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PREDICTIVE FACTORS FOR SPASTICITY AMONG ISCHEMIC STROKE PATIENTS

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Abstract – Spasticity is a determining for functional loss following ischemic stroke. **Objective:** To detect possible predictive factors for its occurrence. **Method:** Demographic, clinical and tomographic data on 146 stroke patients were analyzed. **Results:** Spasticity was noted more frequently among patients who underwent physiotherapy (p<0.0001; OR=19.4; 95% CI: 4.4–84.5), those who underwent such treatment for long periods (p=0.028; OR=4.80; 95% CI: 1.1–8.3) and those with manual work (p=0.041; OR=2.2; 95% CI: 1.02–4.6), lower income (p=0.038), pain complaints (p<0.0001; OR=107.0; 95% CI: 13.5–847.3), appearance of pain at the same time as spasticity (p<0.0001), previous vascular disease (p=0.001; OR=4.2; 95% CI: 1.7–10.3), muscle weakness (p<0.0001; OR=91.9; 95% CI: 12.0–699.4), extensive lesions as seen on tomography (p=0.01) and lesions affecting more than one cerebral lobe (p=0.018). Manual work had a relative risk of 2.9; previous stroke 3.9, and extensive lesion 3.6. **Conclusion:** Spasticity affected 25% of the patients, and was associated with: manual work, previous stroke, extensive lesions, decrease in individual income, underwent physiotherapy, underwent physiotherapy for longer period, pain complaints, the pain started simultaneously with the spasticity, presented changes in strength.

KEY WORDS: spasticity, stroke, cerebral infarct, physiotherapy.

Fatores preditivos para espasticidade após acidente vascular cerebral

Resumo – A espasticidade é fator determinante para perda funcional após o acidente vascular cerebral isquêmico (AVCI). **Objetivo:** Detectar possíveis fatores preditivos para a ocorrência da espasticidade. **Método:** Foram analisados dados demográficos, clínicos e tomográficos de 146 pacientes pós-AVCI. **Resultados:** Na análise univariada a espasticidade foi notada com maior freqüência em pacientes que realizaram fisioterapia (p<0,0001; OR=19,4; 95% CI: 4,4–84,5), com maior tempo de duração desse tratamento (p=0,028; OR=4,80; 95% CI: 1,1–8,3) e que realizavam trabalho braçal (p=0,041; OR=2,2; 95% CI: 1,02–4,6), renda menor (p=0,038), referência de dor (p<0,0001; OR=107,0; 95% CI: 13,5–847,3) e seu aparecimento simultâneo à espasticidade (p<0,0001), acidente vascular cerebral (AVC) pregresso (p=0,001; OR=4,2; 95% CI: 1,7–10,3), fraqueza muscular (p<0,0001; OR=91,9; 95% CI: 12,0–699,4), lesão tomográfica extensa (p=0,01) e lesão afetando mais de um lobo cerebral (p=0,018). Na análise de regressão multivariada a atividade braçal apresentou risco relativo de 2,9; acidente vascular cerebral prévio com risco relativo de 3,9 e lesão tomográfica extensa risco relativo de 3,6. **Conclusão:** A espasticidade afetou um quarto da população estudada e esteve associada ao trabalho braçal, AVC pregresso, lesões tomográficas extensas, diminuição da renda individual, realização de fisioterapia, realização de fisioterapia por um período maior, presença de dor, surgimento da dor simultânea à espasticidade e alteração da força.

PALAVRAS-CHAVE: espasticidade, acidente vascular cerebral, infarto cerebral isquêmico, fisioterapia.

Every year, around the world, stroke affects about 2,000 out of every million people[1]. In England, around 100,000 people a year have their first stroke and 30,000 have recurrent stroke[2-4]. In Brazil, the absolute number of hospitalizations due to stroke ranged between 198,705 and 295,596 per annum between 1994 and 1997, and it was estimated that 25% were recurrent cases[5,6]. Over this period, the costs involved in these patients increased by

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9.7%[7]. After suffering a stroke, 80% of these patients present motor impairment. Of all the sequelae that persist for months, spasticity is the principal determining factor for functional loss, and it is considered to be a priority among therapeutic interventions[8]. The incidence of spasticity ranges from 19 to 39%, and it is not known exactly at what point it emerges following the stroke[9,10]. Independent of the type of intervention carried out, the prognosis for recovering from spasticity is poor and requires a multidisciplinary approach[8]. In Brazil, there is no statistical data on patients who develop spasticity following stroke[11].

Spasticity is characterized by increased resistance to stretching the muscles, and this is seen clinically when attempts are made to flex or extend limbs[12]. Following a stroke, the alpha motor neurons become hyperexcitable and, under the influence of different types of descending fibers, this favors an increase in the excitability of the stretching reflex along the muscle[8]. and also contributes towards the adaptation processes in the muscle fibers following the stroke[15,16]. The roles of the ascending and descending neural paths and of the muscle in the development of spasticity is not well understood, and individuals' particular conditions may possibly interfere in this. Despite the clinical importance of and growing interest in studying spasticity, some questions still lack definitive explanations, especially those relating to predictive factors. Since there is no consensus regarding the pathophysio logy associated with the phenomenon of spasticity, there is justification for conducting studies that deal with factors that are possibly predictive of spasticity, from clinical-tomographic and demographic factors to epidemiological and social factors[1,2,8,9,17,18].

Such an approach constitutes the objective of the present study.

METHOD

Patients and location

The present research is an observational study of 146 consecutive patients observed between 2002 to 2005 as outpatients in a stroke protocol. Every patient had clinical and tomographical diagnosis of ischemic stroke. Base data was collected on admission to the study included: sex, age, ethnicity/color; marital status; schooling level; occupation/work; status within professional occupation; individual income following the ischemic stroke; social class; sports activities; undergoing physiotherapy; length of time undergoing physiotherapy; associated diseases; presence of pain; the time when the pain appeared; evaluation of tonus; muscle strength test; the hemisphere injured; number of lesions; extent of the lesion; cerebral topography; involvement of the white matter; involvement of the cerebral cortex; vascular territory and existence of spasticity was verified after one year of follow up.

Patients presenting hemorrhagic stroke were excluded. Demographic, clinical and tomographic data were analyzed. All the patients who took part in the study signed a consent statement. The study had previously been approved by the local Research Ethics Committee (process number: 0525/02).

Demographic data

With the aim of identifying possible risk factors associated with spasticity, we analyzed the following variables: (1) gender; (2) age: elderly (>60 years) or non-elderly (<60 years); (3) ethnicity: color/race as declared by patients themselves; (4) marital status; (5) schooling level; (6) occupation/work: manual (≥4 hours) or non-manual (<4 hours); (7) status within professional occupation according to the criteria of the IBGE classification (Brazilian Institute for Geography and Statistics): employed, unemployed, domestic worker, self-employed, employer, unpaid work, worker producing for own use or worker producing for own consumption[19]; (8) individual income following the ischemic stroke; (9) social class[20]; (10) sports activities; (11) undergoing physiotherapy and (12) length of time undergoing physiotherapy.

Clinical data

The principal clinical variables analyzed in this study were: (1) associated diseases (hypertension, diabetes, coronary disease, previous stroke); (2) presence of pain associated with spasticity (this was deemed to be present when the patients complained about pain; in the event of communication difficulty, body language suggestive of pain that was observed by the investigator or reported by the family was taken into account); (3) the time when the pain appeared, or when it was perceived, classified as pre-spasticity, concomitant with spasticity or post-spasticity; (4) previous stroke; (5) evaluation of tonus, as measured on the modified Ashworth scale[21]; and (6) strength assessment, as measured using a muscle strength test[22].

Tomographic data

Tomographic data were obtained from 84 of the 146 patients who took part in this study. The tomographic data on the other 62 patients were not analyzed because of loss or damage to the films. The tomographic data were evaluated with regard to: (1) the hemisphere injured (side injured); (2) number of lesions: single or multiple; (3) extent of the lesion: extensive (>5 cm) or not extensive (<5 cm)[23]; (4) cerebral topography: frontal, parietal, temporal, occipital, thalamic, cerebellar, insular or more than one lobe; (5) involvement of the white matter; (6) involvement of the cerebral cortex; and (7) vascular territory. Two investigators analyzed the tomographic data, and possible discrepancies in the analyses were resolved by a third investigator or by means of a meeting to establish a consensus.

Allocation to groups

The patients whose clinical assessments showed spasticity of grades 1 to 4 on the Ashworth scale were allocated to the spastic group. The patients whose clinical assessments showed

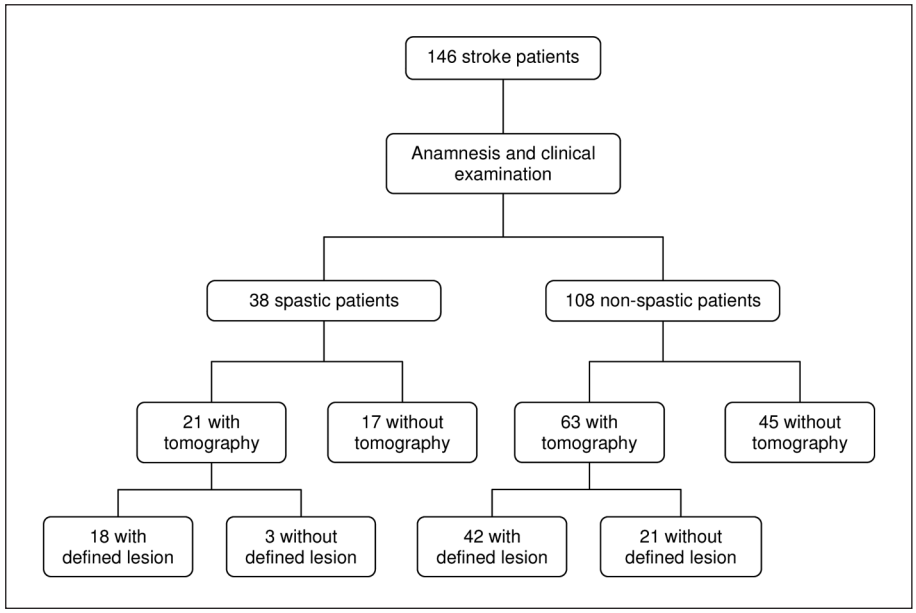

Figure. Flow diagram for patient allocations.

grade 0 (zero spasticity) on the Ashworth scale were allocated to the non-spastic group. There were 108 patients in the non-spastic group and 38 patients in the spastic group (Figure).

Statistical analysis

The chi-squared test was applied to investigate the associations between the following categorical variables: sex, age, ethnicity/color, marital status, schooling level, occupation/work, professional occupation, individual income, social class, sports activities, presence of previous vascular disease, undergoing physiotherapy, pain, time when pain appeared, changes in strength and hemisphere injured. This test was also utilized for all the variables relating to the tomographic data: hemisphere injured, number of lesions, extent of the lesion, lobes affected, white matter, cortex and vascular topography. For the numerical variables, the Mann-Whitney test (expressed as the median and 25^{th} and 75^{th} percentiles) were used for the following variables: number of people living in the home, individual income following ischemic stroke, time taken for hospital attendance, start of physiotherapy attendance and total duration of physiotherapy.

A univariate analysis, chi-square and t tests, was performed to select variables for further inclusion in multivariate (logistic regression) analysis to detect predictors for spasticity. In univariate analysis a p value <0.10 was assumed for inclusion in logistic regression. Relative risks were calculates using CI 95%.

RESULTS

Demographic characteristics

The patients were aged between 25 and 88 years (mean=64.1 years), and there were 77 women and 69 men. The time since their ischemic stroke was greater than or equal to one year and less than five years, and there were patients with and without spasticity. The patients with and without spasticity did not differ with regard to gender, age, ethnicity, color, marital status, schooling level, professional category, social class or sports activities (Table 1). Before their strokes, 19 patients (50%) in the spastic group and 34 patients (31.5%) in the non-spastic group were performing manual activities (p=0.041; OR=2.2; 95% CI: 1.02–4.6). Following their strokes, 19 spastic patients (50%) and 36 non-spastic patients (33.6%) experienced a decrease in individual income (p=0.038).

Analysis of the continuous data relating to demographic characteristics and the development and consequences of the disease (Table 2) showed that the spastic patients experienced a drop in individual income of 37.5% of their minimum salary, in relation to the non-spastic individuals (p=0.067). The spastic patients underwent physiotherapy for seven months longer than did the non-spastic individuals (p=0.001).

Clinical data

Thirty-six spastic patients (94.7%) and 52 non-spastic patients (48.1%) underwent physiotherapy (p<0.0001; OR=19.4; 95% CI: 4.4–84.5). Thirteen spastic patients (36.1%) and eight non-spastic patients (15.6%) underwent physiotherapy for more than one year (p=0.028; OR=4.80; 95% CI: 1.1–8.3) (Table 3).

Nineteen spastic patients (50%) and only one non-spastic patient (0.9%) presented pain (p<0.0001; OR=107.0; 95% CI: 13.5–847.3). The pain started simultaneously with the spasticity in 17 patients (94.4%) (p<0.0001). Thirteen spastic patients (37.1%) and also 13 non-spastic patients (12.3%) had had previous stroke (p=0.001; OR=4.2; 95% CI: 1.7–10.3).

Table 1. Demographic characteristics of the study population.

Variables	Spastic patients (38) n (%)	Non-spastic patients (108) n (%)	Total (146) n (%)	p
Sex				0.251
Female	17 (44.8)	60 (55.5)	77 (52.7)	
Male	21 (55.2)	48 (44.5)	69 (47.3)	
Age				0.936
Elderly	24 (63.1)	69 (63.8)	93 (63.7)	
Non-elderly	14 (36.8)	39 (36.2)	53 (36.3)	
Ethnicity / race				0.573
White	14 (36.8)	52 (48.1)	66 (45.2)	
Black	7 (18.4)	17 (15.7)	24 (16.4)	
Yellow	3 (7.8)	4 (3.7)	7 (4.79)	
Brown	12 (31.5)	26 (24)	38 (26.0)	
Indigenous	2 (5.2)	9 (8.3)	11 (7.53)	
Color				0.228
White	14 (36.8)	52 (48.1)	66 (45.2)	
Non-white	24 (63.1)	56 (51.8)	80 (54.8)	
Marital status				0.624
Single	2 (5.2)	10 (9.2)	12 (8.2)	
Married	27 (71)	65 (60.1)	92 (63.0)	
Divorced	1 (2.6)	6 (5.5)	7 (4.79)	
Widowed	8 (21)	27 (25)	35 (24.0)	
Schooling level				0.279
Illiterat	3 (17.8)	14 (12.9)	17 (11.6)	
Elementary incom.	15 (39.4)	32 (29.6)	47 (32.19)	
Elementary comp.	9 (23.6)	39 (36.1)	48 (32.87)	
High school incom.	3 (7.8)	2 (1.8)	5 (3.42)	
High school comp.	7 (18.4)	16 (14.8)	23 (15.75)	
University-level	1 (2.6)	5 (4.6)	6 (4.10)	
Occupation/work				0.041
Manual	19 (50)	34 (31.5)	53 (36.3)	(OR=2.2; 95% CI: 1.02–4.6)
Non-manual	19 (50)	74 (68.5)	93 (63.7)	
Professional status				0.822
Unemployed	1 (2.63)	6 (5.55)	7 (4.8)	
Employed	6 (15.79)	13 (12.04)	19 (13.0)	
Domestic worker	2 (5.26)	8 (7.4)	10 (6.8)	
Self-employed	3 (7.89)	14 (12.95)	17 (11.6)	
Unpaid worker	2 (5.26)	3 (2.78)	5 (3.42)	
Retired	24 (63.16)	64 (59.26)	88 (60.3)	
Individual income[a]				0. 038
Decreased	19 (50)	36 (33.6)	55 (37.7)	
Increased	1 (2.6)	11 (10.2)	12 (8.3)	
Unchanged	18 (47.3)	60 (55.5)	78 (53.4)	
Social class				0.165
A2	1 (2.6)	0 (0)	1 (0.68)	
B_1	1 (2.6)	8 (7.4)	9 (6.16)	
B_2	3 (7.8)	11 (10.1)	14 (9.58)	
C	17 (44.7)	56 (51.8)	73 (50.0)	
D	15 (39.4)	33 (30.5)	48(32.87)	
E	1 (2.6)	0 (0)	1 (0.68)	
Sports activities				0.881
Yes	8 (21)	24 (33.3)	32 (21.9)	
No	30 (8.9)	84 (77.7)	114 (78.08)	

[a]Data not obtained from one non-spastic patient; OR, odds ratio; CI, confidence interval.

Table 2. Demographic and physiotherapy data.

Variable	Spastic patients Mi (25%–75%)	Non-spastic patients Mi (25%–75%)	p
Individual income following stroke	1.25 ms (1.0–2.5)	2.0 ms (1.0–2.75)	**0.067**
Start of physiotherapy	4 weeks (1–10)	2 weeks (2–4)	**0.137**
Total duration of physiotherapy	12 months (3.5–>12)	5 months (3–8)	**0.001**

Mi: Median; 25th to 75th percentile; ms: minimum salary.

Table 3. Clinical, physiotherapeutic and medication data.

Variables	Spastic patients (38) n (%)	Non-spastic patients (108) n (%)	Total 146 n (%)	p
Physiotherapy				<0.001
Yes	36 (94.7)	52 (48.1)	88 (60.27)	(OR=19.4; 95% CI: 4.4–84.5)
No	2 (5.2)	56 (51.8)	58 (39.7)	
Physiotherapy > one year[a]				0.028
Yes	13 (36.1)	8 (15.6)	21 (24.2)	(OR=4.80; 95% CI: 1.1–8.3)
No	23 (63.8)	43 (84.3)	66 (75.8)	
Associated diseases				0.192
Yes	29 (76.3)	70 (64.8)	99 (67.8)	
No	9 (23.6)	38 (35.1)	47 (32.19)	
Presence of pain				< 0.001
Yes	19 (50)	1 (0.92)	20 (13.7)	(OR=107.0; 95% CI: 13.5–847.3)
No	19 (50)	107 (99)	126 (86.3)	
Onset of pain[b]				< 0.001
Before spasticity	0 (0.0)	1 (100)	1 (5.0)	
At same time	17 (94.4)	0 (0)	17 (85.0)	
Following spasticity	1 (5.6)	0 (0)	1 (5.0)	
Previous stroke[c]				< 0.001
Yes	13 (37.1)	13 (12.3)	26 (18.6)	(OR=4.2; 95% CI: 1.7–10.3)
No	22 (62.9)	92 (87.7)	114 (81.4)	
Changes in strength				<0.001
Yes	37 (97.3)	31 (28.7)	68 (46.57)	(OR=91.9; 95% CI: 12.0–699.4)
No	1 (2.6)	77 (71.2)	78 (53.42)	
Hemisphere lesion				0.416
Right	21 (55.3)	47 (43.5)	68 (46.57)	
Left	15 (39.4)	51 (47.2)	66 (45.20)	
Both	2 (5.3)	10 (9.3)	12 (8.21)	

[a]The total number of patients who underwent physiotherapy for more than one year was 51 non-spastic and 36 spastic individuals; [b]Obtained from one non-spastic and 18 spastic patients; [c]Obtained from 105 non-spastic and 35 spastic patients; OR, odds ratio; CI, confidence interval.

Thirty-seven spastic patients (97.3%) presented changes in strength, while this was observed in 31 non-spastic patients (28.7%) (p<0.0001; OR=91.9; 95% CI: 12.0–699.4).

Tomographic data

From tomography, it was found that 20 patients (23,8%) presented extensive lesions and, among these, seven (35%) presented lesions in more than one cerebral lobe, and 13 (65%) in single lobe. Among these 20 patients with extensive lesions, 10 were spastic (47.7%) and 10 were non-spastic (15.8%) (p=0.01). Among the eight patients with lesions in more than one lobe, five were spastic (23.8%) and three were non-spastic (4.76%) (p=0.018) (Table 4).

Forty patients presented non-extensive lesions (47.6%). Among these, 39 patients (97.5%) had a lesion in one lobe and only one patient (2.5%) has lesions in more than one lobe.

Multivariate analysis

Multivariate regression analysis showed relative risk of

Table 4. Tomographic data.

Variables	Spastic patients (21) n (%)	Non-spastic patients (63) n (%)	p
Hemisphere			0.493
Right	9 (42.9)	14 (22.2)	
Left	7 (33.3)	19 (30.2)	
Both	2 (9.5)	9 (14.3)	
No defined lesion	3 (14.3)	21 (33.3)	
Number of lesions			0.618
Single	13 (61.9)	29 (46)	
Multiple	5 (23.8)	13 (20.7)	
No defined lesion	3 (14.3)	21 (33.3)	
Extensive lesion			0.010
Yes	10 (47.7)	10 (15.8)	
No	8 (38)	32 (50.8))	
No defined lesion	3 (14.2)	21 (33.3)	
Extensive lesion in			0.174
One lobe	5 (24%)	8 (12.7%)	
More than one lobe	5 (24%)	2 (3.2%)	
Non-extensive lesion in			< 0.001
One lobe	8 (38%)	31 (49.2%)	
More than one lobe	0 (0%)	1 (1.58%)	
Lobes			
Frontal	9 (42.8)	19 (30.1)	0.231
Parietal	6 (28.6)	17 (27)	0.212
Temporal	1 (4.76)	3 (4.76)	0.240
Occipital	1 (4.76)	5 (7.94)	0.180
Thalamic	1 (4.76)	0 (0)	0.065
Cerebellar	1 (4.76)	2 (3.17)	0.244
Insular	0 (0)	1 (1.59)	0.193
More than one lobe	5 (23.8)	3 (4.76)	0.018
White matter			0.116
Yes	17 (80.9)	35 (55.5)	
No	1 (4.76)	7 (11.1)	
No defined lesion	3 (14.3)	21 (33.3)	
Cerebral cortex			0.112
Yes	12 (57.1)	21 (33.3)	
No	6 (28.6)	21 (33.3)	
No defined lesion	3 (14.3)	21 (33.3)	
Middle cerebral artery			0.232
Yes	16 (76.2)	36 (57.1)	
No	2 (9.5)	6 (9.5)	
No defined lesion	3 (14.3)	21 (33.3)	

2.9 for manual work, 3.9 for previous stroke, and 3.6 for extensive lesion on tomography (Table 5).

Table 5. Multivariate analysis for risk factors for spasticity.

Factors	Relative risk	p
Manual work	2.9	0.013
Previous stroke	3.9	0.005
Extensive lesion	3.6	0.017

DISCUSSION

Spasticity affected 25.8% of the patients in this study. There is no consensus regarding the frequency of this sequelae, and some controversy results from the subjectivity of spasticity assessments and diagnoses, and regarding the different times after the ischemic stroke at which the assessments are made[9,10,24].

Individuals' own characteristics such as their physical constitution and their functional constitution (the way in which they use their muscles) seem to have an influence

on the development of spasticity[16]. This was seen in our study, in which the patients who had been doing manual activities before their strokes had a relative risk for spasticity of 3. This difference may have been caused by existing modifications and adaptations in the muscle cells, in relation to the size, distribution and contractile properties of the muscle fibers[14-16,25]. Following their strokes, individuals presented reductions in fiber diameters, with some preservation of the proportions of fiber types and fine structure. At successive stages following stroke (3–17 months), the main change is in relation to reduction in volume, progressive atrophy and reduction in the number of muscle fibers due to loss of mobility in the affected limb[16,25].

Spasticity was not associated with gender, age, race/color, marital status, schooling level, status within professional occupation, social class or sports activities. Our initial hypothesis was that constitutional and functional characteristics relating to sex, age and ethnicity might be associated with differences in the prevalence of spasticity, due to alterations in the proportions, quantities, distribution and type of muscle fibers at different ages[14,16,25,26].

It is possible that factors like the sample size and self-declared color may have contributed towards not detecting these variables in our study. The Brazilian population presents a high degree of miscegenation, and specific coverage of this question should be considered in new studies.

Stroke is more prevalent among less privileged social classes[27], and 87% of our patients were from classes C, D and E. This is because both the context of this study (University Hospital that serves preferably a population of underprivileged social classes), as references found in studies that compare industrialized societies, and not industrialized[19].

This created difficulty in making comparisons between the groups, because of the small number of patients from classes A and B. The impact of stroke is significant at all levels of human activity, independent of social class, and it was not different among individuals from less privileged social classes, for whom family incomes that were already very low presented significant reductions following their strokes (37.5%; Tables 1 and 2)[12].

Spasticity was more frequent among the patients with pain, previous vascular disease and muscle weakness, and among those undergoing physiotherapy, and in relation to longer duration of the physiotherapeutic program.

During this study, the physical therapy care was associated with outpatient care when the patients had significant functional loss in the first visit after the ictus.

At that time it was scheduled the start of physical therapy intervention. Patients were included in the Department of Physiotherapy outpatient clinic or they would be directed to another department of physical therapy near their residence, according to their convenience.

The patients who presented severe motor and functional loss, as a consequence of extensive neurological lesion, revealed significant relation between spasticity and physiotherapeutic program.

This can be explained by the systematic of attendance, in which almost all the patients in acute stage, had adhered the Physiotherapy Service.

The patients with greater strength deficit started treatment at an earlier stage. Spastic patients with greater functional deficit[17,19,25] adhered more constantly to the treatment, started two weeks earlier and, on average, continued with treatment for seven months longer (Tables 1 and 2). Recurrent cerebrovascular conditions have also been associated with greater functional losses. We observed a risk 3.9 times greater for developing spasticity in those who had previous stroke, in agreement to the literature[6,28].

Shoulder pains are a frequent complication, occurring in 21–72% of stroke cases. Their main causes are adhesive capsulitis (50%) and subluxation of the shoulder (44%), followed by injury of the rotator cuff, shoulder-hand syndrome[30] and heterotrophic calcification[8]. Spasticity may cause both limitation of the range of movement and decreased strength, and may induce adhesive capsulitis and subluxation. Exaggerated response reflexes to skin stimuli may cause pain in the flexor muscles and spasms in the extensor muscles that interfere with positioning and transfers, thus causing increased waking up and sleep disturbances, among other consequences[8].

The majority of our patients (95%) complained of pain associated with spasticity and correlated its start with the emergence of their spasticity. This percentage of patients in whom the pain started at the same time as their spasticity may have been overestimated because this was a retrospective study that took into account memory-dependent data, since the perception of pain is subjective and also spasticity does not begin instantly.

In our study, extensive lesions (relative risk 3.6) seen on tomography and involvement of more than one cerebral lobe predominated in the patients with spasticity, although there is no consensus regarding the relationship between the size and number of lesions seen on tomography and the occurrence of spasticity[17,18,23].

Imaging studies among stroke patients have indicated that the motor and pre-motor areas are probably responsible for the development of spasticity and for the loss of strength[17,18,30]. Moreover, lesions in the lentiform nucleus, thalamus and cerebellum are associated with prolonged flaccidity.

Perhaps the principal weakness of this study resulted from the choice of a retrospective design and the consequent difficulties that are typical of this model, such as memory bias, non-standardized tomography examinations and the small number of patients from higher socioeconomic classes.

In conclusion, our study demonstrated that 25.8% of the stroke patients presented spasticity that significantly affected their income. The risk factors associated with the occurrence of spasticity were manual professional activities, previous history of stroke, extensive lesions seen on tomography.

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