LETTER TO THE EDITOR

To The Editor

Neuropsychological Symptoms After Anterior Cerebral Artery Ischemic Stroke

Keywords: Stroke, Anterior cerebral artery (ACA), Neglect, Pusher phenomenon, Subjective visual vertical

Clinical deficits after ischemic strokes can reflect neurotopological localization of neuronal function. It was shown – based on stroke lesion analysis – that spatial visual hemineglect (HN) and tilt of subjective visual vertical (SVV) were due to lesions affecting the right superior temporal cortex and the insula.[1] These regions are also part of a distributed cortical vestibular network associated with a disturbed vertical perception and pusher phenomenon (PP). Nevertheless, with respect to lesion location, the anterior cerebral artery (ACA) territory is rarely affected in these patients.[1,2] Therefore, the present case report depicts a unique description of a patient presenting vestibular, spatial, and postural deficits due to lesions in the ACA territory.

We report on a 67-year-old man, with no medical history of neurological disease, who developed a paresis of the left lower extremity. Initial computer tomography (CT) scan with CT angiography showed a pericallosal artery occlusion. Magnetic resonance imaging (MRI) three days after stroke onset depicted the acute ischemia in the vascular territory of the right ACA (Figure 1). Seventeen days after stroke onset, the patient had a severe hemiparesis (leg proximal and distal: 0/5; arm proximal and distal 3–4/5 (MRC scaling), dysarthria, as well as a central facial palsy. The muscle reflexes such as the brachioradialis reflex, the biceps reflex, the patellar reflex, and the Achilles reflex were hyperreflexic on the left side; a positive Babinksi response was obtained on the left side. Sensory testing revealed no abnormalities concerning pain, temperature, touch, or vibratory sensation. Due to the paresis, deficits were seen in the coordination such as in the finger-to-nose testing; a dysmetria could not be detected. The patient was unable to stand and walk because of the severe paresis of the lower extremity; thus, heel-to-shin testing and gait testing could not be conducted.

Bells test and calculation of Center of Cancellation (CoC) score (0.428) revealed a severe spatial HN.[1] In addition, a severe contraversive PP (score of 3 on the Scale for Contraversive Pushing (SCP)),[3] which was tested in a sitting position, a contralesional tilt of SVV[1] with a deviation of 6.3°, and a visual extinction[4] could be measured. SVV was measured by means of a luminous rod presented 1.55 m above the ground. The patient wore special goggles with which he was solely able to see the luminous rod. The patient was instructed to sit upright and to keep his head upright. The vertical head position was water level controlled to achieve highest accuracy. The SVV apparatus was positioned 1.50 m in front of the patient. There were three starting positions for the luminous rod: 20°, 30°, and 40° from vertical, each clockwise, and counterclockwise. The patient's task was to verbally direct the experimenter to orient the luminous rod until "it is exactly vertical." Each starting position was performed twice in a random order. The experimenter rotated the rod back and forth following the subject's instructions until the patient was certain that it was exactly vertical. The individual values of the SVV were averaged over the 12 measurements indicating the angle between the SVV and the objective gravitational vertical in the patient's roll plane.[1] Anosognosia for hemiparesis or disturbed limb ownership was not obtained.[4] The cognitive parameters of the Montreal Cognitive Assessment (MoCA) showed a score of 20 out of 30 indicating cognitive deficits.[5] Deficits in the MoCA were seen in particular in the delayed recall – indicating a retrieval deficit – and the clock drawing tests (Figure 2); the latter indicated visuospatial deficits.

From day 17 to day 68 after stroke onset, the patient resided in an inpatient rehabilitation clinic where he received neuropsychological and physical therapy including optokinetic stimulation and therapy to improve spatial perception.

In a second testing, 68 d after stroke, neglect (CoC score of 0.016),[1] PP (SCP score of 0),[3] SVV (2.1°),[1] and cognitive deficits (MoCA score of 28) normalized.[5]

Lesions of ACA ischemic strokes are usually not associated with the abovementioned neuropsychological phenomena.[1,2,6] There is only one case in the literature with PP, HN, and disturbed SVV with a similar lesion.[2]

Our present case indicates that under certain circumstances and detailed investigations, ACA lesions might indeed provoke vestibular, spatial, and postural deficits. Usually, these signs are highly associated with lesions of the insular cortex.[7] The present and the previous cases[2] indicate that parts of the brain which belong to the ACA territory might be part of a cortical vestibular network and functionally related to the insular cortex, for example, via the superior longitudinal fasciculus which connects frontal, parietal, and insular regions or the adjacent superior occipitofrontal fascicle.[7,8] It was shown that lesions of both the superior longitudinal fasciculus as well as the inferior occipitofrontal fascicle are affected in acute stroke patients with HN as well as in patients with tilt of SVV.[1,9] Thus, these white matter tracts seem to be involved in the vestibulo-cortical network for the perception of verticality in the roll plane as well as in the visuospatial system.

The patient was informed of the intent to publish his case report and gave his written consent. The examination was approved by the ethics committee of the Landesärztekammer Rheinland-Pfalz (#837.032.17 (10,866)) and was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

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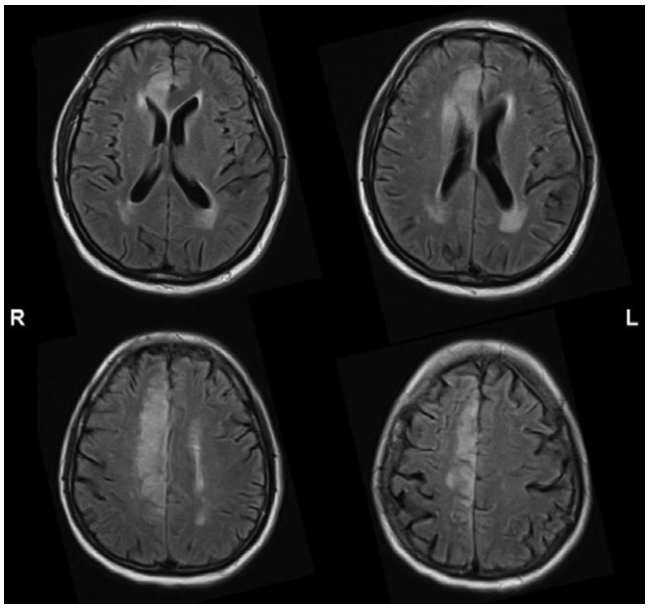

Figure 1: *Horizontal T2-fluid-attenuated inversion recovery (FLAIR)-weighted MRI scan three days after stroke onset showed an extended stroke in the territory of the ACA but no ischemic lesions in middle or posterior cerebral artery territory (slice thickness: 5 mm).*

Figure 2: *Clock drawing of the patient showing visuospatial deficits.*

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Statement of Authorship

MK: conduction of experiments and writing of the MS. AS: design of experiment and writing and supervision of the MS. HC: conduction of the vestibular and neuropsychological testing. FB: writing of the MS, supervision, and financing. FZ: writing of the MS and supervision. BB: writing of the MS, conduction of testing, and supervision.

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