

Tactile Hallucination and Delusion Following Acute Stroke: a Case Report

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ABSTRACT

Tactile hallucination and delusion following acute stroke: a case report

Although many psychiatric disorders, especially depression, may be seen after stroke, development of psychosis is rare. Post-stroke psychiatric disorders are associated with the affected brain regions and may appear with different symptoms. Although psychotic symptoms have been reported in association with strokes in different brain areas, temporoparietooccipital lesions have a higher possibility of leading to the development of secondary psychosis than those in other brain areas. This article aims to present a case of tactile hallucination and delusion in a previously healthy man that developed after ischemic stroke with right temporoparietooccipital involvement.

Keywords: Delusion, ischemic stroke, pathophysiology, tactile hallucination

ÖZET

Akut inme sonrası gelişen taktıl halüsinasyon ve delüzyon: Olgu sunumu

Inme sonrası, başta depresyon olmak üzere birçok psikiyatrik bozukluklar görülebilmese karşın psikoz gelişimi nadirdir. İnme sonrası gelişen psikiyatrik bozukluklar, etkilenen beyin alanları ile ilişkilidir ve farklı belirtiler ile karşımıza çıkabilir. Beyinde farklı inme lokalizasyonlarında psikotik belirtilerin geliştiği bildirilmektedir. Ancak, temporoparietookspital alan lezyonlarında diğerlerine göre daha yüksek oranda ikincil psikoz gelişme olasılığı bulunmaktadır. Bu makalede, sağ temporoparietookspital tutulumlu iskemik inme sonrası gelişen ve delüzyonel olarak yorumlanan taktıl halüsinasyon olgu sunumunun yapılması amaçlanmıştır.

Anahtar kelimeler: Delüzyon, iskemik inme, patofizyoloji, taktıl halüsinasyon



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INTRODUCTION

Nowadays, after cardiac diseases and cancer, stroke is the third largest cause for mortality. Despite a reduction of its incidence and mortality thanks to risk control over the last years, stroke is still one of the most important reasons for death (1). After stroke, a number of psychiatric conditions can be seen, such as depression, most of all, but also anxiety disorders, behavioral disorders, apathy, mania, and psychosis (2-4). Compared to other psychiatric presentations, mania and psychosis occur more rarely (4,5) (Table 1). Psychiatric complications developing after stroke affect not only the patient's social life negatively, but impact on their entire quality of life and the rehabilitation process (6,7).

The relation between a cerebrovascular event and secondary psychosis was first described by Westphal in 1879. A 42-year-old male patient, developing left hemiplegia and left homonymous hemianopsia before his death, reported the sight of bright colors and hallucinations of a sword hanging above his head that might come down at any moment. Westphal described the aspect of the patient in that moment as "fixing his eyes on the ceiling as if he was seeing something scary". At autopsy, brain atrophy and an involvement of the posterior section of the right hemisphere were observed (8).

Physical and psychiatric symptoms developing after stroke show correlations with the affected brain regions (9,10). Anatomically, it has been established that stroke lesions in the temporoparietal/temporoparietooccipital

Table 1: Neuropsychiatric presentations accompanying stroke (4,5)

Syndrome	Prevalence	Clinical signs
Depression	35%	Depressive mood, reduced appetite, weight loss, insomnia, anergy, anhedonia, social withdrawal
Anxiety Disorder	25%	Increased anxiety, unease, physical signs like palpitation, sweating, difficulties to concentrate or to fall asleep
Mania	Rare	Raised mood, reduced need to sleep, flight of ideas, increased speed and amount of speaking, grandiosity
Psychosis	Rare	Hallucinations and delusions
Apathy	20%	Indifference and negligence (independent from depression)
Pathologic affect	20%	Inappropriate laughing and crying attacks
Catastrophic reaction	20%	Anxiety attacks developing in states of physical and cognitive inadequacy, crying, aggressive behavior, swearing, rejection and compensatory self-praise
Anosognosia	24%	Denial of post-stroke deficits without concern

section, and particularly in the right rather than the left hemisphere, have a higher probability to lead to the development of secondary psychosis (11-13). In addition, psychosis cases with effects from the deep subcortical structures can be found in the literature (14-16). After stroke, a number of psychiatric disorders can develop, but psychosis is relatively rarely seen (9,16). Case reports about the development of psychosis after stroke are quite rare in the literature, and data about psychotic disorders related to stroke are limited (17).

Aim of this report is to present a case of acutely developing tactile hallucinations, interpreted as delusional, after an infarction of the right middle cerebral artery (MCA) border region, and to discuss psychiatric presentations that may occur in relation to stroke in the light of information from the relevant literature.

CASE

H.O., a 61-year-old right-handed man, was admitted to the emergency room with complaints of forgetfulness and unusual behavior. After examination, the patient was admitted with a diagnosis of acute right MCA infarction. The patient provided written informed consent during his inpatient treatment.

Around 3 months earlier, the patient had suffered a transitory episode of dysmnesia while talking to his daughter on the phone, not remembering who she was. He described a moment of short-term confusion experienced the day before presenting to the hospital, while praying in the mosque: "I was praying in the mosque, and everyone was prostrating, but I was

standing up; I was confused..."

It was learned that the following morning he had started to search for someone who had pushed him by his neck, trying to make him fall down, later pushing him on the stairwell in his own house. As the patient said: "That morning, it was as if someone put his hands on the back of my neck. I grabbed with my hand, as if I was holding his finger. I asked my wife 'is there anyone?'" His family reported that the patient had opened the door of his house and was searching for someone on the staircase, angrily shouting "Someone pushed me from the bed to the ground".

The patient was known to have suffered from hypertension for 10 years; he was regularly using irbesartan 300mg/day and hydrochlorothiazide 12.5mg/day. No other disease was found, and the patient used neither alcohol nor any other psychoactive substance.

The patient was examined psychiatrically and neurologically. In the psychiatric examination, he appeared appropriate for his age, with sufficient self-care and anxious affect. He showed full cooperation and orientation, speed and amount of speaking were normal, associations were linear and goal-directed. During the patient interview, occasional blocks in his speech were observed. Other than the acute tactile hallucination during the seizure and the subsequent delusional state, no further active psychotic signs were found in the patient. Abstract thinking was preserved and reasoning in the test complete.

In the neurological examination, the patient's eyes were spontaneously open and aligned to the center

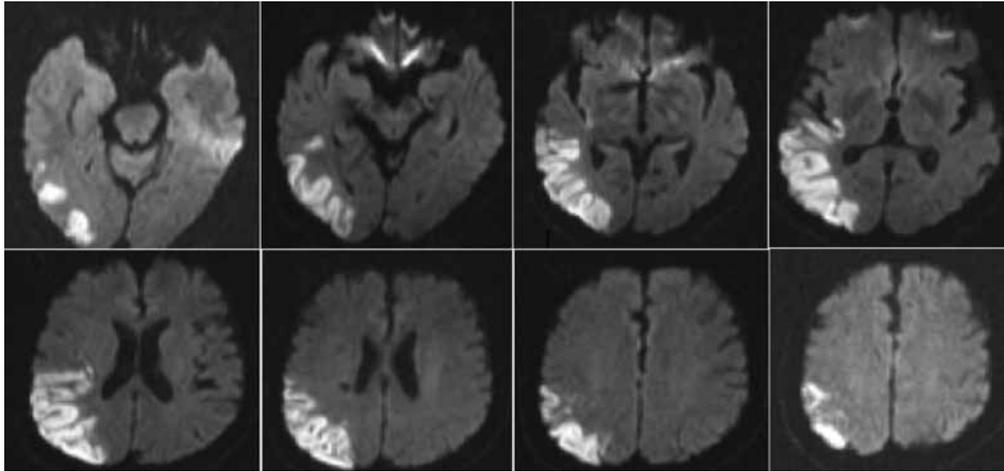


Figure 1: Cranial MRI consistent with border zone infarction

line. Other than a frust paresis in the right upper extremity, there were no motor deficits. Plantar responses were bilaterally flexor.

The patient's laboratory tests (hemogram, biochemical tests and sedimentation) were normal.

In the diffusion brain MRI, a cortico-subcortical diffusion restriction at the level of the temporooccipital lobe in the upper section was observed, showing partly continuity towards the parietal lobe, consistent with acute infarction. The result was consistent with a border zone infarction (Figure 1).

Bilateral carotid and vertebral artery Doppler USG examination found multiple millimeter-thick local parietal fibrocalcific plaque formations in the carotid and its branches, which did not cause hemodynamic changes.

With a diagnosis of acute ischemic cerebrovascular disease, the patient was started on antiaggregant and anticoagulant therapy. The antihypertensive treatment for the patient's hypertension was continued. During monitoring, vital signs and clinical state appeared stable. At discharge, the patient's self-care was assessed as good, his affect adequate, cooperation and orientation complete, association goal-directed, perception normal. The patient showed residual signs of an acute psychotic presentation developed secondarily to an ischemic attack; he still stated that the state experienced during the attack was real. Other than that, no active psychotic signs were found. Abstract thinking and test reasoning were evaluated as complete. Insight was partly present.

DISCUSSION

While after stroke a number of neuropsychiatric disorders can be seen, especially depression, reports about acute psychotic attacks are quite rare in the literature (4,9).

While the risk of developing psychosis is higher in temporoparietooccipital lesions, secondary psychosis has also been reported with focal lesions in other brain regions. Depending on the localization of these lesions, differences in the psychotic presentation can be observed (9,11,18) (Table 2). Kumral et al. (19) did a study with 15 patients, reporting that the great majority of persecutory and jealousy-type delusions developing after stroke were related to the right posterior temporoparietooccipital region.

In right hemisphere lesions, secondary psychosis is more likely to develop than in hemisphere lesions. This has been identified as a risk factor for secondary psychosis (13,25,26). Some sources, however, state that both right and left hemisphere lesions can result in psychotic symptoms (27,28).

Generally, a strong correlation is found between the lateralization of the lesion and the related neuropsychiatric clinical presentations. Cases of mania and alexithymia almost always have right hemisphere lesions. Anxiety, sexual behavior disorder, dissociative state and palinopsia cases are reported to show isolated right hemisphere or bilateral rather than isolated left

Table 2: Cases of cerebrovascular lesions and accompanying psychotic presentations

Author	Psychotic Presentations	Localization of Lesion
Kumral and Ozturk, 2004 (19)	Jealousy or persecutory or somatic delusions	Mostly lenticular, thalamic, and medullar lesions in the right posterior temporoparietooccipital cortex
Nagaratnam and O'Neile, 2000 (20)	Tactile hallucinations and bizarre delusions	Left temporoparietooccipital ischemic stroke
Beniczky et al., 2002 (21)	Complex visual and tactile hallucinations	Right temporoparietooccipital ischemic stroke
Narumoto et al., 2005 (22)	Persecutory delusions	Bilateral caudate ischemic infarction (caput caudatum lesion)
Nye and Arendts, 2002 (23)	Episodic olfactory hallucinations	Hemorrhagic left uncus lesion
Berthier and Starkstein, 1987 (24)	Sporadic auditory and sensory hallucinations accompanied by complex visual hallucinations Reduplicative paramnesia	Wide right frontotemporoparietal ischemic infarction
Barboza et al., 2013 (17)	Delayed persecutory-type delusion	Right frontotemporoparietal lesion
Peroutka et al., 1982 (31)	Complex hallucinations and delusions	Right temporoparietooccipital lesion
Levine and Finklestein, 1982 (32)	Delayed psychosis: Hallucinations and in some cases delusions and agitation	Right temporoparietooccipital lesion

hemisphere lesions. Visual hallucinations and psychosis cases have been reported equally for both hemispheres (18). Cutting (25) proposed the hypothesis that there are similarities between right hemisphere lesions and psychosis, and that schizophrenia might primarily be a right-brain dysfunction. However, some researchers assert that in schizophrenia, a dysfunction is evident in the left hemisphere or in both, rather than on the right side (29,30).

Rabins et al. (26) report temporoparietal lesions in five cases with post-stroke atypical psychosis, accompanied by subcortical atrophy. Subcortical atrophy and right hemisphere lesions have been reported as risk factors for psychosis. Some sources have claimed that only the anatomical localization of the lesion alone may not be sufficient to explain the psychosis and that lesion-related seizures might increase the probability for the development of psychosis. Levine and Finklestein (32) reported that in seven out of eight cases with right temporoparietal stroke or traumatic injury psychosis occurred together with a seizure. As a hypothesis, it may be thought that the lesion region causes continuous electrical activity, thus constituting the organic substrate for the psychotic phenomenon (16). The association between post-lesion seizure and acute psychosis is important for the diagnosis and the therapeutic process of the disease.

It is still not precisely known how focal lesions in

which regions constitute what kind of psychotic presentations. In our case, it can be assumed that an ischemic lesion in the right MCA border irrigation area involving the right temporoparietooccipital region triggered the patient's acute psychotic presentation.

The inadequacy of today's approaches to psychiatric diagnostic systems shows that we still need a better understanding of the pathophysiology of psychiatric diseases. A broader collaboration between neurology and psychiatry may point a way towards an understanding of a number of neuropsychopathologies still waiting for an explanation (33). In conclusion;

1. After a stroke, a number of psychiatric presentations can develop, first of all depression. Depending on localization, different clinical presentations can be seen.

2. Psychotic symptoms are rarely seen after stroke. The likelihood of psychotic symptoms is higher with right hemisphere temporoparietooccipital lesions. However, with other localizations, especially in deep subcortical structures, psychosis can also be seen.

3. A good understanding of the psychiatric signs accompanying focal brain lesions can provide important hints for an understanding of the organic reasons for psychiatric diseases.

4. After stroke, seizures can be seen and related neuropsychiatric presentations can occur. This condition may affect diagnosis and treatment process of the disease.

Contribution Categories	Name of Author
Follow up of the case	E.A., B.T.
Literature review	E.A., F.O., B.T.
Manuscript writing	E.A., F.O.
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