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The Prevalence of Dementia Three Months after Stroke and its Risk Factors

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Abstract

Objective: *The aim of this study was to determine the prevalence of post-stroke dementia (PSD) and its possible clinical and sociodemographic risk factors 3 months after the index stroke episode.*

Methods: *Among 147 patients who were hospitalized in the inpatient neurology clinic of Dicle University Faculty of Medicine with a diagnosis of stroke, 106 that met the inclusion criteria were included in the study 3 months after the index stroke. All patients underwent a detailed systemic and neurological examination, as well as a clinical interview in an effort to determine the sociodemographic features, and both vascular and non-vascular risk factors of stroke. Routine laboratory examinations and cranial imaging (computed tomography [CT] or magnetic resonance imaging [MRI]) were also conducted. The functional, clinical, and cognitive status of the patients were evaluated at the time of hospitalization and 3 months later with the Barthel Index, NIH Stroke Scale (NIHSS), and Mini Mental State Examination (MMSE), respectively.*

Results: *Of the 106 patients included in the study, 32 (30.2%) were diagnosed with PSD. Multivariate analyses revealed that increased age, presence of atrial fibrillation, multiple brain lesions, and cognitive and functional status during hospitalization predicted the development of PSD in this group of patients.*

Conclusion: *The results corroborate previous findings that PSD is a common complication of stroke. Early recognition and treatment of PSD risk factors will definitely diminish the burden of stroke on society and help to improve patient quality of life.*

Key Words: *Dementia, post-stroke dementia, risk factors, prevalence*

INTRODUCTION

Stroke is a major global health problem among elderly populations (Mackowiak-Cordoliani et al., 2005). The majority of post-stroke patients that can proceed with their normal daily activities display major residual physical, cognitive, and behavioral changes, which seriously affect social, professional, and family functioning (Leys et al., 2005). One of the most important complications, which results in dependency and has a major affect on the patient's life is post-stroke dementia (PSD). PSD is a term that defines the dementia that develops following a stroke in a patient that did not have dementia prior to the stroke (Pohjasvaara et al., 1998).

PSD involves all cases that develop dementia after a stroke, independent of the etiology (vascular, degenerative, mixed) (Leys et al., 2005). The frequency of dementia was reported to increase 4-12 times after stroke, with a prevalence rate of 30% (Tatemichi et al., 1992; Censori et al., 1996; Kokmen et al., 1996; Henon et al., 1997; Kooten and Kaudstaal, 1998; Barba et al., 2000; Skoog, 2000; Bakar, 2003a; Zhou et al., 2004).

The increasing elderly population in Turkey and the world, and the decrease of post-stroke death due to widespread intensive care demonstrates that the prevalence of PSD and its burden to society will increase in the future (Leys et al., 2005). These findings also highlight the importance of determining the risk factors related to PSD. Despite numerous studies, there's no consensus on the possible risk factors of PSD other than such factors as age and level of education, which have a role in the development of PSD (Bowler and Hatchinsky, 1994; Moroney et al., 1996; Gorelick, 1997; Breteler et

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al., 1998; Ott et al., 1998a). Various possible risk factors that are linked to the development of PSD, such as hypertension, diabetes mellitus (DM), atrial fibrillation (AF), myocardial infarct (MI), and heart failure, are also associated with the development of stroke. Similarly, it is known that degenerative dementias like Alzheimer's disease seen in elderly patients can first emerge after a stroke and that vascular processes can also affect the formation and progression of degenerative dementias (Henon et al., 1995; Hofman et al., 1997). Therefore, determining and controlling these risk factors can also decrease the occurrence of PSD and other degenerative dementias (Henon et al., 1995; Bakar, 2003b).

Based on these points, the present study aimed to determine the prevalence of PSD 3 months after the index stroke episode (the early post-stroke period) and its possible clinical and sociodemographic risk factors.

METHOD

The study included 147 patients (86 men, 61 women) that were hospitalized in the inpatient neurology clinic of Dicle University Faculty of Medicine with a diagnosis of stroke (ischemic or hemorrhagic) between April 2004 and May 2005. Mean age of the sample was 66.5 ± 6.1 years (range: 55-77 years). The diagnosis of stroke was made according to the following World Health Organization (WHO) criteria: Rapidly developing signs of focal (or global) disturbance of cerebral function leading to death or lasting more than 24 h, with no apparent cause other than vascular (Hatano, 1976), and was confirmed with brain imaging techniques in all patients (magnetic resonance imaging [MRI] and/or computed tomography [CT]).

Exclusion criteria were as follows: (1) subdural hematoma, subarachnoid hemorrhage, or posttraumatic hemorrhage; (2) history of a central nervous system disease that could affect cognitive functioning (for example, a tumor, hydrocephaly, head trauma, or Parkinson's disease); (3) a serious illness that could interfere with cognitive functioning (for example vision or hearing problems); (4) unconsciousness post-stroke; (5) serious aphasia. Informed consent was obtained from all patients prior to beginning the study.

The first patient assessments were conducted during hospitalization in the neurology clinic. The second assessments were performed the third month after the stroke by the same doctor. Patients who did not come to the third-month follow-up were excluded.

Of the 147 patients, 39 whose first assessments were completed were excluded, as they did not show up for the third-month follow-up, and 2 others were excluded due to the suspicion of possible dementia before the index stroke. Among the 39 patients that were not followed-up, 23 had died as a result of heart attack, recurrent stroke, or diabetic coma. There was no response from the remaining 16. Therefore, 106 patients (72.1%) (66 men, 40 women) were included in the study 3 months after the index stroke, as they fulfilled the inclusion criteria.

All patients underwent a detailed systemic and neurological examination, routine laboratory examinations (full blood count, electrolytes, blood sugar, lipid levels, and other values), electrocardiography (EKG), chest radiography, and echocardiography (in order to define emboli and other cardiac disorders).

For this study, the lead author conducted detailed clinical interviews with the patients and their close relatives before discharge from the hospital, and data regarding age, sex, marital status, level of education, and socioeconomic status (SES) were obtained. When evaluating SES, patients with a monthly income below the proposed poverty level reported by the Turkish Statistical Institution (429 YTL for the year 2004) were considered low SES and patients with a monthly income above this level were considered as middle SES.

Post-stroke functioning of the patients was evaluated with the Barthel Index, based on patient records and data gathered from patients and relatives (Mahoney and Barthel, 1965). To objectively evaluate the severity of stroke and clinical condition of the patients before stroke, the NIH Stroke Scale (NIHSS, National Institute of Health Stroke Scale) (Brott et al., 1989) was used, and to determine cognitive functioning and the Standardized Mini Mental Examination (MMSE) (Güngen et al., 2002) or Standardized Mini Mental Examination for illiterate subjects (E-MMSE) (Ertan et al., 1999) was used. When the patients were first hospitalized in the clinic and when they were discharged from the clinic their past history of dementia was assessed by anamnesis, clinical findings, files, information gathered from the relatives, and MMSE or E-MMSE findings. In the light of these findings 2 patients were excluded from the study with the suspicion of a past dementia diagnosis according to DSM-IV.

During the same interview, with the support of patient files, current or past cardiac problems (AF, cardiac insufficiency, valve disease, and myocardial infarct), other

Table I. Comparison of the sociodemographics of patients with PSD [PSD (+)] and those without PSD [PSD (-)].

	PSD (+) (n = 32)	PSD (+) (n = 74)	P
Age (years)	69.9 ± 5.3	65.1 ± 5.8	< 0.0001
Duration of education (years)	2.5 ± 3.5	3.7 ± 4.7	N.S.
	n (%)	**n (%)**	
Sex			
Women	17 (53.1)	23 (31)	0.032
Men	15 (46.9)	51 (69)	
Level of education			
No education	23 (71.8)	45 (60.8)	N.S
Educated	9 (28.2)	29 (39.2)	
SEL			
Low	23 (71.8)	53 (71.6)	N.S
Moderate	9 (28.2)	21 (28.4)	
Working			
Not working	29 (90.6)	59 (79.7)	N.S
Working	3 (9.4)	15 (20.3)	
Marital Status			
Single/widow	5 (15.6)	9 (12.1)	N.S
Married	27 (84.4)	65 (87.9)	

SEL: socioeconomic level; N.S: not significant, PSD: post-stroke dementia.

systemic diseases, and past strokes or transient ischemic attacks were assessed and recorded. Other vascular risk factors (smoking, alcohol, hypertension, hyperlipidemia, and coronary or other atherosclerotic diseases) were evaluated and identified based on the following criteria:

1. Hypertension: Arterial blood pressure was evaluated with 2 different measurements after a 10-min rest and both measurements were evaluated. Patients with a history of antihypertensive treatment and those with systolic blood pressure > 140 mm Hg and diastolic blood pressure > 90 mmHg were considered hypertensive.

2. Diabetes Mellitus: Patients with fasting blood sugar > 110 mg/dl or who were taking antidiabetic or insulin treatment.

3. Hypercholesterolemia: Past history or a finding of serum cholesterol levels > 200 mg/dl based on 2 measurements

4. Hypertriglyceridemia: Past history or a finding of serum triglyceride levels > 150 mg/dl based on 2 measurements.

5. Alcohol: Past or current alcohol use.

6. Smoking: Past or current smoking behavior.

Instead of mean values of the laboratory findings, the number of patients with risk or high lipid levels is provided.

In addition to these clinical data, patients were divided into subgroups according to clinical presentation of stroke, clinical findings, and CT or MRI findings. Patients were grouped as ischemic or hemorrhagic according to stroke etiology, and grouped as single or multiple according to the number of brain lesions. Strokes were grouped according to their localization (right, left, or bilateral), area affected (cortical, sub-cortical, or mixed), and lesion size, based on clinical and radiological assessments.

The detailed systemic and neurological assessments of the patients who were followed-up 3 months after the index stroke were repeated, and the related scales were re-administered in order to assess functional, cognitive, and clinical status. Three months post stroke, patients

Table II. Comparison of vascular risk factors and stroke characteristics of patients with PSD [PSD (+)] and those without PSD [PSD (-)].

n (%)	PSD (+) (n = 32)	PSD (-) (n = 74)	p
Vascular risk factors			
Hypertension	28 (87.5)	68 (91.9)	N.S
Diabetes Mellitus	10 (31.3)	20 (27)	N.S
Heart Disease	9 (28.1)	14 (18.9)	N.S
Atrial Fibrillation	10 (31.3)	8 (10.8)	0.01
Myocardial Infarct	7 (21.9)	7 (9.5)	N.S
High cholesterol Level	10 (31.3)	24 (32.4)	N.S
High Triglyceride Level	15 (46.9)	28 (37.8)	N.S
Past Stroke	15 (46.9)	17 (23)	0.014
Past TIA	14 (43.8)	20 (27)	N.S
Smoking	21 (65.6)	57 (77)	N.S
Alcohol use	1 (3.1)	9 (12.2)	N.S
Type of stroke			
Ischemic	21 (65.6)	51 (68.9)	N.S
Hemorrhagic	11 (34.4)	23 (31.1)	
Localization of the stroke			
Left Hemisphere	11 (34.4)	31 (41.9)	N.S
Right Hemisphere	13 (40.6)	31 (41.9)	
Bilateral	8 (25.0)	12 (16.2)	
Number of lesions			
Single	14 (43.8)	54 (73)	0.004
Multiple	18 (56.3)	20 (27)	
Size of lesion			
< 2 cm	17 (53.1)	44 (59.5)	N.S
2-4 cm	9 (28.1)	12 (16.2)	
> 4 cm	6 (18.8)	18 (24.3)	
The affected area			
Cortical	11 (34.4)	33 (44.6)	N.S
Subcortical	15 (46.9)	23 (31.1)	
Mixed	6 (18.8)	18 (24.3)	

TIA: transient ischemic attack; N.S: not significant; PSD: post-stroke dementia.

and their relatives were interviewed to determine if PSD developed, based on changes in MMSE or E-MMSE scores and current condition.

Scales

The scales that were used during the index stroke and 3 months after the stroke were:

1. Barthel Index: The index is frequently used in Turkish and international studies to numerically assess loss of functionality due to stroke; it was developed by Mahoney and Barthel in 1965 (Mahoney and Barthel, 1965; Altın et al., 2006). The scale includes 10 items that measure the following abilities on a scale of 5-15 points; feeding, personal hygiene, bathing, dressing, toilet, bladder control, bowel control, chair/bed transfer, ambulation, and stair climbing. The index is used to detect the degree of independent functionality without receiving verbal or physical help. Help on any level results in an evaluation that the patient is not independent. Direct testing is not necessary and observation or data collection from relatives, nurses, or other caregivers is sufficient (Mahoney and Barthel, 1965). Total score

Table III. Comparison of the functional, cognitive, and neurological condition of patients with PSD [PSD (+)] and those without PSD [PSD (-)].

	PSD (+)	PSD (-)	p
During Hospitalization			
NIHSS	12 ± 4.7	5.6 ± 4.1	< 0.0001
Barthel Index	45.2 ± 31.7	81 ± 15.6	< 0.0001
MMSE	19. ± 4.2	25.4 ± 5.2	< 0.0001
3 Months Post-Stroke			
NIHSS	9.1 ± 5.4	3.3 ± 4.5	< 0.0001
Barthel Index	48.1 ± 36.3	85.2 ± 13.9	< 0.0001
MMSE	20.6 ± 6.6	26.9 ± 4.9	< 0.0001

NIHSS: National Institute of Health Stroke Scale; MMSE: standardized mini mental test; PSD: post-stroke dementia.

ranges between 0 and 100, and higher scores indicate higher degrees of independence.

2. National Institute of Health Stroke Scale (NIHSS): This scale was developed by the U.S. National Institute of Health for use in detailed clinical and drug studies (Brott et al., 1999). This clinical scale is used in the follow-up of stroke patients to determine the severity of stroke. The scale has 11 questions, of which 1 includes 3 sub items, and measures the level of consciousness, conscious answers to questions, response to directions, extraocular muscle movement, visual area, facial palsy, arm and leg motor movement, extremity ataxia, sensory loss, aphasia, dysarthria, and neurological neglect. The items are evaluated with a 3 point scale. The presence of a problem requires an evaluation score of at least 2 points (with one point increases according to the question, between 0-3). The highest possible score is 36 and lower scores indicate better post-stroke clinical presentation.

3. Standardized Mini Mental Test (MMSE) (for literate and illiterate individuals): This scale developed by Folstein et al. (1975) is easy to use and measures the level of cognitive disorder. It includes orientation, registration-attention-calculation, memory, and language tests, and structuring subtests. The Turkish reliability and validity studies of MMSE and E-MMSE were conducted by Güngen et al. (2002) and Ertan et al. (1999), respectively. As the highest score that can be obtained is 30, it was reported to be a reliable and valid tool for mild dementia in Turkish culture (Güngen et al., 2002). The cut off score for mild dementia is reported to be 23/24.

Statistical Evaluations

All statistical evaluations were performed with SPSS v.10.0 (SPSS, Inc., Chicago). The demographics and frequencies are given as numbers and percentages. Student's t-test was used to compare the patients with and without PSD. Chi-square test was used to evaluate categorical variables. If the expected cell values in the 2 × 2 chi square test were < 5, then Fisher's exact chi square test was used. The level of statistical significance for all statistical analyses was accepted as $P < 0.05$.

In order to find the predictors of PSD development, multivariate analysis and backward stepwise logistic regression were used. Having or not having a PSD diagnosis was accepted as the dichotomous dependent variable. On the other hand, variables that were significant in univariate analyses, such as age, sex, AF, presence of a past stroke, number of lesions (single or multiple), and NIHSS, Barthel Index, and MMSE scores during hospitalization were considered as independent variables. In addition to the level of significance, the results of logistic regression analysis were presented by providing the approximate relative risk (odds ratio [OR]) and 95% confidence rate (GA) values.

RESULTS

Of the 106 patients included in the study, 32 (30.2%) were diagnosed with PSD versus 74 (69.8%) that were not. As the number of subjects was low, they were grouped as working and not working. Patients with regular jobs were considered as working and others were grouped as nonworking. Similarly, the patients who are illiterate and who do not have any education beyond literacy were categorized as having no education and all other patients were categorized as having been educated.

The sociodemographic characteristics of the patients

Table IV. Variables that predicted PSD development according to logistic regression analysis.

Variable (n = 106, PSD = 32)	OR	95% CR	P
Age	0.745	0.609-0.910	0.004
Number of lesions (Multiple)	14.383	1.559-132.685	0.019
Atrial Fibrillation	0.056	0.006-0.527	0.012
Barthel Index (DH)	1.164	1.080-1.255	<0.0001
MMSE (DH)	1.254	1.060-1.484	0.008

DH: during hospitalization; OR: odds ratio; CR: confidence rate; MMSE: standardized mini mental test; PSD: post-stroke dementia.

with PSD are given in Table I. When the sociodemographic characteristics of the patients with and without PSD were compared, it was found that the majority of PSD patients were female and older. There were no statistical differences in the duration of education, SES, or working status among the groups ($P > 0.05$).

As the mean age of the females was higher than that of the males (males: 65.5 ± 5.9 years; females: 68.2 ± 6.0 years; $P = 0.026$) the idea arose that the differences in sex and age between groups were the result of the interaction between these 2 variables. When we compared the patients with and without PSD in terms of age while controlling the other variable in multivariate analysis a statistical difference in sex was not evident (for age $P < 0.001$; for sex $P = 0.171$).

The vascular risk factors for the patients with and without PSD are given in Table II. When the patients with and without PSD were compared according to vascular risk factors, AF and past stroke history were significantly more prevalent in patients with PSD ($P < 0.05$). There were no differences in terms of hypertension, smoking and drinking behavior, DM, past cardiac disease, MI, hypercholesterolemia, or history of TIA between the groups ($P > 0.05$). When the patients with and without PSD were compared according to stroke characteristics there were no differences in terms of stroke type, brain location affected, or size of the lesion ($P > 0.05$) (Table II). CT revealed < 1 lesion in more PSD patients (56.3%, n = 18) than in patients without PSD (27%, n = 20) ($P < 0.05$).

In order to compare the functional, cognitive, and neurological status of the patients the Barthel Index, MMSE or E-MMSE, and NIHSS, respectively, were used (Table III). In the PSD patient assessments, both during hospitalization and 3 months following stroke, results of the 3 scales were significantly different than those of the patients without PSD. While NIHSS scores were significantly higher, the Barthel Index, and MMSE scores were significantly lower. These differences continued in the third month.

Following these analyses a logistic regression analysis was conducted in order to detect variables predictive of PSD development. As having or not having PSD was determined as the dichotomous dependent variable, variables with significant differences in the univariate analyses ($P < 0.05$) (age, sex, AF, past stroke history, number of lesions, and scales applied during hospitalization) were taken as the independent variables. The result of the logistic regression showed that gender, number of past strokes, and NIHSS score during hospitalization did not significantly predict PSD development ($P > 0.05$). It was found that the other 5 independent variables (age, AF, number of lesions, Barthel Index score, and MMSE score during hospitalization) significantly predicted the development of PSD in patients with stroke. The results of the logistic regression are provided in Table IV.

DISCUSSION

In this study, which primarily aimed to determine the frequency of PSD in the early post-stroke period (third month) in the largest hospital in Southeastern Anatolia, 32 patients (30.2%) out of 106 were diagnosed with PSD. The 30.2% frequency rate is close to the upper limit previously reported in the literature (range: 5.9%-32%) (Leys et al., 2005). In a study conducted with 486 cases by Pohjasvaara et al. (1997), the frequency of PSD in the third month after the stroke was 31.8%. In the same study the rate of dementia following a first stroke was 28.9%. The prevalence of PSD according to DSM-IV criteria 3 months after stroke was 27.2% in the Chongqing stroke study conducted in Taiwan (Zhou

et al., 2004) and 22.6% in a study conducted in Poland (Klimkowicz-Mrowiec, 2006).

It was reported that the fatality rate in the post-stroke third month is 8.5 times more in patients with PSD than in those without PSD (Desmond et al., 2002; Barba et al., 2002). The reason for the lower prevalence of PSD in long-term studies (more than 3 months post stroke) was reported to be a higher early death rate in these patients (Leys et al., 2005). On the other hand, in another recent study (Altieri et al., 2004) the prevalence of PSD did not show major changes in the years following stroke; the prevalence of PSD 4 years post stroke was 21.5%.

The other aim of the present study was to determine possible risk factors for the development of PSD. We found that age, AF, and multiple lesions in the brain due to stroke, and the state of cognitive functioning during the outstroke period, based on Barthel Index and MMSE scores, respectively, were risk factors predictive of PSD. In contrast, while there were more female patients and patients with a past history of PSD among the patients with PSD, multivariate analysis showed that these 2 variables were not significant.

Similarly to the present study, findings in the literature point to age as an important factor in the development of PSD (Barba et al., 2000). Age, as for dementia, is an important predictor for PSD development (Censori et al., 1996; Barba et al., 2000; Klimkowicz-Mrowiec, 2006). In the present study the mean age of patients with PSD was significantly higher than that of the patients without PSD (69.9 vs. 65.1 years), and age proved to be a predictive factor for PSD. In a study conducted by Barba et al. (2000), while the mean age of PSD patients was 76 years, the mean age of patients without PSD was 65 years.

Although according to the literature sex is generally reported to have an insignificant relationship to the development of PSD (Mackowiak-Cordoliani et al., 2005), there are reports finding that men have a greater risk of developing PSD development than women (Tatemichi et al., 1992; Skoog 2000). In contrast to the literature, the results of our univariate analysis showed that the prevalence of PSD was higher among women than men (42.5% vs. 22.7%). When the analysis was performed by controlling for age, the difference was not significant. It was concluded that this difference resulted from the higher mean age of the women in our sample. Therefore, it can be concluded that there was no difference in the prevalence of PSD according to sex and this finding is consistent with the literature (Zhou et al., 2004; Klimkowicz-Mrowiec et al., 2006).

In recent years the relationship between dementia and level of education has been investigated in many studies. In general, it has been proposed that a high level of education protects patients from PSD and Alzheimer's' disease, or postpones the course of the current condition (Beard et al., 1992; Bonaiuto et al., 1995; Gorelick, 1997; Stern et al., 1999). Although some studies proposed a relationship between low SES and PSD (Pojhasvaara et al., 1998; Desmond et al., 2000), others do not support such a finding (Tatemichi et al., 1990; Henon et al., 2001). The majority of our sample was uneducated (64.1%), of low SES (71.7%), and unemployed (83%). These characteristics of our sample hindered a direct evaluation of the effects of low SES and education level on the development of PSD, which are reported to be significant predictors (Barba et al., 2000; Zhou et al., 2004).

Like sociodemographic factors, data in the literature regarding the relationship between vascular factors and PSD are contradictory. Some studies have reported relationships between hypertension (Tatemichi et al., 1994; Ott et al., 1998b), DM (Ott et al., 1996), smoking (Ott et al., 1998a), MI (Gorelick et al., 1993), and PSD. A recent study conducted with a large sample (Zhou et al., 2004) reported that all vascular risk factors are more prevalent in patients with PSD. In addition, only DM, AF, past stroke history, and regular alcohol use significantly predicted PSD development. Conversely, Barba et al. (2000) failed to find a relationship between PSD and hypertension, DM, MI, cardiac insufficiency, aorta and mitral valve disease, TIA, or smoking.

The relationship between AF and PSD has been reported to be a significant predictor of PSD (Pojhasvaara et al., 1998; Barba et al., 2000; Tang et al., 2004). It was proposed that AF, resulting in thromboembolism and a reduction in cardiac discharge, reduces blood flow to the heart, and that this can explain the cognitive decline in patients (Gorelick, 1997; Ott et al., 1997). In the Rotterdam study, which was conducted with a very large sample, it was reported that AF is more frequent in patients with dementia than in those without (Ott et al., 1997). In the same study it was stressed that AF increases the risk of decline in cognitive functioning in elderly patients and is an important risk factor for vascular dementia (Ott et al., 1997).

It was thought that the number and size of cerebral infarcts in some specific areas of the brain have an important role in the development of dementia (Tatemichi et al., 1992). It was reported that dementia was found in patients with ischemic necroses > 100 ml and was

not found in patients with ischemic necroses < 100 ml (Tatemichi et al., 1992; Pohjasvaara et al., 1998; Gorelick, 1997). Although there was no relationship between lesion size and PSD, the increase in number of lesions were found to be a predictive factor for PSD.

The fact that the majority of our patients who developed PSD had a past history of stroke could be related to this issue. Past strokes can increase the risk of PSD by increasing the number of lesions. Although past history of strokes was not a significant factor in predicting PSD according to our multivariate statistics, many studies reported that a history of stroke is a significant predictor of PSD (Tatemichi et al., 1994; Zhou et al., 2004). In a 10-year ischemic stroke study, Desmond et al. (1998) reported that the presence of a history of stroke is an important risk factor for dementia. In a study conducted with a general population Kokmen et al. (1996) proposed that a second stroke is an important independent predictor for PSD. These findings suggest that the accumulated effects of cerebrovascular disease on the brain may result in an irreversible process.

Differing from the literature there was no relationship between PSD, and stroke localization, or presence of TIA. Some studies reported a higher frequency of PSD in patients with lesions in the left hemisphere (Desmond et al., 1998; Pohjasvaara et al., 1998; Zhou et al., 2004).

Left hemisphere lesions may result in dementia by affecting language and memory functions. Moreover, lesions in this brain area are thought to be an important risk factor for PSD because they affect general cognitive functioning (Pohjasvaara et al., 1998). On the other hand, Tang et al. (2004) proposed that bilateral lesions are more predictive of PSD than lesions in either side of the brain. They also reported that this finding is not compatible with other literature findings; however, when it is considered that bilateral lesions mean more than one lesion in the brain, the finding is then compatible (Tang et al., 2004). Again, in other studies, (Barba et al., 2000), lesions in the dominant hemisphere are more frequent is patients with PSD in comparison to patients without PSD, and anterior region infarcts and frontal lesions can be independent risk factors.

Similar to the literature, it was found that cognitive, neurological, and functional disorders during hospitalization were more significant in PSD patients and that the difference was still evident in the third month following stroke. Analyses we conducted showed that in particular Barthel Index and MMSE scores during examination can be helpful in predicting the development of PSD. Lin et al. (2003), who reported a similar finding, reported that these kinds of scales can be helpful in taking necessary precautions and predicting PSD, but there is also a need for studies conducted with larger samples.

When evaluating the results of our study, some limitations should be considered. First of all, this study evaluated whether patients develop PSD in the early post-stroke period (third month) or not. With the present results it is not possible to detect the development of PSD after the third post-stroke month.

As there was no specialized stroke unit in our department and as we hypothesized that it would be difficult to follow-up stroke patients for periods beyond the third post-stroke month due to fatalities or problems in sustaining communication, this study was limited to patients after the third post-stroke month. This study would have been more easily employed if there had been a specialized stroke unit in our department.

The stroke histories and past dementia symptoms of our patients were obtained from their medical files, when evident, and from information provided by the patients and relatives. Although we made a careful investigation to detect evidence of dementia before stroke, the lack of an objective assessment of premorbid cognitive functioning is a limitation of this study. Another limitation is that the majority of our sample was composed of low SES patients with no health insurance and low education level. This sociodemographic structure hinders both the generalization of the findings to the general population and evaluating the effects of low SES on the development of PSD is important as it might be a significant contributing risk factor (Leys et al., 2005). It should be remembered that the generally low SES of our sample might have influenced the finding of higher rates of PSD than are present in the general population. In addition, exclusion of patients with aphasia and unconscious patients should be considered as a limitation of the study, as this may have resulted in our finding a lower PSD prevalence than actually existed.

The results of our study support that PSD is a frequently seen complication (30%) in stroke patients. Older age, AF, multiple lesions in the brain, and low cognitive functioning during hospitalization are important risk factors that predicted PSD. When evaluated along with the literature findings it is more correct to consider multiple predictive factors that interact with each other in the development of PSD. Early recognition and treatment of the risk factors will definitely diminish the burden of stroke on society and help in improving patient quality of life.

REFERENCES

Altın Ö, Gelecek N, Akdal G et al., (2006) The effect of unilateral visual neglect on mobility status and quality of life in stroke patients. Nörolojik Bilimler Dergisi, 23:264-271.

Altieri M, Di Pierro V, Pasquini M et al., (2004) Delayed poststroke dementia: a 4-year follow-up study. Neurology, 62:2193-2197.

Bakar M (2003a) Poststrok dementia : Epidemiyoloji ve risk faktörleri. dementia Dergisi, 3:15-20.

Bakar M (2003b) Demans gelişiminde inme risk faktörleri. Demans Dergisi, 3:59-63.

Barba R, Martinez-Espinosa S, Rodriguez-Garcia E et al., (2000) Poststroke dementia clinical features and risk factors. Stroke, 31:1494-1501.

Barba R, Morin MD, Cemillan C et al., (2002) Previous and incident dementia as risk factors for mortality in stroke patients. Stroke, 33:1993-1998.

Beard CM, Kökmen E, Offord KF et al., (1992) Lack of association between Alzheimer's disease and education, occupation, marital status, or living arrangement. Neurology, 42:2063-2068.

Bonaiuto S, Rocca WA, Lippi A et al., (1995) Education and occupation as risk factors for dementia: a population based case control study. Neuroepidemiology, 14:101-109.

Bowler JV, Hachinski V (1994) Progress in the genetics of cerebrovascular disease: Inherited subcortical arteriopathies. Stroke, 25:1696-1698.

Breteler MMB, Bots ML, Ott A et al., (1998) Risk factors for vascular disease and dementia. Haemostasis, 28:167-173.

Brott T, Adams HP, Olinger CP et al., (1989) Measurements of acute cerebral infarction: a clinical examination scale. Stroke, 20:864–870.

Censori B, Manara O, Agostinis C et al., (1996) Dementia after first stroke. Stroke, 27:1205-1210.

Desmond DW, Moroney JT, Paik MC et al., (2000) Frequency and clinical determinants of dementia after ischemic stroke. Neurology, 54:1124-1131.

Desmond DW, Moroney JT, Sano M et al., (2002) Mortality in patients with dementia after ischemic stroke. Neurology, 59: 537-543.

Ertan T, Eker E, Güngen C et al., (1999) The Standardized Mini Mental State Examination for illiterate Turkish elderly population. 2nd International Symposium on Neurophysiological and Neuropsychological Assessment of Mental and Behavioral Disorders, 28-30 Ağustos 1999; Bursa.

Folstein MF, Folstein SE, McHugh PR (1975) "Mini-Mental State": a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res, 12: 189-198.

Gorelick PB (1997) Status of risk factors for dementia associated with stroke. Stroke, 28:459-463.

Gorelick PB, Brody J, Cohen D et al., (1993) Risk factors for dementia associated with multiple cerebral infarcts: A case control analysis in predominantly African-American hospital-based patients. Arch Neurol, 50:714-720.

Güngen C, Ertan T, Eker E et al., (2002) Standardize Mini Mental Test'in Türk toplumunda hafif dementia tanısında geçerlik ve güvenilirliği. Türk Psikiyatri Derg, 13:273-281.

Hatano S (1976) Experience from a multi center stroke register: a preliminary report. Bull World Health Organ, 54: 541-553.

Henon H, Godefroy O, Leys D et al., (1995) Early predictors of death and disability after acute cerebral ischemic event. Stroke, 26:392-398.

Henon H, Pasquier F, Duricu I et al., (1997) Preexisting dementia in stroke patients. Baseline frequency, associated factors and outcome. Stroke, 28:2429-2436

Henon H, Durieu I, Guerouaou D et al., (2001) Poststroke dementia: Incidence and relationship to prestroke cogntive decline. Neurology, 57:1216-1222.

Hofman A, Ott A, Breteler MMB et al., (1997) Atherosclerosis, apolipoprotein E, and the prevalance of dementia and Alzheimer's disease in the Rotterdam study. Lancet, 349:151-154.

Klimkowicz-Mrowiec A, Dziedzic T, Slowik A et al., (2006) Predictors of poststroke dementia: Results of a hospital-based study in Poland. Dement Geriatr Cogn Disord, 21:328-334.

Kooten F, Koudstaal PJ (1998) Epidemiology of post stroke dementia. Haemostasis, 28:124-133.

Kökmen E, Whisnant JP, O'Fallon WM et al., (1996) Dementia after ischemic stroke. A population based study in Rochester Minnesota (1960-1984). Neurology, 9:154-159.

Leys D, Henon H, Mackowiak-Cordoliani MA et al., (2005) Poststroke dementia. Lancet Neurol 4:752-759.

Lin JH, Lin RT, Tai CT et al., (2003) Prediction of poststroke dementia. Neurology, 61: 343-348.

Mackowiak-Cordoliani MA, Bombois S, Memin A e ark. (2005) Poststroke dementia in the elderly. Drugs Aging 22:483-493.

Mahoney FI, Barthel DW (1965) Functional evaluation: the Barthel index. Maryland State Med J, 14:61-65.

Moroney JT, Bagiella E, Desmond DW et al., (1996) Risk factors for incident dementia after stroke: Role of hypoxic and ischemic disorders. Stroke, 27:1283-1289.

Ott A, Breteler MMB, De Bruyne MC et al., (1997) Atrial fibrillation and dementia in a population based study: The Rotterdam study. Stroke, 28:316-321.

Ott A, Breteler MMB, Van Harskamp F et al., (1998a) The incidence and risk of dementia: The Rotterdam study. Am J Epidemiol, 147:574-580.

Ott A, Slooter AJC, Hofman A et al., (1998b) Smoking and the risk of dementia and Alzheimer disease in a population based cohort study. The Rotterdam study. Lancet, 351:1840-1843

Ott A, Stolk RR, Hofman A et al., (1996) Association of diabetes mellitus and dementia. The Rotterdam study. Diabetologia, 39:1392-1397.

Pohjasvaara T, Erkinjutti T, Vataja R et al., (1997) Dementia three months after stroke. Baseline freqency and efect of different definitions of Dementia in the Helsinki Stroke Aging Memory Study (SAM) Cohort. Stroke, 28:785-792.

Pohjasvaara T, Erkinjutti T, Ylikoski MA et al., (1998) Clinical determinants of poststroke dementia. Stroke, 29:75-81.

Skoog I (2000) Risk factors for dementia after stroke. Fifth Annual Advances in stroke symposium. Proceedings Highlights. Bermuda, 27-28.

SPSS (1999) Statistical Package for Social Sciences (computer program). Windows version, release 10.0 Chicago, IL, ABD.

Stern Y, Albert S, Tang MX et al., (1999) Rate of memory decline in AD is related to education and occupation: cognitive reserve? Neurology, 53:1942-1947.

Tang WK, Chan SS, Chiu HF et al., (2004) Frequency and determinants of poststroke dementia in a Chinese cohort. J Neurol, 251: 604-608.

Tatemichi TK, Desmond Dw, Mayeux R et al., (1992) Dementia after stroke: Baseline frequency, risks, and clinical features in a hospitalised cohort. Neurology, 42:1185-1193.

Tatemichi TK, Foulkes MA, Mohr JP et al., (1990) Dementia in stroke survivors in the stroke data bank cohort. Prevalence, incidence, risk factors and computed tomographic findings. Stroke, 21:858-866.

Tatemichi TK, Paik M, Bagiella E et al., (1994) Risk of dementia after stroke in hospitalized cohort: Result of longitudinal study. Neurology, 44:1885-1891.

Zhou DH, Wang JY, Li J et al., (2004) Study on frequency and predictors of dementia after ischemic stroke: the Chongqing stroke study. J Neurol, 251:421-427.