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Case study

Clinical recovery from stroke lesions and related outcomes

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ABSTRACT

Stroke lesions affect neurological status and are a critical determinant of treatment. This study investigated stroke lesions in terms of clinical recovery and related outcomes. Twenty-seven stroke patients were assessed via longitudinal observational study. Brain lesions were evaluated using MRI. The Fugl-Meyer Assessment and clinical evaluations were performed monthly between 1 and 6 months after onset. The anterior limb (ICAL) and genu (ICG) of the internal capsule were associated with recovery of the upper limbs in chronic stroke patients. Involvement of the anterior half of the middle third of the corona radiata, the ICAL, the ICG, and the caudate nucleus were related to recovery of the lower limbs. Involvement of the middle third of the corona radiata, the ICAL, the ICG, and the lentiform nucleus were associated with sensory recovery. Clinical recovery from stroke, in terms of motor and sensory function, was related to injury in several white matter areas, such as the corona radiata and internal capsule, and was also associated with the basal ganglia as a gait pattern generator. Clinicians should be aware of stroke lesions, and should design therapeutic strategies accordingly, also with respect to treatment duration.

1. Introduction

Clinical recovery and prognosis in stroke patients is important not only for the patients themselves, but also for their families, neurologists, therapists, and physiatrists. At present, the factors associated with acute stroke are being evaluated in numerous ways. Clinical evaluations, including National Institutes of Health Stroke Scale (NIHSS) scores, early nutritional status, and vascular supply (using susceptibility-weighted imaging protocols), have been applied to evaluate acute prognosis after stroke [1,2]. Although the long-term outcome and clinical prognosis of stroke lesions is being investigated by many researchers, there remains a lack of published data. Thus, the stroke lesions associated with the long-term prognosis or outcome after stoke have not yet been revealed.

Assessing clinical outcome is very important in stroke patients; early prediction of clinical recovery and outcome are also important, for both patients and clinicians. Therefore, this study investigated clinical outcomes in stroke patients with respect to specific lesion locations. We performed a meticulous descriptive analysis of magnetic resonance imaging (MRI) data, as well as a functional evaluation, and investigated the relationship between specific lesions and functional status.

2. Methodology

2.1. Study designs and subjects

This study was approved by the Ethics Committee of the Catholic University of Korea. Written consent was obtained from all subjects according to the Declaration of Helsinki. This study was a small-scale, longitudinal, observational clinical trial. Data from 27 right-handed first-stroke patients, which were recruited single center from March of 2014 to February of 2015, were analyzed. All of the subjects had suffered a supratentorial cerebral infarction or supratentorial ICH and met the following inclusion criteria: (1) a first-ever unilateral stroke; (2) ability to 1-step obey; (3) a Fugl–Meyer Assessment (FMA) score lower than 60 for the upper extremity or lower than 28 for the lower extremity [3]. Exclusion criteria were (1) presence of knee joint effusion determined using US, (2) a history of knee injury or surgery, or history of knee injection within 3 months, (3) a history of inflammatory arthritis or inflammatory myopathy, (4) diabetes mellitus, or (5) peripheral nervous disease. This study was observational study for clinical recovery and outcomes, we did not calculated the exact sample size. Sample size of previous studies vary 11–41 [4,5]. Including prediction for loss of follow up might be 20%. We decided the sample size was over than thirty subjects.

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2.2. Data acquisition

From all subjects, we obtained demographic and brain magnetic resonance imaging (1.5T MRI, Siemens, Germany) data and evaluated motor and sensory impairments clinically. Brain lesions were evaluated, including size, using a high-resolution 1.5-T anatomical MRI protocol (5-mm slice thickness) within 14 days of stroke. All subjects received physical and occupational therapies as neurodevelopmental and task-orientated treatment approaches, respectively. The rehabilitation programs of all subjects commenced within 1 month of stroke onset. Treatment continued for up to 6 months after onset, and consisted of 1–2-h sessions per day for 5 days a week, including both the physical and occupational therapy components [4]. The subjects also received speech therapy as required. The interventions were focused mainly on using and strengthening the affected limb, basic mat activities, symmetric weight-bearing and weight-transfer activities, and gait training; however, these activities were not specifically targeted at a particular purpose.

Regarding the MRI data, both T1- and T2-weighted images were included in the analysis. Brain lesions were identified and evaluated on the MR images, including the size. Lesion size was calculated using a picture archiving communication system (PACS, Marotech, Seoul, Korea), and absolute lesion size (cm3) was determined by multiplying the sum of all lesion areas in each plane by slice thickness. Brain lesions identified by brain CT or MRI scans were classified according to anatomical location as follows: premotor cortex (PMA); supplementary motor area (SMA); primary motor cortex (M1); anterior half of the middle third of the corona radiata (AMCR); posterior half of the middle third of the corona radiata (PMCR); internal capsule, anterior limb (ICAL); internal capsule, genu (ICG); internal capsule, posterior limb (ICPL); basal ganglia, lentiform nucleus (BGLN); caudate nucleus (CN); and thalamus (TH) [5].

Clinical evaluation of motor and sensory capability was performed using the FMA each month after stroke onset, up to 6 months [6]. To determine the clinical outcome, we classified subjects according to their ability or inability to perform isolated movements of the upper or lower limbs, based on FMA data. We considered FMA scores $\geqslant 23$ (for the upper limb) and $\geqslant 19$ (for the lower limb) to indicate recovery of isolated movements, with sensory scores $\geqslant 22$ (equal to or above the median score at 6-months post-onset) indicating recovery of higher sensory function.

2.3. Statistical analysis

Data were analyzed using SPSS for Windows software (v. 12.0; SPSS Inc., Chicago, IL, USA). For dichotomous data, we applied Fisher's exact test to compare the relationship between the brain lesion and recovery during the 6-month post-stroke period. All tests were two-tailed, and p-values $\leqslant 0.05$ were taken to indicate statistical significance.

3. Results

Thirty-one patients were enrolled initially, during experimental period. Four patients were excluded; one patient was excluded for re-attack, three patients were excluded for follow-up loss (transfer to other rehabilitation center at distant location). In total, 27 patients (mean age, 54.4 ± 13.9 years; 14 females, 13 males; Table 1) completed in the study. Twelve of the patients had left hemiplegia (right-sided stroke lesion) and 15 had right hemiplegia (left-sided stroke lesion). The demographic and overall clinical data are listed in Table 1.

Table 1
Demographic data of the participants.

Demographic characteristic (N = 27)	
Sex, M/F (%)	48.1/51.9
Age, y*	54.4 ± 13.9
Side of stroke, R/L (%)	44.4/55.6
Time from stroke to rehab, d*	15.5 ± 6.6
Stroke pathology, hemorrhage/infarction (%)	59.3/40.7
Brain injury location/cause of lesion (n, %)	
ICH, BG	9 (33.3)
ICH, TH	3 (11.1)
ICH, T-P	2 (7.4)
ICH F-T	2 (7.4)
Infarction, MCA	8 (29.6)
Infarction, BG	2 (7.4)
Infarction, internal capsule and PVWM	1 (3.7)

ICH, intracerebral hemorrhage; BG, basal ganglia; TH, thalamus; T-P, temporoparietal; F-T, frontotemporal; MCA, middle cerebral artery; PVWM, periventricular white matter.
* Mean ± SD.

The volume of stroke lesion and involved structure were presented at Table 2. The mean lesion volume of all participants was 36.98 ± 34.70 cm^3. The data on involved structure were divided by PMA, SMA, M1, AMCR, PMCR, ICAL, ICG, ICPL, BGLN, CN, and TH [5,7].

The relationship between stroke lesion and clinical recovery of the upper limb is described in Table 3. Involvement of the ICAL was significantly associated with recovery of the upper limb in the 2–6-month post-stroke period. Involvement of the ICG was significantly associated with recovery of the upper limb in the 3–6-month post-stroke period.

The relationship between stroke lesion and clinical recovery of the lower limb is described in Table 4. Injury to the AMCR was significantly associated with clinical recovery of the lower limb in the 3–4-month post-stroke period, while involvement of the ICAL and ICG was significantly associated with recovery of the lower limb in the 2–6-month post-stroke period. In addition, the CN was significantly associated with recovery of the lower limb in the 5–6-month post-stroke period (Table 4).

The relationship between stroke lesion and recovery of sensory function is described in Table 5. Injury to the AMCR was significantly related to sensory outcome in the 3–4-month post-stroke period, while injury to the PMCR and ICAL was significantly associated with sensory outcome in the 3–6-month post-stroke period. Injury to the ICG was significantly related to recovery of sensory function in the 2–6-month post-stroke period, but involvement of the BGLN was not significantly associated with sensory recovery in the 4–6-month post-stroke period.

Table 2
Brain lesion site and volume.

Lesion site	N (%)
MC	13 (48.15%)
AMCR	12 (44.44%)
PMCR	22 (81.48%)
ICA L	6 (22.22%)
ICG	7 (25.93%)
ICPL	22 (81.48%)
BGLN	21 (77.78%)
CN	3 (11.11%)
TH	7 (25.93%)
The volume of lesion (cm^3)	36.98 ± 34.70

MC, motor cortex; AMCR, anterior half of the middle third of the corona radiata; PMCR, posterior half of the middle third of the corona radiata; ICAL, internal capsule anterior limb; ICG, internal capsule, genu; ICPL, internal capsule, posterior limb; BGLN, basal ganglia, lentiform nucleus; CN, caudate nucleus; TH, thalamus.