical Research of the hospital. Study subjects or their next-of-kin signed a permission to treat form on admission to the hospital.

All patients were admitted to the hospital within 48 hours of onset of symptoms. On admission, demographic characteristics, salient features of clinical and neurological examination and results of laboratory tests (blood cell count, biochemical profile, serum electrolytes and urinalysis), chest radiography, and twelve-lead electrocardiography were recorded. In all patients, brain CT scan was performed within this first week of hospital admission. In patients with negative results, a second CT scan was obtained during hospitalization, or the patients were studied using magnetic resonance imaging (MRI). Overall, 34.6% of patients were studied by angio-MRI.

Statistical methods

Demographic characteristics, clinical events and outcome of patients with hemorrhagic sensorimotor stroke and those with ischemic sensorimotor stroke and remaining patients

with intracerebral hemorrhage were compared using the Studen's t-test for continuous variables and the chi-square (χ^2) test (with Yate's correction when necessary) for categorical variables. Variables plus age (used as a continuous variable with a constant odds ratio for each year) and sex were subjected to multivariate analysis with a logistic regression procedure and forward stepwise selection if $P < 0.10$ after univariate testing. Sensorimotor stroke coded as ischemic = 0, hemorrhagic = 1 were the dependent variables. The effect of variables on the presence of hemorrhagic or ischemic sensorimotor stroke was studied in two multiple regression models in which ischemic and hemorrhagic sensorimotor stroke were the dependent variables, respectively. Odds ratio (OR) and 95% confidence intervals were calculated from the beta coefficients and standard errors. Statistical significance was set at $P < 0.05$.

Results

Hemorrhagic sensorimotor stroke accounted for 9% of all cases of sensorimotor stroke (n = 133) and 2.9% of intracerebral hemorrhage (n = 408). There were 6 men and 6 women with a mean (SD) age of 75.6 (8.9) years. The characteristics of these patients are shown in Table 1. Hypertension was found in 12 (100%) patients, heavy smoking in 3 (25%), obesity in 3 (25%) and previous TIA in 3 (25%). Dysarthria was present in 5 (41.7%) patients, headache in 3 (25%) and altered consciousness in 2 (16.7%). Involvement of the internal capsule was the most frequent topography (75%) followed by the basal ganglia (50%). At the time of hospital discharge, 11 patients had hemiparesis (mild in 7, moderate in 4) and only 1 patient was symptom free.

When the groups of patients with hemorrhagic and ischemic sensorimotor stroke were compared, hypertension, TIA, previous hemorrhagic stroke, obesity, sudden onset, headache at stroke onset, altered consciousness, and internal capsule and basal ganglia involvement were significantly more frequent in the hemorrhagic group (Table 2). For the comparison of hemorrhagic sensorimotor stroke and the remaining patients with intracerebral hemorrhage, hypertension, TIA, obesity, heavy smoking, and internal capsule topography were significantly more frequent in patients with hemorrhagic sensorimotor stroke, whereas altered consciousness, basal ganglia and parietal topography, and ventricular involvement were significantly more common among the remaining patients with intracerebral hemorrhage (Table 2). In-hospital mortality was 0% in patients with hemorrhagic sensorimotor stroke and in patients with ischemic sensorimotor stroke compared with 28.3% in the group of the remaining patients with intracerebral hemorrhage ($P = 0.042$).

In the multivariate analysis, altered consciousness (OR = 17.2, 95% CI 2.2 - 133.3) and basal ganglia topography (OR = 10.3, 95% CI 2.6 - 40.8) were independent variables significantly associated with hemorrhagic sensorimotor stroke.

Discussion

Hemorrhagic sensorimotor stroke accounted for 9% sensorimotor strokes and 2.9% of intracerebral hemorrhages included in the stroke registry. Sensorimotor stroke is a clinical syndrome usually caused by symptomatic lacunar infarcts (62.4% of cases), showing that the lacunar hypothesis in pure motor stroke is clinically valid and useful. However, pathological heterogeneity in patients with sensorimotor stroke was found in 37.6% of patients, a percentage higher than 25% in the study of Gan et al [14]. Therefore, approximately in one of every three cases of sensorimotor stroke, this condition may be associated with an underlying non-lacunar infarct mechanism (ischemic: such as cardiac source of embolus, atherosclerosis, or hemorrhagic), which, in turn may influence management.

In a previous study [15], we found that lacunar syndrome not due to lacunar infarcts were mainly due to small intraparenchymatous hemorrhages or large subcortical infarcts. Moreover, 2.9% of intracerebral hemorrhages in our study presented as sensorimotor stroke. Classically, it was considered unlikely that intracerebral hemorrhage would cause a lacunar syndrome without other neurological manifestations, such as altered alertness, visual field defects, eye movement abnormalities or higher cortical dysfunction [1, 16]. However, Mori et al [17] in their study of 174 cases with recent intracerebral hemorrhage found 19 (10.9%) patients with lacunar syndrome and 7 (4%) presented sensorimotor stroke, a percentage higher to that fund in this study. Before the introduction of CT or MRI, it is likely that small hemorrhages producing lacunar syndrome might be misdiagnosed as lacunar infarctions. Therefore, it is very important to establish a definite aetiological diagnosis to allow a correct classification of sensorimotor stroke into the different stroke subtypes because of relevant therapeutic implications [3].

Hemorrhagic and ischemic sensorimotor stroke cannot be distinguished on the basis of semiological findings at the acute stage of stroke. The present results demonstrate a striking similarity between hemorrhagic and ischemic sensorimotor stroke. In the multivariate analysis, altered consciousness was the only clinical variable independently associated with the hemorrhagic variant. Accordingly, sensorimotor stroke in association with altered mental state at stroke onset was more likely to be of hemorrhagic origin.

The absence of in-hospital deaths indicates a favourable short-term prognosis in hemorrhagic sensorimotor stroke [18, 19]. Finally, the presence of basal ganglia topography (50%) was another independent predictor of hemorrhagic sensorimotor stroke, associated with internal capsule involvement (75%). In a previous study [20], we found that isolated capsular hemorrhage was associated with a higher

Table 2. Comparison of Patients With Hemorrhagic Sensorimotor Stroke (SMS), Patients With Ischemic SMS and the Remaining Patients With Intracerebral Hemorrhage

Data	Hemorrhagic SMS	Ischemic SMS	P -value[a]	Remaining Intracerebral Hemorrhages	P-value[b]
Total patients	12	121		396	
Sex, male	6 (50)	61 (50.4)	0.608	208 (52.5)	0.546
Age, years, mean (SD)	75.58 (8.9)	73.6 (12.3)	0.254	72.56 (12.43)	0.225
Age ≥ 85 years	2 (16.7)			67 (16.9)	1.000
Risk factors					
Hypertension	12 (100)	85 (70.2)	0.019	235 (59.3)	0.002
Diabetes	1 (8.3)	31 (25.6)	0.164	62 (15.7)	0.423
Atrial fibrillation	1 (8.3)	17 (14)	0.495	61 (15.4)	0.433
Cardiac heart failure	1 (8.3)	3 (2.5)	0.318	8 (2)	0.238
Transient ischemic attack	3 (25)	9 (7.4)	0.078	19 (4.8)	0.022
Previous cerebral infarct	0	16 (13.2)	0.200	36 (9.1)	0.325
Previous hemorrhagic stroke	1 (8.3)	0	0.090	27 (6.8)	0.579
Peripheral vascular disease	2 (16.7)	8 (6.6)	0.223	21 (5.3)	0.142
Obesity	3 (25)	8 (6.6)	0.062	15 (3.8)	0.012
Alcohol abuse	1 (8,3)	6 (5)	0.492	16 (4)	0.404
Smoking (>20 cigarettes/day)	3 (25)	13 (10.7)	0.159	31 (7.8)	0.069
Hyperlipidemia	2 (16.7)	26 (21.5)	0.517	46 (11.6)	0.423
Clinical findings					
Sudden onset	7 (66.7)	45 (37.2)	0.048	236 (59.6)	0.431
Headache	3 (25)	9 (7.4)	0.078	137 (34.6)	0.363
Nausea, vomiting	1 (8.3)	2 (1.7)	0.249	95 (24)	0.183
Speech dysorders (dysarthria)	5 (41.7)	39 (32.2)	0.357	140 (35.4)	0.432
Altered consciousness	2 (16.7)	3 (2.5)	0.064	163 (41.2)	0.076
Neuroimaging findings					
Thalamus	3 (25)	11 (9.1)	0.115	84 (21.2)	0.490
Internal capsule[c]	9 (75)	52 (43)	0.034	64 (16.2)	0.000
Corona radiata	0	7 (5.8)	0.508	3 (0.8)	0.914
Pons topography	0	12 (9.8)	0.305	16 (4)	0.615
Basal ganglia	6 (50)	15 (12,4)	0.004	95 (24)	0.049
Parietal topography	0	0	1	85 (21.5)	0.058
Ventricular	0	0	1.000	95 (24)	0.040
Symptom-free at discharge	1 (8.3)	25 (20.7)	0.275	23 (5.8)	0.522
Urinary complications	2 (16.7)	3 (2.5)	0.064	48 (12.1)	0.979
Infectious complications	1 (8,3)	2 (1.7)	0.249	84 (21.2)	0.471
Respiratory complications	2 (16.7)	4 (3.3)	0.092	46 (11.6)	0.593
Length of hospital stay, mean (SD)	18.5 (7.9)	14.9 (8.9)		21.1 (21)	0.125
In-hospital mortality	0	0	1.000	112 (28.3)	0.042

Data are n (%) unless otherwise stated. Comparison between [a]hemorrhagic and ~~iscahemic~~ sensorimotor stroke; [b]hemorrhagic sensorimotor stroke and remaining intracerebral hemorrhages; [c]Isolated (n = 3) or with basal ganglia (n = 6). Total topographies: internal capsule plus basal ganglia 6, internal capsule 3, thalamus 3.

frequency of lacunar syndromes ($P < 0.05$), mainly as pure motor hemiparesis (12.5% of cases) and sensorimotor stroke (8.5%) compared with other intracerebral hematomas, in which pure motor stroke occurred in only 2.9% of cases and sensorimotor stroke in 1.5%.

However, important clinical differences between hem-

orrhagic sensorimotor stroke and the remaining cases of intracerebral hemorrhage were observed. Patients with hemorrhagic sensorimotor stroke showed a higher frequency of internal capsule involvement as well as better outcome (no case of in-hospital death). This may be related to the small size of the lesion as opposed to the remaining patients with intracerebral hemorrhage who showed a higher frequency of altered consciousness, basal ganglia and parietal topography, and ventricular involvement which is consistent with large size of the lesion and a more frequent cortical topography of the hematoma [16, 21].

Conclusion

In summary, hemorrhagic sensorimotor stroke is an infrequent syndrome and accounted for 9% of all cases of lacunar sensorimotor stroke and 2.3% of all cases of intracerebral hemorrhage. The presence of altered consciousness at stroke onset may be a useful sign for distinguishing hemorrhagic from ischemic sensorimotor stroke. There are important differences between hemorrhagic sensorimotor stroke and the remaining intracerebral hemorrhages.

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Disclosure of Conflict of Interest

No conflict of interest.

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