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Lacunar infarction in the very old

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The characteristics of lacunar infarction in patients aged 80 years and more (very old patients, VOP) are poorly studied, and it is difficult to define the mechanism of stroke in VOP.

Purpose. To evaluate whether risk factors, coexisting causes of stroke, clinical features in VOP with first lacunar infarction differ from those in patients aged 45–79 years (younger patients, YP).

Methods. We studied 102 VOP with first lacunar infarction and compared them with 612 YP.

Results. VOP with presumed small vessel disease infarction had a lower frequency of male sex, hypercholesterolemia, smoking, and family history of stroke or heart disease than YP, independently of coexisting sources of embolism. While a potential cardiac source of embolism was more frequent in VOP than in YP (29.4% vs. 11.4%, $p < 0.001$), the frequency of concomitant large vessel disease did not differ between the two groups (7.2% vs. 5.9%, $p > 0.05$). <50% ICA (internal carotid artery) stenosis was an independent predictor for "pure" small vessel disease infarction in both age groups. The association between diabetes and "pure" small vessel disease, seen in YP, failed in the VOP group. The VOP group with "pure" small vessel disease showed a higher frequency of immediately stabilized neurologic deficit than did the YP group (80.3% vs. 68.4%, $p = 0.047$).

Conclusions. The age-related risk factors show that small vessel disease infarction in VOP more frequently shares the features that are common for large vessel disease than in YP. The clinical picture suggests a higher frequency of small vessel disease in VOP compared to YP with small vessel disease and coexisting cardioembolic source.

Key words: lacunar infarction, elderly, atherosclerosis, cardioembolic stroke

INTRODUCTION

Lacunar infarcts in VOP represent a stroke subgroup with controversial risk factors and stroke mechanism. Stroke risk factors, etiology, clinical features in VOP are poorly studied. It is unclear whether VOP with lacunar infarction makes a specific subgroup which differs from the patients aged 45–79 years (younger patients, YP).

Lacunes are caused by several arteriopathies, mainly microatheroma and lipohyalinosis (1–4). The exact relationships between microatheroma and lipohyalinosis and between small vessel and large vessel disease remain uncertain. Moreover, there is an age-related increase in the number of patients with concomitant cardioembolic sources, mainly atrial fibrillation, and defining the etiology of infarction becomes more difficult (5–8). The importance of analyzing small vessel disease in the elderly lies in the nature of the clinical syndromes they produce and the special problems that arise in the diagnosis and defining the etiology of stroke and in the use of different treatments, which depend upon stroke etiology.

We compared 117 VOP with lacunar infarction with 770 YP. Also, association with coexisting potential cardioembolic sources and large vessel disease was analysed.

MATERIALS AND METHODS

We studied 714 patients with first-ever lacunar infarction. A comparison was made between 102 VOP and 612 YP.

All patients were assessed following the standard protocol of the prospective Stroke Registry which includes neurological examination, brain computed tomography (CT), magnetic resonance imaging (MRI), extracranial Doppler ultrasonography, ECG, and standard blood tests. M-mode and B-mode echocardiography were carried out systematically in all patients with an evidence of heart disease.

We evaluated risk factors and vascular concomitants, including hypertension, diabetes mellitus, cigarette smoking, hypercholesterolemia, elevated venous hematocrit, ischemic heart disease, arrhythmia or peripheral vascular disease, and a family history of stroke or heart disease. The characteristics of stroke onset, general

clinical findings, transient ischemic attacks (TIAs), functional disability, and the type and cause of the stroke were evaluated following the protocol of the Stroke Registry. Infarct topography was based on CT or magnetic resonance imaging (MRI).

Infarcts were classified into different groups of presumed etiology according to the results of the investigation.

Presumed "pure" small vessel disease was defined for patients with hypertension or diabetes mellitus and <1.5 cm infarct in the territory of the deep perforators, in the absence of any other etiology.

More than one potential stroke etiology was retained in patients with presumed small vessel disease and associated potential cardioembolic sources or associated large vessel disease as alternative potential causes of stroke.

Atherosclerosis with stenosis (large artery disease). Narrowing of ≥50% of the lumen diameter of the corresponding extracranial artery or large intracranial artery (middle cerebral artery (MCA), posterior cerebral artery (PCA), or basilar artery (BA)), in the absence of any other etiology.

Patients with a potential cardiac source of embolism were divided into two groups, one with isolated cardioembolic stroke in the absence of any other etiology, and one with more than one potential stroke etiology.

A potential cardiac source of embolism: left ventricular akinetic segment (as a sequela of myocardial infarction), acute (<3 months) myocardial infarct, global ventricular hypokinesia, left ventricular or atrial thrombus, left atrial myxoma or other cardiac tumor, patent foramen ovale with a peripheral venous source of embolism; rheumatic heart disease, endocarditis, mitral valve prolapse, prosthetic aortic or mitral valves; atrial fibrillation and sick sinus syndrome.

Classic lacunar syndromes included pure motor hemiparesis, pure sensory stroke, pure sensorimotor stroke, dysarthria-clumsy hand syndrome, and ataxic hemiparesis (9). Pure motor hemiparesis was defined as isolated motor hemiparesis with involvement of at least two of three areas (face/arm/leg). Pure sensory stroke and pure sensorimotor stroke were defined in a similar way. Dysarthria-clumsy hand syndrome was defined as the presence of moderate to severe dysarthria, ipsilateral ataxia, mild (if any) motor weakness, or increased tendon reflexes (Babinski sign) in the absence of sensory deficit. Ataxic hemiparesis was defined as moderate hemiparesis with ataxia.

We studied 714 patients with first-ever lacunar infarction. A comparison between 102 VOP and 612 YP first-ever with lacunar infarction was made. The group with "pure" small vessel disease was analysed separately from those with associated potential cardioembolic sources or large vessel disease as alternative potential causes of stroke. We compared infarction territory, risk factors, vascular investigations, and clinical characteristics in VOP and YP with "pure" small vessel disease. We analysed risk factors and concomitants predicting small vessel disease infarction in the VOP and YP.

To evaluate the role of cardioembolism in VOP with small vessel disease and concomitant potential cardioembolic sources, a comparison was made between patients with ''pure" small vessel disease and those with small vessel disease and coexisting potential cardioembolic sources.

Statistical analysis. For statistical analysis, Fisher's exact test and the χ^2 test were used. Differences were regarded as statistically significant when the p value was less than 0.05; NS in the text denotes a p value >0.05. Multivariate regression analysis was used to determine predictors of small vessel disease stroke in both age groups. Hypertension was excluded from the model because of the large number of patients with hypertension in the small vessel disease group, which could potentially mask information about other risk factors. In the multivariate logistic regression analysis, factors with $p < 0.05$ were considered as independent predictors.

RESULTS

Lacunar infarction occurred in 612 patients in the YP group and 102 in the VOP group. Neuroimaging (CT/MRI) was normal in 26.1% of YP and 23.5% of VOP, NS. The proportion of patients with normal MRI did not differ significantly between the two age groups and was 11.8% of the 110 YP examined by MRI and 5% of the 20 VOP examined by MRI.

The proportion of patients with "pure" small vessel disease was comparable among two age groups (496 (17.2%) in YP and 66 (14.6%) in VOP, NS). In patients with lacunar infarction, a coexisting potential cardiac source of embolism was more frequent in VOP than in YP (29.4% vs. 11.8%, $p < 0.001$), while the proportion of patients with concomitant large vessel disease did not differ between the two age groups (Fig. 1). The main cardiac source of embolism in the VOP group

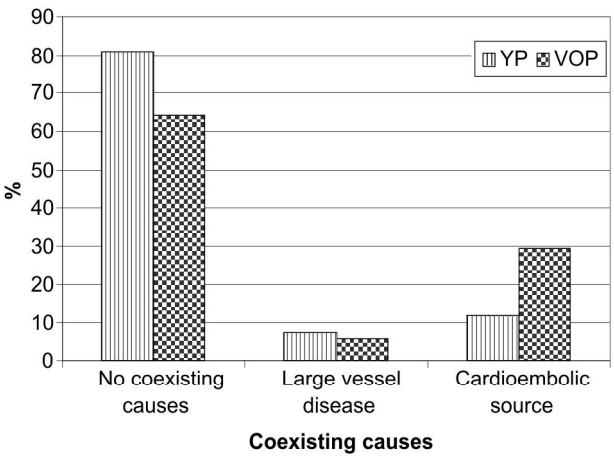

Fig. 1. Coexisting causes of lacunar infarction

was atrial fibrillation (found, respectively, in 20.6% of VOP and 4.1% of YP with lacunar infarction, $p < 0.001$). The proportion of other potential sources of cardioembolism did not differ between the two age groups (10.8% in VOP and 8.7% in YP).

Characteristic of "pure" small vessel disease infarction

Topography of infarcts (Table 1). Neuroimaging (CT/MRI) was normal in 26.7% of all patients, a similar result being seen in the two age groups. MRI showed areas of infarctions in all 12 VOP cases examined, but was negative in 11 of the 83 YP (13.3%) patients examined ($p > 0.05$). Bilateral stroke was more common in VOP than in YP (18.2% and 9.1%, respectively, $p = 0.021$). The localization of infarcts did not differ significantly between the two age groups.

Risk factors and medical history. Table 2 summarizes the main risk factors and previous medical histories for both age groups. There was a statistically significant male predominance in the YP group compared to the VOP group (57.7% and 40.9%, respectively, $p < 0.01$).

Hypercholesterolemia, smoking, elevated hematocrit, and a family history of heart disease and stroke were less frequent in VOP than in YP.

A previous TIA was noted at approximately the same frequency in both groups; however, the proportion of contralateral TIA was greater in the VOP group (75% versus to 27.8% in the YP group, $p < 0.001$). In patients with previous TIAs, the higher frequency of contralateral TIA in the VOP group as compared to YP correlated with a higher frequency of bilateral <50% ICA stenosis in VOP vs. YP (87.5% vs. 47.8%, $p = 0.031$).

Although the frequency of <50% ICA stenosis was greater in VOP with first stroke than in YP (38.9% vs. 26.9%, $p < 0.001$), the higher proportion of <50% ICA stenosis among VOP vs. YP with small vessel disease did not reach statistical significance.

Table 1. **Results of neuroimaging about the territory of infarct**

	45–79 years (n = 496) (%)	≥ 80 years (n = 66) (%)	p value
Subcortical:			
Single internal capsular or striatum	62.9	60.6	NS
Single thalamus	7.9	10.6	NS
Multiple	11.7	13.6	NS
Brainstem	17.5	15.2	NS
CT and/or MRI negative	26.8	25.8	NS
Side:			
left	36.1	27.3	NS
right	28.0	28.8	NS
bilateral	9.1	18.2	0.021

Risk factors for infarction due to small vessel disease. After exclusion of hypertension, logistic regression analysis (Table 3) revealed that <50% ICA stenosis was an independent predictor for small vessel disease infarction in both age groups. The association between diabetes and small vessel disease (OR 1.4, $p < 0.005$) seen in YP failed in the VOP group.

Clinical features. The clinical features are presented in Table 4. The VOP group showed a higher frequency of immediately stabilized neurologic deficit than the YP group (80.3% vs. 68.4%, $p = 0.047$). The frequency of immediately stabilized neurologic deficit did not differ significantly between VOP with "pure" small vessel disease and other stroke etiologies (80.3% vs. 82.3%). However, in VOP patients with multiple lesions, an immediately stabilized neurologic deficit was found in only 33.3% (3/9) of patients versus 82.4% of patients with infarcts due to another etiology ($p < 0.01$).

Pure motor hemiparesis was present in more than 40% of patients. Face, arm, and leg involvement on one side was the most common type of weakness distribution and was recorded in 27.8% of YP and 24.2% of VOP. Isolated monoparesis was found at a significantly higher frequency in the VOP group (6.1% vs. 1.6%, $p < 0.05$). Moreover, in the VOP group, isolated monoparesis was more common in the small vessel disease group than

Table 2. **Main risk factors and previous medical history in patients with "pure" small vessel disease**

Age	45–79 years (n = 496) (%)	≥80 years (n = 66) (%)	p value
Sex (male)	57.7	40.9	<0.01
Hypertension	96	97	NS
Cardiac history:	11.5	15.2	NS
Angina pectoris without myocardial infarction	7.7	13.6	NS
Previous myocardial infarction (>3 months)	3.2	1.5	NS
Diabetes mellitus/hyperglycaemia	25.4	18.2	NS
Hypercholesterolemia	29.8	16.7	0.026
Smoking	33.5	13.6	0.001
current	25.2	6.1	0.001
former	8.3	7.6	NS
Migraine	1.6	0	NS
Claudication	4	1.5	NS
Hematocrit > 45	19.2	7.6	0.021
Family history of stroke or heart disease	9.3	1.5	0.032
Previous TIA	18.2	12.1	NS
ipsilateral	13.1	3	0.018
other territory	5	9.1	NS
<50% ICA* stenosis	46.4	54.6	NS

* Internal carotid artery.

Table 3. **Results of multivariate logistic regression analysis for "pure" small vessel disease infarction prediction in VOP and YP**

	45–79 years (n = 2889) Odds ratio and 95% confidence interval (CI)	p value	≥80 years (n = 452) Odds ratio and 95% confidence interval (CI)	p value
Sex (female)	1.3 95%CI(1–1.6)	0.012	1.2 95%CI(0.7–2.2)	NS
Diabetes mellitus/ hyperglycaemia	1.4 95%CI(1.1–1.8)	<0.005	0.9 95%CI(0.5–1.8)	NS
Hypercholesterolemia	1.2 95%CI(1–1.5)	NS	1 95%CI(0.5–2.2)	NS
Smoking	0.6 95%CI(0.5–0.8)	<0.001	1 95%CI(0.4–2.2)	NS
Claudication	0.6 95%CI(0.4–0.9)	0.022	0.3 95%CI(0.04–2.4)	NS
Hematocrit > 45	1.1 95%CI(0.9–1.5)	NS	0.6 95%CI(0.2–1.4)	NS
Family history of stroke or heart disease	1.1 95%CI(0.8–1.4)	NS	1.1 95%CI(0.11–10)	NS
Previous ipsilateral TIA	0.8 95%CI(0.6–1.1)	NS	0.18 95%CI(0.04–0.8)	0.021
<50% ICA stenosis	2.7 95%CI(2.1–3.3)	<0.001	1.9 95%CI(1.1–3.2)	0.022

Table 4. **Clinical features in patients with "pure" small vessel disease**

	45–79 years (n = 496) (%)	≥80 years (n = 66) (%)	p value
Abrupt nonprogressive onset	68.4	80.3	0.047
Reduction of consciousness:	4.4	6	NS
Clinical syndromes:			
Pure motor stroke	40.3	43.9	NS
Restricted hemiparesis	12.5	19.7	NS
One area involved	3.4	9.1	0.029
Face	1.8	3	NS
Monoparesis	1.6	6.1	0.02
Two areas involved	9.1	10.6	NS
Face-arm	5.4	7.6	NS
Arm-leg	1	3	NS
Face-arm-leg	27.8	24.2	NS
Pure sensory stroke	3.6	3	NS
Incomplete	0.8	0	NS
Face-arm-leg	2.8	3	NS
Sensorimotor stroke	15.3	12.1	NS
Incomplete	3	1.5	NS
Face-arm-leg	12.3	10.6	NS
Dysarthria-clumsy hand	2	0	NS
Ataxic hemiparesis	3.6	6.1	NS
Lacunar syndromes	60.9	56.1	NS
Hemianopia	3	0	NS
Aphasia	8.1	7.6	NS
Cephalgia	7.7	4.6	NS
Activity of daily living at 1 month:			
No disability	22.8	27.3	NS
Mild disability	48.6	40.9	NS
Moderate disability	24.8	25.8	NS
Severe disability	3.2	6.1	NS
Death	0.6	0	NS

in the another stroke etiology group (6.1% and 1.8%, $p < 0.05$), while in the YP group it occurred at a similar frequency in patients with infarction due to small vessel disease or another etiology (1.6% and 2.4%, NS). In all four VOP patients with monoparesis, CT was normal, while in 2/2 patients with isolated face involvement CT showed lesions in the subcortical area. In the 8 YP patients with isolated monoparesis, CT/MRI was negative in 4 and positive in 4 (single subcortical lacunes). In 7 of the 9 YP patients with face involvement, CT was positive and showed lesions in the subcortical area (3 cases) or brainstem (1 case), and multiple lesions in the deep territory (3 cases), whereas in 2 patients CT was negative.

The outcome of stroke did not differ significantly between the two age groups (Table 4).

Role of coexisting arterial or cardiac sources of embolism in VOP

Single and multiple infarction. Multiple old deep lesions were not seen in patients with coexisting arterial or cardiac source of embolism, but were seen in 13.6% of patients with "pure" small vessel disease. Brainstem lesion was found in 13.3% of patients with more than one stroke etiology (vs. in 15.2% of patients with "pure" small vessel disease).

Risk factors. The main differences (Table 5) are associated with more pronounced risk factors related to atherosclerotic disease in patients with more than one stroke etiology. Coexisting peripheral and coronary artery disease was found more frequently in patients with more than one stroke etiology than in those with "pure" small vessel disease, particularly in patients with coexisting potential cardiac source of embolism the respective figures being 13.3% and 1.5% ($p = 0.032$) for peripheral artery disease and 16.7% and 1.5% ($p = 0.011$) for myocardial infarction.

The coexisting arterial or cardiac sources of embolism do not change the difference between VOP and YP risk factor profiles mainly associated with atherosclerosis. We found that VOP with small vessel disease had a lower frequency of male sex (39.7% vs. 61.8%, $p < 0.001$), hypercholesterolemia (21.6% vs. 31.4%, $p = 0.045$), smoking (12.8% vs. 35.1%, $p < 0.01$) and family history of heart disease and stroke (2% vs. 9.8%, $p < 0.005$) than YP, although the difference between the proportion of patients with elevated hematocrit did not reach statistical significance (12.8% vs. 20.4%, NS).

Table 5. **Comparison of risk factors in VOP with "pure" small vessel disease and in VOP with more than one stroke etiology**

	Pure small vessel disease (n = 66) (%)	More than one stroke etiology (n = 36) (%)	p value
Sex (male)	40.9	33.3	NS
Hypertension	97	100	NS
Diabetes mellitus/hyperglycaemia	18.2	27.8	NS
Hypercholesterolemia	16.7	30.6	NS
Smoking	13.6	13.6	NS
Claudication	1.5	11.1	0.036
Hematocrit >45	7.6	22.2	0.03
Family history of stroke or heart disease	1.5	2.8	NS
Myocardial infarction	1.5	16.7	<0.005
Previous TIA	12.1	27.8	0.047
<50% ICA stenosis	54.5	47.2	NS

Clinical importance of a potential cardiac source of embolism. The main findings are shown in Table 6. There was a trend for previous TIAs to be more frequent in patients with small vessel disease associated with cardioembolic source than in those with "pure" small vessel disease. There was also a trend for the duration of TIAs to be shorter (61 ± 14.4 minutes vs. 84.3 ± 10.4 minutes), the number of TIAs to be greater (2.1 ± 0.7 vs. 1.4 ± 0.2), and the period between TIA and stroke to be shorter (49.1 ± 18 weeks vs. 60.1 ± 15.6 weeks) in patients with coexisting cardioembolic source, although it did not reach statistical significance. The type of stroke onset did not differ significantly between patients with and without a coexisting cardioembolic source. In contrast, cortical signs, particularly hemianopia, were more frequent in patients with a potential cardioembolic source than in those with "pure" small vessel disease ($p < 0.05$).

In YP with a coexisting cardioembolic source, there was a higher frequency of patients with non-progressive stroke (81.9% and 68.4%, respectively, $p = 0.019$) and cortical signs (33.3% and 16.7%, $p < 0.005$), and a trend to a lower frequency of patients with previous TIA (13.9% and 18.2%, NS).

In YP, the proportion of patients with either features of nonprogressive onset, cortical signs or multiple

Table 6. **Clinical features in VOP with "pure" small vessel disease and in VOP with a concomitant potential cardioembolic source**

	Pure small vessel disease (n = 66) (%)	Small vessel disease+potential cardioembolic source (n = 30)	p value
Previous TIA	12.1	23.3	NS
Abrupt nonprogressive onset	80.3	70	NS
Three areas involved:			
Pure motor hemiparesis	24.2	16.7	NS
Pure sensory stroke	3	0	NS
Pure sensorimotor stroke	10.6	13.3	NS
Ataxic hemiparesis	6.1	3.3	NS
Dysarthria clumsy hand	0	0	NS
Restricted pure hemiparesis:	19.7	10	NS
one area	9.1	3.3	NS
two areas	10.6	6.7	NS
Cortical signs:	15.2	40	0.017
aphasia	7.6	16.7	NS
apraxia	10.3	13.3	NS
agnosia	9.1	16.7	NS
hemianopia	0	16.7	0.002
Outcome			
no disability or mild disability	68.2	50	NS
moderate to severe disability	31.8	46.7	NS
death	0	3.3	NS

lesions on CT/MRI was higher at a coexisting potential cardioembolic source than in with "pure" small vessel disease (87.7% vs. 74.8%, $p < 0.05$), which was not the case in VOP (76.7% vs. 90.6%, NS).

DISCUSSION

We found a more frequent coexistence of small vessel disease and potential cardioembolic source in VOP vs. YP due to the higher frequency of atrial fibrillation in the VOP vs. YP. The similar results in the elderly are recorded in (8, 10). It is a not specific feature of lacunar stroke and is associated with the age-related increase of stroke patients with a potential cardioembolic source (11, 12) Moreover, these results are supported by the population-based studies showing an increasing frequency of atrial fibrillation with age (13–15). The proportion of patients with coexisting large vessel disease was equally frequent in both age groups and was in agreement with the comparable proportion of patients with >50% ICA stenosis among stroke patients with hypertension. Although the higher proportion of patients with coexisting potential cardioembolic sources reduces the relative proportion of "pure" small vessel disease, the proportion of patients with "pure" small vessel disease is comparable among the two age groups with first infarcts.

We found that VOP with small vessel disease had a lower frequency of male sex, hypercholesterolemia, smoking and family history of heart disease and stroke than YP, independently of coexisting arterial or cardiac sources of embolism. The lower frequency of these factors in VOP can, in part, be explained by premature death or earlier first stroke in patients with more risk factors. The lower frequency of male sex and hypercholesterolemia has been discussed also in (10).

Diabetes mellitus significantly differentiated small vessel disease infarction from other infarct subtypes in the YP group and did not play a significant role in VOP. Patients with diabetes are at a higher risk of macro- and microangiopathy and thereby of stroke and myocardial infarction. Diabetic stroke patients usually are younger than nondiabetic (16, 17). Diabetes worsens the stroke outcome independently on stroke subtype (17, 18). Diabetes associated with hypertension is a major but maybe not an independent risk for lacunar infarction (9, 16, 19). The premature death of patients with diabetes and earlier first stroke associated with diabetes decreases with age the proportion of patients with first stroke secondary to diabetes. Moreover, with age, the increasing proportion of patients with more than one cause of stroke and thereby the coexistence of diabetes mellitus with other risk factors in the elderly mask the role of diabetes as an independent predictor for lacunar stroke. The results of previous reports (9) that diabetes was a significant predictor of lacunar infarctions in the elderly don't contradict our results because part of those patients had experienced a previous stroke in the younger age while we analysed only patients with the first stroke. The association between diabetes and multiple deep (but not single) lacunes which may be caused by recurrent stroke (20) supported a possible higher interrelation between diabetes and stroke in general (which may also be recurrent) than between diabetes and the first stroke.

In both our VOP and YP groups, <50% stenosis of the ICA was an independent predictor of small vessel disease infarction, which is in agreement with reports (10, 21, 22–26). It is possible that ICA atherosclerosis and occlusion of intracerebral small vessels in many hypertension patients is the same process and many first strokes do not occur while the process is limited to the large vessels before intracerebral small vessels are involved, i. e. until atherosclerosis spreads more distally in the arterial system, resulting in a stenosis or occlusion of small vessels with first-ever stroke independent on age.

In patients with small vessel disease and a potential cardioembolic source, the clinical picture suggests a higher frequency of small vessel disease in VOP than in YP. The similar frequency of previous TIA in VOP with "pure" small vessel disease or with small vessel disease and potential cardioembolic sources and the lack of multiple infarctions, which are more common in patients with cardioembolic stroke (27, 28, 29), seems to favour small vessel disease infarction rather than cardioembolism (1–3). Moreover, the proportion of patients with atypical clinical features, such as nonprogressive onset and incomplete pure hemiparesis, did not differ between VOP with "pure" small vessel disease or with concomitant potential cardioembolic sources, suggesting that a concomitant cardiac source of embolism does not change the etiology of stroke. However, in some patients, a higher frequency of cortical signs favours cardioembolism (1–3) as a potential cause of infarct. On the other hand, the proportion of patients with at least one of the features of nonprogressive onset, cortical signs, and multiple lesions on CT/MRI, common to cardioembolism, was greater in YP with concomitant cardiac source of embolism than in YP with "pure" small vessel disease ($p < 0.05$), while, in VOP, these signs were equally frequent in the two groups, suggesting that small vessel disease is a potential cause of stroke in VOP with a concomitant potential cardioembolic source, whereas cardioembolism is a more likely stroke mechanism in YP with a concomitant potential cardioembolic source than in YP.

CONCLUSIONS

The age-related risk factor profile implies that small vessel disease infarction in VOP more frequently shares the features which are common for large vessel disease than in YP. The clinical picture suggests a higher

frequency of small vessel disease in VOP compared to YP with small vessel disease and a coexisting cardioembolic source.

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LABAI SENŲ PACIENTŲ LAKŪNINIAI INSULTAI

S a n t r a u k a

Vyresnių negu 80 metų (LSP) pacientų lakūninio insulto ypatybės blogai ištirtos, o šio insulto mechanizmas dažnai yra neaiškus. Šio darbo tikslas – palyginti LSP lakūninio insulto rizikos veiksnius, gretutines insulto priežastis, klinikines ypatybes su jaunesniais pacientais (JP).

Medžiaga ir metodai. Palygintos 102 LSP ir 612 JP, pirmą kartą persirgusių insultu, charakteristikos.

Rezultatai. Lakūniniu insultu persirgę LSP rečiau ($p < 0,05$) rūkė, rečiau turėjo padidintą cholesterolio kiekį, šeimyninę insulto ar infarkto anamnezę, tarp jų, priešingai negu JP, mažiau buvo vyrų. Galima gretutinė kardioembolinė insulto priežastis dažnesnė LSP negu JP atveju (29,4% ir 11,4%, $p < 0,01$), o esanti stambių arterijų patologija nesiskyrė abiejose grupėse (7,2% LSP ir 5,9% JP, $p > 0,05$). Tik JP cukrinis diabetas buvo patikimas lakūninio insulto prognostinis veiksnys. <50% atvejais vidinės miego arterijos susiaurėjimas buvo nepriklausomas lakūninio insulto prognostinis veiksnys abiejose amžiaus grupėse ($p < 0,05$). Nors kardioembolija galėjo būti abiejų amžiaus grupių ligonių, turinčių galimą kardioembolinį šaltinį, insulto priežastimi, LSP klinika dažniau negu JP buvo būdingesnė smulkių kraujagyslių ligai, o ne kardioemboliniam insultui.

Išvada. LSP lakūninių insultų rizikos veiksniai, esanti patologija skyrėsi nuo JP. LSP lakūniniai insultai dažniau negu JP pasižymėjo stambiųjų kraujagyslių ligai būdingomis savybėmis.

Raktažodžiai: lakūniniai insultai, kardioembolija, seni pacientai, aterosklerozė