ACTA INFORM MED. 2012 Dec; 20(4):242-243

doi: 10.5455/aim.2012.20.242-243
Received: 15 September 2012
Accepted: 23 November 2012

GERSTMANN'S SYNDROME IN ACUTE STROKE PATIENTS

Sanela Zukic[1], Zamir Mrkonjic[2], Osman Sinanovic[1], Mirjana Vidovic[1], Biljana Kojic[1]
Department of Neurology, University Clinical Center Tuzla, Tuzla, Bosnia and Herzegovina[1]
Faculty of Education and Rehabilitation, Tuzla, Bosnia and Herzegovina[2]

Coresponding author: Sanela Zukic, MD, Msc. Department of Neurology, University clinical center Tuzla, Trnovac 1, Tuzla, Bosnia and Herzegovina. E-mail: nellaz@bih.net.ba

Original paper
ABSTRACT
Objective: Gerstmann in 1924. observed in a few patients a concomitant impairment in discriminating their own fingers, writing by hand, distinguishing left from right and performing calculations. He claimed that this tetrad of symptoms constituted a syndromal entity, assigned it to a lesion of the dominant parietal lobe. Since than, Gerstmann`s syndrome (GS) was enigma for neuropsychologists. The aim of this study was to analyze frequency and clinical features of GS among acute stroke patients. **Patients and methods:** We prospectively analyzed 194 acute stroke patients (average age 65±11.06 years, male 113 (58.2%), female 81 (41.8%) hospitalized at Department of Neurology, University Clinical Center Tuzla, during the six mounths in 2010. For clinical assessment of agraphia, alexia and acalculia we used Minessota Test for Differential Diagnosis of Aphasia's. **Results:** Among these acute stroke patients, 59 (30.40%) had alexia, agraphia and acalculia or different combinations of these disorders. Two patients (3.4%) had agraphia and acalculia associated with other part of tetrad of GS: finger agnosia and left-right disorientation. They both where men, right handed, and cranial computed tomography scan showed ischemic lesion in the left parietal and left temporoparietal lobe. **Conclusion:** Gerstmann`s syndrome is rare clinical entity, and has the high value in localization and the lesion is mainly localized to angular gyrus of the dominant hemisphere.
Key words: Gerstmann`s syndrome, acute stroke.

1. INTRODUCTION

Gerstmann in 1924. observed in a few patients a concomitant impairment in discriminating their own fingers, writing by hand, distinguishing left from right and performing calculations. He claimed that this tetrad of symptoms constituted a syndromal entity, assigned it to a lesion of the dominant parietal lobe (1). There are a little data about frequency of GS among stroke patients. Dr Benton has studied cases over a period of >15 years, and concluded that, in rank order, the frequency of the individual components is dyscalculia, then dysgraphia, right–left disorientation and, least commonly, finger-agnosia. The two least common symptoms tend to occur together most often, whereas other combinations are apparently more random (2). However, because its occurs so rarely in pure form, GS in fact be less useful as a diagnostic indicator of focal parieto-temporo-occipital disease than in some other symptoms combinations (3). Fathermore, some authors are not in agreement with Gerstmann`s postulate of damage to a common cognitive function underpinning clinical semiology. Their evidence from intact functional neuroanatomy suggests that pure forms of Gerstmann's tetrad do not arise from lesion to a shared cortical substrate but from intraparietal disconnection after damage to a focal region of subcortical white matter (4).

The aim of this study was to analyze frequency and clinical features of GS among acute stroke patients.

2. PATIENTS AND METHODS

We prospectively analyzed 194 acute stroke patients (average age 65±11.06 years, male 113 (58.2%), female 81 (41.8%) hospitalized at Department of Neurology, University Clinical Center Tuzla, during the six mounths in 2010. The patients were evaluated in the first week of stroke, during the acute phase of disease. Diagnosis was based on clinical, neurological and neuroradiological findings (computed tomography and/or magnetic resonance). For clinical assessment of agraphia, alexia and acalculia we used Minessota Test for Differential Diagnosis of Aphasia.

3. RESULTS

Among these acute stroke patients, 59 (30.40%) had alexia, agraphia and acalculia or different combinations of these disorders. In most of the stroke patients thay were associated (59.3%) (Figure 1). Only two of patients (3.4%) had agraphia and acalculia associated with other part of tetrad of GS: finger agnosia and left-right disorientation. They both where men, right handed. In the absens of aphasia, apraxia, or other neuropsychological impairment (intelligence, memory and attention), thay demonstrated all four Gerstmann symptoms. Cranial computed tomography scan showed isch-

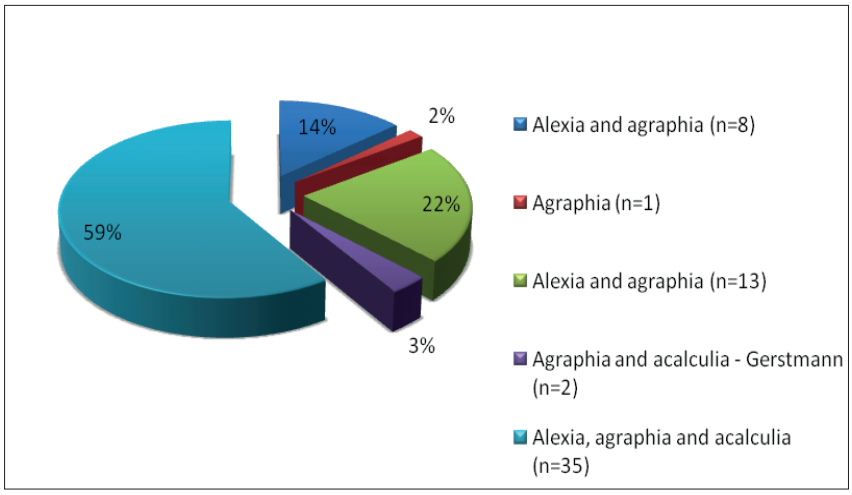

Figure 1. Distribution of frequency of alexia, agraphia and acalculia

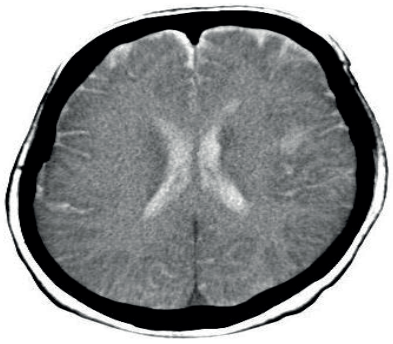

Figure 2. Ischemic stroke in left parietal lobe (computed tomography) in a patient with Gerstmann` syndrome

emic lesion in the left parietal and left temporoparietal lobe (Figure 2).

Ando et al. (5) indicates that Gerstmann syndrome can be caused by not only dysfunction of the left angular gyrus, but also of the left middle frontal gyrus in the dominant hemisphere.

In association with other impairments such as aphasia, apraxia or sensorimotor deficits, Gerstmann's syndrome has been described in numerous brain lesions, however, when accompanying deficits were moderate or absent, Gerstmann's syndrome was produced by left parietal disease in right-handed patients (6, 7).

Rusconi et al. (4) tested Gerstmann`s hypothesis by performing in depth functional and structural neuroimaging in a series of a healthy subjects, and postulated that the Gerstmann tetrad is not functional, but structural. A lesion of separate but spatially convergent fiber pathways involved in the four domain would then cause this syndrome by way of disconnection. These findings shed an interesting light on the clinical consequences of damage to the dominant parietal lobe. Neither the constituent symptoms of Gerstmann syndrome nor lesion to this region of white matter are uncommon, but their selective association in pure Gerstmann syndrome is a seldom clinical event.

4. CONCLUSION

Gerstmann`s syndrome is rare clinical entity and regarding the localizing value these case reports have also confirmed Gerstmann` statement that syndrome is associated with damage to the dominant parietal lobe. Undoubtedly, GS as enigma will continue to intrigue both, clinical neurologists and reseachers in neuropsychology.

Conflict of interest: none declared.

REFERENCES

1. Rusconi E, Pinel P, Dehaene S, Kleinschmidt A. The enigma of Gerstmann` syndrome revisited: a telling tale of the vicissitudes of neuropsychology. Brain. 2012; 13: 320-332.
2. Compston A. The enigma of Gerstmann's syndrome. *Brain. 1965;* 1966: 89; 183-198.
3. Benton AL. Gerstmann`s syndrome. Arch Neurol. 1992; 49: 445-447.
4. Rusconi E, Pinel P, Eger E, LeBihan D, Thirion B, Dehaene S, Kleinschmidt A. A disconnection account of Gerstmann syndrome: functional neuroanatomy evidence. Ann Neurol. 2009; 66(5): 654-662.
5. Ando Y, Sawada M, Morita M, Kawamura M, Nakano I. Incomplete Gerstmann syndrome with a cerebral infarct in the left middle frontal gyrus. Rinsho Shinkeigaku. 2009; 49(9): 560-565.
6. Tohgi H, Saitoh K, Takahashi S, Takahashi H, Utsugisawa K, Yonezawa H, Hatano K, Sasaki T. Agraphia and acalculia after a left prefrontal (F1, F2) infarction. Journal of Neurology, Neurosurgery and Psychiatry. 2005; 58: 629-632.
7. Kovacevic L, Kapidzic A, Sinanovic O. Gerstman syndrome in ischemic stroke. Neurologia Croatia. 2005; 54, Suppl (2): 125.