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Clinical Study

Splenium or parahippocampus involvement and its relationship to cognitive decline in posterior cerebral artery infarction

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ABSTRACT

Although cognitive impairment after a posterior cerebral artery (PCA) infarct is frequently observed, the important functional areas associated with cognitive decline, other than the thalamus, have not been determined. We investigated the locus or loci that might induce cognitive decline after a PCA infarct. Forty-one patients with unilateral PCA infarctions involving only the occipital lobe or the occipital lobe plus other PCA areas were included. All subjects received a mini-mental status examination (MMSE) within 2 months of onset; 43.9% had cognitive impairment. The severity of cognitive impairment was not associated with left hemisphere lesion location, sex, age, education level, or the time between stroke and the MMSE assessment. Only the lesion volume was negatively correlated with MMSE score. Lesion location analysis revealed that an occipital plus splenial or parahippocampal lesion contributed to a decline in MMSE, which suggests that parahippocampal or splenial involvement with an occipital lesion is associated with the cognitive decline seen after PCA infarction.

1. Introduction

Posterior cerebral artery (PCA) territory infarcts account for 5% to 10% of all strokes.[1] Cognitive impairment after PCA infarcts is observed frequently and diverse neuropsychological deficits including dysphasia, amnesia, pure alexia, dyscalculia, visual neglect, and visual agnosia are described.[1–5] A lesion localization study showed that vascular dementia was associated with PCA infarction of the paramedian thalamic or inferior medial temporal areas, and with lesions of the parieto-temporal or temporo-occipital territories.[6] However, apart from the thalamus, other strategic areas within the PCA territory associated with cognitive decline have not been investigated.[7,8]

The development of MRI has allowed improved localization of lesion topography. The areas frequently involved in PCA infarcts are the occipital lobe, posterior ventral temporal lobe (fusiform and parahippocampal gyrus), thalamus, corpus callosum splenium, posterior cingulate gyrus, and retrosplenial cortex.[1,9]

We aimed to determine the locus or loci that might be involved in cognitive impairments after PCA infarction. We performed a lesion localization study in patients with PCA territory infarcts using mini-mental status examination (MMSE).[10] The advantages of a MMSE are that it requires only 5 min to 10 min to evaluate the patient's global cognitive state, and that the test is highly reliable between individual scorers and between tests.[11]

2. Methods

2.1. Patients

The study included 41 patients (26 men and 15 women) with unilateral PCA infarctions involving the cerebral cortex. The mean age (±standard deviation [SD]) was 61.59 ± 14.15 years (range: 29–87 years) with a mean (±SD) of 9.24 ± 4.32 years of education (range: 0–16 years). From April 2003 to September 2003, subjects were recruited consecutively from acute stroke units of four general hospitals in Seoul, Korea. Inclusion criteria were right-hand preference as assessed by Edinburgh Handedness Inventory,[12] stroke onset less than 60 days prior, alertness, and cooperation. Exclusion criteria were prior stroke, aphasia severe enough to interfere with test taking, and a history of cognitive impairment. A complete neurological examination was performed and a neurologist, who was blinded to lesion location, assessed the MMSE. Cognitive impairment was defined as a score below 24 points on the MMSE. This study was performed in accordance with the ethical standards of the Declaration of Helsinki. All subjects provided informed consent.

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2.2. Lesion analysis

In each scan section, the lesion boundary on CT scan (2/41) or T2-weighted MRI (39/41) was visually identified and outlined using a manual pixelwise method with the aid of a picture archiving and communication system (PACS) workstation (General Electric Medical Systems, Cincinatti, OH, USA). Lesion volume (in cm^3) was calculated by multiplying the area of lesions in consecutive CT scan slices by the slice thickness, and in consecutive MRI slices by the slice thickness plus the interslice gap distance. A neurologist blinded to the patient's clinical status measured the lesion volume.[13] To define lesion location, two neurologists, unaware of clinical findings, coded the involved lesion as the occipital lobe, temporal fusiform gyrus, parahippocampal gyrus, corpus callosum splenium, thalamus, or posterior cingulate gyrus.

2.3. Statistical analysis

We used an independent *t*-test and multiple linear regression models to investigate the relationship between sex, PCA lesion side and volume, age, years of education, time of stroke onset to MMSE test, severity of cognitive impairment (as indicated by continuous MMSE scores), and the presence of a lesion following infarction. To assess the relationship between lesion location and cognitive impairment (as categorized MMSE scores), we performed multiple logistic regression. All statistical analyses were conducted using the Statistical Package for the Social Sciences version 13.0 (SPSS; Chicago, IL, USA). $P < 0.05$ was set as a two-tailed statistical significance level.

3. Results

3.1. Variables affecting MMSE score

The mean time interval (±SD) between onset of infarction and brain imaging was 3.71 ± 3.23 days. Mean time (±SD) from onset of infarction to the MMSE test was 13.90 ± 14.84 days. The mean MMSE score was 22.98 ± 6.01. Twenty-four patients had right PCA infarction and 17 had left involvement. Motor power was normal in all patients, except for two patients with mild hemiparesis, which was above grade 4 on the Medical Research Council Scale for motor testing. Eighteen (43.9%) had cognitive impairment, defined as a MMSE score below 24 points. Cognitive decline after left and right PCA infarction occurred in 11 of 24 (45.8%) and 7 of 17 (41.2%) patients, respectively. Cognitive impairment severity (MMSE score) did not differ between right and left PCA infarctions (mean ± SD; right: 22.75 ± 6.22, left: 23.29 ± 5.87, $t = 0.28$, $p = 0.779$) or by sex ($t = 1.46$, $p = 0.153$). Impairment did not appear to be influenced by the interval between stroke onset and MMSE assessment ($t = 1.85$, $p = 0.072$), age ($t = -0.93$, $p = 0.358$), or level of education

Table 1
Demographics and clinical features of patients with PCA infarction ($n = 41$)

Case Sex/age (years)	Onset (days)	Hemisphere	MMSE (total)	Stroke location	CT scan or MRI	Lesion volume (cm^3)
1. M/58	4	Left	25	O, Fu, PH, Th	MRI	57.61
2. M/73	7	Left	17	O	MRI	3.86
3. F/35	4	Left	29	O	MRI	5.34
4. M/70	59	Left	27	O, Spl	MRI	26.07
5. M/36	49	Left	29	O	MRI	11.29
6. M/30	19	Left	25	O, Spl, Th	MRI	22.28
7. F/29	17	Left	30	O, CG	MRI	39.25
8. M/67	29	Left	25	O, Spl	MRI	15.68
9. M/54	9	Left	27	O, Fu, PH, Spl, Th	MRI	60.60
10. M/70	16	Left	28	O, Fu, PH	MRI	26.45
11. M/71	12	Left	24	O, Fu, PH, Spl	MRI	5.07
12. M/64	3	Left	29	O	MRI	9.02
13. F/38	1	Left	15	O, Fu, PH, Spl, Th, CG	MRI	46.74
14. M/57	8	Left	9	O, Spl, Th, MB	MRI	9.68
15. F/61	8	Left	18	O, PH	MRI	70.56
16. F/56	1	Left	19	O, Fu, PH, Th, MB	MRI	21.96
17. M/53	5	Left	19	O, PH, Spl	MRI	16.54
18. M/44	1	Right	27	O	MRI	7.14
19. M/49	20	Right	27	O, Spl	MRI	17.50
20. M/87	6	Right	25	O, CBLL	MRI	15.97
21. M/45	3	Right	29	O	MRI	15.18
22. F/69	6	Right	27	O	MRI	32.80
23. M/63	59	Right	30	O, Fu, PH	MRI	36.74
24. M/69	33	Right	27	O	CT	31.35
25. M/59	20	Right	25	O	MRI	21.26
26. M/70	21	Right	24	O, PH, Spl	MRI	46.89
27. M/73	16	Right	26	O, Fu, PH	MRI	40.23
28. F/57	26	Right	27	O	MRI	41.91
29. F/80	4	Right	15	O, PH, Th, CBLL	MRI	38.49
30. F/68	7	Right	24	O, Spl, CG, CBLL	MRI	48.43
31. M/73	34	Right	13	O, Fu, PH, Spl, Th	MRI	68.10
32. F/70	5	Right	17	O, Fu, PH, Spl, Th	MRI	99.34
33. F/69	1	Right	27	O, Fu	MRI	26.47
34. M/54	11	Right	24	O, Fu, PH, Spl, Th	MRI	45.92
35. F/68	4	Right	24	O, Fu, PH, Spl, Th	MRI	22.48
36. M/65	8	Right	30	O, Spl	MRI	33.07
37. F/71	3	Right	14	O, Fu, PH, Th	MRI	41.06
38. M/83	1	Right	25	O, CBLL	MRI	1.63
39. M/77	6	Right	7	O, Fu, PH, Spl, CG	MRI	68.89
40. F/66	8	Right	17	O, Fu, PH	MRI	61.34
41. F/75	16	Right	15	O, Fu, SplC)	CT	17.94

CBLL = cerebellum, CG = cingulate gyrus, F = female, Fu = fusiform gyrus of temporal lobe, M = male, MB = midbrain, MMSE = Mini-mental status examination, O = occipital lobe, Onset = time from stroke onset to MMSE test, PH = parahippocampal gyrus, Spl = splenium, Th = thalamus.