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Insula and aphasia

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Abstract. A 59-year-old woman developed impaired speech initiation as the result of a left anterior insular infarct. Findings from this case suggest that dominant hemisphere anterior insular lesions impair the speech initiation loop.

Key words: Insula – Nonfluent aphasia – Initiation

Introduction

Studies of Broca's aphasia reveal that many patients have damage to the insula [6, 9]. Other investigators from Wernicke [10] to date have suggested that insular lesions produce a conduction aphasia, no language disturbance, or according to Marie [5], anarthria. However, because isolated insular lesions with good documentation are rarely reported, the role of the insula in language has not been clearly defined. Furthermore, analysis of insular language function in the past has not taken into full account the architectonics of this region. The following case serves to illustrate the presence of speech impairment caused by an infarct restricted to the anterior insula.

Case report

A 59-year-old, right-handed black woman with a history of childhood rheumatic fever, hypertension, and cigarette use awoke one morning in July 1990 with difficulty in speaking. On admission her blood pressure was 160/96 mm Hg with a normal cervical vascular and cardiac examination. She was fully oriented and attentive but demonstrated difficulty in initiating speech and pronouncing words. She displayed aberrant linguistic prosody with occasional phonemic and rare semantic paraphasias in speech. She made similar paraphasic errors in reading. She displayed difficulty with the use of function words in her writing. Her verbal comprehension was impaired on sentences whose meaning was dependent on syntax. She had trouble naming low-frequency words and parts of objects. Repetition was poor. However, during the interview when she became angry at a family member, speech fluency, initiation, pronunciation, and prosody were almost normal. Praxis, memory, constructions, right-left orientation, finger naming, geography, and executive function testing were normal. Her cranial nerves were normal except for slight right lower facial weakness, and her motor system was normal. Reflexes were brisker in the right lower extremity than in the left with a right extensor plantar response. Sensory testing, coordination, and gait were normal.

An admission computed tomographic scan of her head was normal. A left carotid angiogram showed slight atherosclerotic changes of the left cavernous internal carotid artery and M1 segment. An echocardiogram showed slight left ventricular hypertrophy, 3+ aortic insuffiency; a bubble study showed no evidence of a right-to-left shunt. Holter monitoring was normal. MRI of the head obtained 7 days after admission showed a small infarct deep in the left anterior insula with pressure on the extreme capsule and claustrum and bilateral putaminal lacunar infarcts (Fig. 1a). We concluded that she had a small vessel infarct or branch occlusion involving insular branches of the superior division of the left middle cerebral artery.

Over the following week all her language deficits improved. Speech initiation, however, remained affected, with mild difficulty in speech pronunciation and prosody. The Western Aphasia Battery [4] was administered at this time. Subtest scores included spontaneous speech = 19.0 (20), fluency = 9.0 (10), comprehension = 10.0 (10), repetition = 7.8 (10), naming = 8.5 (10). (Normal scores on the latter four subtests are 10, 9.9, 9.8, and 9.5, respectively). Her score on the repetition subtest was the result of failure to repeat the last test sentence. The remainder of her performance was consistent with preserved repetition. On the Naming subtest she was able to name all of the 20 objects. Her score reflects her poor performance on the word fluency subsection. She produced the names of only 5 animals in 1 min. Her aphasia quotient was 90.6 (100, normal score = 98.4), reading and writing was 15.3 (20). She made no paralexic errors on this subsection. Her performance was felt to be consistent with her pre-stroke baseline. Praxis was 9.7 (10), and construction was 6.4 (10). The last score was secondary to a low performance on the calculation section. She had always had difficulty with mathematics, and we felt her performance reflected her pre-stroke baseline. Overall, other than impaired speech intiation and subsequently impaired verbal fluency, this patient demonstrated no language disturbance. However, though she produced grammatically correct sentences, an adequate evaluation of her syntactic processing was not performed. Therefore, this case cannot shed light on the insula's role, if any, in syntax.

She was discharged to her home with instructions to take one aspirin a day. A follow-up neurologic examination 1 month later revealed she continued to have difficulty with speech initiation without evidence of any language disturbance. Speech pronunciation and prosody were normal. MRI of the head performed 5 months after her stroke demonstrated a deep left anteroinferior

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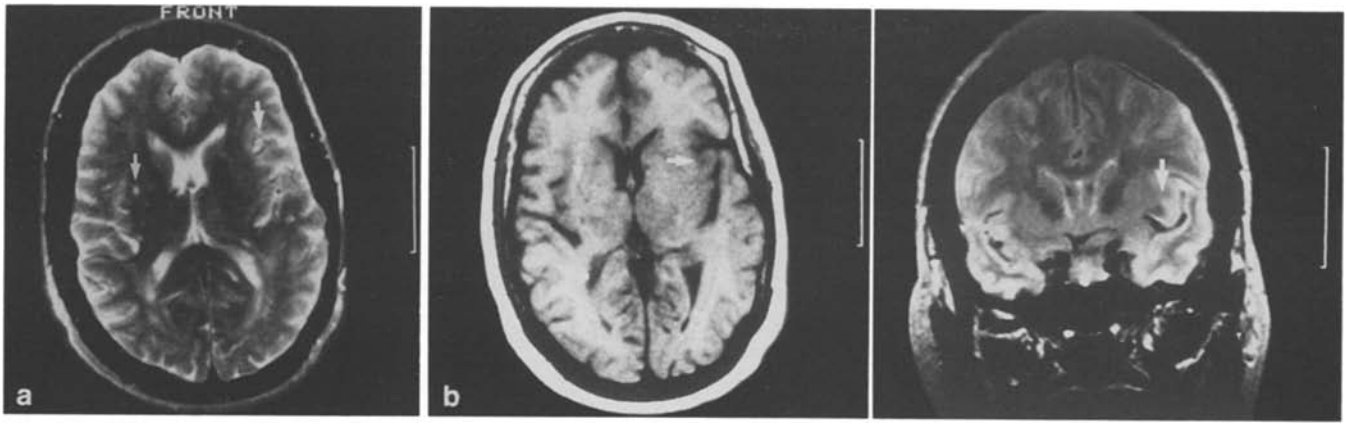

Fig. 1. a MRI scan of the head performed 7 days after the patient's stroke. T2-weighted images demonstrate areas of high signal intensity in the left anterior insula and right putamen consistent with infarcts. *Bar:* 5 cm. **b** MRI scan of the head performed 5 months after the patient's stroke. T1-weighted images show a low signal intensity in the left anterior insula. T2-weighted coronal images demonstrate an area of high signal intensity in the left anteroinferior insula. *Bar:* 5 cm

insular infarct (Fig. 1b) unchanged from the previous study. Her difficulty with speech initiation remained unchanged, as well. She died unexpectedly 7 months after her stroke from unrelated causes.

Discussion

The aphasia of this patient was characterized by difficulty in speech fluency, initiation, pronunciation, linguistic prosody, comprehension of syntax-dependent sentence meaning, naming, and repetition, which dramatically improved over the following days, leaving her with a relatively isolated deficit in speech initiation. In addition to the left anterior insular infarct, MRI showed small bilateral putaminal lacunes. Although basal ganglia infarcts can produce aphasia, they tend to involve the anterior and superior aspects of the putamen, often in conjunction with damage of the anterior limb of the internal capsule [2]. They also tend to be larger in size than lacunar infarcts. Alexander et al. [1] have argued that isolated putaminal lesions produce word-finding difficulty or no language disturbance at all. The consensus, therefore, is that whereas putaminal injury may or may not result in language disturbances, lacunar infarctions of the putamen do not. In addition, based on the patient's initial examination and MRI findings of a new infarct in the insula, the lacunar infarcts are old. Therefore, the putaminal lacunes in this patient are likely to be unrelated to her aphasia, as they do not explain her clinical findings. It is also unlikely that pressure on the extreme capsule and claustrum produced these speech and language deficits, as difficulty with initiation still persisted 5 months after the acute event when whatever edema had been present should have resolved.

The insula's role in language may be better understood by examining its histology and connections. The insula has been divided into three regions based on cytoarchitectonic studies in Old World monkeys and in man [7, 8]. In an anterior-ventral to posterior-dorsal gradient, the insula changes from agranular cortex with three cellular layers to six-layered dysgranular cortex with clustered granule cells in layer 4 and then to granular cortex with layers 2 and 4 both granular and fully demarcated. Based on connectivity, the insula may be divided into an anteroventral (anterior) and a posterodorsal (posterior) region, as well. Connections of the anterior insula, where this patient's lesion was located, include agranular orbitofrontal, frontal opercular, prepiriform, ventral temporopolar, anterior cingulate cortex, amygdala, thalamic ventroposterior medial, medial dorsal, centromedian-parafascicular, midline, and reticular nuclei.

The initiation of behavior, including speech, depends on both appropriate motivation and arousal-activation. Because the anterior insula has reciprocal projections with the limbic (cingulate and amygdala) and reticular (thalamic centromedian-parafascicular and reticular nuclei) systems, as well as with the perisylvian speech area (frontal opercular region), it would seem to be an important area in the speech initiation process. In accordance with this hypothesis is the observation that lesions in other regions of this system (i.e., cingulate, frontal opercula, thalamus) produce deficits in speech initiation.

During a moment of anger the patient's aphasia nearly completely resolved. Although the mechanism underlying this improvement remains speculative, it is possible that the limbic-reticular-cortical networks that mediate emotion may have input into the speech cortex that does not depend on the insula or at least the portion of insula damaged in this patient.

Alexander et al. [1] have reported two patients with infarcts of the insula and adjacent extreme/external capsule region. Both of these patients had a mild fluent aphasia with word-finding difficulty, rare phonemic paraphasias, and agraphia. The discrepancy between their patients and this patient may be related to the location of their patients' lesions. In their paper, a CT scan of one of these patients shows an infarct involving anterior and posterior insula that relatively spared the anteroinferior region, which had been injured in the patient reported

here. The aphasia seen in the patients of Alexander et al. may have been secondary to dysgranular/granular insular cortex damage or damage to white matter.

The anatomic distinction between the location of this patient's infarct and those of Alexander's may relate to the vascular supply involved. The insular gyri are supplied by branches of the cortical divisions of the middle cerebral artery as they travel over this region. The short insular gyri are fed by the superior division, while the long insular gyri and transverse temporal gyri receive branches from the inferior division. In some instances the superior division may send branches to the entire insula with no contribution from the inferior division [3], or the superior division may feed the short and long gyri while the inferior division supplies the transverse temporal gyri (personal observation).

Although this case provides evidence that the anterior insula should be included in the anatomic and functional network that mediates speech, further examples of isolated insular lesions will not only help to increase our confidence that the insula is a speech area, but may also better define its role.

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