

Case Report of an Astasia-Abasia Syndrome After Operation of a Left Frontal Glioblastoma Multiforme

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Received: July 02, 2018; Published: September 26, 2018

Abstract

Astasia-Abasia syndrome is an inability to stand (Greek: astasia) and walk (Greek: abasia). We report of a case of postoperatively occurring astasia-abasia syndrome in a patient with left frontal localized glioblastoma multiforme and infiltration of the anterior portions of the corpus callosum. Before and directly after operation there were no focal neurological deficit, motor function (muscular strength, coordination) and the depth and surface sensibility were completely inconspicuous, no abnormalities in the orientated neuropsychological examination (mini-mental state), affect, no signs of content or formal mental disorders. Early rehabilitation was carried out from the second postoperative day onwards, without any effect to the symptoms. The patient died on the 8th day after operation by pulmonary embolism.

Keywords: Astasia; Abasia; Glioblastoma Multiforme; Infarction Corpus Callosum

Introduction

Astasia-abasia syndrome is an inability to stand (Greek: astasia) and walk (Greek: abasia). This term is regularly used when no focal neurological deficit is found, which is responsible for this severe function disorder, e.g. in stasobasophobia (Greek stasis = standing, base walking, Greek phobos = fear) as fear of falling. It is also used in cases of these symptoms in association with focal neurological diseases. The syndrome was first described in 1888 by Paul Blocq in psychiatric patients who showed a normal function of the legs in the bed during the examination, but were not able neither to stand nor to walk [1]. Blocq noted that the syndrome was often accompanied by other psychogenic symptoms such as psychogenic paralysis, seizures and tremor. Following modern nomenclature the so-called "hysterical abasia" is a dissociative movement disorder. The term "psychogenic gait disorders" stands for the complete inability to stand and walk. It is also used by some authors in organically induced disorders, such as severe polyneuropathies, cerebellar diseases or severe paralysis after stroke, Parkinson's disease and multiple sclerosis [2,3]. The term "Astasia-Abasia" is also used by some authors in organically induced disorders, such as severe polyneuropathies, cerebellar diseases or severe paralysis after stroke, Parkinson's disease and multiple sclerosis [2,3]. Descriptions of the syndrome in the literature are almost exclusively in the form of case reports with partly very heterogeneous basic diseases as etiological basis [4,5]. The findings of some studies of patients with conversion disorders suggest that alterations in regional brain perfusion may accompany these conversion symptoms. Functional brain imaging may offer a means of elucidating the neural correlates of conversion disorder [6]. In addition to these heterogeneous case descriptions of organic basic diseases and, on the other hand, dissociative movement disorders, we find in the literature a further group of cases with different lesions of the central nervous system [7-9]. In addition to these heterogeneous case descriptions of dissociative movement disorders, we find in the literature a further group of cases with different lesions of the central nervous system [7-9]. One of these patients is the casuistry described as follows. Despite the inconspicuous examination the patient was unable to stand or walk.

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Materials and Methods

The 67-year-old lady suffered a generalized convulsive spasm seizure, which then led to diagnosis of a left-frontal space requirement tumor with a diameter of 6 - 8 cm, surrounded by a moderate edema and infiltration of the anterior parts of the corpus callosum with otherwise inconspicuous findings after imaging diagnostics (CCT and MRI) and consequently to hospitalization. The patient had no focal neurological deficit during the initial examination. After the operative removal of the frontal space requirement tumor, no focal neurological deficit was found in the neurological examination. Both, the motor function (muscular strength, coordination) and the depth and surface sensibility were completely inconspicuous. There were no abnormalities in the orientated neuropsychological examination (mini-mental state: fully 30 points), affect was balanced clinical examination showed an balanced affect, no signs of content or formal mental disorders, a planned neuropsychological examination was prevented by death. Despite the inconspicuous neurological examination (cranial nerves, motor activity, sensibility, muscular reflexes, coordination) patient was unable to stand or walk. Postoperative MRI showed a subtotal resection of the tumor with an increasing perifocal edema. Due to the severity of this dysfunction early rehabilitation (physiotherapy, occupational therapy, bed cycling) was carried out from the second postoperative day onwards, with the emphasis on a stand and gait training with an average of 300 therapy minutes per day. On the 8th postoperative day, the patient unfortunately died as a result of a fulminant pulmonary embolism, so that we can only look back on a brief rehabilitative therapy course. Despite intensive rehabilitation, the syndrome showed no improvement during the 7-day therapy period.

Discussion

Uniform etiology of the Astasia-Abasia Syndrome is still difficult to find. Apart from the heterogeneous basic diseases from different subject areas [4,5], as well as the description of psychogenic disturbances [6,10,11], we also find case descriptions of an astasia abasia syndrome due to different hereditary diseases [7-9]. In our case we had no advice for a psychogenic disorder, neither in actual clinical symptomatic nor in anamnesis. A common feature with our case description is found in two cases, which call a syndrome of the corpus callosum. [8,9]. Our casuistry is probably the next similar case of a patient with the left frontal and the corpus callosum infiltrating astrocytoma [8] reporting the case of a 55 year old woman suffering from an astrocytoma of the corpus callosum, probably originating in the middle part of the commissure and subsequently involving the whole structure, beyond which it extended very little. They describe the symptoms and signs, consisting initially of a progressive isolated astasia-abasia, in the absence of any psychological disturbance, and subsequently consisting of an appraxic disorganisation involving particularly gestural activity of the left upper limb. Commonly all descriptions have that patients are unable to stand or walk without a focal neurological deficit or evidence of mental disorders. Kumral., et al. [9] reports a patient who had a callosal infarct, showed by CT and MRI, without injury to the adjacent hemispheres. Angiography revealed an occlusion of both internal carotid arteries. Besides interhemispheric disconnection with unilateral left agraphia, left tactile anomia, left ideomotor apraxia and left ear auditivoverbal extinction, the patient showed signs of left hemineglect and astasia-abasia, both persistent. Kataoka., et al. [12] reports a patient with an infarction of a region nearby, the posterior region of Gyrus cinguli, here only astasia without abasia. Little is known about the prognosis of the organically induced syndrome, in general the psychogenic induced astasia abasia syndromes are considered to be prognostically favorable. From the observation of the first 7 day of therapy of our patient unfortunately no improvement could be achieved despite a high therapy intensity. This gave the impression that this lack of ability of standing and walking seems to be more treatment-resistant than in other cases based on focal neurological deficits such as paresis, coordination disorders etc. Problematic in the course of the therapy was the fact that commonly used therapy concepts for patients with acquired brain damage (Bobath, Vojta, PNF...) could only be used with difficulty, since no functional impairment could be detected during lying or sitting.

Conclusion

In summary the astasia abasia syndrome is still quite unknown in etiology and prognosis and seems to be of rare frequency. Searching for the terms astasia and abasia Medline database shows only few references. Therefore it will be important to publish more case studies to evaluate this syndrome.

Conflict of Interest

There are no financial interest or conflict of interest.

Bibliography

- 1. Okun M and P Koehler. "Paul Blocq and (psychogenic) Astaia Astasia Abasia". Movement Disorders 22.10 (2007): 1373-1378.
- 2. Boogaarts H., et al. ""Recumbent" Gait: Relationship to the phenotype of Astasia-Abasia?" Movement Disorder 22.14 (2007): 2121-2122.
- 3. Snijders A., et al. "Neurological gait disorders in elderly people: clinical approach and classification". Lancet Neurology 6.1 (2007): 63-74.
- 4. Lafforgue P., et al. "Astasia-abasia revealing a primary Sjögren's Syndrome". Clinical Rheumatology 12.2 (1993): 261-264.
- 5. Shiota J., et al. "Antemortem diagnosis of Marchaifava-Bignami Disease". Rinsho Shinkeigaku 29.6 (1989): 701-706.
- 6. Yazici K and L Kostakoglu. "Cerebral blood flow changes in patients with conversion disorder". *Psychiatry Research: Neuroimaging Section* 83.3 (1998): 163-168.
- 7. Aimard G., et al. "Does Astasia-Abasia of parietal origin exist?" Presse Médicale 14.31 (1985): 1666.
- 8. Laroche C et al. "Astasia-Abasia. Unilateral left-sided apraxia and touch disorder in an astrocytoma of the Corpus Callosum. A clinic-pathological report". Annales de Médecine Interne 127.1 (1976): 1-10.
- 9. Kumral E., et al. "Callosal infarction after bilateral occlusion of the internal carotid arteries with hemineglect syndrome and astasia-abasia". Revue Neurologique 151.3 (1995): 202-205.
- 10. Mota A. "Psychogenic Astasia-Abasia: a case report and a review of the literature". European Psychiatry 33 (2016): S325.
- 11. Stankovic M., et al. "Astasia-Abasia and Ganser Syndrome in a preadolescent girl: a case report". Srpski Arhiv za Celokupno Lekarstvo 143.7-8 (2015): 446-450.
- 12. Kataoka H., et al. "Novel representation of Astasia associated with posterior cingulate infarction". Stroke 37.1. (2006): e3-e5.

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