PAPER

Clinical study of 35 patients with dysarthria-clumsy hand syndrome

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Objectives: Although dysarthria-clumsy hand syndrome (DCHS) is a well known and infrequent lacunar syndrome, there are few data regarding the spectrum of associated clinical characteristics, anatomical site of lesion, and aetiopathogenetic mechanisms. We report a clinical description of this subtype of lacunar stroke based on data collected from a prospective acute stroke registry.

Methods: From 2500 acute stroke patients included in a hospital based prospective stroke registry over a 12-year period, 35 patients were identified as having DCHS.

Results: DCHS accounted for 1.6% of all acute stroke patients (35/2110), 1.9% of acute ischaemic stroke (35/1840), and 6.1% of lacunar syndromes (35/570) admitted consecutively to a neurology department and included in the stroke registry over this period. The results supported the lacunar hypothesis in 94.3% of patients (n = 33). Atherothrombotic and cardioembolic infarction occurred in only one patient each (2.9%). No patient with DCHS had an intracerebral haemorrhage. Outcome was good (mortality in hospital 0%, symptom free at discharge 45.7%). After multivariate analysis, absence of limitation at discharge, limb weakness but not cerebellar-type ataxia, and internal capsule (40%), pons (17%), and corona radiata (8.6%) location were significantly associated with DCHS.

Conclusions: DCHS is a rare cerebrovascular syndrome, and supports the criteria of the lacunar hypothesis. The majority of patients in this study had internal capsule infarcts. The prognosis is good with striking similarity compared with other types of lacunar strokes. There are important differences between DCHS and non-lacunar strokes. Internal capsule and pons are the most frequent cerebral sites.

Dysarthria-clumsy hand syndrome (DCHS) is the most uncommon and poorly studied of the classic lacunar syndromes.[1 2] Some authors have used the labels "dysarthria-clumsy hand" and "ataxic hemiparesis" interchangeably, not adhering to Fisher's original descriptions,[3 4] and thus confusing the clinical distinction. Additionally, DCHS is infrequently individualised in different prospective stroke registries and little is known regarding the frequency and natural history of this disorder. While the lacunar hypothesis is supported in most lacunar syndromes,[5 6] it has not been tested in DCHS, probably because of its infrequent presentation, despite the fact that most of these cases are caused by a lacunar infarction, although cases secondary to intracerebral haemorrhage in pontine[7] or cerebellar[8] sites have been reported. The sites of lesions responsible for DCHS are not definitely established, although based on a study in six patients, it is assumed that pons is the main site; however, cases of DCHS have been also described in lacunar infarctions of the anterior limb of the internal capsule, genu of the internal capsule, or corona radiata.[10–12] Therefore, a clinical study of 35 patients with DCHS collected from a prospective stroke registry was carried out, in order to assess: (*a*) the frequency of DCHS caused by different stroke subtypes and (*b*) differential demographic, clinical, neuroimaging, and outcome data of DCHS compared with patients with other lacunar syndromes and with those with non-lacunar stroke.

METHODS

Between January 1986 and December 1997, the data of 2500 stroke patients admitted consecutively to the Department of Neurology of Sagrat Cor (an acute care 350 bed hospital in Barcelona, Spain) were collected prospectively in a stroke registry.[13] For the purpose of this study, patients with transient ischaemic attack (n = 328), subarachnoid haemorrhage (n = 35), and spontaneous subdural haematoma (n = 27) were excluded. The study population consisted of 2110 patients with acute ischaemic (n = 1840) or haemorrhagic (n = 270) stroke. Subtypes of stroke were classified according to the Cerebrovascular Study Group of the Spanish Society of Neurology,[14] which is similar to the National Institute of Neurological Disorders and Stroke classification[15] and has been used by our group in previous studies.[16–20] Subtypes of stroke included 553 patients with atherothrombotic infarcts, 484 lacunar infarcts, 468 cardioembolic infarcts, 248 infarctions of undetermined origin, 87 infarctions of unusual aetiology, and 270 intracerebral haemorrhages. Definitions of cerebrovascular risk factors and lacunar syndromes (pure motor stroke, pure sensory stroke, sensorimotor stroke, ataxic hemiparesis, DCHS, and atypical lacunar syndromes) were those used in recent studies.[18–20]

For the purpose of this hospital based prospective study, 570 consecutive patients with lacunar syndromes (secondary to lacunar, n = 484 or non-lacunar cerebral infarcts, n = 86) were collected. There were 277 patients with pure motor stroke, 99 with pure sensory stroke, 81 with sensorimotor stroke, 23 with ataxic hemiparesis, 35 with DCHS, and 55 with atypical lacunar syndromes. Atypical lacunar syndromes included isolated dysarthria (n = 32); hemichorea-hemiballismus (n = 5); isolated hemiataxia (n = 5); unilateral (n = 2) or bilateral (n = 3) paramedial thalamic infarct syndrome; pure motor hemiparesis with transient subcortical aphasia n = 4); and pure motor hemiparesis with transient internuclear ophthalmoplegia (n = 4).

Patients selected as having DCHS (n = 35) met the following criteria:[9] (*a*) dysarthria without dysphasia; (*b*) unilateral "central" facial weakness with ipsilateral

Abbreviations: DCHS, dysarthria-clumsy hand syndrome

clumsiness appearing as a cerebellar-type ataxia (dysmetria, dysrhythmia, dysdiadochokinesia, gait ataxia), or with mild or no weakness; and (*c*) no sensory symptoms or signs. Other lacunar syndromes included all patients with lacunar stroke with the exception of DCHS (n = 535). Non-lacunar stroke included all patients whose clinical picture did not conform to the preceding subgroups (n = 1540).

All patients were admitted to the hospital within 48 hours of onset of symptoms. On admission, demographic characteristics, salient features of clinical history and neurological examination, results of routine laboratory tests, chest radiography, and twelve lead electrocardiography were recorded. In all patients, brain CT scans were performed within the first week of hospital admission. Patients with negative results had a second CT during their stay in hospital or were studied by MRI. Other investigations included angioMRI (51% of patients), echo Doppler of the supra-aortic trunks (43%), arterial digital subtraction angiography (8%), B mode echocardiography (40%), and lumbar puncture (4%).

Demographic variables included age and sex. All other findings were dichotomised as present *v* absent. Anamnestic findings comprised history of hypertension, diabetes, myocardial infarction or angina, rheumatic heart disease, congestive heart failure, atrial fibrillation, smoking (>20 cigarettes/day), alcohol misuse (>80 g/day), intermittent claudication, transient ischaemic attack, previous cerebral infarction, hyperlipidaemia, nephropathy, cirrhosis or chronic liver disease, chronic obstructive pulmonary disease, and age ⩾85 years. Clinical variables were sudden onset of symptoms (minutes), headache, dizziness, seizures, nausea or vomiting, altered consciousness (drowsy, stuporous, comatose), limb weakness (hemiparesis or hemiplegia; Babinski's sign not mandatory), sensory symptoms, hemianopia, aphasia or dysarthria, ataxia, and cranial nerve palsy. Neuroimaging variables comprised internal capsule, basal ganglia, cerebellum, mesencephalon, pons, middle cerebral artery, and basilar artery. Outcome variables were mortality in hospital, degree of clinical disability at discharge, cardiac events (acute myocardial infarction, heart failure, tachyarrhythmia), respiratory events (pulmonary embolism, atelectasis or respiratory infection), urinary events, vascular events, and infectious complications.

Statistical analysis

Demographic characteristics, clinical events, and outcome of patients with DSCH were compared with those of patients with lacunar syndromes and patients with non-lacunar stroke. Univariate and multivariate analysis were performed. In the univariate analyses, continuous variables were compared with Student's *t* test and categorical variables with the χ^2 test (with Yates' correction when necessary). Statistical significance was set at $p<0.05$.

In the comparison of DCHS and other lacunar syndromes, variables related to DCHS in the univariate analysis plus age (used as a continuous variable with a constant odds ratio (OR) for each year) and sex were studied in a multiple linear regression model based on demographics, risk factors, clinical data, neuroimaging, and outcome variables, a total of eight variables. In the comparison of DCHS and non-lacunar strokes, two multiple linear regression models were set. The first predictive model, with 11 variables, was based on demographic, vascular risk factors, and clinical data. The second predictive model was based on demographic, risk factors, and clinical and neuroimaging data, and had 16 variables. In all cases, DCHS (coded as absent = 0, present = 1) was the dependent variable. The level of significance was set as 0.15, and the tolerance level at 0.0001. The maximum likelihood approach was used to estimate weights of the logistic parameters.[21] OR and 95% confidence intervals (CI) were calculated from the beta coefficients and standard errors. The hypothesis that the logistic model adequately fitted the data was tested by means of the goodness of fit χ^2 test.[22] The SPSS-PC+[23] and the BMDP[24] computer programs were used for statistical analyses.

RESULTS

The 35 patients with DCHS accounted for 1.6% of all cases of acute stroke, 1.9% of ischaemic stroke, and 6.1% of lacunar sydromes. Males made up 71% of patients, with a mean (SD) age of 71.3 (10.5) years. Hypertension and diabetes were the main cardiovascular risk factors in 62.9% and 34.3% of patients, respectively. Sudden onset of symptoms (minutes) was recorded in 35.6% of patients. In 71.5% of the cases (n = 25), neuroimaging studies confirmed the brain lesion sites; these were the internal capsule in 40% of cases (in the anterior limb, genu or near the genu), pons in 17.1% (paramedian rostral lesions), corona radiata in 8.6%, basal ganglia in 2.9%, and thalamus in 2.9%.

With regard to stroke subtype, lacunar infarction was diagnosed in 33 patients and non-lacunar stroke in the remaining two (atherothrombotic infraction, n = 1; cardioembolic infraction, n = 1). No case of DCSH secondary to haemorrhagic stroke was recorded. Outcome of DCSH was good, with a 0% mortality rate in hospital and 45.7% of patients having no neurological disability at discharge. The mean length of hospital stay was 11.7 days. There were no significant differences in demographic data, risk factors, clinical variables, or outcome between 25 patients with DCHS and confirmed sites, and the remaining 10 patients in which the site of the lesion was not confirmed.

When the groups of DCHS and other lacunar syndromes (n = 535) were compared, limb weakness (but not cerebellar-type ataxia), speech disturbances, corona radiata and basilar artery involvement, and freedom from symptoms at discharge were significantly more frequent in DCHS group (table 1); only limb weakness and absence of limitation at discharge appeared to be significant variables related to DCHS after multivariate analysis (table 2). However, the comparison between the groups of DCHS and non-lacunar stroke (n = 1540) showed important differences, particularly in respect to the unfavourable outcome of non-lacunar stroke (higher mortality in hospital, longer hospital stay, and fewer symptom-free patients at discharge) (table 1). After multivariate analysis, the significant variables related to DCHS appeared to be hypertension, diabetes, limb weakness, pons sites, corona radiata involvement, internal capsule involvement, and freedom from symptoms at discharge (table 2).

DISCUSSION

In this prospective hospital-based stroke registry, DCHS accounted for 6.1% of lacunar syndromes, 1.9% of ischaemic infarcts, and 1.6% of acute strokes. These findings are consistent with the North American Symptomatic Carotid Endarterectomy Trial, in which DCHS accounted for 4.6% of the 283 possible lacunar strokes and 7.1% of 210 probable lacunar strokes.[25] In agreement with previous studies, DCHS is the most infrequent classic lacunar syndrome.[26 27] In an investigation of 68 consecutive patients with sudden onset dysarthria due to a single infarction confirmed by MRI or CT, DCHS syndrome was observed in eight patients.[12] To our knowledge, the present series of 35 patients with DCHS is the largest reported in the literature.

This study shows that DCHS was caused by a lacunar infarct in 94% of patients and by other stroke subtypes in 6%. Therefore, the lacunar hypothesis[5 6] was justified in 94% of patients. A main finding of the study is that different topographies of lesions may cause DCHS, which is in contrast

Table 1 Results of univariate analysis. Comparison of patients with dysarthria-clumsy hand syndrome (DCHS) with patients with other lacunar syndromes and with patients with non-lacunar stroke

	DCHS	Other lacunar syndromes	p value*	Non-lacunar stroke	p value†
Total patients	35	535		1540	
Sex, male	25 (71.4)	317 (59.2)	NS	731 (47.5)	<0.05
Age, years, mean (SD)	71.34 (10.52)	72.64 (10.64)	NS	74.0 (12.73)	NS
Age≥85 years	4 (11.4)	63 (11.8)	NS	271 (17.6)	NS
Risk factors					
Hypertension	22 (62.9)	391 (73.1)	NS	727 (47.2)	<0.05
Diabetes	12 (34.3)	147 (27.5)	NS	264 (17.8)	<0.02
Atrial fibrillation	6 (17.1)	79 (14.8)	NS	493 (32)	<0.05
Clinical findings					
Limb weakness	33 (94.3)	403 (75.3)	<0.005	1188 (77.1)	<0.01
Dysarthria	35 (100)	185 (34.6)	<0.001	847 (55)	<0.001
Cerebellar-type ataxia	6 (17.1)	32 (6)	NS	99 (6.4)	NS
Dizziness	0	6 (1.1)	NS	80 (5.2)	<0.02
Nausea, vomiting	1 (2.9)	20 (3.7)	NS	168 (10.9)	<0.1
Altered consciousness	0	14 (2.6)	NS	437 (28.4)	<0.001
Subtype of stroke					
Lacunar infarct	33 (94.3)	451 (83.8)	NS	0	<0.001
Atherothrombotic infarction	1 (2.9)	34 (6.3)	NS	518 (33.6)	<0.05
Cardioembolic infarction	1 (2.9)	20 (3.7)	NS	447 (29)	<0.05
Infarction of unusual cause	0	2 (0.4)	NS	85 (5.5)	NS
Infarction of unknown cause	0	8 (1.5)	NS	240 (15.6)	NS
Intracerebral haemorrhage	0	20 (3.7)	NS	250 (16.2)	NS
Neuroimaging findings					
Internal capsule	14 (40)	180 (33.6)	NS	227 (14.7)	<0.001
Thalamus	1 (2.9)	81 (15.1)	<0.03	107 (6.9)	<0.001
Basal ganglia	1 (2.9)	42 (7.8)	NS	286 (18.6)	<0.01
Pons	6 (17.1)	37 (6.9)	<0.04	64 (4.2)	<0.02
Corona radiata	3 (8.6)	26 (4.8)	<0.01	11 (0.7)	<0.01
Basilar artery involvement	6 (17.1)	37 (6.9)	<0.04	99 (6.4)	<0.05
Outcome					
Symptom free at discharge	16 (45.7)	129 (24.2)	<0.005	211 (13.7)	<0.001
Severe disability at discharge	0	13 (2.4)	NS	209 (13.6)	<0.02
Urinary complications	0	18 (3.3)	NS	187 (12.1)	<0.02
Infectious complications	1 (2.9)	22 (4.1)	NS	271 (17.6)	<0.02
Length of hospital stay, mean (SD)	11.71 (5.75)	12.64 (8.45)	NS	20.55 (25.1)	<0.02
Mortality in hospital	0	4 (0.7)	NS	340 (22.1)	<0.001

*DCHS *v* other lacunar syndromes.
†DCHS *v* non-lacunar stroke.
Percentages in parentheses or otherwise stated.

to the criteria of Glass *et al*,[9] who reported that the majority of patients with DCHS had pontine infarcts. In 25 patients with confirmed sites by neuroimaging studies, the lesion was found in the internal capsule in 40% of patients (in the anterior limb, genu, or near the genu), while pons lesions were found only in 17% of patients (in the paramedian rostral sites). Corona radiata, basal ganglia, and thalamus were other uncommon sites. However, the present results are in agreement with the initial descriptions of DCHS. Fisher[28] stated that the best recognised association of DCHS has been with lacunes of the anterior limb of the internal capsule. Spertell and Ransom[10] described a case of DCHS with a low density lesion near the genu. Capsular genu syndrome was also found to be associated with dysarthria.[29] With regard to pontine base infarction, in a clinical radiological correlation study, Kim and associates[30] observed that lesions located in the paramedian rostral pons tended to produce DCHS. In our series, the six cases of pontine infarction showed paramedian rostral pons lesions. The corona radiata is another rather uncommon site also previously mentioned by Urban *et al*.[12]

Therefore, different lesion sites may cause DCHS, possibly because this syndrome is due to a partial involvement of the motor fibre along the course of the pyramidal tract secondary to a small cerebral lacunar infarct that disrupts the corticospinal fibers independently of the sites of the lesion. An alternative or complementary explanation may be that although supratentorial small, deep infarcts can be seen on imaging in DCHS, there is a possibility that the clinical syndrome could be secondary to a tandem non-imaged lesion in the pons. Future studies using diffusion weighted imaging would allow more precise discrimination of the sites of lesions in DCHS with higher sensitivity and specificity.

From a clinical point of view, this study highlights the favourable outcome in DCHS and, as expected, the difference between DCHS and non-lacunar stroke. More interestingly, however, is the comparison between DCHS and other lacunar syndromes. Limb weakness but not cerebellar-type ataxia, speech disturbances, corona radiata and basilar artery involvement, and freedom from symptoms at discharge were significantly more frequent in patients with DCHS, but after multivariate analysis, only limb weakness and absence of functional disability at discharge were significant predictors of DCHS. Unilateral pyramidal signs in the form of mild weakness is a well known distinguishing feature of DCHS, being absent in the majority of atypical lacunar syndromes and in other lacunar syndromes, such as pure sensory stroke.[2 4] In the literature, differentiation between ataxic hemiparesis and DCHS is not always clear, but in accordance with Fisher[1] and Mohr & Martí-Vilalta,[4] patients with obvious ataxia and definitive weakness were classified as having ataxic hemiparesis and were not included among

Table 2 Results of multivariate analysis

Variable	β	SE (β)	Odds ratio (95% CI)
DCHS *v* other lacunar syndromes*			
Limb weakness	1.796	0.738	6.03 (1.42 to 25.59)
Absence of limitation at discharge	1.045	0.358	2.84 (1.41 to 5.73)
DCHS *v* non-lacunar stroke			
Demographic, vascular risk factors and clinical data†			
Absence of limitation at discharge	1.799	0.359	6.04 (2.99 to 12.22)
Limb weakness	1.740	0.738	5.70 (1.34 to 24.23)
Diabetes mellitus	0.976	0.375	2.65 (1.27 to 5.53)
Hypertension	0.758	0.365	2.13 (1.04 to 4.36)
Female sex	−0.991	0.386	0.37 (0.17 to 0.79)
Demographic, vascular risk, clinical, and topographic data‡			
Corona radiata	2.467	0.780	11.79 (2.56 to 54.33)
Internal capsule	2.305	0.419	10.03 (4.41 to 22.78)
Absence of limitation at discharge	1.597	0.392	4.94 (2.29 to 10.66)
Pons	1.569	0.546	4.80 (1.65 to 14.01)
Limb weakness	1.512	0.755	4.54 (1.03 to 19.92)
Diabetes mellitus	0.897	0.403	2.45 (1.11 to 5.40)
Hypertension	0.767	0.387	2.15 (1.01 to 4.60)
Female sex	−0.807	0.406	0.45 (0.20 to 0.99)
Basal ganglia	−2.838	1.051	0.06 (0.01 to 0.46)

*β = −4.638; SE (β) = 0.739; goodness-of-fit $\chi^2 = 0.011$; df = 2; $P = 0.995$; area under the ROC curve = 0.680.
†β = −6.073; SE (β) = 0.795; goodness-of-fit $\chi^2 = 7.357$; df = 8; P = .499; area under the ROC curve = 0.777; sensitivity 68.5%; specificity 68.7%; correct classification 68.7%.
‡β = −6.402; SE (β) = 0.835; goodness-of-fit $\chi^2 = 3.73$; df = 8; P = .893; area under the ROC cirve = 0.858; sensitivity 71.4%; specificity 83.9%; correct classification 83.6%.

patients with DCHS. DCHS presents characteristic and well-differentiated clinical and semiological features, and the absence of neurological disability in 46% of patients indicates that DCHS is the lacunar syndrome with the most favorable outcome.

In summary, DCHS is a rare cerebrovascular syndrome (6% of lacunar strokes) and, in the majority of patients, is due to small vessel disease as the stroke mechanism at capsular or pontine sites. The majority of patients in our study had internal capsule infarcts. The prognosis is good with striking similarity to clinical features of other lacunar syndromes. There are important differences between DCHS and non-lacunar strokes in demographic and risk factors, clinical, sites of lesion, and outcome.

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