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Frequency, Determinants, and Consequences of Anosognosia in Acute Stroke

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Anosognosia is a well-known symptom after stroke but its frequency following acute stroke is not known and knowledge of its impact on functional outcome is limited. This prospective study included 566 consecutive, unselected, acute stroke patients. Anosognosia was evaluated on acute admission using the test of Bisiach et al. (1986), stroke severity with the Scandinavian Neurological Stroke Scale (SNSS), and activities of daily living (ADLs) with the Barthel Index (BI). Multiple linear and logistic regression analyses were done to find the influence of anosognosia on the outcome of stroke *per se*. The frequency of anosognosia was 21% on acute admission. The lesion was located in the right hemisphere in 81% of the patients. Anosognosia was seen more frequently following cortical vs. subcortical lesions but showed no significant relationship to any of the four cortical lobes. The presence of anosognosia *per se* predicted 11.5 points less in discharge BI, increased the likelihood of death during the hospital stay by a factor of 4.4, and reduced the likelihood of discharge to independent living in survivors by 0.43. Anosognosia is common in acute stroke, has a profound influence on the prognosis, and indicates patients needing special encouragement and assistance with mobilization. **Key Words:** Anosognosia—Stroke—Cerebrovascular disorders—ADL—Prognosis.

Introduction

The term *anosognosia* was introduced by Babinski to describe patients who ignored their hemiplegia [cit. by Cutting (1)]. This and related phenomenona have also been described as denial of illness (2) and unawareness of disease (3). In stroke it is common to use the term to describe patients who are unaware of hemiplegia or hemianopia. Studies of the frequency of anosognosia following stroke have generally evaluated small selected groups, They have not been community-based and were not conducted in the acute phase of stroke. A wide range in frequency has been reported: from 17% to 59% (1,4–6). The frequency reported in right hemisphere strokes ranges from 28% to 85% (1,4–9) and in left hemisphere strokes from 0% to 17% (1,4–6). These wide ranges are probably due to differences in definition of anosognosia, patient selection, and varying time of examination after stroke. Anosognosia is known to have a strong association with right hemisphere lesions (1,2,5) and some association with reduced general intellectual function (1,10,11) but beyond this not much is known about what determines the symptom. The consequences for functional outcome have only been investigated late after stroke in selected samples (12,13). The present study was undertaken to find the frequency of anosognosia, its determinants, and its consequences for functional outcome in a prospective and consecutive study of a large number of unselected, acute stroke patients.

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Methods

Definition of Stroke

Stroke was defined according to World Health Organization criteria (14): rapidly developed clinical signs of focal disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin. Patients with subarachnoid bleeding were not included.

Inclusion

This study is part of the Copenhagen Stroke Study described in detail elsewhere (15). The setting is community-based and includes all hospitalized patients from a well-defined catchment area, regardless of the age of the patient, the severity of the stroke, and the condition of the patient prior to the stroke. Eighty-eight percent of all stroke patients are hospitalized in the Copenhagen area (16). All patients were transferred on acute admission to the same 60-bed stroke unit, where all stages of acute care, work-up, and rehabilitation took place. A total of 1,014 patients with acute stroke were admitted during the study period from January 1, 1992, to September 30, 1993 [mean age was 74.5 years, SD 10.9; 451 males (44.5%) and 563 females (55.5%)].

Exclusion

Excluded were 448 patients who 1) were not admitted to the hospital within the first week after stroke onset (76); 2) were unconscious on admission (57); or 3) were unable to cooperate because of aphasia (217) or general weakening in the acute stage (98). Patients with aphasia were not generally excluded but most patients with moderate or severe aphasia were unable to cooperate with the test for anosognosia. Excluded patients were older (mean age 75.4 years, SD 10.6) than those included (mean age 73.7 years, SD 11.1, $t = 2.5$, $p = 0.014$), and the percentage of females was higher among the excluded patients (61%) than among the included (52%, $\chi^2 = 8.0$, $p = 0.005$). The excluded patients also had a lower neurologic score [mean Scandinavian Neurological Stroke Scale (SNSS) score 27.0, SD 19.0, compared with 43.4, SD 11.6, $t = -16.0$, $p < 0.001$], had a higher percentage of left hemisphere lesions (66% compared with 50%, $\chi^2 = 25.1$, $p < 0.00001$), and had higher mortality (35% compared with 10%, $\chi^2 = 97.3$, $p < 0.00001$).

Mean duration from stroke onset to admission was 14 hours in the remaining 566 patients with acute stroke: 71% were admitted within 24 hours, 82% within 48 hours, and 89% within 72 hours.

Procedures

A history of previous strokes was obtained on admission. The hospital register containing information on diagnosis from former admissions was also studied. Information concerning comorbidity was obtained on admission and included other disabling disease apart from previous strokes (amputation, multiple sclerosis, heart failure, latent or persistent respiratory insufficiency, parkinsonism, etc.). Length of hospital stay was registered and a corrected length of hospital stay was computed as the length of hospital stay minus days spent in hospital for nonmedical reasons after completing rehabilitation, i.e., waiting time for nursing home.

Type, size, and localization of the stroke lesion were determined by visual inspection of CT scans. The size was measured as the largest diameter of the lesion. All scans were evaluated by the same radiologist, who was blinded to patient data. CT scans were done by a Siemens Somatom DR scanner.

Anosognosia was assessed on admission using the test procedures described by Bisiach et al. (3). According to these procedures, the diagnosis of anosognosia is based on the patient's inability to recognize limb weakness and visual field defects. The anosognosia score was dichotomized and scored as present for any rating below maximum.

Initial stroke severity was assessed with the SNSS (17,18) on admission. The SNSS evaluates level of consciousness; eye movement; motor strength in arm, hand, and leg; orientation; aphasia; facial paresis; and gait. The total score ranges from 0 to 58 points. The two neuropsychological items of this scale—aphasia and orientation—were used separately in the analyses, and because of this a purely neurologic score, not including these items, was computed and used in all analyses (ranging from 0 to 42 points). The aphasia score was used in categorical form—whether any aphasia was present or not. Normal orientation was defined as orientation correct for time (month), place (hospital), and person (birthday); mild disorientation as two correct answers; moderate disorientation as one correct answer; and severe disorientation as no correct answer. We assigned the values from 3 (normal orientation) to 0 (severe disorientation) for the orientation score.

Activities of daily living (ADLs) were assessed using the Barthel Index (BI) (19), which evaluates ten different abilities and ranges in total score from 0 to 100 points. Patients included in the study were assessed during the first week after admission and then subsequently

every week during hospital stay by the nursing and training staff.

Rehabilitation

Rehabilitation based on the Bobath technique was given daily to all patients by nursing staff, physiotherapists, and occupational therapists within the stroke unit. Rehabilitation was completed within the unit. Patients were discharged when the rehabilitation team decided that further improvement in function was unlikely.

Statistics

Comparisons for continuous data were carried out with Student's nonpaired t-test and categorical tables were analyzed with the $\chi 2$ test. Univariate correlations were performed using the Pearson product–moment correlation. To evaluate the relative importance of multiple influences on outcome variables, multiple linear regression and multiple logistic regression analyses were performed. To retain as much information as possible, backward stepwise regression was first performed, and then all independent variables with $p < 0.2$ were entered into forward stepwise regression. The explanatory power of the multiple linear regression models was judged by Adjusted R^2. The required two-tailed significance level for all tests was set to 0.05 for all statistical tests. All analyses were carried out using the SPSS for Windows 6.0 statistical package (20).

Ethics

The study was approved by the Ethics Committee of Copenhagen, approval number V. 100.2263/91.

Results

Frequency and Patient Characteristics

Basic patient characteristics appear in Table 1. Anosognosia was found in 118 patients (21%). Frequency of anosognosia according to age group appears in Table 2. For 403 patients admitted within 24 hours of stroke onset, anosognosia was found in 19%, and for 101 patients admitted within 24 to 72 hours, anosognosia was found in 21%. The frequency of anosognosia was significantly higher, 31%, in 62 patients admitted from 3 to 7 days of stroke onset ($\chi^2 = 4.0$, $p = 0.044$), although there was no significant difference in mean SNSS score on admission (43.5, SD 11.3, and 42.7, SD 14.0, $t = -0.41$, $p = 0.68$). The opposite tendency was found for aphasia. Aphasia was found in 20% of the patients admitted within 3 days of stroke and in 16% admitted from within 3 to 7 days of stroke. The frequency of a right hemisphere stroke was 50% for patients admitted within 7 days and 57% for patients admitted later.

Table 1. *Basic patient characteristics*

N	566
Age, years (SD)	73.7 (11.1)
Sex, male	274 (48%)
Handedness, right	501 (93%)
Lesion side, left	257 (49%)
Mortality, in hospital	54 (10%)
Prior stroke	122 (22%)
Comorbidity	106 (20%)
SNSS on admission, mean	43.4 (11.6)
Neurologic severity, mean (SD)	30.0 (10.1)
Barthel Index on admission	61.9 (38.4)

SNSS: Scandinavian Neurological Stroke Scale
Neurologic severity: SNSS on admission excluding orientation and aphasia score

Table 2. *Frequency of anosognosia according to age group*

Age Group	N	Anosognosia (%)
< 60 years	55	7 (13%)
60–69 years	110	17 (16%)
70–79 years	215	48 (22%)
> = 80 years	186	46 (25%)

Determinants of Anosognosia

There was no significant difference in age or sex distribution between patients with and without anosognosia. Anosognosia was associated with more severe strokes. Patients with anosognosia had a lower initial neurologic score (24.3, SD 11.4 compared with 31.9, SD 9.3, $t = 6.1$, $p < 0.001$) and a lower initial functional (BI) score (39.8 compared with 72.2, SD 33.2, $t = 7.4$, $p < 0.001$), and they were older (mean age 75.9 years, SD 9.5 compared with 73.1 years, SD 11.4, $t = -2.4$, $p = 0.016$).

They also had a higher percentage of right hemisphere lesions (81% compared with 42%, $\chi^2 = 53.6$, $p < 0.00001$). The frequency of anosognosia was 36% in patients with right hemisphere lesions and 9% in patients with left hemisphere lesions. The frequency of anosognosia was the same (21%) whether or not the patient had had a previous stroke.

Premorbid dementia was known to be present in 11% with anosognosia (13 patients) but only in 4% without anosognosia (16 patients). Patients with anosognosia had a lower mean orientation score on admission (2.1, SD 1.1) than patients without anosognosia (2.7, SD 0.7, $t = 5.7$, $p < 0.001$). This relation could be caused by a third factor, e.g., stroke severity. A multiple logistic regression analysis was done with the presence of anosognosia as the dependent variable and neurological score, age, sex, previous strokes, comorbidity, side of stroke lesion, and orientation score on admission as the independent variables. The resulting model correctly classified 85% of the cases for presence of anosognosia; it included neurologic score, side of stroke lesion, and orientation score. A 1-point increase in orientation score reduced the likelihood of anosognosia by a factor of 0.58 (odds ratio, 95% CI: 0.44 to 0.76). The influence of the side of the lesion was much larger: a right-sided lesion increased the likelihood of anosognosia by a factor of 6.6 (odds ration 95% CI: 3.7 to 11.6).

Impaired orientation was not a necessary precondition for anosognosia: 61 (52%) of the patients with anosognosia had unimpaired orientation. Nor was it sufficient for anosognosia to occur; 67 (54%) of the patients with impaired orientation did not have anosognosia. The relation between anosognosia and impaired orientation was similar for patients with right- and left-sided lesions: 47% of the patients with right hemisphere lesions and anosognosia had impaired orientation, as did 54% of the patients with left hemisphere lesions ($\chi^2 = 0.37$, $p = 0.54$).

We found no relation between anosognosia and handedness in patients with left hemisphere lesions: 95% of the patients with anosognosia and a left hemisphere lesion were right-handed, as were 95% of those without anosognosia. There was also no relation between the side of the lesion and gender in patients with anosognosia: a left hemisphere lesion was found in 19% of the men with anosognosia and in 20% of the women with anosognosia ($\chi^2 = 0.02$, $p = 0.88$).

A CT scan was done in 88% of the patients. Basic characteristics appear in Table 3. Median duration from stroke onset to CT scan was 10 days; for patients without visible focal lesions it was 11 days. There was no significant difference in the distribution of stroke subtypes (hemorrhage/infarct) between patients with (hemorrhage: 9%) and without anosognosia (hemorrhage: 5%, $\chi^2 = 3.4$, $p = 0.07$). Patients with anosognosia had larger stroke lesions (mean diameter 53.7 mm, SD 28.3) than patients without (30.0 mm, SD 21.2, $t = 106.3$, $p < 0.001$), and their stroke lesions more often involved the cortex (71% compared with 30%, $\chi^2 = 41.8$, $p < 0.00001$).

The frequency of anosognosia was not significantly related to lesions in either the frontal, temporal, parietal, or occipital lobe (in patients with cortical involvement; Table 4). There was also no association of anosognosia to lesions restricted to either the frontal, temporal, parietal, or occipital lobe (Table 4). An association of anosognosia with thalamic and especially the lateral thalamic region was found in patients with purely subcortical lesions (Table 5). Very few patients had lesions restricted to a single subcortical area, and anosognosia in lesions restricted to the thalamus did not occur (Table 5).

Influence on Functional Outcome

The presence of anosognosia in surviving patients was univariately associated with a poorer functional outcome (lower BI score), a longer stay in hospital, and a smaller proportion of patients returning to independent living after discharge (Table 6). Because anosognosia was also associated with more severe strokes, lower initial functional status, and older age, multivariate analyses of the influence of anosognosia on the outcome were carried out to determine the influence of anosognosia.

A multiple linear regression analysis of discharge BI was carried out with age, sex, side of stroke lesion, presence of a previous stroke, presence of comorbidity, initial BI score, initial neurologic score, and presence of anosognosia as the independent variables. The resulting model included initial BI score, initial neurologic score, presence of comorbidity, and presence of anosognosia. The model explained 61% of the variance in discharge BI ($F = 182.8$, $p < 0.0001$), and the presence of anosognosia independently accounted for 11.5 points less in discharge BI score (95% CI: −16.2 to −6.9).

Table 3. *CT data*

CT performed, N	500
No focal lesion on CT %	35%
Infarct %	60%
Hemorrhagic %	5%
Lesion size, mm (SD)*	35.8 (25.2)
Cortical involvement %*	40%

* For patients with visible lesions on CT scans

Table 4. *Association between anosognosia and lesion localization in patients with cortical involvement on CT scan*

	Lobe involved in stroke lesion				Lesion restricted to one lobe			
	Total number	*Without anosognosia N=72*	*With anosognosia N=55*	*Statistics*	*Total number*	*Without anosognosia N=72*	*With anosognosia N=55*	*Statistics*
Frontal lobe	62	34 (47%)	28 (51%)	NS	20	14 (19%)	6 (11%)	NS
Temporal lobe	53	27 (38%)	26 (47%)	NS	6	3 (4%)	3(6%)	NS
Parietal lobe	67	36 (50%)	31 (56%)	NS	14	8 (11%)	6 (11%)	NS
Occipital lobe	35	19 (26%)	16 (29%)	NS	23	12 (17%)	11 (20%)	NS

A similar multiple linear regression analysis of length of rehabilitation was carried out with the same independent variables as the prior analysis. The final model included initial BI and presence of comorbidity, but not anosognosia.

A multiple logistic regression analysis was performed on discharge to independent living including the same independent variables as well as marital status. The final model included BI score on admission, marital status, and presence of anosognosia, and correctly classified 91.4% of the cases. The presence of anosognosia reduced the likelihood of discharge to independent living by a factor of 0.43 (odds ratio, 95% CI: 0.19 to 0.96).

Mortality in patients with anosognosia (24%) was higher than in patients without (6%, $\chi^2 = 34.8$, $p < 0.00001$), but patients who died during hospital stay could not be included in these analyses in a reasonable way. An additional analysis was carried out to investigate whether anosognosia had any independent predictive value on mortality, using the same covariates. The result was that anosognosia independently increased the risk of dying during hospital stay by a factor of 4.4 (odds ratio, 95% CI: 1.8 to 10.8).

Discussion

This study of 566 patients reports the frequency of anosognosia in acute stroke. Previous studies have generally been based on small and selected samples (1,4, 5,7–9); one study included only hemiplegic patients (1), and others included only patients with right hemisphere lesions (7–9). These studies have generally found higher frequencies than the frequency of 21% reported here except Stone et al. (6), who found anosognosia in 28% of patients with right hemisphere stroke and 5% of patients with left hemisphere stroke (corresponding to 17% of all stroke patients). That study was not community-based, the population was not large (171, of whom 116 were assessable), and the patients were not assessed before 2 to 3 days after stroke.

Anosognosia for different symptoms is dissociable (3), and it is not unusual for patients to acknowledge that they had a stroke without acknowledging hemiplegia (1). A precise definition is therefore necessary. We operationally defined it according to the test method of Bisiach et al. (3), defining anosognosia as the lack of recognizing hemiplegia or hemianopia. In contrast to a number of other studies, we have chosen to compute the frequency of anosognosia based on the total number of stroke patients rather than in relation to patients with hemiplegia or hemianopia only. We included patients who by definition could not have anosognosia, as they had no hemiplegia or hemianopia. We did so because we wanted to know the extent of the problem in the total population of acute stroke patients.

A limitation of the present study is that 36% of the 881 patients who were admitted within the first week of stroke onset, and who were not unconscious, were untestable. Most of these (69%) had aphasia. Very little is known about anosognosia in aphasic patients, and the interest has been focused on awareness of paraphasic speech, not on anosognosia for hemiplegia and hemianopia (21,22). A left hemisphere localization did not preclude anosognosia in our study, and anosognosia was found to be associated with lesion size. Although the frequency of anosognosia in left hemisphere stroke patients is somewhat higher than what can be determined by a test that inevitably has to rely on verbal communication, it must still be assumed to be lower than in patients with a right hemisphere stroke. Accordingly, the frequency of anosognosia in all stroke patients may be somewhat lower than the 21% found in the testable patients.

A surprising finding was the higher frequency of anosognosia in patients admitted later than 3 days after stroke. It could be that some of these patients were slow to seek help because of their anosognosia, and our data

Table 5. *Association between anosognosia and lesion localization in patients without cortical involvement of CT scans*

	Area involved in stroke lesion				Lesion restricted to one area			
	Total number	Without anosognosia N=171	With anosognosia N=24	Statistics	Total number	Without anosognosia N=171	With anosognosia N=24	Statistics
Anterior thalamus	5	3 (2%)	2 (8%)	NS	0	0	0	—
Medial thalamus	9	7 (4%)	2 (8%)	NS	1	1 (1%)	0	NS
Lateral thalamus	16	11 (6%)	5 (21%)	$\chi^2 = 5.8$ $\chi^2 = 0.016$	1	1 (1%)	0	
All thalamic areas	17	12 (7%)	5 (21%)	$\chi^2 = 5.0$ $\chi^2 = 0.025$	5	5 (4%)	0	NS
Putamen	41	38 (22%)	3 (13%)	NS	11	10 (9%)	1 (7%)	NS
Globus pallidus	44	39 (23%)	5 (21%)	NS	8	7 (6%)	1 (7%)	NS
Caudate nucleus	13	11 (6%)	2 (8%)	NS	5	5 (4%)	0	NS
Anterior internal capsule	50	40 (23%)	10 (42%)	NS	25	22 (19%)	3 (20%)	NS
Posterior internal capsule	53	44 (26%)	9 (38%)	NS	25	24 (21%)	1 (7%)	NS

Table 6. *Univariate analysis of outcome in surviving patients in relation to presence of anosognosia on admission*

	With anosognosia	Without anosognosia	Statistics
Functional status (BI) at discharge, mean (SD)	57.1 (40.3)	87.7 (22.9)	$t = 6.6$, $p < 0.001$
Length of rehabilitation, mean days (SD)	37.0 (30.5)	26.1 (25.8)	$t = 3.5$, $p < 0.001$
Discharge to independent living	62%	91%	$\chi^2 = 50.8$, $p < 0.00001$

BI: Barthel Index
Length of rehabilitation: length of hospital stay excluding time spent waiting for nursing home

suggest that the dramatic symptom of aphasia will cause earlier attention. Other explanations are possible but it should be noted that the patients were similar in terms of neurologic severity of stroke.

Anosognosia was associated with larger stroke lesions, most often involving the cortex. No particular association of anosognosia with localization of the stroke lesion in any of the cerebral lobes was found, apart from the well-known strong association with right-sided stroke lesions. A particular association with parietal lobe lesions as reported by Bisiach et al. (3) and Starkstein et al. (5) could not be confirmed. Our results were more in accordance with those of Hier, Mondlock, and Caplan (8) and Levine, Calvanio, and Rinn (10), who also did not find an association between anosognosia and parietal lesions.

Anosognosia had some association with thalamic involvement, but not with basal ganglia involvement. The absolute numbers were small and the result should be interpreted with caution. Previous reports have found an association of thalamic lesions with anosognosia (3,5) but the findings concerning basal ganglia lesions have been contradictory (3,5).

We found anosognosia to be associated with reduced orientation and presumably reduced general intellectual function, which is in accordance with what has been found in acute hemiplegics by Cutting (1) and for persistent anosognosia by Levine et al. (10). We also found that reduced orientation was neither necessary nor sufficient for anosognosia to occur. These findings are in line with the existing literature, indicating that reduced general cognitive function or confusion has often, but by no means always, been found with anosognosia (11).

The pathophysiology of anosognosia is not known. The strong association with right hemisphere lesions and the weaker association with reduced general cognitive function lends credibility to a two-component model: 1) a right hemisphere lesion makes it harder to notice hemianopia or hemiparesis, which could be either the result of a disconnection of the primary, sensory, and motor areas of the right hemisphere from the language centers in the left hemisphere or caused by some more general association of the right hemisphere with awareness; and 2) the sensory or motor deficit, which is not automatically registered, has to be discovered through reasoning about information the person inevitably will get that something is wrong (23). A relation of anosognosia to emotional indifference toward the illness (anosodiaphoria) has been noted (1). It could also be that the patient with anosognosia lacks the motivation to discover disturbed functions and that the association of anosognosia with right hemisphere lesions is caused by impaired right hemisphere emotional processing (24).

Anosognosia was shown to have an independent effect on functional outcome. Although the presence of anosognosia did not prolong the rehabilitation period, it did influence the functional level achieved after completed rehabilitation. It was associated with 11.5 points less in BI score after the influence of other variables had been accounted for. The presence of anosognosia was also related to a dramatic decrease in the likelihood of independent living after completed rehabilitation. The prognostic significance of anosognosia was underlined by the finding that anosognosia was a strong independent predictor of mortality.

Early mobilization may be one of the factors behind the reduction in life-threatening medical complications found in stroke units (25). It is, then, possible that the anosognosic patient is less active and more prone to complications following immobility. It is important to emphasize early mobilization of the anosognosic patient.

Our results are in line with the few existing studies in this area. Gialanella and Mattioli (12) assessed anosognosia in a small and selected group of patients with left hemiplegia one month after stroke and found anosognosia to be a negative prognostic factor for motor and functional recovery. They used only univariate analyses. Sundet et al. (13) lumped together involuntary crying and denial of illness as pathologic emotional reactions in a study of selected right hemisphere stroke patients assessed approximately five months post-stroke.

They found the presence of pathologic emotional reactions to be a more important predictor of self-reported functional status than other neuropsychological variables in a multiple regression analysis.

Anosognosia is probably the least studied of the major neuropsychological symptoms in stroke. It is important to assess for research and for evaluation of patients. It is common in acute stroke, of importance for rehabilitation and functional outcome, and identifies patients who require special devices or assistance in order to allow adequate mobilization.

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