

Single Case – General Neurology

Dysarthria-Facial Paresis and Rostral Pontine Ischemic Stroke

Edison K. Miyawaki^a Douglas R. Wilcox^a Andrew W. Kraft^a
Jeffrey P. Guenette^b

^aDepartment of Neurology, Mass General Brigham, Harvard Medical School, Boston, MA, USA; ^bDepartment of Radiology, Mass General Brigham, Harvard Medical School, Boston, MA, USA

Keywords

Dysarthria · Facial paresis · Pons · Ischemic stroke

Abstract

We describe an acute, postoperative dysarthria-facial paresis. While the rare stroke syndrome has been described previously, we present an under-described clinical nuance to its presentation with a particularly clear imaging correlation. A 78-year-old, right-handed man with a past medical history of aortic stenosis presented after a transcatheter aortic valve replacement. Immediately postoperatively, no neurological deficits were noted. That evening, he described his speech as “drunken.” He was later noted to have a right lower facial droop in addition to the speech change. His speech exhibited labial, lingual, and (to a lesser degree) guttural dysarthria. At the patient’s request due to claustrophobia, he received 2 mg of oral lorazepam prior to cranial imaging. Afterwards, he was sleepy but arousable, yet was unable to put pen to paper when asked to write. Right lower facial paresis persisted, but he now demonstrated a right pronator drift, which resolved after 14 h without other evolution to his clinical examination. Brainstem lesions above the level of the pontine facial nucleus may present with central facial paresis contralateral to the lesion. An associated dysarthria may have both labial and lingual features in the absence of tongue or pharyngeal weakness. Our review of reported cases of dysarthria in isolation, dysarthria in combination with facial paresis, and facial paresis finds that all presentations may result from cortical, subcortical, or brainstem involvement. Stroke mechanisms are most commonly thromboembolic or small-vessel-ischemic in either the anterior or posterior circulations.

© 2022 The Author(s).
Published by S. Karger AG, Basel

Correspondence to:
Edison K. Miyawaki, edison_miyawaki@hms.harvard.edu

Introduction

Dysarthria-facial paresis is an uncommon variant of pure motor hemiparesis. Responsible lesions in one small series ($n = 13$) included infarcts of corona radiata with or without involvement of the basal ganglia, of basal ganglia with extension to internal capsule, and infarcts in the rostral pons and basis pontis; pontine lesions accounted for 5 of the 13 [1]. In a prospective study of 2,500 acute strokes over a 12-year period, dysarthria-facial paresis was observed in 12 cases; an additional 9 cases presented as isolated dysarthria [2]. We report a case of dysarthria-facial paresis associated with a high pontine stroke contralateral to the side of the facial paresis. The motor examination transiently evolved after use of a benzodiazepine, then reverted to isolated dysarthria and facial paresis.

Case Report/Case Presentation

A 78-year-old, right-handed man with a past medical history of aortic stenosis presented after a transcatheter aortic valve replacement. Immediately postoperatively, no neurological deficits were noted. That evening, he described his speech as “drunken.” The following morning, he was noted to have a right lower facial droop in addition to the speech change. At no point during the hospitalization, did the patient describe a headache. Neurologic examination found an intact sensorium without dysnomia, paraphasia, or disturbance in repetition aside from slurred words; he followed multistep commands without difficulty. Visual fields were full. Cranial nerve examination was notable for a lower right facial paresis at rest and with activation. Forehead wrinkle and eyelid closure were symmetrical. His speech exhibited labial, lingual, and (to a lesser degree) guttural dysarthria, with particular difficulty articulating “ma-ma-ma” and “la-la-la.” Eye movements were full without nystagmus, lateropulsion, internuclear ophthalmoparesis, or skew deviation on cover/uncover testing. The pupils were symmetrical in both a well-lit and darkened room. The palate elevated promptly and symmetrically. The tongue protruded in the midline. Motor examination found no pronator drift or focal deficit on confrontational testing. Sensory examination was intact to vibration and temperature on the face and throughout the body. Deep tendon reflexes were symmetrical; toes were downgoing. There was no appendicular dysmetria. The patient and his wife gave their written informed consent both to publish their case and to use full-face images to illustrate the eyes and forehead on both sides (Shown in Fig. 1).

At the patient’s request due to claustrophobia, he received lorazepam (2 mg orally) prior to cranial magnetic resonance (MR) imaging. Afterwards, he was sleepy but arousable, yet was unable to put pen to paper when asked to write. Right lower facial paresis persisted, but he now demonstrated a right pronator drift. Imaging results are shown in Figure 2. A postoperative echocardiogram found no source of clot. The following morning, his pronator drift was no longer present. He continued to exhibit facial asymmetry at rest and with activation.

Discussion/Conclusion

Our case is of interest for three reasons. First, a deficit initially restricted to the face appeared to blossom in the context of lorazepam sedation. The benzodiazepine, whose half-life is approximately 12 h, appeared responsible for a new deficit (arm weakness) that was not present before it was given. When examined again, 14 h after the pronator drift had first been noted, the patient was no longer sleepy, and his arm weakness was no longer present. Serial examination argued against stroke progression. There was no subsequent



Fig. 1. **a** Subtle flattening of the right nasolabial fold at rest. **b** The asymmetry is clearer with activation. Written permission was obtained to use these specific photographs.

deterioration in his neurological examination. During his admission, at the time of his initial evaluation before his MR imaging, his National Institutes of Health Stroke Scale (NIHSS) score was 2 (1 for the facial paresis on task 4; 1 for the dysarthria on task 10). At the time of discharge, his NIHSS scale remained 2. But, of note, within 10 days after first evaluation, the patient's deficits improved on 81 mg of aspirin per day, in addition to 10 mg of pravastatin (three times per week) that he had been taking prior to his valve replacement. Given the perioperative timing of the patient's presentation, even in the absence of evidence for a cardiac source of clot on his postoperative echocardiogram, an embolic mechanism was deemed most likely for his stroke.

Second, the facial paresis related to a rostral pontine lesion contralateral to the paresis. Size of the lesion by imaging might have predicted more of a clinical motor deficit on first examination, although diffusion tensor imaging of the corticospinal tract at different levels of the pons in normal persons suggests that corticospinal tract occupies more overall space, with less fractional anisotropy, in rostral versus caudal pontine locales [3]. An implication is that rostral pontine lesions may present with formes frustes of contralateral, pure motor hemiparesis, as our case illustrates.

Third, we were interested in the relationship between types of dysarthria and a brainstem stroke. Fisher's early report on dysarthria-clumsy hand syndrome, which our case resembles, described 3 patients [4]. All three had unilateral facial weakness. One of the three had a slight deviation of the tongue to the right, but the nature of the dysarthria is described only as moderately slurred speech. In the remaining two, difficulty in uttering "la-la-la" is mentioned

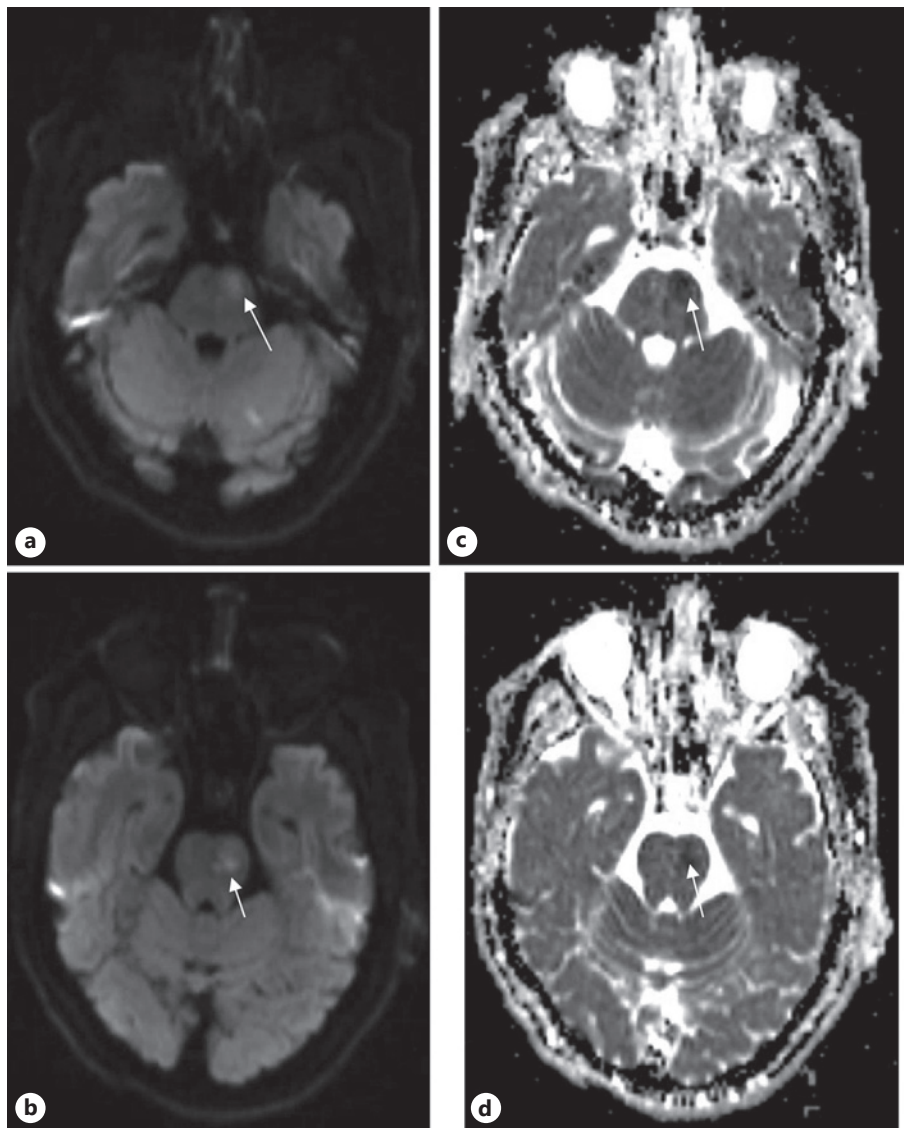


Fig. 2. Diffusion-weighted b1000 images (a, b) and associated apparent diffusion coefficient maps (c, d) show a focus of diffusion restriction in the anterior brainstem at the junction of the midbrain and pons. Minimal-associated T2 fluid-attenuated inversion recovery signal abnormality was present. These findings, all indicated with arrows, are consistent with an acute infarct. See text for additional commentary.

specifically. We observed that our patient’s articulation problem was more likely labial or lingual than guttural; no tongue deviation or palatal abnormality was noted over the course of serial examinations prior to his discharge from hospital. After discharge, his deficits cleared after 2 weeks.

In the pre-MR literature, isolated unilateral, lower facial palsy has been reported as a lacunar syndrome with responsible lesions identified in contralateral internal capsule or corona radiata; in 4 of 5 cases in an early report, dysarthria or a speech disturbance was present [5]. Presence of dysarthria may not help distinguish a brainstem from a higher localization in cases of isolated lower or central facial weakness: in a prospective study of 101 consecutive cases of sudden-onset dysarthria radiographically evaluated by DWI, a majority (63%) had supratentorial lesions, often in multiple locations [6].

Based on an EMBASE review from January 2015 to December 2021, using the search terms “dysarthria,” “facial paresis,” and “stroke,” we identified additional abstracts or papers to help elucidate stroke localizations and mechanisms associated with isolated dysarthria, dysarthria and central facial paresis, or isolated central facial paresis.

In a cohort of 879 patients presenting with NIHSS scores of 3, 9 (0.4%) exhibited central facial paresis with or without dysarthria [7]. Seven of the 9 patients had flow-limiting thromboembolic, mid-to-distal M1-segment/proximal M2-segment middle cerebral artery disease; the authors argued that thromboembolism, rather than small-vessel-associated lacunar infarction, might account for the majority of cases with presentations similar to our case.

In a large study examining stroke outcomes in England, Wales, and Northern Ireland from 2013 to 2015 [8], authors focused on impairments in verbal communication, whether related to aphasia or dysarthria or both. Specifics of the clinical examinations of 88,974 stroke survivors are not available in the report, but the authors interestingly observe that stroke severity rather than type of stroke (intracerebral hemorrhage vs. infarction) is associated with likelihood of communication impairment. “Mild” stroke more commonly affected articulation rather than language (26% with dysarthria vs. 17% with aphasia among those with NIHSS scales <5 at time of presentation).

In another large study of stroke outcome (2012–2014) in the Republic of Korea [9], among 3,929 patients seen at 6 months after their first stroke, 149 (0.4%) had either isolated facial palsy or isolated dysarthria (30 with facial palsy; 110 with dysarthria). Ischemic infarction accounted for the preponderant majority (84.6% in the facial palsy group; 90% in the dysarthria group); stroke localizations were not reported.

Utilizing a database of all consecutive acute ischemic strokes (2011–2014) in three hospitals in Japan [10], 65 of 2,216 patients (3.1%) presented either with “pure” dysarthria or dysarthria-facial paresis. The authors were specifically interested in first strokes in which a single lesion involved internal capsule and/or corona radiata. Thirteen patients with pure dysarthria and 18 with dysarthria-facial paresis were included in their analysis. Compared to the dysarthria-facial paresis group, pure dysarthria was associated with *left* subcortical lesions (in 12 of 13 cases) and less stroke volume – the latter as assessed by an ABC/2 method applied to diffusion-weighted imaging (DWI) [11]. Patients with dysarthria-facial paresis exhibited larger lesions (median 828 vs. 285 mm³) either in the left or right hemisphere, often involving medial-posterior putamen and the genu and posterior limb of the internal capsule; lesions involving corona radiata were more medial with involvement of the caudate body in the dysarthria-facial paresis group compared to the pure dysarthric cases. Twenty-nine of the 31 patients were reevaluated 90 days after stroke onset; in 21 (72%), deficits had resolved. Given the selection criteria, these data do not reflect other possible foci of stroke.

In reports of dysarthria and/or facial paresis associated with other localizations, neighborhood signs were often observed, including (in a series of midbrain ischemic infarction or hemorrhage) limb paresis and ipsilateral oculomotor palsy [12], dysphagia in patients with dysarthria as a presenting feature [13], and (in a series of lateral medullary infarctions) truncal ataxia and palatal weakness [14]. In a single case report [15], supranuclear facial palsy (albeit with accompanying homolateral arm and leg weakness) was observed in a case of basilar dissection and high pontine infarction.

In the present report, we describe a case of dysarthria-facial paresis with MR correlation. We found no evidence for stroke on inspection of DWI and apparent diffusion coefficient sequences in the anterior circulation. MR angiography did not evince either M1- or M2-segment middle cerebral artery compromise. In the posterior circulation, aside from the pontine finding shown in Figure 2, a very small area of DWI brightness is also apparent in the left cerebellar hemisphere, perhaps as a consequence of the same embolic process that gave rise

to the pontine lesion. Absent a cardiac source on postoperative imaging, and in the context of a periprocedural stroke, we opted for treatment with low-dose aspirin alone, without an additional antiplatelet drug or other anticoagulation.

Brainstem lesions above the level of the pontine facial nucleus may present with central facial paresis contralateral to the lesion. An associated dysarthria may have both labial and lingual features in the absence of tongue or pharyngeal weakness. Dysarthria and facial paresis together, in the absence of other clinical deficits, have traditionally been considered uncommon, dating to early reports of pure motor hemiparesis and other “lacunar” syndromes. Low, but variable percentages (0.4–3.1%), have been reported in the literature since 2015. Accompanying deficits aside from dysarthria and central facial paresis may clue the clinician to nonpontine localizations, and the particular combination described in this report has been associated with infarction in various locations in both anterior and posterior circulations, although clinical examination details are not always specified in large prospective and retrospective series. Types of pure motor hemiparesis exist, and, as this case illustrates, the examination may serially reveal variant aspects, whether as a result of stroke evolution or other factors, including toxic-metabolic contributions.

Acknowledgments

The authors acknowledge Drs. Kelly Rogers, Lena Liu, Sarah Abou Alaiwi, all at Mass General Brigham, Boston, and Harvard Medical School students Susan Gonzales and Natalie Sadlak for their care of the patient.

Statement of Ethics

The authors have complied with the guidelines for human studies and conducted themselves ethically in accordance with the World Medical Association Declaration of Helsinki. This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Written informed consent was obtained from the patient and his wife for publication of the details of his medical case and all accompanying images.

Conflict of Interest Statement

The authors report no conflicts of interest.

Funding Sources

No funding was received.

Author Contributions

E.K.M. wrote the first draft of the paper which was reviewed by each coauthor (D.R.W., A.W.K., J.P.G.), all of whom contributed to the clinical care of the patient, the literature review, and editing of the manuscript; all authors' textual changes and additions were incorporated. J.P.G. contributed the radiographic images and their interpretation.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries may be directed to the corresponding author.

References

- 1 Kim JS. Pure dysarthria, isolated facial paresis, or dysarthria-facial paresis syndrome. *Stroke*. 1994;25(10):1994–8.
- 2 Arboix A, López-Grau M, Casanovas C, García-Eroles L, Massons J, Balcells M. Clinical study of 39 patients with atypical lacunar syndrome. *J Neurol Neurosurg Psychiatry*. 2006;77(3):381–4.
- 3 Seo JP, Jang SH. Characteristics of corticospinal tract area according to pontine level. *Yonsei Med J*. 2013;54(3):785–7.
- 4 Fisher CM. A lacunar stroke. The dysarthria-clumsy hand syndrome. *Neurology*. 1967;17(6):614–7.
- 5 Huang CY, Broe G. Isolated facial palsy: a new lacunar syndrome. *J Neurol Neurosurg Psychiatry*. 1984;47(1):84–6.
- 6 Kumral E, Celebisoy M, Celebisoy N, Canbaz DH, Calli C. Dysarthria due to supratentorial and infratentorial ischemic stroke: a diffusion-weighted imaging study. *Cerebrovasc Dis*. 2007;23(5–6):331–8.
- 7 Sands KA, Shahripour RB, Kumar G, Barlinn K, Lyerly MJ, Haršány M, et al. Central facial palsy as manifestation of middle cerebral artery ischemia. *J Neuroimaging*. 2016;26(5):499–502.
- 8 Mitchell C, Gittins M, Tyson S, Vail A, Conroy P, Paley L, et al. Prevalence of aphasia and dysarthria among inpatient stroke survivors: describing the population, therapy provision and outcomes on discharge. *Aphasiology*. 2021;35(7):950–60.
- 9 Chang WH, Sohn MK, Lee J, Kim DY, Shin Y-I, On G-J, et al. Impact of central facial palsy and dysarthria on quality of life in patients with stroke: the KOSCO study. *NeuroRehabilitation*. 2016;39(2):253–9.
- 10 Yamaguchi N. The clinical characteristics of stroke-induced dysarthria: a retrospective study. *J Neurol Sci*. 2017;381(Suppl 1):1124.
- 11 Sims JR, Gharai LR, Schaefer PW, Vangel M, Rosenthal ES, Lev MH, et al. ABC/2 for rapid clinical estimate of infarct, perfusion, and mismatch volumes. *Neurol*. 2009;72(24):2104–10.
- 12 Tanaka K, Yamada T, Torii T, Yoshimura T, Takase K-I, Togao O, et al. Pure dysarthria and dysarthria-facial paresis syndrome due to internal capsule and corona radiata infarction. *BMC Neurol*. 2015;15(1):184.
- 13 Yin L, Sun Q, Ma C. Clinical and imaging analyses of pure midbrain stroke in 21 cases. *Eur J Neurol*. 2015;22(Suppl 1):779.
- 14 Ogawa K, Suzuki Y, Oishi M, Kamei S. Clinical study of 46 patients with lateral medullary infarction. *J Stroke Cerebrovasc Dis*. 2015;24(5):1065–74.
- 15 Mittal S, De Georgia M. Pontine infarct due to isolated basilar artery dissection causing supranuclear facial palsy. *Neurol*. 2015;84(Suppl 14):P5156.