Auditory Spatial Deficits in the Early Stage of Ischemic Cerebral Stroke

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Background: Clinical research, together with computed tomography/magnetic resonance imaging findings, proves that ischemic stroke (IS) that damages auditory pathways can cause hearing loss and impairment of higher auditory processes such as sound localization. The goal of the study was to find possible correlations between the IS risk factors, ischemic lesion volume and localization, neurologic status, and the sound localization capability in acute IS patients. *Methods:* We consecutively enrolled 61 IS patients into the study. The control group consisted of 60 healthy volunteers. All neuro-otological evaluations were performed up to 30 days from the incidence of stroke. All the subjects underwent the horizontal minimum audible angle test (HMAAT) and standard tonal and speech audiometric assessments. *Results:* HMMAT results were significantly worse in the IS patients and were present in 82.0% of the patients. There were more patients with unilateral disturbances than with bilateral ones (54.1% versus 27.9%). It was the characteristics of the ischemic lesions that correlated strongly with the sound localization deterioration, that is, their bilateral (the 90° azimuth, $P = .018$; the 180°, $P = .002$), multiple (the 45°, $P = .020$; the 180°, $P = .007$; the 225°, $P = .047$), and lacunar character (the 90°, $P = .015$; the 225°, $P = .042$). Differences in the types of HMAAT results were significant for lesions in the frontal and the temporal lobe ($P = .018$ and $P = .040$). In addition, worse sound localization ability was more common in patients with poor speech discrimination and the bilateral sensorineural hearing loss. We have not found statistically significant correlations for other analyzed factors such as the cortical/subcortical character of the lesions, the patients' neurologic status, and cerebrovascular risk factors. *Conclusions:* Sound localization impairment is common in IS patients and it is the multiple, bilateral, and lacunar character of the ischemic lesions that seems to be strongly positively correlated with the disturbance of the sound localization ability. **Key Words:** Ischemic stroke—lacunar stroke—sound localization impairment—spatial hearing.

Ischemic cerebral stroke can cause not only various otological symptoms such as the sensorineural hearing loss, tinnitus, or vertigo but also central auditory processing disorders. One of the central auditory processing disorders is the impairment of sound localization ability.[1-11] This type of impairment was reported in different types of brain tissue damage, especially in case of ischemic lesions, both of the right and left cerebral hemisphere.[12-21] Anatomic structures responsible for hearing and sound localization are organized on different central nervous system (CNS) levels and spread from the brainstem to the cortex. Thus, auditory dysfunction in stroke patients varies depending on the localization and volume of ischemic lesions damaging the auditory pathways.

The nuclei of the trapezoid corpus, nuclei of the lateral lemniscus, superior nucleus of the oliva, and the nucleus

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of the inferior colliculus of the tectal lamina constitute the anatomic basis of sound localization in the mechanism of interaural time delay and intensity.[22] Pathologic vascular or neoplastic processes of these regions, although rare, impair sound localization substantially because they restrict the broad stream of information going to the upper levels of the CNS.[10,15,23] Functional magnetic resonance imaging (fMRI) studies proved that tasks demanding sound localization in healthy subjects cause activation of lower part of the inferior parietal lobule and posterior parts of the middle and inferior frontal gyri, bilaterally.[24-26] In addition, it was proved that sound localization process activates the planum temporal and parietotemporal operculum,[27,28] and it seems that it is the temporal lobe that is most important in the sound localization on the cortical level.[29] Thus, most of research focuses on the cortical–subcortical lesions of this region,[2,4,8,10,12-21,30,31] and on the correlations between the impairment of sound localization and lesions in the right, left, or both hemispheres.

The goal of this study was to find components of the ischemic cerebral stroke that correlate with the most profound impairment of the sound localization process.

Materials and Methods

Stroke Patients

Between May 2006 and January 2011, we consecutively enrolled 61 ischemic stroke (IS) patients into the study–33 men and 28 women admitted to the Adults' Neurology Department of the Medical University of Gdańsk, Poland. The average age of the patients was 56.2 ± 17.3 years (range, 21-80 years). All subjects were right-handed. All neuro-otologic evaluations were performed during the early stage after the incidence of stroke (up to 30 days, average, 10 ± 7 days). The diagnosis of stroke was based on the World Health Organization criteria in patients with neurologic symptoms lasting at least 24 hours. The exclusion criteria were: age older than 80 years, patients with previous history of stroke (but not transient ischemic attack), serious general state, dementia, neurodegenerative disorders, other previously identified neurologic diseases, patients without logical verbal contact due to aphasia, psychotic symptoms, visual spatial neglect syndrome tested with the line bisection test and the nonverbal shape cancellation task,[32,33] conductive or mixed type hearing loss, asymmetric sensorineural hearing loss, and history of ear surgery.

According to the Food and Drug Administration classification of the hearing loss (the pure middle tone average [PMTA] for values at 500, 1000, 2000, and 4000 Hz in the better ear), 40 subjects had hearing within normal limits and 21 presented the sensorineural hearing loss (17 mild and 4 moderate).

Control Group

The control group consisted of 60 age-matched subjects, 28 men and 32 women. The average age of the group was 53.1 ± 19.2 years (range, 21-80 years), and it consisted of healthy volunteers (mainly students and members of the hospital medical staff). The exclusion criteria for the control group were previously identified neurologic diseases, diabetes, circulatory insufficiency, alcoholism, smoking, use of medications affecting the CNS, history of noise exposure at work, hearing disorders including the conductive and the mixed type hearing loss, and history of ear surgery. All subjects underwent otological and neurologic examination. According to the Food and Drug Administration classification, 3 subjects had sensorineural mild hearing loss. All of them were aged older than 60 years, and their hearing loss was because of cochlear presbycusis.

Neurologic Examination and Localization of Ischemic Lesions

Initial quantitative assessment of the patients was performed on the first day of the hospitalization, no more than 48 hours from the incidence of stroke. Full neurologic examination was performed in each of the IS patients. All of the IS patients received standard stroke unit treatment. During the hospitalization in the Department of Neurology, laboratory tests, electrocardiogram, chest X-ray, and ultrasound of the carotid and vertebral arteries were performed. In certain cases, echocardiogram and internal medicine and cardiological or vascular surgery consultations were necessary. The National Institutes of Health Stroke Scale (NIHSS) was used to examine patients neurologically.[34] History of hypertension, coronary heart disease, diabetes, and hyperlipidemia was taken. The presence and the localization of ischemic lesions were identified by MRI (3.0 T). On the basis of the previously mentioned measures, patients were divided into stroke etiological subgroups: large-artery atherosclerosis, cardioembolism, small-artery disease, other etiologies, and mixed etiologies. Depending on the lateralization of ischemic lesions, they were divided into the right-sided, the left-sided, and the bilateral ones. In addition, localization of the lesions was further described as of (1) the frontal lobe; (2) the temporal lobe; (3) the parietal lobe; (4) the occipital lobe; (5) the midbrain; (6) the diencephalon; and (7) the cerebellum. This classification does not include lesions localized at the frontoparietal and the temporo–parieto–occipital border. In such cases, lesions were classified to 1 of the 7 groups on the basis of the localization of the biggest part of the lesion. To localize the lesions properly, radiologic tables of anatomy by Weir et al[35] were used. Furthermore, the lesions were divided according to the classification by Ruff et al[19] into the lesions of the right anterior hemisphere, the right posterior hemisphere, the left anterior hemisphere, the left posterior hemisphere, the cerebellum, and of the brainstem. According