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Transcortical mixed aphasia with left frontoparietal lesions

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Abstract We present a case of transcortical mixed aphasia following a left frontoparietal infarct caused by vasospasm after subarachnoid haemorrhage. Although CT showed low-density areas in the left frontal lobe and basal ganglia, single photon emission CT revealed a wider area of low perfusion over

the entire left hemisphere, except for the left perisylvian speech areas. Hence, transcortical mixed aphasia may be caused by the isolation of perisylvian speech areas due to disconnection from surrounding areas.

Key words Aphasia · Cerebral blood flow · Speech area

Introduction

We present a case of transcortical mixed aphasia following a left frontoparietal infarct caused by vasospasm after subarachnoid haemorrhage.

Case report

A 59-year-old, right-handed woman experienced an attack of headache and vomiting on 12 July 1994. On July 16 CT demonstrated subarachnoid haemorrhage in the suprasellar cistern, and cerebral angiography revealed an aneurysm at the junction between the left internal carotid and posterior communicating arteries. Although neck clipping was successfully performed via a frontotemporal approach on the day of admission, she suffered a period of unconsciousness due to vasospasm 1 week after the onset of symptoms. A cerebral angiogram obtained during this period revealed spasm of the left internal carotid artery.

By 3 weeks, she was alert but manifested a mild hemiparesis and aphasia. Spontaneous speech was markedly reduced, and she often demonstrated echolalia. Her object naming, verbal comprehension, reading and writing were also severely disturbed. This was in contrast to the full preservation of her ability to repeat phonemes and short sentences (5–6 words).

The Western aphasia battery (WAB) was performed 6 weeks following the onset of symptoms. Her spontaneous speech score was 6 of 20, comprehension 1.8, repetition 10, naming 0.6, reading 0.6, and writing 1.4 of 10. Therefore, her language deficit was classified as a transcortical mixed aphasia. Her performance intelligence quotient (IQ) on the Wechsler adult intelligence scale-revised (WAIS-R) was 66, and her score on Raven's coloured progressive matrices was 21 of 36.

CT obtained 1 month later revealed infarcts in the territory of the left anterior cerebral artery (Fig. 1). On a cerebral blood flow (CBF) study, ^{99m}Tc HM-PAO single photon emission CT (SPECT) performed 6 weeks after the onset of symptoms demonstrated reduced perfusion in the whole left hemisphere, except for the left perisylvian speech areas (Fig. 2).

Discussion

Transcortical mixed aphasia has rarely been reported. Bogousslavsky et al. [1] reported four cases of transcortical mixed aphasia in 1200 patients with acute cerebrovascular disorders. This condition is caused by anterior and posterior lesions of the left hemisphere when the perisylvian speech area is intact [2].

Although we could not find any reason for the posterior anatomical isolation of the speech area on plain CT, SPECT revealed an area of low perfusion involving the entire left hemisphere, except for the left perisylvian speech areas. Therefore, it is clearly important to investigate such lesions by CBF studies.

Our results suggest that transcortical mixed aphasia may be caused by the isolation of perisylvian speech areas due to disconnection from surrounding areas.

References

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Fig.1 CT obtained 1 month after the onset of symptoms demonstrates infarcts in the left frontal lobe and basal ganglia

Fig.2 SPECT shows decreased cerebral blood flow in the entire left hemisphere, except for the left perisylvian speech area

