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Commentary on the paper by Walsh et al. “Bilateral cerebellar stroke presenting with acute dysphonia and late palatal tremor”

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The case report by Walsh et al. described the development of spasmodic dysphonia (SD) secondary to cerebellar stroke. The patient was presented with hyperfunctional, hyperadductive voice disorder affecting supraglottis, including ventricular (false) cords. Speech was incomprehensible with strained-strangled quality and occasional voice arrests. The diagnosis of SD was made based on these speech symptoms, hyperfunctional supraglottis visualized with fiberoptic laryngoscopy, the lack of benefits from voice and speech therapy, and some improvement of voice problems following unilateral botulinum toxin injection into the thyroarytenoid muscle.

As such, this is an atypical SD case due to several reasons. Laryngeal spasms in SD primarily affect (true) vocal cords rather than ventricular cords, although supraglottic hyperadduction involving ventricular cords may also be present. While SD patients have problems with coordinating voice onset and offset during connected speech due to SD-specific voice breaks, they usually are able to sustain sound, and their speech remains comprehensible. The lack of response to speech therapy and the benefits from botulinum toxin injections into the laryngeal muscles are typical but not specific to SD only. On the other hand, speech therapy is beneficial in patients with supraglottic hyperadduction, whereas persistent supraglottic squeeze may be managed more successfully with botulinum toxin injections into the supraglottic musculature¹.

This case report further suggests that cerebellar lesion without functional, structural or dopaminergic abnormalities in any other brain regions may cause SD. Conversely, previous studies have shown relief of dystonia symptoms following cerebellar lesions and cerebellectomy^{2, 3}, and SD has been characterized as a brain network disorder, involving abnormalities in the basal ganglia, thalamus, sensorimotor cortex, and cerebellum^{4, 5}.

In conclusion, the diagnosis of SD in this patient is complicated by unusual clinical presentation, atypical diagnostic criteria, and the presence of cerebellar pathology without abnormalities in other dystonia-related brain regions. The differential diagnosis is more likely to include other cerebellar disorders, such as spinocerebellar atrophy, other movement disorders, such as chorea, and muscle tension dysphonia, as a compensatory voice disorder secondary to cerebellar pathology.

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