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ORIGINAL ARTICLE

Demographic Datas in Lenticulostriate Artery (LSA) Infarcts

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Abstract

Introduction: Lenticulostriate arteries (LSA) are main truncal branches of middle cerebral artery. Their number changes between 6 to 12 and they supply n.lentiformis, external part of n.caudatus, anterior and dorsal parts of internal capsule and limited parts of globus pallidus. LSA infarcts constitute 1-2% of all cerebral ischemic accidents.

Methods: In the present study we investigated the demographic data and etiologic risk factors of 143 patients (68 males, 75 females) who were diagnosed with the LSA infarction and treated in our inpatient clinic between the years 2013-2018.

Results: There was no difference between etiology of stroke and prognosis (p=0.206). A correlation was found between smoking and early-term prognosis (p<0.001 rho: 0.458). Univariate analysis showed that there was no correlation between the patients with poor functional outcome and their age, sex, hypertension, diabetes mellitus, coronary artery disease or hypercholesterolemia. The patients with poor functional outcome had more commonly current smoking history, higher initial NIHSS score and higher mRS scores.

Discussion and Conclusion: LSA infarcts have been seen relatively less common compared to other vascular territory infarcts and etiologically related to atherotrombosis and cardioembolism. In this study we showed a similar distribution of risk factors with the literature reports by evaluating the demographic data of LSA infarct patients.

Keywords: Atherothrombotic; cardioembolic; LSA infarcts.

LSA infarct strokes constitute 1-2% of all ischaemic strokes and the diameter of the infarcts change between 2 to 7cm[1]. Clinically they present contralateral hemiparesis with equally affected upper and lower extremities and sometimes aphasia and anosognosia[2,3].

Striatocapsular infarctions are caused by the simultaneous occlusion of more than one orifice among the immediately adjacent small, long, lenticulostriate arteries. In contrast to the original concept that striatocapsular infarctions occur nearly exclusively as a result of embolic occlusion of the proximal middle cerebral artery (MCA)[4-6], a significant proportion of this type of infarction also occurs as a result of in situ thrombosis of the MCA. According to the previous reports[1-3,7], the most frequent causes of striatocapsular infarcts are artery-to-artery embolism from the internal carotid artery (ICA) and cardiogenic embolism, as well as atherosclerotic disease in the MCA at the origin of the lenticu-lostriate arteries.

The identification of stroke subtype is valuable for both the practicing clinicians and an optimal design of clinical stroke trials because the aetiology of ischaemic stroke affects patient management, outcome and prognosis[8,9]. In addition, the treatment of intracranial atherosclerotic disease may be different from that of extracranial ICA disease[10] as

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well as cardiogenic embolism. However, the TOAST (Trial of Org 10172 in Acute Stroke Treatment) system, which is the most widely used stroke classi-fication, is insufficient for differentiating between in situ thrombotic striatocapsular infarction or car-dioembolism, although the system is highly accepted among the physicians[10-12]. In contrast, small, deep infarctions of the lenticulostriate territory have been found in patients with atherosclerotic MCA disease[13-15] as well as those with extracranial embolic sources[16,17]. There has been no clear description about the relationship between lesion patterns of acute striatocapsular infarcts and stroke mechanisms.

In the present study, we investigated the demographic data and aetiologic subtypes of LSA infarcts in acute ischaemic stroke (AIS) patients.

Materials and Methods

In this study we investigated the demographic data and etiologic risk factors of 143 patients (68 males, 75 females) retrospectively who were diagnosed with LSA infarction and treated in our inpatient clinic between the years 2013-2018.

The classification of subtypes of stroke was determined based on the TOAST system. This study in-cluded patients with cardioembolism, large artery atherosclerosis, or stroke of undetermined aetiology due to two or more identified causes or due to negative evaluation. Significant stenosis of ICA was defined as stenosis >50% or occlusion of the artery relevant to the infarction. Infarct locations were deter-mined by using MRI scan. Stroke severity of patients were classified according to National Institute of Health Stroke Scale (NIHSS) as mild (<8), moderate (8-14) and severe (>14). Prognosis of pa-tients was classified according to modified Rankin Scale (mRS) as good prognosis (mRS:0-2) and bad prognosis (mRS:3-6).

Results

In this study, we wanted to share the demographic data and detectable aetiologic risk factors of 143 patients (68 M, 75 F) who were diagnosed with LSA infarction in our clinic between 2013 and 2018. The mean age of the patients was 69.5±13.5 (M: 65.36±12.7, F: 73.32±13.23) and 20 patients were under 55 years of age. Male to female (M/F) percentages of patients were 47.6/52.4. Female/male ratio was 1.1 in AIS group.

79 of the patients (55.2 %) had left LSA infarction and 64 of the patients had right LSA infarction. Of the patients, 91 had hypertension (HT) (63.6 %), 42 had diabetes (DM) (29.4 %), 26 had HT + DM (18.1 %), 20 had ischaemic cerebrovascular disease (14 %), 11 had congestive heart failure (CHF) (11.7 %) and 66 had hypercholesterolemia (46.2 %) (Table 1).

In the present study on aetiology; atrial fibrillation (AF) was detected in 43 patients (30.1 %), and cardiac pathologies that could be the source of cardiac embolism in 16 patients in echocardiography. (Apical thrombus in 1 patient, hypokinetic segments in 14 patients and low ejection fraction (EF), rheumatic valve disease in 1 patient). Five of 16 patients were also present in the AF group. Atheroma plaques (70% and above) were detected in 8 patients (5.6 %) in the carotid vertebral Doppler USG leading to severe stenosis on the symptomatic side. In 1 patient, total occlusion was observed on the symptomatic side and atheroma plaques were found to cause moderate stenosis (50-70%) in 19 patients (13.3 %). A total of 28 patients had large vessel disease (>50% stenosis), and 7 of these patients had additional cardiac pathology. In 19 patients, carotid stenosis was less than 50% on the symptomatic side. In addition, intimal medial thickening was reported in 77 patients. Six patients had moderate carotid stenosis on the symptomatic side and AF coexistence, and four patients had advanced carotid stenosis on the symptomatic side and cardiac hypokinetic segment. Echocardiography revealed 5 cases with patent foramen ovale. Eighteen patients (10.5%) showed that EF was below 50% as a result of echocardiography.

Of the 143 patients, it was accepted that 21 had atherothrombotic aetiology, 44 had cardioembolic aetiology, 7 had more than one aetiology, and 71 had stroke of undetermined aetiology. Number of patients with favourable outcome were 103 (72%) and poor outcome were 40 (27%) (Table 2).

No relation was found between the side selection (right or left) and AF in the LSA infarct patients (x^2 test p: 0.168). There was no difference between aetiology of stroke and prognosis (x^2 test p: 0.206). A correlation was found between smoking and early-term prognosis (p<0.001 rho: 0.458). Univariate analysis showed that there was no correlation between

Table 1. Baseline characteristics

	n=143 (%)
Age, yr	69.5±13.5
Sex, men	68 (47.6)
Hypertension	91 (63.6)
Diabetes	42 (29.4)
Hypercholesterolemia	66 (46.2)
Current smoking	50 (35.1)
Coronary artery disease	23 (16.1)
Initial NIHSS	5.37±3.8
mRS at 1st month	1.98±1.5

Table 2. Subtype and stroke mechanisms

	n=143(%)
Stroke subtype	
Large artery atherosclerosis*	21 (14.7)
Cardioembolism	40 (28.0)
Two or more causes*	7(4.9)
Negative evaluation (stroke of undetermined aetiology)	75 (52.4)

the patients with poor functional outcome and their age, sex, HT, DM, coronary artery disease or hypercholesterolemia. Higher initial NIHSS scores and higher mRS scores. The patients who had poor functional outcomes showed traits like more common smoking and had (Table 3).

Discussion

The classification of subtype of stroke was determined based on the TOASTsystem. This study inclu-ded patients with cardioembolism (27%), large artery atherosclerosis (14%), or stroke of undetermined aetiology due to two or more identified causes (0.4%) or due to negative evaluation (52%). Similar to other vascular territory infarctions, many striatocapsular infarcts are frequently included in the "unde-termined aetiology" category according to the TOAST classification.

Arterial hypertension, the single most frequent stroke risk factor, showed a high prevalence in all stroke subtypes, except for combined other etiologies. In the NINCDS Stroke Data Bank, hyperten-sion and diabetes mellitus were more common in lacunar and atherothrombotic stroke than in cardio-embolic stroke[18]. The Oxfordshire Community Stroke Project found no differences between lacu-nar and cardioembolic stroke regarding hypertension or diabetes mellitus[19]. Similar to previous studies hypertension (63.6%) was the most remarkable aetiologic relationship with LSA infarcts.

In previous studies it had been shown that the current smoking and hypercholesterolemia are mainly associated with stroke due to large-artery atherosclerosis[20-23]. In our study a correlation was found between smoking and early-term prognosis (p<0.001 rho: 0.458). The patients with poor functional outcome had more commonly a history of current smoking, higher initial NIHSS score and higher mRS scores.

There was no difference between aetiology of stroke and prognosis (p=0.206) in contrast with the previous reports. In a lot of previous clinical studies, cardioembolic stroke (with AF) was associated with the highest mortality rate and worst functional outcome after 90 days and the most severe acute neurological deficit[24-26] and patients with lacunar stroke had the mildest deficits and the best prog-nosis among all subtypes[9,27].

In our study there was no relation found between the side selection (right or left) and AF in the LSA infarct patients (p=0.168). Univariate analysis showed that there was no correlation between the patients with poor functional outcome and their age, sex, HT, DM, coronary artery disease or hypercholesterolemia.

In this study, we aimed to show whether there exists a specific relationship of LSA infarcts with demographic data and aetiologic factors or not. We concluded that studies including larger group of patients are necessary to detect any specific relation between these factors and LSA territory infarcts.

Ethical Committee Approval: Retrospective study.

Peer-review: Externally peer-reviewed.

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Table 3. Univariate Analysis of Factors Associated with a Poor Outcome at 1 Months

	Patient with favourable outcome (n=103) (%)	Patient with poor outcome (n=40) (%)	p
Age, yr	68.86±11.6	72.0±11.7	0.342
Sex, men (%)	51 (49)	17 (42)	0.45
Hypertension (%)	68 (66)	23 (57.5)	0.342
Diabetes (%)	29 (28.1)	13 (32.5)	0.609
Hypercholesterolemia (%)	48 (46.6)	18 (45)	0.863
Current smoking (%)	22 (21.4)	28 (70.0)	<0.001
Coronary artery disease (%)	14 (13.6)	9 (22.5)	0.193
Initial NIHSS	4.08±2.96	8.70±3.99	<0.001
mRS	1.20±0.9	3.97±0.8	<0.001

Conflict of Interest: None declared.

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