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. 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. 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 (Figure 1). Seventeen days after stroke onset, the patient had a severe hemiparesis (leg proximal and distal: 0/5; arm proximal

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), PP (SCP score of 0), SVV (2.1°), and cognitive deficits (MoCA score of 28) normalized.

Lesions of ACA ischemic strokes are usually not associated with the abovementioned neuropsychological phenomena. There is only one case in the literature with PP, HN, and disturbed SVV with a similar lesion. 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. The present and the previous cases 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. 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. 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.

ACKNOWLEDGMENTS

The present study was supported by a grant from the Deutsche Forschungsgemeinschaft, DFG (BA 4097-1) to B.B. and F.B.

DISCLOSURES

Dr. Zipp reports grants from DFG, grants from BMBF, during the conduct of the study; grants from DFG, grants from BMBF, grants from PMSA, grants from MPG, grants from
Genzyme, personal fees from Merck Serono, personal fees from Roche, personal fees from Sanofi-Aventis, personal fees from Celgene, personal fees from ONO, personal fees from Octapharma, outside the submitted work.

**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.

*Michael Kim  
Department of Neurology and Neuroimaging Center (NIC) of the Focus Program Translational Neuroscience (FTN), University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany

*Annette Spreer  
Department of Neurology and Neuroimaging Center (NIC) of the Focus Program Translational Neuroscience (FTN), University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany

Hannah Cuvenhaus  
Edith-Stein-Fachklinik, Bad Bergzabern, Germany

Frank Birklein  
Department of Neurology and Neuroimaging Center (NIC) of the Focus Program Translational Neuroscience (FTN), University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany

Frauke Zipp  
Department of Neurology and Neuroimaging Center (NIC) of the Focus Program Translational Neuroscience (FTN), University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany

Bernhard Baier  
Department of Neurology and Neuroimaging Center (NIC) of the Focus Program Translational Neuroscience (FTN), University Medical Center of the Johannes Gutenberg University Mainz, Mainz, Germany

Edith-Stein-Fachklinik, Bad Bergzabern, Germany

*These authors contributed equally to this work.

**Correspondence to:** Bernhard Baier, Department of Neurology, Edith-Stein Fachklinik, Wiesenstraße 25, 76887 Bad Bergzabern, Germany. Email: baierb@uni-mainz.de

**REFERENCES**