

Study of Clinical Spectrum, Topographic Correlations and Frequency of Post Stroke Movement Disorders in Adults

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Abstract: Methods: We reviewed consecutive patients with involuntary abnormal movements (IAMs) following a stroke who were included in the Madras Institute of Neurology, RGGGH, Stroke Registry and they were followed up for at least one year after the onset of the IAM. We determined the clinical features, topographical correlations and frequency of movement disorders associated with stroke.

Results: Of 1500 patients with stroke 56 developed movement disorders up to one year after the stroke. Patients with chorea were older and the patients with dystonia were younger than the patients with other IAMs. In patients with isolated vascular lesions without IAMs, surface lesions prevailed but patients with deep vascular lesions showed a higher probability of developing abnormal movements. One year after onset of the IAMs, 12 patients (21.4%) completely improved their abnormal movements, 38 patients (67.8%) partially improved, four did not improve (7.1%), and two patients with chorea died. In the nested case-control analysis, the patients with IAMs displayed a higher frequency of deep lesions (63% v 33%), than patients without IAMs. OR 3.38, 95% CI 1.64 to 6.99, $p < 0.001$. Patients with deep haemorrhagic lesions showed a higher probability of developing IAMs (OR 4.8, 95% CI 0.8 to 36.6).

Conclusions: Chorea is the commonest movement disorder following stroke and appears in older patients. Involuntary movements tend to persist despite the functional recovery of motor deficit. Deep vascular lesions are more frequent in patients with movement disorders.

Keywords: post stroke movement disorders, vascular cause for movement disorders

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I. Introduction

Involuntary abnormal movements (IAMs) caused by strokes are relatively common, ¹⁻⁴ and chorea, ³⁻¹⁵ tremor, ¹⁶⁻²⁹ dystonia, ³⁰⁻⁴³ parkinsonism, ⁴⁴⁻⁵⁴ and myoclonus ^{10,35,55,56} have all been associated with both infarcts and cerebral haemorrhage. IAMs may occur as part of the symptomatology of acute stroke ^{1-11,13,14,16,17,20-30,32-37,39,42-54,56}; they may be delayed or progressive. ^{3,12,15,18,19,31,38,40,41,55}

We reported 56 patients from our prospective and retrospective Stroke Data Registry Cohort who developed IAMs. Our Stroke Data Registry is an observational study that collects clinical, laboratory, radiological, and follow up data of all patients with acute stroke, admitted to the Department of Neurology or to the medical department of Rajiv Gandhi government general hospital, Chennai. They all were examined and treated by the neurologist from the Madras institute of Neurology.

II. Objective

The objective of this study was to analyse the clinical features, topographical correlations, follow up, and frequency of movement disorders associated with stroke.

III. Methods

Between march 2016 and march 2018, a total of 1500 consecutive stroke patients were included in the Stroke Data Registry; 30 of them had been admitted to non-neurological services. For this report, we selected all patients who had IAMs post stroke. After reviewing the case histories, which included the description and classification of the IAMs, and the videotapes that were made for all patients, two neurologists (F A and N C), who were blind to the imaging results, independently confirmed the diagnosis and the type of abnormal movement. We determined the time between stroke and IAM onset, the evolution of the abnormal movement and the motor deficit, the presence of behavioural abnormality, and mortality. All the patients with stroke were

carefully followed up for more than 12 months, and when patients developed an IAM they were followed up for at least one year after the onset of the IAM.

Retrospective analysis of patients attending the movement disorder OPD and also in Neurology Review OPD had also been done.

Stroke was defined as the rapid development of signs of focal or global disturbance of cerebral function, lasting over 24 hours or leading to death, without any apparent cause.^{57,58} We used the definitions and guidelines for the diagnostic classification of stroke recommended by the World Health Organization (WHO), with the different subtypes based on schemes developed by the Pilot Bank of Stroke Data.^{59,60} Computed tomography (CT) or magnetic resonance imaging (MRI) had to show an ischaemic lesion or evidence of parenchymal, ventricular, or subarachnoid bleeding corresponding to the clinical picture. During the first 30 days after stroke, the following risk factors were evaluated on the basis of our Registry^{58,61}: high blood pressure, diabetes, cardiac disease, previous stroke, hyperlipidaemia, smoking, haematological disorders (in patients under 45 years of age), and carotid artery disease.

We recognised four types of IAM in our patients:

- chorea—defined as an arrhythmic involuntary movement, which intrudes in a sudden, brief, and non-repetitive fashion¹⁴
- dystonia—defined as an abnormal movement characterised by sustained muscular contractions, frequently causing twisting and repetitive movements or abnormal posturing⁶²
- tremor—defined as a rhythmic oscillation of a body part⁶³
- parkinsonism—defined as the presence of bradykinesia and at least one of the following symptoms: muscular rigidity, rest tremor, or postural instability.^{64–66}

Chorea, dystonia, and tremor were classified as focal (affecting a single part of the body), segmental (affecting two or more adjacent parts of the body), multifocal (affecting more than one part of the body), unilateral (affecting ipsilateral arm and leg), or generalised. We classified tremor as predominantly at rest, postural, or kinetic. Behavioural disorders were categorised in terms of confusion, abulia, and disinhibition. A CT or MRI was done for all patients in the first week after the involuntary movements appeared.

To evaluate the patients with parkinsonism we used the Unified Parkinson's Disease Rating Scale (UPDRS III)⁶⁷ motor score at follow up; to evaluate tremor, we used the Tremor Rating Scale of Fahn, Tolosa and Marin, part C, Disability Assessment Scale,⁶⁸ a total of 28 points, 7 items; for dystonia, we used the Fahn and Marsden Dystonia Scale, section II, Disability Scale,⁶⁹ a total of 30 points, 7 items; and for chorea, the Marsden and Schachter Scale,⁷⁰ a total of 23 points, 5 items. We evaluated each patient twice in the first month after the onset of the abnormal movements, then every month during the first year, and every three months thereafter. We considered improvement to be partial when, in their last evaluation, the patients showed an improvement by more than 1 point in each item of the scale in the signs and symptoms of parkinsonism, tremor, dystonia, and chorea, compared with the first evaluation.

The *t* test, Kruskal–Wallis test, and χ^2 test were used for statistical comparison of age, sex, and time between stroke and IAM onset among the subgroups with chorea, dystonia, tremor, and parkinsonism. We used the analysis of variance to compare age among the four groups of movement disorders.

Using the Stroke Data Registry, we carried out two nested case–control analyses. In the first, we determined the difference in the frequency of surface and deep vascular lesions among the groups; in this analysis we included the 56 cases with IAMs and two controls for each case, matched for age and sex. The χ^2 test was used to compare the imaging features of patients with surface and deep lesions with and without IAMs. In the second case–control analysis, we included 35 patients with deep vascular lesions who had IAMs and one control for each case with deep vascular lesions without IAMs, matched by age and sex. This analysis was performed to determine the risk of developing IAMs related to the ischaemic or haemorrhagic lesion. Odds ratios (OR) and 95% confidence intervals (CI) were calculated.

IV. Results

4.1. Patients' characteristics

Fifty nine of the 1500 patients (3.9%) included in the Stroke Data Registry developed IAMs after stroke. Three patients with pre-stroke tremor were excluded. The average age (SD) of the included 56 patients (3.7%) (22 men, 34 women) was 63.3 (18.1) years (range 17–90 years). Chorea was the commonest movement disorder (35.7%), and the patients who had chorea were older (74.5 (8.1) years) than the patients with other IAMs ($p=0.0009$) (table 1). Patients with chorea had the shortest time interval between diagnosis and IAM onset (4.3 (3.6) days) ($p<0.05$) (table 1) and patients with parkinsonism had the longest interval (117.5 (97.3) days) ($p<0.05$). In seven patients, the abnormal movements started on the first day of the stroke. The latest onset of an IAM was 10 months after stroke in a patient with parkinsonism.

Table 1: Demographic characteristics of the 56 patients with post-stroke abnormal movements

	Abnormal movement			
	Chorea	Dystonia	Tremor	Parkinsonism
*Significant difference between age of patients with chorea and age of patients with other types of movement (p=0.0009; analysis of variance).				
†Significant difference when comparing time between diagnosis and start of abnormal movement (p<0.05; Kruskal–Wallis test).				
No. of patients	20	16	14	6
Age in years (SD)	74.5 (8.1)*	47.9 (20.9)	62.8 (14.4)	62.3 (17.8)
Sex (male/female)	7/13	5/11	6/8	4/2
Time in days (SD) between diagnosis of stroke and start of abnormal movement	4.3 (3.6)†	15.7 (19.9)	18.7 (12.8)	117.5 (77.3)†

4.2. Clinical features and follow up of IAM patients

Fifteen of the 18 patients with focal chorea or hemichorea (83.3%) (table 2) had the motor deficit on the same side as the abnormal movement. Two did not have motor deficit and the remaining one had a contralateral motor deficit. Fifteen patients with chorea (75%) improved partially, two (10%) improved completely, one did not show any improvement and two died.

Table 2: Abnormal movement and imaging features of the 20 patients with post-stroke chorea

Patient no./sex/age (years)	Clinical features	CT scan or MRI
Type of chorea: S, spontaneous; A, intensified by action; BM, with ballistic movement.		
CT, computed tomography; MRI, magnetic resonance imaging.		
1/M/85	Left hemichorea S, A	Bilateral temporal infarct
2/F/74	Right hemichorea S, A, BM	Right paramedian thalamic infarct, left occipital calcification
3/F/70	Left hemichorea S, A, BM	Left posterolateral thalamic lacunar infarct
4/M/77	Left hemichorea S, A, BM	Right paramedianputaminothalamic capsular infarct
5/F/67	Right hemichorea S, A, BM	Left posterolateral thalamic infarct
6/M/80	Generalised chorea S, A	Bilateral cortical temporal infarcts
7/F/77	Right hemichorea S, A	Bilateral lenticular infarcts and bilateral frontal temporal infarcts
8/F/74	Left hemichorea S, A	Bilateral external capsular infarcts
9/M/75	Right hemichorea S, A	Left putaminocapsulo-thalamic haemorrhage
10/F/75	Right hemichorea S, A	Left frontotemporal haemorrhagic infarct and right striatal lacunar infarct
11/M/58	Right hemichorea S, A	Left posterolateral thalamocapsulo-lenticular haemorrhage
12/F/75	Left hemichorea S, A	Bilateral corona radiata infarcts
13/F/76	Left hemichorea S, A	Right thalamocapsular infarct
14/F/90	Right hemichorea S, A, BM	Left pallidal capsular infarct
15/M/78	Generalised chorea S, A, BM	Bilateral thalamic haematoma
16/M/71	Left hemichorea S, A	Bilateral lenticular and right thalamic infarcts
17/F/71	Right hemichorea S, A	Bilateral lenticular-striatal infarcts
18/F/83	Right hemichorea S, A, BM	Right cerebellar and left thalamic infarct
19/F/72	Left foot chorea S, A, dystonia	Right corona radiata and right lateral pons infarcts
20/F/53	Left hemichorea S, A, BM	Right posterolateral thalamic and subthalamic infarct

Eight of the 11 patients with focal or unilateral tremor (72.7%) (table 3) showed motor deficit ipsilateral to the abnormal movement. In four patients (28.5%) the tremor disappeared completely and in nine (64.2%) partially.

Table 3: Abnormal movement and imaging features of the 14 patients with post-stroke tremor

Patient no./sex/age (years)	Clinical features	CT scan or MRI
Type of tremor: R, rest; P, postural; K, kinetic.		
CT, computed tomography; MRI, magnetic resonance imaging.		
1/M/72	Right hemitremor P	Left frontal infarct
2/F/73	III left cranial nerve palsy, left dysmetria, upper right limb tremor P, K, rubral type	Left mesencephalic haematoma
3/F/73	III left cranial nerve palsy, left hemitremor R, P, K	Right paramedianthalamo-mesencephalic infarct
4/F/74	Upper limbs tremor R, P	Right ventrolateral thalamic infarct
5/M/57	Upper left limb tremor P	Right parieto-occipital haematoma
6/M/58	Upper left limb tremor P	Left putaminocapsular haematoma
7/F/43	Upper right limb tremor P, K, dystonia	Left frontotemporal infarct
8/M/76	Head and upper left limb tremor R, P, K	Right lenticular haematoma
9/F/76	Upper right limb tremor P	Left parietal, right semioval, and bilateral pontine infarcts
10/F/74	Upper limbs, voice, head and orthostatic tremor R, P, K	Hydrocephalus, subarachnoid haemorrhage

11/M/25	Ataxia, right dysmetria, head, voice, upper left limb tremor R, P, dystonia, myoclonic jerk	Left posterior thalamo-mesencephalic haematoma
12/F/55	Upper left limb tremor P, K	Subarachnoid and ventricular haemorrhage
13/F/63	Ataxia, left dysmetria, upper limbs with predominance of upper right limb tremor R, P, K, dystonia	Subarachnoid haemorrhage and hydrocephalus
14/M/50	Left dysmetria and hemiataxia, tongue and upper left limb tremor P, K	Left cerebellar and lateral medullary infarcts

In 10 of the 15 patients with focal or hemidystonia (66.6%) (table 4), the motor deficit was ipsilateral to the involuntary movement. In five patients (31.2%) the dystonia disappeared completely and in 10 partially (62.5%).

Table 4: Abnormal movement and imaging features of 16 patients with post-stroke dystonia

Patient no./sex/age (years)	Clinical features	CT scan or MRI
Type of dystonia: S, spontaneous; A, intensified by action; M, mobile dystonic spasms causing repetitive dystonic posture; F, fixed, present at rest and action but not intensified by action		
CT, computed tomography; MRI, magnetic resonance imaging.		
1/F/68	Cranial and right foot tremor S, dystonia	Left putaminal capsular infarct
2/F/65	Right hemidystonia, cranial dystonia S, M	Bilateral temporal infarct
3/M/32	Left hemidystonia S, M	Right putaminal capsular infarct
4/F/60	Oromandibular, right cervical dystonia S, M	Left parieto-occipital haematoma
5/M/27	Left hemidystonia S, A, M	Right pallidocapsular lacunar, left caudate head and right thalamic medial infarcts
6/F/71	Blepharospasm S, M	Left lenticular lacunar infarct
7/F/86	Abulia, blepharospasm S, M	Lenticular infarcts and bilateral radiate corona
8/M/36	Cranial lingual dystonia S, A, M	Right frontal haematoma
9/F/25	Upper left limb dystonia S, A, F	Right putaminocapsular infarct
10/M/24	Generalised dystonia S, M	Left globuspallidus infarct
11/F/17	Left hemidystonia S, M	Right parietal haematoma
12/F/63	Left foot dystonia A	Right paramedian thalamic and lenticulocapsular infarcts
13/F/40	Right hand dystonia S, A	Left medial and lateral pontine haematoma
14/F/40	Right hemidystonia A, M	Left medial and lateral infarct of pons and right striatal infarct
15/M/36	Oromandibular, left hand dystonia, S, A, M and tremor	Right frontal haematoma
16/F/77	Left foot dystonia S, A, F	Right posterolateral thalamocapsular haematoma

The signs and symptoms of parkinsonism developed rapidly, starting unilaterally on the same side of the hemiparesis in three cases and in one bilaterally with ipsilateral predominance to the motor deficit (table 5). One recovered spontaneously after two years. At the start of levodopa therapy, between one and eight weeks after onset of parkinsonism, five patients showed a moderate response and one did not respond. After six months of therapy, the response was poor in all five patients. Four of the five patients had early fluctuations and dyskinesias between one and nine months after starting levodopa. Parkinsonism was progressive in five patients. None of our patients had a history of parkinsonism prior to stroke.

Table 5: Abnormal movement and imaging features of the six patients with post-stroke parkinsonism

Patient no./sex/age (years)	Clinical features	CT scan or MRI
Signs of parkinsonism: Rig, rigidity; Bra, bradykinesia; Tre, tremor (R, rest, P, postural); Dyst, dystonia.		
CT, computed tomography; MRI, magnetic resonance imaging.		
1/F/76	Bilateral parkinsonism with predominance of lower limbs Rig+Bra+TreP	Bilateral lenticular and pons infarcts
2/M/66	Bilateral parkinsonism with predominance of lower limbs Rig+Bra	Right frontal and left parietal infarct
3/M/71	Right hemiparkinsonism Rig+Br+Tre R, P	Left mesencephalic and pons infarct
4/M/80	Hemiparkinsonism with predominance of upper right limb Rg+Br+Dyst	Left caudocapsular infarct
5/M/54	Upper left limb parkinsonism Rig+Br+TreP	Right subcortical frontal infarct
6/F/27	Bilateral parkinsonism with predominance of left limbs Rig+Br+TreP	Bilateral lenticular infarcts

Fifty patients (89.2%) had a motor deficit (table 6), out of which there was complete improvement in 19 cases (38%). The abnormal movement improved totally in 12 patients (21.4%) and partially in 38 cases (67.8%) (table 6).

Table 6: Clinical features of the 56 patients with post-stroke abnormal movements

	Abnormal movements				Total patients n (%)
	Chorea n (%)	Tremor n (%)	Dystonia n (%)	Parkinsonism n (%)	
*Significant differences (p<0.01) when comparing the groups.					
Total patients	20 (35.7)	14 (25)	16 (28.5)	6 (10.7)	56 (100)
Clinical features					
Abulia	–	–	2 (12.5)	3 (50)	5 (8.9)
Disinhibition	1 (5)	2 (14.2)	–	–	3 (5.3)
Somnolence	10 (50)	4 (28.5)	5 (31.2)	–	19 (33.9)
Confusion	5 (25)	6 (42.8)	2 (12.5)	–	13 (23.2)
Coma	2 (10)	2 (14.2)	1 (6.2)	–	5 (8.9)
Motor deficit*	18 (90)	14 (100)	12 (75)	6 (100)	50 (89.2)
Upper limb	–	–	–	1 (16.6)	1 (1.7)
Hemi body*	17 (85)	13 (92.8)	12 (75)	4 (66.6)	46 (82.1)
Bilateral	1 (5)	1 (7.1)	–	1 (16.6)	3 (5.3)
Improvement of motor deficit					
Partial	14 (70)	7 (50)	5 (31.2)	4 (66.6)	30 (53.5)
Total*	4 (20)	7 (50)	7 (43.7)	1 (16.6)	19 (33.9)
None	–	–	–	1 (16.6)	1 (1.7)
Improvement of abnormal movement					
Partial	15 (75)	9 (64.2)	10 (62.5)	4 (66.6)	38 (67.8)
Total*	2 (10)	4 (28.5)	5 (31.2)	1 (16.6)	12 (21.4)
None	1 (5)	1 (7.1)	1 (6.2)	1 (16.6)	4 (7.1)
Deaths	2 (10)	–	–	–	2 (3.5)

4.3. Type and aetiology of vascular lesions in patients with IAMs

Thirty nine patients had an infarct, 14 a parenchymal haemorrhage, and three a subarachnoid haemorrhage. We found large and medium sized vessel atherothrombosis in seven of the 39 patients with an infarct (17.5%), small vessel occlusion (lacunes) in 25 patients (64.1%), embolism of cardiac origin in six (15.3%), and in one patient the cause of infarction remained unknown. Arterial hypertension was the cause of intracerebral haemorrhage in 11 (78.5%) of the patients with parenchymal haemorrhage, eight of whom (72.7%) had deep lesions.

4.4. Lesion location in the patients with IAMs

We found isolated lesions in 23 (43.3%) of the 53 patients with parenchymal lesions (haemorrhage or infarct), located at the surface in seven (30.4%). In 30 cases (56.6%), the lesions were multiple or large enough to be located in more than one location.

4.5. Correlation of isolated lesions with topography of the IAMs

The abnormal movement was focal or unilateral in 17 (73.9%) of the 23 patients with contralateral isolated lesions (tables 2–5). Four patients had chorea (23.5%), four had tremor (23.5%), seven dystonia (41.1%), and two parkinsonism (11.7%). In 16 (69.5%) of the 23 patients with isolated lesions, the stroke was ischaemic. Seven patients showed thalamic infarcts (43.7%), of whom five had chorea, one dystonia, and one tremor. Six patients had lentiform infarcts (37.5%), of whom five had dystonia and one chorea. One patient with parkinsonism had a caudate nucleus infarct; one patient with parkinsonism and another with tremor had isolated surface infarcts (12.5%). Seven of the 23 patients with isolated lesions had haemorrhages (30.4%), of whom three with dystonia showed surface lesions (42.8%); in two patients with tremor (28.5%) the lesions were lentiform and in two patients (28.5%), one with tremor and the other with dystonia, the haemorrhages were located in the brain stem.

4.6. Correlation of unilateral lesions with side of the IAM

Thirty four patients had unilateral lesions (60.7%): 10 patients with chorea (29.4%), 10 with tremor (29.4%), 11 with dystonia (32.3%), and 3 with parkinsonism (8.8%). Focal or unilateral chorea correlated with a contralateral lesion in nine patients (90%) (table 2), of which five were in the thalamus (55.5%). Seven patients with focal or unilateral tremor (70%) (table 3) showed a contralateral lesion, three of them had surface lesions (42.8%). One patient with tremor had an ipsilateral cerebellar lesion. Eleven patients with focal or unilateral

dystonia had a contralateral lesion (100%) (table 4), four of which were located in the lentiform nucleus (36.3%). One patient with focal dystonia had a lesion in the pons. Focal or unilateral parkinsonism correlated with a contralateral lesion in three patients (100%) (table 5), located in the mesencephalon-pontine, caudocapsular, or subcortical frontal area.

4.7. Correlation of lesions with behavioural disorders

Confusion was the most frequent behavioural disorder (table 6); in six cases it was related to thalamic lesions, in three cases to surface lesions, in two cases to lentiform nucleus lesions, and in two cases to subarachnoid haemorrhage. Abulia was related to two frontal lesions, one in the caudate and two in the lentiform nucleus. Disinhibition was infrequent.

4.8. Nested case-control analysis

When we compared the 56 stroke patients with IAMs with the 112 stroke patients without IAMs, infarcts prevailed in both groups (70% in each). Additionally, the patients with IAMs showed a higher frequency of deep lesions—involving basal ganglia, capsule, diencephalon, and mesencephalon—than those patients without IAMs (63% v 33%, $p < 0.001$). Surface infarcts were found more frequently in patients without IAMs than in the cases with IAMs (16% v 43%, $p < 0.001$). The probability of developing IAMs was three times higher when the vascular lesion was deep (OR 3.38, 95% CI 1.64 to 6.99).

In the second analysis comparing the 35 patients with IAMs who had deep lesions in the basal ganglia, thalamus, and brain stem with the 35 stroke patients without IAMs but with deep vascular lesions, we found that the patients who had IAMs showed a higher frequency of haemorrhaging (22.8% v 5.1%, $p = 0.04$). Among these patients, the probability of developing IAMs was four times higher (OR 4.8, 95% CI 0.8 to 36.6), although it was not statistically significant.

V. Discussion

In our series, the age at onset was different in the four types of IAM. Patients with chorea were the oldest, whereas those with dystonia were the youngest. There is already some clinical evidence that damage to the brain early in life tends to lead to dystonia rather than to other movement disorders. For example, young onset Parkinson's disease tends to present with dystonia rather than parkinsonism.⁷⁵ This is probably due to changes in neuronal development related to age or brain plasticity, as has been demonstrated in experimental focal cortical lesions, inducing changes in the adjacent cortex and in the contralateral hemisphere.⁷⁶

The late start of parkinsonism post stroke might be artefactual and related to the spontaneous onset of this common disorder or may result from deafferentation, which is indicated by secondary or transsynaptic degeneration, or certain functional changes in neuronal activities and their connecting structures.¹⁶ The partial recovery could be related to a plastic reorganisation or reinnervation.^{16,76}

High blood pressure and heart disease were the principal risk factors for stroke, as in our previous reports and others in the literature.⁵⁸⁻⁶¹ Three of our patients who had ischaemic lesions had diabetes.

Ischaemic^{2,3,7-11,13-24,26,28-36,38-40,42-46,48-54,71,73} orhaemorrhagic,^{1-6,12,25,27,37,41,55,56,71,73} supratentorial^{1-16,18-21,26-34,36,38,39,41-46,48,50,51,53-56,71-75,77,78} or infratentorial,^{17,22-25,35,37,40,47,49,52} and surface^{15,19-21,32,46,74} or deep^{1-14,16-18,22-31,33-45,47-56,71-73} vascular lesions have been described in patients with abnormal movements. The presence of an isolated lesion was correlated with an involuntary movement in less than half of our patients. Lentiform nucleus lesions were the most frequent in our dystonic patients. This contrasts with a recent report of patients with acute infarction limited to the lentiform nucleus where dystonia was not found in any patient⁷⁷; as emphasised by the authors of this study, this could be related to the fact that, in this series, the presence of movement disorders was not a selection criterion. Our results coincide with previous reports in which the dystonia was most frequently related to lentiform nucleus lesions, particularly when involving the putamen,^{2,36,41,71,78} being less frequently associated with lesions of the brain stem,^{35,37,40,79} spinal cord, and cerebellum.^{40,80-82} Dystonia of the hand secondary to a pontine haemorrhage, as seen in one of our patients, has not been reported. Two patients with foot dystonia had a severe proprioceptive deficit. The dystonia of both worsened when they walked up or down hills, that is, when they significantly modified the joint position of the foot, which could be related to proprioceptive dysfunction.⁷³

As described in the literature,^{19,20,74} two patients with upper limb tremor, especially of the hand, had cortical lesions. This suggests that cortical strokes may modulate the sensorimotor circuitry and produce movement disorders.^{19,20}

As in previous reports, in our patients with chorea, the most frequent lesions were in the thalamus,^{3,9,13,14,71} surface,¹⁵ and lentiform nucleus.^{5,6,10,11} One of the eight patients with hemiballismus had subthalamic lesions, one had a pallidal lesion, and in the six remaining patients there were thalamic lesions. Lesions of the subthalamic nucleus^{1,3,4,9} presumably reduce the inhibitory output of the globus pallidus on the thalamus by diminishing the normal excitatory drive to the internal segment of the globus pallidus. This

disinhibition gives rise to excessive excitatory drive to the cortex, which is expressed as a contralateral hyperkinetic movement.^{13,71}

Vascular parkinsonism has been associated with unilateral^{48,50,51,53} or bilateral^{44-49,52,54} basal ganglia infarcts in the striatum or lentiform nucleus. In our patients we found unilateral and bilateral infarcts of the basal ganglia at the mesencephalic and frontal levels. The parkinsonian symptoms could be due to vascular lesions disrupting the interconnecting fibre tracts between the basal ganglia, the thalamus, and the motor cortex that leads to disruption not only of sensorimotor integration,^{46,48,49} but also of descending reticular pathways to the major centres of the brain stem.⁴⁹

Two forms of vascular parkinsonism have been suggested: one with acute onset, associated with basal ganglionic infarcts, and another with insidious progression, possibly associated with more diffuse subcortical white matter ischaemia.^{46,83} This approach does not yet include frontal surface vascular lesions that could produce parkinsonism as was seen in two of our patients and in a previous study.⁷⁴

Our findings strongly suggest that six of our patients had vascular parkinsonism. Five patients developed parkinsonism three months post stroke and one patient 10 months post stroke. As in previous reports,^{46,84,85} the distribution of parkinsonism in our patients at start was ipsilateral to the motor deficit or was bilateral with predominance in the lower limbs. One of our cases with a caudocapsular infarct improved spontaneously.⁴⁵

Most previously reported cases of vascular parkinsonism have shown no response to levodopa.^{46,54} In five patients, we noticed an initial moderate response at the start of therapy with levodopa as in previous reports.^{44-46,84} Our cases showed motor complications at an early stage.

In agreement with previous reports,⁷¹ abulia in our patients was most frequently associated with lesions in the surface and lentiform and caudate nuclei lesions, and disinhibition was infrequent. Both behavioural syndromes can be produced by damage either to the prefrontal cortex or in the massive projection from prefrontal cerebral cortex to the caudate nucleus and the reciprocal striatopallido-thalamocortical projections back to the prefrontal cortex via the “complex” basal ganglia circuits.⁷¹ In our patients, confusion was the most frequent behaviour disorder and their lesions were most frequently located in the thalamus and surface.

The case-control analysis showed that deep lesions were more frequent in patients with IAMs and could condition a higher risk of developing IAMs. The higher frequency of deep lesions in patients with IAMs could be due to the anatomical location of the basal ganglia. The IAMs could be related to deep lesions, especially haemorrhagic ones, although the risk associated with them had no statistical significance, which could be related to the number of patients studied.

From the results of our study, we conclude that lesions involving the basal ganglia (but almost always with surrounding deep white matter) most commonly cause movement disorders. Furthermore, most patients with isolated or unilateral lesions developed contralateral IAMs. Correlation between site of lesion and type and laterality of IAM, however, is difficult to establish. In general, accepted models of basal ganglia circuitry do not fit well with clinical observations of patients with basal ganglia lesions. One explanation is that CT and MRI do not establish the full extent of pathology and, even more importantly, do not show the distant functional effects of such lesions. This may be especially important in patients with stroke in whom ischaemic lesions concurrent with or prior to the last vascular event cannot be detected by current imaging techniques.

VI. Conclusion

The recognition of movement disorders in the setting of stroke can be important in localizing the lesions and in suggesting an underlying etiology. Most strokes associated with movement disorders involve small vessel branches of the middle or posterior cerebral arteries since these supply the basal ganglia, the usual pathological site. The movement disorders can appear acutely at the time of the stroke or they can have a delayed onset. The most commonly observed disorders are hemiballism-hemichorea and dystonia, but other hyperkinetic and hypokinetic disorders can occur as well. The movement disorders may need to be a target for therapy since they can contribute to disability. Involuntary movements tend to persist despite the functional recovery of motor deficit.

VII. Limitations

Our study on post-stroke frequency of movement disorders (3.7%) is difficult to compare with other studies, since most reports are of isolated cases or series of patients with a given type of abnormal movement or anatomical lesion.^{1-56,71-74}

References

- [1]. Kelman H. Hemiballismus: a clinicopathologic study in two cases. *J NervMentDis* 1945;101:363–71.
- [2]. Marsden CD, Obeso JA, Zarranz JJ, et al. The anatomical basis of symptomatic hemidystonia. *Brain* 1985;108:463–83.
- [3]. Diersen G, Gioino G. Correlación anatómica del hemibalismo. *Revista Clínica Española* 1961;82:280–305.
- [4]. Martín TP, Akoek NS. Hemichorea associated with a lesion of corpus Luysii. *Brain* 1934;57:504–5.
- [5]. Jones HR, Baker RA, Kott S. Hypertensive putaminal hemorrhage presenting with hemichorea. *Stroke* 1985;16:130–1.
- [6]. Chang MH, Chiang HT, Lai PH, et al. Putaminal petechial haemorrhage as the cause of chorea: a neuroimaging study. *J NeurolNeurosurg Psychiatry* 1997;63:300–3.
- [7]. Kase CS, Maulsby GO, de Juan H, et al. Hemichorea-hemiballism and lacunar infarction in the basal ganglia. *Neurology* 1981;31:452–5.
- [8]. Tabaton M, Mancardi G, Loeb C. Generalized chorea due to bilateral small, deep cerebral infarcts. *Neurology* 1985;35:588–9.
- [9]. Lee MS, Marsden CD. Movement disorders following lesions of the thalamus or subthalamic region. *MovDisord* 1994;9:493–507.
- [10]. Klawans HL, Moses H, Nausieda PA, et al. Treatment and prognosis of hemiballism. *N Engl J Med* 1976;295:1348–50.
- [11]. Srinivas K, Rao VM, Subbulakshmi N, Braskaran J. Hemiballism after striatal hemorrhage. *Neurology* 1987;37:1428–9.
- [12]. Melamed E, Korn-Lubetzki I, Reches A, et al. Hemiballismus: detection of focal hemorrhage in subthalamic nucleus by CT scan. *Ann Neurol* 1978;4:582.
- [13]. Dewey RB, Jankovic J. Hemiballism-hemichorea: clinical and pharmacological findings in 21 patients. *Arch Neurol* 1989;46:862–7.
- [14]. Kurlan R, Shoulson I. Differential diagnosis of facial chorea. In: Jankovich J, Tolosa E, eds. *Advances in Neurology, Facial Dyskinesias*. New York: Raven Press, 1988;49.
- [15]. Lopez Arlandis JM, Burguera JA, Coret F, et al. Movimientos anormales de origen vascular. *Neurología* 1989;4:316–22.
- [16]. Miwa H, Hatori K, Kondo T, et al. Thalamic tremor: case reports and implications of the tremor: generating mechanism. *Neurology* 1996;46:75–9.
- [17]. Otto S, Buttner T, Schols L, et al. Head tremor due to bilateral thalamic and midbrain infarction. *J Neurol* 1995;242:608–11.
- [18]. Ghika J, Bogousslavsky J, Henderson J, et al. The “jerky dystonic unsteady hand”: a delayed motor syndrome in posterior thalamic infarctions. *J Neurol* 1994;241:537–42.
- [19]. Kim JS. Delayed onset hand tremor caused by cerebral infarction. *Stroke* 1992;23:292–4.
- [20]. Kim JS. Writing tremor after discrete cortical infarction. *Stroke* 1994;25:2280–2.
- [21]. Dethy S, Luxen A, Bidaut LM, et al. Hemibody tremor related to stroke. *Stroke* 1993;24:2094–6.
- [22]. Okuda B, Tachibana H, Sugita M, et al. Bilateral internuclear ophthalmoplegia, ataxia and tremor from midbrain infarction. *Stroke* 1993;24:481–2.
- [23]. Okuda B, Tachibana H. Rubral tremor. *Neurology* 1996;46:288–9.
- [24]. Ferbert A, Gerwig M. Tremor due to stroke. *MovDisord* 1993;8:179–82.
- [25]. Mossutto-Agatiello L, Puccetti G, Castellano AE. “Rubral tremor” after thalamic hemorrhage. *J Neurol* 1993;241:27–30.
- [26]. Martínez Peñeque B, Balseca A, Martí-Masso JF, López de Munain A, et al. Temblor rubro tras lesión vascular estalámica. *Rev Neurol* 1998;26:80–4.
- [27]. Schlitt M, Brown JW, Zeiger HE, et al. Appendicular tremor as a late complication of intracerebral hemorrhage. *SurgNeurol* 1986;25:181–4.
- [28]. Ferbert A, Gerrig D. Non-rubral tremor of vascular origin. *MovDisord* 1991;6:276.
- [29]. Lee MS, Lee SA, Heo JH, et al. A patient with a resting tremor and a lacunar infarction at the border between the thalamus and the internal capsule. *MovDisord* 1993;8:244–6.
- [30]. Grimes JD, Hassan MN, Quarrington AM, et al. Delayed onset posthemiplegic dystonia: CT demonstration of basal ganglia pathology. *Neurology* 1982;32:1033–5.
- [31]. Keane JR, Young JA. Blepharospasm with bilateral basal ganglia infarction. *Arch Neurol* 1985;42:1206–8.
- [32]. Jacob PC, Pratap CR. Blepharospasm and jaw closing dystonia after parietal infarcts. *MovDisord* 1995;10:794–802.
- [33]. Russo LS. Focal dystonia and lacunar infarction of the basal ganglia: a case report. *Arch Neurol* 1983;40:61–2.
- [34]. Demierre B, Rondot P. Dystonia caused by putamino-capsulo-caudate vascular lesions. *J NeurolNeurosurg Psychiatry* 1983;46:404–9.
- [35]. Day TJ, Lefroy RB, Mastaglia FL. Meige’s syndrome and palatal myoclonus associated with brain stem stroke: a common mechanism? *J NeurolNeurosurg Psychiatry* 1986;49:1324.
- [36]. Obeso JA, Giménez-Roldán S. Clinicopathological correlation in symptomatic dystonia. *Adv Neurol* 1988;50:113–22.
- [37]. Muñoz JE, Tolosa E, Saiz A, et al. Upper limb dystonia secondary to a midbrain hemorrhage. *MovDisord* 1996;11:96–9.
- [38]. Schwartz M, De Deyn PP, Van den Kerchove M, et al. Cervical dystonia as a probable consequence of focal cerebral lesion. *MovDisord* 1995;10:797–8.
- [39]. Moïho ES, Factor SA. Basal ganglia infarction as a possible cause of cervical dystonia. *MovDisord* 1993;8:213–16.
- [40]. Jankovic J, Shutish C. Blepharospasm associated with brain stem lesions. *Neurology* 1983;33:1237–40.
- [41]. Pettigrew LG, Jankovic J. Hemidystonia: a report of 22 patients and a review of the literature. *J NeurolNeurosurg Psychiatry* 1985;48:650–7.
- [42]. Camal A, Greene P, Khandji A. Paroxysmal kinesigenic dystonic choreoathetosis associated with thalamic infarcts. *MovDisord* 1990;5:235–8.
- [43]. Powers JM. Blepharospasm due to unilateral diencephalic infarction. *Neurology* 1985;35:283–4.
- [44]. Chang CM, Yu YL, Ng HK, et al. Vascular pseudoparkinsonism. *Acta Neurol Scand* 1992;86:588–92.
- [45]. Tolosa E, Santamaría J. Parkinsonism and basal ganglia infarcts. *Neurology* 1984;34:1516–18.
- [46]. Winikates J, Jankovic J. Clinical correlates of vascular parkinsonism. *Arch Neurol* 1999;56:98–102.
- [47]. Defer GL, Remy P, Malapert D, et al. Rest tremor and extrapyramidal symptoms after midbrain hemorrhage: clinical and 18 F-Dopa PET evaluation. *J NeurolNeurosurg Psychiatry* 1994;57:987–9.
- [48]. Kulisevsky J, Berthier ML, Avila A, et al. Unilateral Parkinsonism and stereotyped movements following a right lenticular infarction. *MovDisord* 1996;11:752–4.
- [49]. Kulisevsky J, Avila A, Berthier ML. Bipolar affective disorder and unilateral Parkinsonism after a brainstem infarction. *MovDisord* 1995;10:802.
- [50]. Friedman A, Kang UJ, Tatemichi TK, et al. A case of Parkinsonism following striatal lacunar infarction. *J NeurolNeurosurg Psychiatry* 1986;49:1087–8.
- [51]. Tison F, Duche B, Loiseau P. Syndrome hemiparkinsonien vasculaire. *Rev Neurol (Paris)* 1999;149:565–7.
- [52]. De la Fuente Fernández R, López JM, Rey del Corral P, de la Iglesia Martínez F. Peduncular hallucinosis and right hemiparkinsonism caused by left mesencephalic infarction. *J NeurolNeurosurg Psychiatry* 1994;57:870.

- [53]. Feñeñon G, Houeñto JL. Unilateral Parkinsonism following a large infarct in the territory of the lenticulostriate arteries. *MovDisord* 1997;12:1086–90.
- [54]. Murrow RW, Schweiger GD, Kepes JJ, et al. Parkinsonism due to a basal ganglia lacunar state: clinicopathological correlation. *Neurology* 1990;40:897–900.
- [55]. Gatto EM, Zurrú MC, Rugilo C, et al. Focal myoclonus associated with posterior thalamic hematoma. *MovDisord* 1998;13:182–4.
- [56]. Donat JR. Unilateral asterixis due to thalamic hemorrhage. *Neurology* 1980;30:83–4.
- [57]. Kotila M. Declining incidence and mortality of stroke? *Stroke* 1984;15:255–9.
- [58]. Alarcón F, Hidalgo F, Moncayo J, et al. Cerebral cysticercosis and stroke. *Stroke* 1992;23:224–8.
- [59]. World Health Organization. Cerebrovascular diseases: prevention, treatment, and rehabilitation. World Health Organ Tech Rep Ser 1971:469.
- [60]. Kunitz SC, Gross CR, Heyman A, et al. The pilot stroke data bank: definition design and data. *Stroke* 1984;15:740–6.
- [61]. Alarcón F, Vanormelingen K, Moncayo J, et al. Cerebral cysticercosis as a risk factor for stroke in young and middle-aged people. *Stroke* 1992;23:1563–5.
- [62]. Fahn S. Concept and classification of dystonia. In: Fahn S, Marsden CD, Calne DB, eds. *Advances in Neurology, Dystonia 2*. New York: Raven Press, 1988:58.
- [63]. Findley LJ. Tremors: differential diagnosis and pharmacology. In: Jankovic J, Tolosa E, eds. *Parkinson's disease and movement disorders*. Baltimore, Munich: Urban and Schwarzenberg, 1988.
- [64]. Hughes AJ, Daniel SE, Kilford L, et al. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study. *J Neurol Neurosurg Psychiatry* 1992;55:181–4. Post-stroke movement disorders: report of 56 patients 1573

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