Isolated Dysarthria in Stroke- A Clinical Study

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Summary

Background and Purpose: Dysarthria is a speech disorder that is common in cerebral lesions. Pure dysarthria is a very rare form of lacunar syndromes. The purpose of this study is to clarify the clinical picture and the anatomic site of the lesions causing pure dysarthria.

Methods: Twenty-nine patients with sudden onset of pure dysarthria due to acute ischemic or hemorrhagic stroke confirmed by cranial computed tomography and/or magnetic resonance imaging were included. Dysarthria was evaluated within the first 72 hours after stroke onset and at the end of the first week. Dysarthria was graded as mild, moderate or severe.

Results: Neuroimaging showed supratentorial lesions (86.2%) which were located in corona radiata in 13 patients, frontal lobe in 3, posterior limb of the internal capsule in 2, external capsule in 2, thalamus in 2, insular area in 2 and parietal cortex in 1 patient. In 4 patients (13.8%), the infratentorial region was affected. In all patients with infratentorial involvement, the lesions were located in the paramedian pontin area. Supratentorial and infratentorial lesions were also more frequent on the left.

Conclusions: In our study corona radiata involvement was more frequent in patients with pure dysarthria than the other regions. Pure dysarthria was more frequent in corona radiata lesions due to selective involvement of corticobulbar tract.

Key words: Dysarthria, lacunar syndrome, corona radiata, left

İnmede İzole Dizartri- Klinik Çalışma

Özet

Amaç: Dizartri sliklikla serebral lezyonlarla ortaya çıkan bir konuşma bozukluğuudur. Saf dizartri lümen sendromların nadir görülen bir formdur. Çalışmanın amacı izole dizartrili olgularda klinik tabloyu ve saf dizartriye neden olan lezyonların anatomik lokalizasyonlarını belirlemektir.


Sonuçlar: Nörografjielde supratentorial lezyonlar (86.2%); 13 hastada korona radiatada, 3 hastada frontal lobta, 2 hastada internal kapsüllerin arka bacağında, 2 hastada eksternal kapsülde, 2 hastada talamusta, 1 hastada insuler alanda, 1 hastada parietal kortekste izlenmiştir. Dört hastada (13.8%), infratentoriyal bölge etkilenmiştir. İnfratentoriyal tutulumu olan tüm hastalar, paramedian pontin infarkt şeklinde prezentе olmuştur. Supratentoriyal and infratentoriyal lezyonlar sol tarafta daha sık idi.
Tartışma: Çalışmamızda saf dizartrili hastalarda korona radiata tutulumu diğer bölgelere göre daha sıktır. Korona radiata lezyonlarında kortikobulbar liflerin selektif tutulabilmesinden dolayı saf dizartri daha sık görülebilir.

Anahtar Kelimeler: Dizartri, laküner sendrom, korona radiata, sol

INTRODUCTION
Dysarthria is a speech disorder characterized by dysfunction in the initiation, control, and coordination of the articulatory structures involved in speech output\(^\text{(4,14)}\). Patients having isolated dysarthria exhibit intact cortical language functions including comprehension, reading and writing with normal auditory functions, although the speech is inarticulate\(^\text{(4)}\).

Dysarthria is common in cerebral lesions of different origin and location\(^\text{(11)}\). Isolated dysarthria caused by stroke, termed "pure dysarthria" (PD), is very rare, since sudden onset of PD is usually associated with other neurological deficits\(^\text{(2,12)}\).

There is little information about its anatomic specificity, spectrum of associated clinical characteristics, etiologic mechanisms, and its prognosis. The purpose of this study is to clarify the clinical picture of the pure dysarthria syndrome and to determine the anatomic localization of the lesion by using cranial computed tomography (CT) and/or magnetic resonance imaging (MRI).

MATERIAL AND METHODS
Between June 2006 and September 2014, the data of stroke patients admitted to the Department of Neurology of Atatürk Training and Research Hospital were collected retrospectively. Patients with transient ischemic attack, subarachnoid hemorrhage, and spontaneous subdural hematoma were excluded. Only acute ischemic or hemorrhagic stroke patients who had PD without any other sensory-motor dysfunction constituted the study group. Patients with stroke in the subacute or chronic stage, and patients with concomitant disorders such as disturbance of consciousness, aphasia, anarthria and dementia were not included.

We recorded 29 patients (17 males, 12 females) with sudden onset of PD due to acute ischemic or hemorrhagic stroke confirmed by CT and/or MRI. All the patients were right handed.

The age, gender, family history of stroke, history of cigarette smoking, history of previous stroke; presence of hypertension, hyperlipidemia, diabetes mellitus, coronary artery disease, atrial fibrillation was noted.

The diagnostic work up (Carotis Doppler ultrasonography, CT angiography, MR angiography, echocardiography etc.) done to determine the etiology of the stroke was evaluated and patients were classified according to the Trial of Org 10172 in acute stroke treatment (TOAST).

Dysarthria is routinely classified as mild, moderate or severe in our clinic and recorded in the patient’s files. The examination of dysarthria\(^\text{(4,10,11)}\) had been performed by two experienced neurologists and an assistant of neurology (MÇ, TÖ, TM) independently within the first 72 h after stroke onset. Dysarthria was reevaluated and recorded at the end of the first week. Articulation was evaluated on the basis of various samples, namely, spontaneous speech, repetition of sentences and words, reading a short story, and rapid iteration of syllables (pa/ta/ka). All these data were taken from the patient’s files.

Location of the lesion was identified by CT (n=29) and MRI (n=26) scans. CT was performed with a Hitachi W950SR X-Ray CT (spiral) System and MRI with a General Electric Vectra Model 1.5 T.
RESULTS

Mean age of the 29 patients (17 males and 12 females) was 68.8 ± 10.2 years (range 37 to 91 years). Twenty-four (82.8%) of our patients had hypertension, 17 (58.6%) had hyperlipidemia, 10 (34.5%) had diabetes mellitus, 11 (37.9%) had a history of cigarette smoking, 7 (24.1%) had a history of myocardial ischemia, 3 (10.3%) had atrial fibrillation, 8 (27.6%) had a history of previous stroke and 4 (13.8%) had a family history of stroke (Table-1).

Twenty-eight patients with PD had ischemic stroke, only one patient had hemorrhagic stroke. Twenty-eight patients were assigned a subtype, cardioembolic 4 (14.3%), small vessel disease 21 (75.0%) and undetermined 3 (10.7%). Large artery atherosclerosis wasn’t determined.

The lesions responsible for dysarthria were located in the supratentorial region in 86.2%, and infratentorial region in 13.8% of the patients. Supratentorial strokes were located in the corona radiata in 13, frontal lobe in 3 (one was hemorrhagic stroke), posterior limb of the internal capsule in 2, external capsule in 2, thalamus in 2 (posteromedial thalamus and anterolateral thalamus), insular area in 2 and parietal cortex in 1 patient (Table-2).

In 4 patients (13.8%), the infratentorial region was affected. In all patients with infratentorial involvement, the lesions were located in the paramedian pontine area. (Table-2).

The lesions were located in the left side in 79.3% (n=23) and in the right side in 20.7% (n=6) of the patients. Supratentorial lesions were more frequent on the left side (80.0%). Infratentorial infarcts were also more frequent on the left side (75.0%).

Dysarthria was evaluated separately within 72 hours of the attack and at the end of the first week. It was graded as mild, moderate or severe. At the initial examination 20 of 29 patients had moderate and 9 had mild dysarthria. There was no change at the end of the first week in 14 of 20 patients with moderate dysarthria. Four patients had regressed to mild dysarthria. In two cases speech was normal at the end of the first week. Six of nine patients with mild dysarthria speech became normal at the time of reevaluation. In 3 of them no change was seen.

Table 1: Risk factors, cause of stroke of patients with pure dysarthria.

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Pure dysarthria (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age ± SD, years</td>
<td>68.8 ± 10.2</td>
</tr>
<tr>
<td>Age range years</td>
<td>37-91</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>17/12</td>
</tr>
<tr>
<td>Hypertension</td>
<td>24 (82.8%)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>17 (58.6%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>10 (34.5%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>11 (37.9%)</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td>7 (24.1%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>3 (10.3%)</td>
</tr>
<tr>
<td>History of stroke</td>
<td>8 (27.6%)</td>
</tr>
<tr>
<td>Family history of stroke</td>
<td>4 (13.8%)</td>
</tr>
</tbody>
</table>
DISCUSSION

Dysarthria due to stroke is most often associated with other neurological deficits such as hemiparesis, hemiataxia, clumsiness of one hand, central facial paresis, and tongue deviation (8).

Dysarthria has been reported in different localizations of ischemic stroke. The lesions responsible for dysarthria were located in the supratentorial region in 45.6% and in the infratentorial region in 54.4% of patients in a study of Urban et al. (15). In another study the lesions responsible for dysarthria were located in the supratentorial area in 62% and in the infratentorial area in 31% and in both areas in 7% of patients (11).

Pure dysarthria is extremely rare, as shown in a study of 227 patients with lacunar infarcts in whom PD was noted in only 0.4% (3). In another study, PD was present in 3.6% of 55 patients with dysarthria (4).

Pure dysarthria syndrome was first described by Fisher along with other lacunar syndromes such as ataxic hemiparesis, dysarthria-clumsy hand, and pure sensory, pure motor strokes (5,6). Fisher defined PD as a variant of lacunar syndromes, caused by a pontine base infarction (5,6,12). The boundaries of PD still remain unclear, because clinical features of reported cases of PD were not uniform, with variations of mild concomitant deficits (12).

Different reports about the localization of lesions associated with PD were presented (12). The lesions responsible for the PD were located in three different anatomic structures in Ichikawa and Kageyama’s series: the anterior capsule, the genu and the bulbar motor cortex. They had suggested that this syndrome was caused by the disruption of one of the following parts of the motor systems necessary for articulation: the frontopontine fibers in the anterior capsule, the corticobulbar fibers in the genu, or the bulbar motor cortex (8). Other lesion sites associated with PD have included the basal ganglia, anterior parts of the posterior limb of the internal capsule, paramedian pontine base and cortical regions supplied by the middle cerebral artery (10,12).

Ozaki et al. had presented five cases with PD due to anterior internal capsule and/or
corona radiata infarction. They had suggested damage to frontopontine fibres and anterior thalamic radiations which were located in this region that might play an important role for the development of PD\(^{(13)}\). In the corona radiata, the motor fibers may be more loosely packed than in the external capsule, and selective involvement of the corticobulbar fibers with sparing of the corticospinal tracts may be possible\(^{(5)}\). Similarly, the infarcts were most frequently located in the corona radiata (44.8\%) in our study. But also we have seen PD in the lesions of external capsula in 6.9\% of patients.

There are also patients reported with paramedian pontine infarcts, suggesting that lesions in these areas may also involve corticobulbar fibers without affecting the motor fibers for extremities\(^{(5)}\). In our study, the paramedian pontine region was affected in 13.8\% (n=4) of patients.

Pure dysarthria due to cortical stroke is considered rare. The small size of the infarct may make it undetectable by CT or conventional MRI. Diffusion-weighted MRI has been shown to be more sensitive in identifying acute, small ischemic lesion than conventional MRI\(^{(9)}\). In our study, cortical lesions were present in 6 of 29 patients (20.7\%). One of 6 patients was presented with dysarthria from cortical frontal hemorrhage.

Six patients with dysarthria as their isolated or major symptom from a small cortical stroke has been reported. Five had infarction and one had hemorrhage. In the patients with ischemic stroke, the lesions were identified by diffusion-weighted MRI but not by T-2 weighted MRI. The lesions were located lateral to the precentral knob\(^{(9)}\).

Akiyuki et al. reported that a patient who presented only with dysarthria had a small limited cortical infarction located at the left middle frontal gyrus. They suggested that an isolated middle frontal gyrus lesion can cause PD by secondary compression of the cortical areas related or connected to the corticobulbar tract\(^{(16)}\). The analysis of the lesion location showed that 50.0\% (3/6) of all cortical infarcts leading to dysarthria were located on the frontal knob in our study.

Hiraga et al. reported that a patient who developed PD had an acute cortical infarction in the insular cortex. They suggested that the role of the insula in language has been difficult to assess clinically due to the rarity of pure insular strokes\(^{(5)}\). In our study, insular infarcts were found in only 2 patients.

Okuda et al. suggested that frontal cortical hypoperfusion, particularly in the anterior opercular and medial frontal regions, plays an important role in the development of PD\(^{(12)}\).

Ichikawa and Kageyama had suggested that the PD syndrome can be accepted as a lacunar syndrome like pure motor hemiparesis, ataxic hemiparesis, and other lacunar conditions\(^{(8)}\).

Pure dysarthria can arise from either lacunar or cortical infarction without clinically evident differences. As it is the case for PD, however, other lacunar syndromes can result from cortical lesions as well\(^{(12)}\).

Alexander and Wildgeuber suggested that right-sided lesions would not cause dysarthria\(^{(1,5,18)}\). But in literature some studies reported that dysarthria in right-sided lesions\(^{(4,8,11,13,14,15)}\). Consequently dysarthria is more frequently associated with left sided extracerebellar lesions\(^{(11,13,14,15)}\).

Urban et al. had found that 88.7\% of extracerebellar infarcts leading to dysarthria were located in the left hemisphere and 11.3\% on the right side. Supratentorial infarcts were found more often on the left (89.5\%) and brainstem infarcts were also more frequently on the left side (86.7\%) in Urban's study\(^{(14)}\).

Ozaki et al. had suggested that the lesion in the dominant hemisphere might be
responsible for the development of PD. However, in one of their patients the lesion was in the non-dominant hemisphere (13). The analysis of the lesion side in our study showed that 82.8% of all lesions leading to PD were located on the left and 17.2% on the right side. Since it has previously been shown that a lesion of the cortico-lingual pathway is crucial in the pathogenesis of dysarthria in stroke, a possible explanation might be a more dominant descending pathway from the left motor cortex (14). In our study supratentorial and also infratentorial lesions were more frequent on the left than on the right side.

Different reports about the side of cerebellar infarction associated with dysarthria are presented (4,15). In our study, acute cerebellar stroke wasn’t determined.

In conclusion, the infarcts were most frequently located in the corona radiata (13/29) in our study. In 2 patients, the internal capsule was affected. Both supratentorial and infratentorial lesions were more frequent on the left side (23/29). These findings indicate the importance of the left sided cortico-bulbar tracts for articulation (14).

Our study had limited number of patients who were followed only for one week. The second limitation can be the usage of diffusion weighted imaging with low Tesla which may be insufficient to show small cortical lesions. Further studies including higher number of patients would contribute to clarify the underlying mechanism of dysarthria.

CONFLICT OF INTERESTS
There is no conflict of interests

DISCLOSURE
Nothing to disclose

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