

A Case of Pure Word Mutism: Don't Be Fooled!

A case of pure word mutism, often called "small Broca's aphasia," in a 44-year-old woman with a psychiatric history and a long history of unresolved, vague neurologic symptoms is presented. Although hysterical reaction was high on the list of differential diagnoses due to the patient's medical history, cranial computed tomography performed in the emergency department revealed an infarct in the distribution of the middle cerebral artery, which probably caused her condition. The patient improved slightly during hospitalization and was discharged on the sixth hospital day with a moderate expressive aphasia for which she received intensive speech therapy. [Linett LM: A case of pure word mutism: Don't be fooled! Ann Emerg Med November 1989;18:1233-1236.]

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INTRODUCTION

Speaking, as a means of communication, is so common it is often taken for granted. Pure word mutism, or "verbal apraxia,"¹ is an unusual clinical entity in which the patient exhibits a complete expressive aphasia despite intact comprehension and undisturbed writing ability. It is believed to arise from a lesion that disconnects the motor cortex for speech from lower centers.²

A case of pure word mutism associated with right-hand numbness and hemiparesis is presented. The patient's medical history included atypical migraine headache and unresolved vague neurologic symptoms. She had been referred for psychiatric evaluation. Although hysterical reaction was strongly considered, cranial computed tomography (CT) revealed an infarction in the distribution of the middle cerebral artery, which probably caused her symptoms.

CASE REPORT

A 44-year-old, right-handed woman presented to the emergency department with the acute onset of aphasia and right-hand weakness and numbness. The patient was otherwise healthy and had been taking alprazolam during the past year for anxiety and panic attacks. Her husband provided additional history by saying that his wife had telephoned him at work earlier in the day and her speech had been "garbled and slow" and she was complaining of a "numb feeling in her right hand, like it was asleep." When her husband arrived home shortly thereafter, his wife was totally unable to speak, although otherwise asymptomatic.

At the time of initial evaluation in the ED, the patient was able to write legibly and coherently on a notepad. When she attempted to speak, however, she became increasingly frustrated and began to cry. She was not able to whisper, hum, or mouth words either spontaneously or in imitation. Her comprehension appeared intact in that she could follow simple written and verbal instructions. Questions were answered by nodding to indicate yes or no. Acts such as licking lips and rolling the tongue were easily performed, as were chewing and swallowing. She denied headache, diplopia, photophobia, stiff neck, or syncope. She nodded yes to subjective numbness and weakness of the right hand.

Vital signs were entirely within normal limits as was the complete non-neurologic assessment. Indirect laryngoscopy showed normal larynx and vocal cord movement. On neurologic evaluation, the patient was alert and

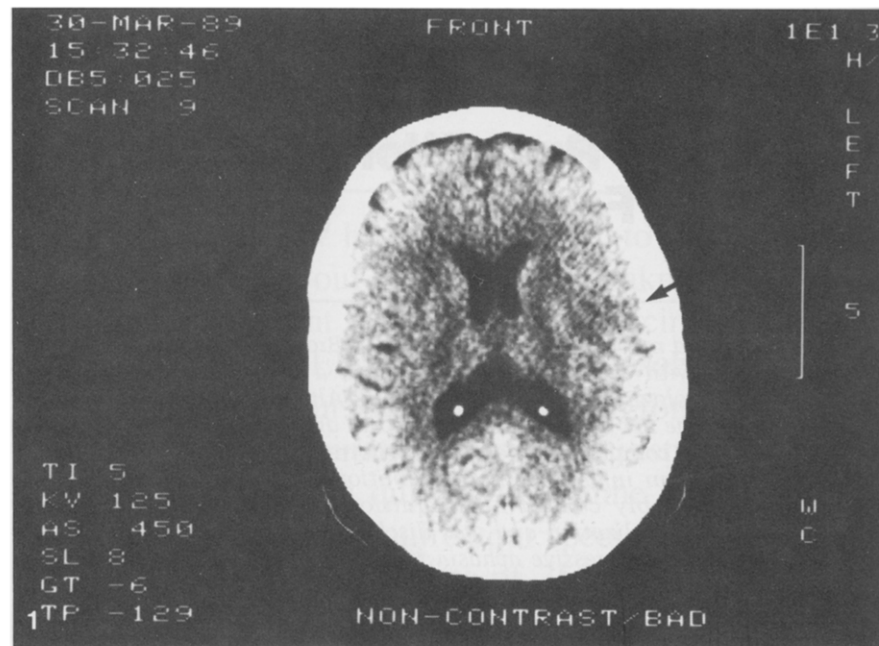
FIGURE 1. Nonenhanced cranial CT scan showing an early nonhemorrhagic infarct of the left temporal and parietal regions (arrow).

oriented (by writing) to person, place, and time. Cranial nerves were intact without visual field deficit. She exhibited a 3/5 grip of the right hand as compared with a 5/5 grip of the left as well as a diminished response to pinprick and light touch on the right hand only. Reflexes were symmetrical, and plantar responses were flexor. Gait and cerebellar testing were normal.

A review of her medical records revealed that the patient had a several-year history of atypical migraine headaches and had been seen on numerous occasions by her neurologist for vague complaints of numbness and paresthesia of the lips, face, arms, and legs, for which no etiology had been found. She had been referred for psychiatric testing and evaluation, but those records were not available.

Initial laboratory results revealed a WBC count of 8,300/mm³ (74% segmented neutrophils, 11% band forms, 9% lymphocytes, 3% monocytes, and 3% eosinophils). Platelets were 123,000/mm³; hemoglobin, 13.6 g/dL; and hematocrit, 40.2%. Electrolytes, BUN, and glucose were normal. Chest radiograph, ECG, and urinalysis were within normal limits. Although a hysterical reaction was considered part of the differential diagnosis, a CT scan was considered necessary to rule out other pathology. With some reluctance, the patient agreed to the study provided no contrast agent was used. The scan revealed an early nonhemorrhagic infarct of the left temporal and parietal regions in the distribution of the middle cerebral artery (Figure 1).

The patient was hospitalized with a diagnosis of left cerebrovascular accident. Magnetic resonance imaging of her head the next day showed a dominant signal abnormality involving the left insular cortex as well as scattered gray matter signal abnormalities in the high left parietal region, which are suspicious of a cortical infarction (Figure 2). There were also scattered white matter signal abnormalities consistent with a demyelinating process believed to be of separate etiology. Other hospital test-



ing included waking EEG, lumbar puncture, noninvasive carotid echography, and rheumatologic evaluation, all of which were essentially within normal limits.

The patient's speech improved slightly during her hospitalization. She was able to say simple words and begin phrases, although slowly and with much difficulty. Based on the improvement of her condition, she was discharged on the sixth hospital day for intensive speech therapy follow-up.

DISCUSSION

The dominance of one cerebral hemisphere, most often the left, emerges with speech and right hand preference, particularly for writing. A lack of development or loss of cerebral dominance as a result of disease entails a disturbance of both of these traits.³ Although speech and language are closely interwoven functions, they are not strictly synonymous. A language dysfunction is always a reflection of a brain abnormality in the dominant cerebral hemisphere. A speech disorder may or may not have a similar origin — it may be due to abnormalities in different parts of the brain or to extracerebral mechanisms.²

Even in left-handed individuals, the left hemisphere is usually the most important hemisphere for speech. Anatomically, in 65% of

brains, part of the temporal lobe appears to be larger in the left hemisphere than its counterpart (an area located behind the transverse gyrus), and this area is believed to have particular importance for speech. In addition, Broca's area, located in the region of the operculum of the frontal lobe, is a vital anatomic speech area. Other parts of the left cerebral cortex in the temporal and parietal lobes are also considered crucial.^{4,5}

In the classification of aphasic disorders, there are three major syndromes and a multitude of less-common entities.⁶

First, global or total aphasia usually results from an infarction in the distribution of the internal carotid artery or middle cerebral artery; tumor and trauma are other possibilities. The result is often a large lesion located in the language area of the major cerebral hemisphere. By definition, all aspects of speech and language are affected. Patients are unable to read and write, and their comprehension is poor for spoken and written words. This type of aphasia is the most severe and has the worst prognosis.⁷

Second, in Wernicke's (sensory or central) aphasia, disruption of the entire array of language is evident. This "fluent" aphasia occurs due to infarction of the lower division of the middle cerebral artery (tumor and infection are other possible causes) result-

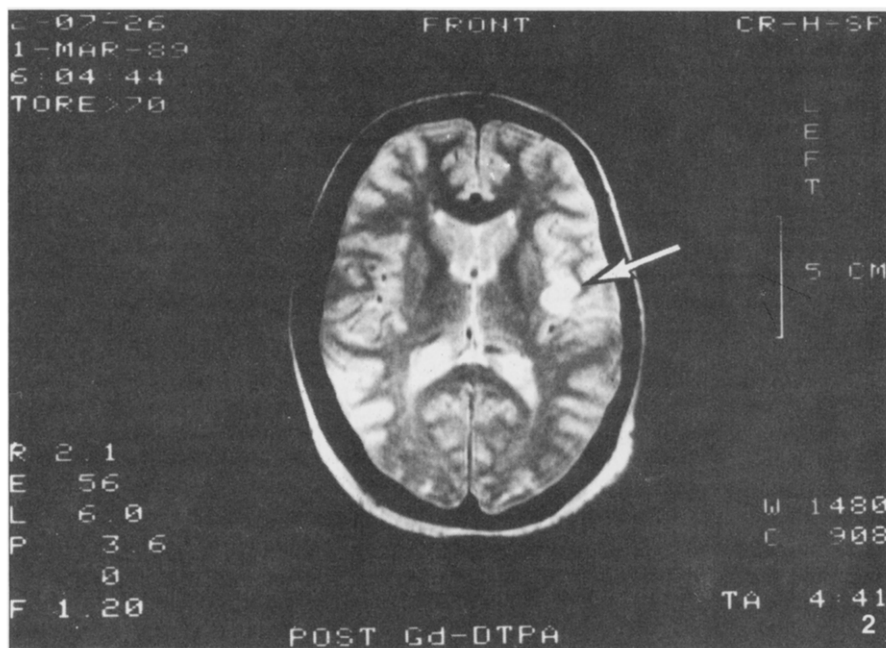


FIGURE 2. Magnetic resonance imaging showing a dominant signal abnormality involving the left insular cortex as well as scattered gray matter signal abnormalities in the high left parietal region suspicious of a cortical infarction (arrow).

ing in lesions of the posterior perisylvian structures of the parietal and temporal lobes. Total incomprehension of spoken speech as well as the inability to read or repeat aloud results, although speech is fluent. Alexia and agraphia are common components of this syndrome.⁸

Finally, Broca's or motor aphasia involves infarction or hemorrhage in the distribution of the superior frontal branch of the middle cerebral artery. The result is a cortical and subcortical lesion of the prefrontal and frontal regions. This so-called non-fluent aphasia involves failure of the motor aspects of speaking and writing. Although there usually exists a less-obvious impairment in language comprehension than in Wernicke's, patients may run the gamut from virtually normal comprehension to severe difficulty in understanding spoken and written language. Associated clinical symptoms often include weakness or numbness of the lower part of the face and arm on the opposite (right) side and, occasionally, the leg as well. Visual field disturbance is uncommon.⁶

A closely related syndrome, pure word mutism, which this patient exhibited, also results in wordlessness but leaves writing undisturbed. This is a rare clinical entity believed to be anatomically a disconnection of the motor cortex for speech from lower centers. In mutism, a syndrome

closely allied to what Mohr has called "small Broca's aphasia," the patient loses all capacity to speak but retains the ability to write, understand spoken words, and read silently with comprehension.² It may occur in patients with gross dementia, bilateral basal ganglion lesions,⁹ pseudo-bulbar palsy,¹⁰ or the syndrome of akinetic mutism.¹¹ In these instances, the underlying cause is typically self-evident. Other causes include psychotic illness,¹² elective mutism (relatively common in children),¹³ and hysteria.¹⁴

This type of speech disorder is usually transitory with language restored to normal within a few weeks or months. When speech returns, language may be syntactically complete, showing neither loss of vocabulary nor agrammatism, or there may be varying degrees of dysarthria, anomia, and paraphasic substitutions. Terms such as aphemia,^{15,16} cortical dysarthria,¹⁷ and pure word dumbness¹⁸ have been used to describe this disorder. Many authors currently regard it as an apraxia of speech.¹⁹⁻²¹

This patient was somewhat unique in that she had no prominent orobuccolingual dyspraxia on initial evaluation. She exhibited the common reactions of frustration, the so-called catastrophic reaction²² and, later while hospitalized, withdrawal. These patients often withdraw totally from all interpersonal commu-

nication for varying amounts of time.

SUMMARY

This case is noteworthy in that it demonstrates that localized infarctions may cause discrete lesions in certain language functions in the brain and that the syndrome of mutism may be mistaken for hysteria.

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