Pure Dysarthria, Isolated Facial Paresis, or Dysarthria–Facial Paresis Syndrome

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Background and Purpose  Pure dysarthria, isolated supranuclear facial paresis, and their combination without somatic motor dysfunction are rarely encountered clinical syndromes and have not yet been clearly characterized.

Methods  Thirteen patients (9 men, 4 women; aged 33 to 72 [mean, 56] years) with unilateral strokes who developed dysarthria with or without facial paresis but without somatic motor dysfunction were reviewed in addition to case reports from previous literature.

Results Computed tomographic scan and/or magnetic resonance imaging showed infarcts on the corona radiata in 4 patients, basal ganglia abutting the internal capsule in 3, basal ganglia–corona radiata in 1, pontine base in 3, and cortical–subcortical bulbar motor area in 2. The dysarthria and facial paresis were usually mild and transient, and either one was likely to be unnoticed.

Conclusions  It is suggested that pure dysarthria or isolated facial paresis syndrome be considered as an extreme continuum of dysarthria–facial paresis syndrome, which is likely to be a variant of dysarthria–clumsy hand syndrome. (Stroke. 1994;25:1994-1998.)

Key Words  • cerebrovascular disorders • dysarthria • facial paralysis

Bilateral strokes involving the pyramidal tract may produce supranuclear or pseudobulbar palsy without significant somatic motor dysfunction. 1-3 However, recent reports have shown that unilateral lesions may cause supranuclear dysarthria with or without lower facial paresis, without significant motor weakness. 4-10 These case reports have been described under various headings, including isolated facial palsy, 4 supranuclear facial palsy, 6-8 pure dysarthria, 5,9 and isolated dysarthria. 10 Patients with similar clinical syndromes were also included in an article titled “Capsular Genu Syndrome,”7 causing further confusion in understanding this syndrome. Moreover, those reports were small in patient number, and the lesion localization for this syndrome needs to be more clearly addressed. Therefore, I present my experience of 13 patients with unilateral strokes who presented with pure dysarthria or dysarthria–facial paresis without somatic motor dysfunction. I also review the previously reported cases.

Subjects and Methods

At Asan Medical Center, I encountered 39 patients with stroke who presented with sudden dysarthria (with or without facial paresis) without sensorimotor dysfunction of the extremities between January 1989 and January 1994. All underwent brain computed tomographic (CT) scan and/or magnetic resonance imaging (MRI). Twenty patients had distinct bilateral lesions and presented features of previously described pseudobulbar syndrome. 1-3 Three showed diffuse and multiple small infarcts that were difficult to correlate with the symptoms. The remaining 16 patients showed unilateral lesions in the imaging study that were considered appropriate for the patients’ symptoms. However, 3 of them were excluded: 1 had a history of stroke, which probably had occurred in the side contralateral to the new lesion; the other 2 patients, although they were without limb weakness or clumsiness at the time of examination, recalled transient weakness of the limb at the onset of stroke.

Results

Thirteen patients (9 men and 4 women, aged 33 to 72 [mean, 56] years) were studied. All were examined within 5 days of stroke onset except for 3 (patients 1, 5, and 9). Table 1 summarizes the details of each patient, including the risk factors for stroke. All complained of dysarthria. The degree of dysarthria was usually mild or moderate; none had unintelligible speech. Eight had mild lower facial paresis (often manifested as flattening of the nasolabial fold). The presence of facial paresis remained unclear in 1 patient (patient 9), who had had residual facial paresis from previous Bell’s palsy. Three had transient dysphagia of mild degree, and 2 showed mild tongue deviation. Although none had arm or finger weakness or ataxia on examination, patient 2 had brisk deep tendon reflexes in the elbow and patient 5 felt slight clumsiness on gait.

CT and MRI were performed in 6 and 11 patients, respectively, and showed unilateral infarcts in all cases. The infarcts were located in the corona radiata in 4 patients (anterior portion in 2, posterior portion in 2), basal ganglia in 3 (adjacent to the anterior limb of the internal capsule in 2 and posterior limb of the internal capsule in 1), basal ganglia–corona radiata in 1, pontine base in 3, and cortical or subcortical bulbar motor area in 2. Figs 1 through 3 present radiological findings of patients 6, 10, and 13. Except for 3 (patients 7, 11, and 12), the infarcts were less than 1.5 cm in their longest diameter. Three patients underwent...
TABLE 1. Summary of 13 Cases

<table>
<thead>
<tr>
<th>Patient/Sex/Age, y</th>
<th>Risk Factors</th>
<th>Dysarthria</th>
<th>Facial Paresis</th>
<th>Other</th>
<th>Lesion Location</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/60</td>
<td>HT, CA</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>R BG-P, IC</td>
<td></td>
</tr>
<tr>
<td>2/M/52</td>
<td>HT, DM, SM</td>
<td>+</td>
<td>-</td>
<td>↑ DTR in right elbow</td>
<td>L BG-P, CR</td>
<td>Angio: NL</td>
</tr>
<tr>
<td>3/M/63</td>
<td>DM, SM, CA</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>L P, CR</td>
<td></td>
</tr>
<tr>
<td>4/M/33</td>
<td>SM</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>L BG-A, IC</td>
<td>Angio: NL</td>
</tr>
<tr>
<td>6/F/67</td>
<td>HT, DM</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>L A, CR</td>
<td></td>
</tr>
<tr>
<td>7/M/53</td>
<td>DM, SM</td>
<td>+</td>
<td>+</td>
<td>Mild DP, LP</td>
<td>L BG-A, IC</td>
<td>Angio: NL</td>
</tr>
<tr>
<td>8/F/42</td>
<td>SLE, aCL</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>L A, CR</td>
<td></td>
</tr>
<tr>
<td>10/M/51</td>
<td>SM, LA</td>
<td>+</td>
<td>+</td>
<td>Mild DP</td>
<td>R pons</td>
<td></td>
</tr>
<tr>
<td>11/M/62</td>
<td>HT, DM</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>R pons</td>
<td></td>
</tr>
<tr>
<td>12/M/59</td>
<td>HT, DM, SM, HL</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>L frontal cortex</td>
<td></td>
</tr>
<tr>
<td>13/M/53</td>
<td>DM, SM, CA</td>
<td>+</td>
<td>+</td>
<td>Mild DP, slight LP, PP</td>
<td>L frontal subcortex</td>
<td>DS: NL</td>
</tr>
</tbody>
</table>

HT indicates hypertension; CA, cardiac disease; R, right; BG, basal ganglia; P, posterior; IC, internal capsule; DM, diabetes mellitus; SM, cigarette smoking; ↑, increased; DTR, deep tendon reflex; L, left; CR, corona radiata; angio, angiogram; NL, normal; A, anterior; HL, hyperlipidemia; MRA, magnetic resonance angiogram; MCA occl, middle cerebral artery occlusion; DP, dysphagia; LP, lingual paresis; SLE, systemic lupus erythematosus; aCL, positive anticardiolipin antibody; DS, duplex scanning; LA, positive lupus anticoagulant; and PP, palatal paresis.

conventional angiogram and 1 underwent magnetic resonance angiogram, which showed complete occlusion of the proximal middle cerebral artery in 1 patient (patient 5) and normal findings in 3. Two underwent carotid duplex scanning; 1 (patient 13) showed normal findings, whereas another (patient 9) showed diffuse atherosclerotic plaque in the carotid system. Follow-up lasted 2 to 24 (average, 6) months. The neurological symptoms (dysarthria and facial paresis) resolved or markedly improved, usually in several months. However, patients 9 and 12 developed right hemiparesis associated with aggravated dysarthria 5 and 3 months after the initial event, respectively. CT scan identified a new infarct in the left corona radiata in patient 12; patient 9 refused imaging studies.

Discussion

Our 13 patients showed dysarthria with or without facial paresis but without motor weakness or ataxia in the extremities. Patients with similar clinical syndromes have been previously reported under various titles. However, as seen in Table 2, most of them had a combination of dysarthria and facial paresis, suggesting that these cases may be categorized in the same spectrum of clinical syndromes. The facial paresis in this syndrome is generally mild and transient and therefore...
may be unnoticed by conventional neurological testing, especially when the patients are not examined early enough. It should be noted that some of the previously reported patients with pure dysarthria or isolated facial paresis and our 2 patients with pure dysarthria (patients 1 and 5) were not examined in an acute stage of stroke.

Nevertheless, 4 patients described by Ozaki et al. and 4 by Ichikawa and Kageyama were reported to have dysarthria only. Furthermore, Donnan et al. found 1 patient with dysarthria alone among 62 patients with lacunar stroke, and Arboix et al. identified 4 patients with isolated dysarthria among 670 stroke patients. Last, Kashiwara and Matsumoto briefly reported 4 patients with isolated transient dysarthria among 63 patients with capsular infract, although the presence or absence of facial paresis was not mentioned.

Therefore, it seems that pure dysarthria in a strict sense may be produced by unilateral minor strokes. However, the patients described as having isolated facial paresis generally shared dysarthria, and the presence of pure supranuclear facial paresis syndrome without dysarthria, although reported, remains unconvincing. The 2 patients described by Melo et al. as having isolated facial paresis among 255 patients with pure motor stroke also had dysarthria. Although Arboix et al. and Donnan et al. described 6 and 1 patients, respectively, with motor dysfunction restricted to facial muscles in their series, they did not describe the presence or absence of dysarthria or other lower cranial nerve dysfunction. Therefore, pure dysarthria and isolated facial paresis syndrome, if they exist, should be quite rare. Furthermore, subtle signs of facial paresis (such as flattening of the nasolabial fold) or a mild degree of dysarthria may present equivocally and thus be neglected or unnoticed. Therefore, it may be reasonable to consider these syndromes as a continuum of dysarthria-facial paresis syndrome, which, as previous authors have suggested, may also be regarded as a variant of dysarthria-clumsy hand syndrome. Indeed, patients with similar clinical symptoms were classified as having dysarthria-clumsy hand syndrome in a recent study of lacunar stroke.

According to our data and previous reports, dysphagia, palatal paresis, and lingual paresis are uncommon and, if present, are mild and transient in this syndrome. It is noteworthy that the 2 patients with dysphagia in the series of Huang and Broe had bilateral lesions. However, dysphagia is often a part of so-called capsular genu syndrome, in which distinct facial-lingual paresis is characteristically present with occasional masseter-palatal-laryngeal weakness. It seems that patients with capsular genu syndrome tend to present mild but definitive weakness in the extremities. In conjunction with this, all 3 patients who had weakness of the fingers among 9 patients described as pure dysarthria syndrome showed lesions in the genu area of the corona radiata. However, a few patients with capsular genu syndrome with normal somatic motor function were also reported who usually had lesions on the superior part of the internal capsule (or corona radiata). These rare cases overlap with dysarthria-facial paresis syndrome.

Our patients and previously reported cases with dysarthria-facial paresis syndrome tend to have small strokes on the corona radiata or basal ganglia abutting a portion of the internal capsule. In the corona radiata, the motor fibers may be more loosely packed than in the internal capsule, and a selective involvement of the corticobulbar fibers with sparing of the corticospinal tracts may be possible. In conjunction with this, in a clinical-radiological study of 63 patients with capsular infarct, all 4 who had isolated transient dysarthria showed infarcts in the superior portion of the internal capsule (or corona radiata). The lesions in the basal ganglia slightly abutting the internal capsule may also involve corticobulbar fibers selectively.

In the present study, infarcts on the subcortical-cortical bulbar motor area and in the paramedian pontine base were identified in 2 and 3 patients, respectively. Previously, Ichikawa and Kageyama described 1 patient with a lesion on the cortical bulbar motor area,
Table 2. Reported Cases With Dysarthria and/or Facial Paresis Without Significant Limb Weakness

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>No. of Cases</th>
<th>Symptoms and Signs</th>
<th>Lesion Site</th>
<th>Designation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huang and Broe (1984)</td>
<td>5</td>
<td>FP (5)</td>
<td>A, CR (4)</td>
<td>Isolated FP</td>
</tr>
<tr>
<td>Huang et al (1988, case 5)</td>
<td>1</td>
<td>FP (1)</td>
<td>Pons (1)</td>
<td>Pure supranuclear facial palsy</td>
</tr>
<tr>
<td>Hopf et al (1990)</td>
<td>2</td>
<td>FP (2)</td>
<td>Pons (2)</td>
<td>Supranuclear palsy</td>
</tr>
<tr>
<td>Bogousslavsky and Regli (1990, cases 3 and 5)</td>
<td>2</td>
<td>FP (2)</td>
<td>G, CR (1)</td>
<td>Capsular genu syndrome</td>
</tr>
<tr>
<td>Arboix et al (1991)</td>
<td>4</td>
<td>DA (2)</td>
<td>G, IC (1)</td>
<td>Isolated dysarthria</td>
</tr>
<tr>
<td>Ichikawa and Kageyama (1991)</td>
<td>9</td>
<td>DA (9)</td>
<td>A, IC or</td>
<td>Pure dysarthria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FP (5)</td>
<td>A, CR (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increased reflexes (5)</td>
<td>G, CR (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abnormal finger-tapping (3)</td>
<td>Frontal cortex (1)</td>
<td></td>
</tr>
</tbody>
</table>

FP indicates facial paresis; DA, dysarthria; DP, dysphagia; LP, lingual paresis; A, anterior portion; CR, corona radiata; P, posterior portion; IC, internal capsule; G, genu portion; and CSO, centrum semiovale. Values in parentheses are number of patients.

and Hopf et al reported 2 patients with paramedian pontine infarcts, suggesting that lesions in these areas may also involve the corticobulbar fibers without affecting the motor fibers for extremities. When Fisher originally described pure dysarthria syndrome as one of the lacunar syndromes, he suggested the pontine base as a responsible site.

The size of the lesion in our series was generally less than 1.5 cm in its longest diameter, suggesting that dysarthria-facial paresis syndrome is most often caused by lacunar stroke. Unfortunately, angiographic study was performed only in 4 patients. Nevertheless, the presence of large arterial occlusion (patient 5), cardiac arrhythmia (patient 13), and diffuse atherosclerotic vessels (patient 9) along with the cortical localization of 2 patients suggests that stroke mechanisms other than lacunae may also be responsible for this syndrome. It is noticeable that 5 of 9 patients described by Ichikawa and Kageyama had a lesion size greater than 1.5 cm in diameter. Finally, although the clinical course of the patients with dysarthria-facial paresis syndrome is generally benign, 2 of our patients developed subsequent major strokes in several months.

In summary, dysarthria-facial paresis syndrome, a probable variant of dysarthria-clumsy hand syndrome, consists of a combination of dysarthria and lower facial paresis but without definitive somatic motor dysfunction. Pure dysarthria or isolated facial paresis syndrome may be considered as an extreme of this syndrome, which is attributed to the small unilateral strokes involving the corticobulbar tract but strategically sparing the corticospinal fibers in the regions of the corona radiata, basal ganglia–internal capsule, subcortical-cortical motor area, or paramedian pontine base.

References


Pure dysarthria, isolated facial paresis, or dysarthria-facial paresis syndrome.

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doi: 10.1161/01.STR.25.10.1994

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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