Original Article

Isolated dysarthria-facial paresis syndrome: A rare clinical entity which is usually overlooked

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Background and Aims: The aim of the study is to search the lesion localization of the pure isolated facial paresis-dysarthria syndrome in patients who were admitted to our neurology clinic in a prospective study. **Methods:** Over a period of six years, the patients who had no prominent sensorimotor dysfunction were examined by neurologists and underwent computerized tomography (CT) and/or magnetic resonance imaging (MRI). **Results:** Eleven patients out of more than 2000 had the aforementioned clinical picture. Lacunar infarctions were identified at the corona radiata in nine patients, and at the internal capsule in two patients. As reported previously, facial paresis was usually mild and temporary. Six of our eleven patients were seen at the outpatient clinic one month later. Four of them had completely recovered and the other two had mild dsyarthria without any facial paresis. The other five could not be reached after leaving the hospital. **Conclusions:** Dysarthria-facial paresis is a rare clinical entity and possibly a variation of dysarthria-clumsy hand syndrome, and we suggest that pure facial paresis (FP) and pure dysarthria should be considered as very extreme examples of this syndrome.

Key Words: Capsular genu, dysarthria, facial paresis, lacunar syndromes

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.[1] However, the site of the lesion responsible for dysarthria could not be identified on CT scans and pontine base was the suggested lesion site.[1] Following this report, some authors presented cases with unilateral lacunar infarctions without significant somatic motor dysfunction using the diagnostic term of isolated facial paralysis, pure dysarthria, facial paresis-dysarthria (FP-DA) syndrome and capsular genu syndrome. In our report, we present 11 cases of FP-DA and discuss the lesion topography.

Materials and methods

Between January 1995 and December 2000, the data of stroke patients admitted consecutively to the Department of Neurology of Ataturk Training and Research Hospital were collected prospectively. For the purpose of this study, patients with transient ischaemic attack, subarachnoid hemorrhage, and spontaneous subdural hematoma were excluded. Only acute ischaemic or hemorrhagic stroke patients who had FP-DA without any other sensory-motor dysfunction constituted the study group. All patients were examined by the first author within the first 48 h of onset of symptoms and underwent CT scans and/or MRI. There were six men and five women aged 48–83 years with a mean of 66. Table 1 summarizes the clinical data of the patients as well as the risk factors.

Results

All patients complained about dysarthria. The degree of dysarthria was usually mild to moderate, and all patients had a mild lower facial paresis. Lacunar infarctions were located at the corona radiata in nine patients. In two patients, internal capsular lacunes were present. In one of them, the lacune was located at the anterior part internal capsule touching the adjacent caudate nucleus head and in the other, posterior part of the internal capsule and the adjacent globus pallidus were involved. As reported previously, facial paresis was usually mild and temporary. Six of our eleven patients were seen at the outpatient clinic one month later. Four of them had completely recovered and the other two had mild dysarthria without any facial paresis. The other five could not be reached after leaving the hospital.

Discussion

All of our patients had both FP and DA. This may be due to early examination by clinicians who were aware of this syndrome and symptoms were recognized before they disappeared. As expected, most of the patients recovered well. Of risk factors, hypertension accounted for 100% and was higher than

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Table 1: Summary of the cases

Sex	Age	Risk factors	Dysarthria	Facial paresis	Other	Lesion location
Male	57	HT, SM	+	+ (Right)	-	Left HCN, AIC
Female	83	HT, DM	+	+ (Right)		Left GP, PIC
Male	67	HT, HL	+	+ (Left)		Right CR
Female	48	HT	+	+ (Right)		Left CR
Male	59	HT, SM, CD	+	+ (Right)		Left CR
Female	65	HT	+	+ (Right)		Left CR
Female	67	HT, CD	+	+ (Right)		Left CR
Male	78	HT, DM, SM	+	+ (Left)		Bilateral CR
Male	71	HT, AF	+	+ (Right)	Right Babinski sign +	Bilateral CR
Female	60	HT	+	+ (Right)		Left CR
Male	71	HT	+	+ (Left)		Bilateral CR

HT: Hypertension, SM: Smoking, HCN: Head of the caudate nucleus, AIC: Anterior internal capsule, DM: Diabetes mellitus, GP: Globus pallidus, PIC: Posterior internal capsule, HL: Hyperlipidemia, CA: Corona radiata, CD: Coronary heart disease, AF: Atrial fibrillation

previously reported by Kaul *et al*. in a study which also included lacunar syndromes of different types.[2] Even they constituted a minority of our cases. Some patients only had CT scans. High-resolution MRI could be more precise for the lesion localization.

Dysarthria and supranuclear facial paralysis are well-known characteristics of ischaemic stroke with prominent limb weakness and sensory deficit, but isolated FP, isolated DA or FP-DA cases without significant sensorimotor deficit are very rarely reported (Table 2 summarizes patients with similar clinical syndromes that have been previously reported by different authors[3]-[17]). In these cases, FP is usually mild and temporary, and if neurological examination is not performed at earlier stages subtle signs of facial paresis (such as flattening of nasolabial folds) might disappear without being noticed, and other symptoms such as very slight dysarthria may remain unnoticed. Therefore, it is proposed that the two syndromes should be considered as a continuum dysarthria-facial paresis syndrome, which, as previous others have suggested, may also be regarded as a variant of dysarthria-clumsy hand syndrome.[9],[18] A variant of DA-FP shows additional lingual paresis, dysphagia and weakness of one hand, which was first described by Bogousslavsky and Regli. This syndrome is characterized by acute stroke limited to genu of the internal capsule on one side and characterized with dysarthria, contralateral faciolingual and to a lesser extent masseter-palatal-pharyngeal-laryngeal-weakness. There is no limb involvement and mild weakness is limited to the hand.[17] Ozaki *et al*. suggested that this syndrome should be classified on the basis of specific syndromes rather than the anatomic sites involved for practical reasons, and they proposed using the conventional terms, 'pure dysarthria' and 'faciolingual paresis.'[6]

In FP-DA syndrome, anatomical localizations of the lacunar lesion have been reported in the anterior limb or superior part of the genu portion of the internal capsule and/or in the corona radiata adjacent to the internal capsule, pontine base, or in the cortex.[3]–[6],[8]–[12],[16],[19] In the corona radiata, the motor fibers may be more loosely packed than in the external cap-

Table 2: Reported cases with dysarthria and/or facial paresis without significant limb weakness

References	No. of Patients	Facial paresis	Dysarthria	Dysphagia	Other	Localization
3	2	1	1			IC genu/ant
4, 5	5	5	4	2		4 CR, 1 AIC
6, 7	5	1	5			CR/AIC
4, 5	1	1				Left pons
8	2	2	2			Pons
9	9	5	9		Deep tendon reflex R	4 AIC, CR-4 G, CR-1 AIC
10	13	13	8	3	1Deep tendon reflex, 2LP,1PP, 1gait difficulty	3 BG-IC, 5 BG-CR, 3 pons, 2 FC, SC
11	37	3	4			Basis pontis
12	1	1				Basis pontis
13	7	5	7		3 LP	4CR, 2 genu-PIC
14	12	3	12	2		IC-CR
15	68	40	68			
16	6	4	4	3	5 clumsy tongue movements, sensory symptoms	Cortex
17	6	6	5	3	Tongue, weakness, masseter finger, hand weakness	Capsulargenu

IC - Internal capsule, CA - Corona radiate, AIC - Anterior internal capsule, BG - Basal ganglia, LP - Lingual paresis, PP - palatal paresis, FC - Frontal cortex, SC - Subcortical, PIC - Posterior internal capsule

sule, and selective involvement of the corticobulbar fibers with sparing of the corticospinal tracts may be possible. The lesions in the basal ganglia slightly abutting the internal capsule may also involve corticobulbar fibers selectively.[19] 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. 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 pure dysarthria.[14] Urban *et al*. also reported that interruption of the corticolingual pathways to the tongue is crucial in the pathogenesis of isolated dysarthria after extracerebellar stroke based on transcranial magnetic stimulation and HMPAO-SPECT studies of patients with isolated dysarthria syndrome.[13] In our study, lesions were in the corona radiata (nine patients) and internal capsule (two patients). We did not have any patients with pontine lesions. It was reported that in most cases, the lesions were located in the left hemisphere, thus suggesting that the lesion in the dominant hemisphere might be responsible for the development of pure dysarthria, as it was the case in our patients.[5],[6],[8],[13],[14][16],[19] As reported previously, the lesion of the corticolingual pathway is crucial in the pathogenesis of dysarthria in stroke.[15] Alexander and Wildgeuber suggested that right-sided lesions would not cause dysarthria.[15],[20],[21]

In conclusion, DA-FP is a rare clinical entity and possibly is a variation of dysarthria-clumsy hand syndrome, and we suggest that pure FP and pure dysarthria should be considered as very extreme examples of this syndrome. Future studies using diffusion weighted imaging would allow more precise discrimination of the sites of lesions in FP-DA with higher sensitivity and specificity.

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