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Dystextia and dystypia as modern stroke symptoms: A case series and literature review

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ABSTRACT

Stroke recognition remains a barrier to care in cerebrovascular disease. Despite an increasing reliance on digital communication, the clinical utility of deficits relating to technology remains unexplored. Dystextia and dystypia, terms used to refer to impairments in texting and typing, respectively, may serve as modern indicators of stroke and provide information regarding stroke duration, symptomatology, and etiological diagnosis. In this report, we describe two cases in which dystextia and dystypia were involved in stroke presentation and perform a literature review surrounding these signs. Four out of six cases identified on literature review in which stroke etiology was described, in addition to both of our presented cases, were found to be embolic in origin. While shared lesion topography involving the left posterior upper insular cortex and superior longitudinal fasciculus was identified in our cases, additional research is required for proper symptom-lesion mapping. Further characterization of dystextia and dystypia, and their corresponding localization, may assist in stroke diagnosis and guide investigations.

1. Introduction

Human communication is shifting from speaking to texting and typing at an unprecedented speed with a multifold impact on society. Among the effects of this change, we are witnessing an evolution in our recognition and understanding of stroke. Texting is prevalent among those older than 50, the demographic at the highest risk of stroke [1]. In this context, dystextia and dystypia (deficiencies in texting and typing, respectively) [2,3] have emerged as signs of neurological injury.

While a medical approach to agraphia has been established [4], no such framework for dystextia and dystypia exists. The clinical and anatomical characterization of dystextia and dystypia may not only allow for the identification of neurological injury, but may also provide clues regarding the nature and chronology of the symptoms in question. Here, we perform a review of literature surrounding these signs and present two cases which illustrate the importance of these *modern* clinical presentations.

2. Case reports

Case 1. A 47-year-old right-handed woman with a history of smoking and no vascular risk factors had been last seen well at 08:00. At 08:50, she erratically texted her daughter (Fig. 1a), who then called her. Upon noting word finding difficulties, the patient was taken to the hospital, where a hyperacute stroke protocol was initiated.

Significant findings on examination included mild right-sided nasolabial fold flattening, anomia with paraphasias, difficulty with repetition and reading, and impaired fluency. A National Institutes of Health Stroke Scale score of 5 was assigned, with one point allocated each for level of consciousness questions, level of consciousness commands, right facial palsy, and two points for language.

A computed tomography angiogram (CTA) demonstrated perfusion mismatch in the left middle cerebral artery (MCA), and recombinant tissue-type activating plasminogen was administered. Symptoms improved thereafter, although fluency, repetition, reading, and writing difficulties persisted.

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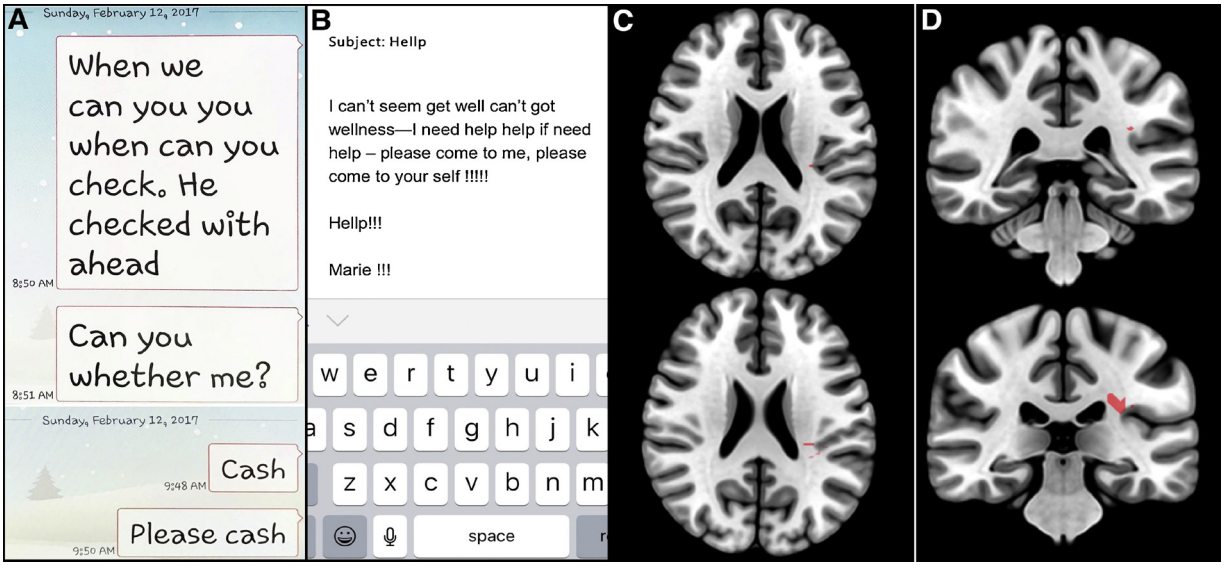

Fig. 1. Patient text messages and emails demonstrative of dystextia and dystypia. Lesion overlap of areas of infarction corresponding to cases 1 and 2 is also demonstrated. a) case 1, b) case 2. In c) and d), areas in red demonstrate shared infarction territory involving the posterior insula and white matter portions corresponding to the trajectory of the superior longitudinal fasciculus. Overlap imaging was generated using Statistical Parametric Mapping 12 clinical toolbox software (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

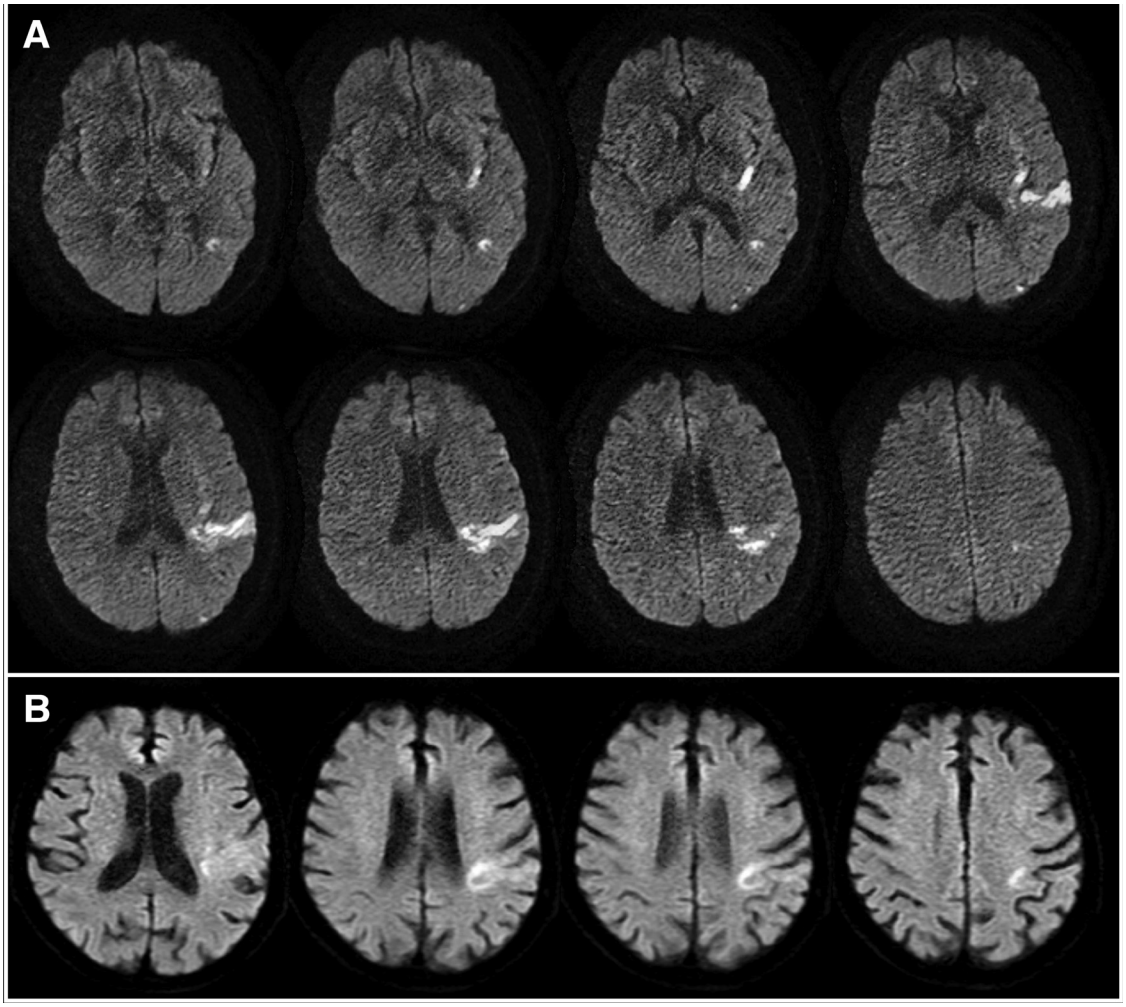

Fig. 2. Diffusion weighted imaging sequences from patients with dystextia and dystypia; a) case 1, b) case 2.

Follow-up magnetic resonance imaging (MRI) demonstrated foci of restricted diffusion in the left posterior insula, temporal operculum, subcortical white matter, and frontal, occipital, and parietal cortices (Fig. 2a). Etiological investigations were unremarkable apart from a patent foramen ovale on echocardiogram.

Case 2. A 90-year-old right-handed woman with atrial fibrillation developed apparent sudden-onset confusion and motor difficulty at 21:00 and contacted her family. Her email appeared to reflect perseveration and anomia, and her speech was described as being garbled (Fig. 1b). She was taken to the hospital and found to have word finding difficulties and dysarthria.

A CTA demonstrated low attenuation in the left subinsular cortex with thrombus in the insular branch of the MCA. Intravenous thrombolysis was not administered as this patient had initially been taken to a non-stroke centre and elapsed the 4.5-hour treatment window.

Follow-up MRI revealed an ischemic infarct in the left posterior insular cortex and subcortical white matter of the left frontoparietal parenchyma (Fig. 2b). The patient was anticoagulated with warfarin 5 mg daily, and two months later, continued to have expressive aphasia, dystypia, and agraphia.

3. Literature review

A summary of the reports identified during literature review is provided in Table 1 of the supplementary file. In total, six cases of dystextia, four cases of dystypia, and one case involving both deficits were identified. Among them, 64% involved aphasia and 82% were secondary to stroke. The two cases not attributable to stroke were, as per the authors of the original reports, secondary to migraine headache and iatrogenic (post deep brain stimulation). Aphasia occurred in 86% and 40% of cases involving dystextia and dystypia, respectively. Apraxia was documented in only two out of the 11 cases found in the review.

As two of the cases involved non-stroke etiologies, and three others did not discuss the etiology of the reported stroke, a stroke mechanism was assigned to only six of the 11 patients. One case was attributable to small vessel occlusion, two were the result of large vessel atherosclerosis, two were classified as cardioembolic, and one was deemed to be of unknown etiology. The left posterior insula was involved in 3 cases among 9 in which stroke topography was reported, and 8 out of 9 of these cases reported lesions involving the left anterior cerebral circulation.

4. Discussion

We report three main findings based on our cases. First, dystextia and dystypia could serve as important indicators of acute stroke, prompting the activation of urgent stroke care protocols. Second, although limited by the anecdotal cases hereby presented, qualitative lesion mapping suggests a common site of injury in the left posterior upper insula and white matter tracts corresponding to the left superior longitudinal fasciculus (Fig. 1c, d). Third, an embolic stroke mechanism was identified, consistent with the presumed cortical localization of language deficits.

In circumstances where patients are unaccompanied, dystextia and dystypia may be the sole identifiable signs resulting in emergency care. Although the time of onset is indeterminable based on the time of message delivery, it would indicate that symptoms were present at that time. Additionally, patients with transient ischemic attack often have difficulty describing their symptoms post event resolution, which is clinically problematic. Messages from patients with dystextia and dystypia offer objective information to complete this task, although they must be interpreted within the context of spell checking software and socially acceptable grammatical and spelling errors.

Currently, the cerebral regions responsible for dystextia and dystypia remain uncertain. While our cases, and three of those found on literature review, demonstrate shared lesion topography in the left posterior insula, large cohorts with a variety of lesions are required for mapping of these symptoms. Preliminarily, our observations in these two cases and findings on literature review may encourage further research on the relationship between dystextia, dystypia and the perisylvian cortex.

Lastly, as found in our cases and in four of six cases with an assigned stroke mechanism on literature review (Table 1, Supplemental file), dystextia and dystypia are likely cortical symptoms indicating underlying embolic mechanisms. This is similar to reports regarding isolated aphasia [5]. Accordingly, these symptoms should prompt investigations regarding sources of artery to artery or cardiac embolism.

5. Conclusion

Dystextia and dystypia may serve as modern indicators of stroke. Preliminary observations prompt further investigations on the role of the left posterior upper insular cortex in these deficits, likely as a proxy of the white matter tracts underlying the perisylvian region. Systematic research is needed for proper lesion-symptom mapping. The development of an approach to dystextia and dystypia and their inclusion into the neurological palette of stroke symptoms may be of clinical utility.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.clineuro.2019.02.001.

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