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Parkinsonism and progressive atypical psychiatric symptoms in the post-stroke subacute period

Post-stroke subakut evrede görülen progresif atipik psikiyatrik belirtiler ve parkinsonizm

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ABSTRACT

Stroke has a significant impact on the psychological and physical health of patients. Approximately 1%–4% of patients, movement disorders develop in the early post-stroke period. While chorea appears within hours of a stroke, post-stroke dystonia and tremor may develop after several months. Depression is generally observed in the post-stroke period; however, reports of atypical psychiatric symptoms, such as psychosis, in the early subacute-stroke period are rare. Herein, we demonstrate the case of a patient in whom parkinsonism symptoms and atypical psychiatric findings were found, which progressed to 3 and 2 weeks, respectively, after ischemic stroke.

Keywords: Stroke, Parkinsonism, Movement Disorder, Behavioral Disorders, Psychosis

ÖZET

İnmenin, hastaların psikolojik ve fiziksel sağlığı üzerinde önemli etkisi vardır. İnme sonrası erken dönemde yaklaşık %1-4 oranında hareket bozukluğu görülür. İnme sonrası genellikle depresyon görülürken, erken subakut dönemde nadiren psikoz benzeri atipik psikiyatrik belirtiler olabileceği belirtilmekteydi. Burada iskemik stroke sonrası ikinci haftada atipik psikiyatrik bulgular, üçüncü haftada parkinsonizm semptomlarının başlayarak progresif seyir gösterdiği bir vakayı sunmayı amaçladık.

Anahtar Kelimeler: İnme, Parkinsonizm, Hareket Bozukluğu, Davranış Bozuklukları, Psikoz

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INTRODUCTION

Stroke is a common neurological disease engendered by vascular causes. Stroke is characterized by the rapid development of clinical symptoms produced by focal or global disturbances of cerebral function, with the symptoms lasting longer than 24 hours (Investigators, 1988). In recent years, dramatic improvements in amelioration and sequelae rates are achieved thanks to the stroke centers that have become widespread, in our territory (Çıplak, Adıgüzel & Akalın, 2022).

Movement disorders are extremely rare in the early post-stroke period, and previous studies have reported them in only approximately 1%–4% of the cases (Suri et al., 2018). Parkinsonism is a disease mainly associated with neurodegeneration; however, it could also be attributed to iatrogenic and vascular pathologies or trauma (Handley, Medcalf, Hellier, & Dutta, 2009). Although psychiatric symptoms such as depression (most common), mania, generalized anxiety disorder, and emotional lability have been reported (Srivastava, Agarwal & Gautam, 2017), atypical psychotic symptoms similar to schizophrenia have rarely been reported in the post-stroke period (Lo Buono et al., 2019).

Although the average latency interval of post-stroke parkinsonism is shown to be 117.5 days in previous studies, it was observed on the 21st day in this study (Ghika-Schmid, Ghika, Regli & Bogousslavsky, 1997). Herein, we present a case with parkinsonism findings, which appeared 3 weeks post-stroke and progressed with atypical motor and psychiatric findings.

CASE

A 78-year-old right-handed man unremarkable history of health conditions, except for atrial fibrillation and diabetes mellitus, was admitted to the hospital with the complaint of sudden onset of slurred speech and numbness and weakness in the right arm and leg. Neurological examination identified dysarthric speech, muscle strength of 4/5 in the upper right and lower extremities, and Babinski sign on the right. Brain diffusion-weighted magnetic resonance imaging (DW-MRI) detected restricted diffusion compatible with acute ischemia, starting from the head of the left caudate nucleus and continuing up to the level of the corona radiata (Figure 1). He was admitted to the neurology department for 15 days and discharged with the prescription of acetylsalicylic acid (100 mg/day) and clopidogrel (75 mg/day). In the follow-up evaluation 3 weeks post-stroke, the patient exhibited slow bilateral reactions and tremors, which were especially prominent while moving the right arm, difficulty in daily life and self-care activities, disorganized speech and behaviors, and difficulty in walking. On neurological examination, the patient demonstrated poor facial expression, dominant bilateral resting tremor, and bradykinesia in the upper right extremity, short step length, and postural instability. He was subsequently diagnosed with subacute post-stroke parkinsonism. Treatment was initiated with levodopa (100 mg) and benserazide (25 mg) with a daily dose of 2 × ½, with an aim of gradually increasing the dose to 4 × 1 daily at the end of 2 weeks.

Psychiatric assessment of the patient revealed behavioral changes, such as defecation without removing the clothes, walking around the house naked, introversion, decreased speech, overeating, insomnia, soliloquy, visual hallucinations, and occasional skepticism, starting from 21 days post-stroke. Prior to this, the patient had an extroverted personality and good social communication skills and functionality and had shown no history of psychiatric illnesses. The examination of the patient's mental state revealed that he made occasional eye contact and that his appearance was appropriate for his sociocultural level; however, his self-care behavior was observed to be mediocre. Furthermore, the patient displayed normal orientation, apathy, and a lack of spontaneous speech. He showed a diminished capability of abstract thinking, insight, and reasoning. The psychological symptoms were recorded as consistent and not fluctuating, and because the laboratory findings were normal, delirium was excluded. The patient was treated with escitalopram (10 mg/day) and olanzapine (2.5 mg/day) and was followed up in the neurology and psychiatry clinic; however, reassessment was not possible because of the patient's death 3 weeks after the examination.

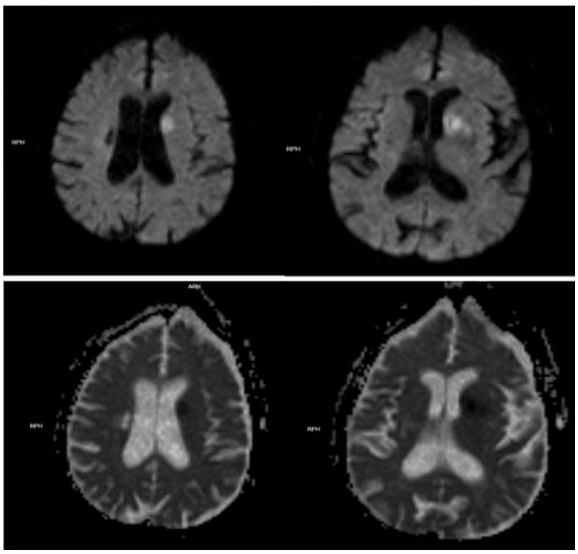

Figure 1. Diffusion restriction in the left caudate nucleus on brain DWI-MRI sequences.

DISCUSSION

Movement disorders (dystonia, chorea, and parkinsonism) are rare complications of strokes. Although post-stroke parkinsonism accounts for 22% of secondary movement disorders, approximately only 1%–4% of strokes cases develop parkinsonism. Dystonia, observed in 23.2% of all stroke cases, is the most frequent post-stroke movement disorder. The frequency of parkinsonism is high in ischemic stroke cases (17.4%), and chorea (17.4%) and hemorrhagic stroke cases present with a high prominence of dystonia (45.5%) and tremors (19.7%).

Chorea develops within hours of a stroke, whereas post-stroke dystonia and tremors may develop after several months. While motor deficits might improve post-stroke in some patients, involuntary movements may occur due to pathological neural connections. Contrary to previous studies, the present case manifested secondary parkinsonism symptoms (e.g., tremors) within weeks and not months.

Suri et al. examined 284 patients with post-stroke movement disorders and showed that lesions in the basal ganglia, thalamus, cerebellum, and most frequently, the posterolateral thalamus (22.5%) were the causes for such disorders (Suri et al., 2018). Lesions in the putamen (19%) and caudate nucleus (14%) have also been reported as the frequent causes of movement disorders. In this case, the lesion was present in the caudate nucleus. Secondary parkinsonism may be influenced by the anatomical localization and size of the infarction, thalamocortical connections, or the genetic predisposition of the person (Bonelli & Cummings, 2022). The frontal–subcortical circuits connecting the frontal cortex, basal ganglia, and thalamus process information from various regions of the brain, mainly the dorsolateral, prefrontal, orbitofrontal, and medial frontal regions. Ischemia of the dorsolateral prefrontal region deteriorates executive functions, problem-solving, and abstract thinking and leads to impulsivity, apathy, and personality changes. Furthermore, disinhibition and behavioral disorders are seen in cases of orbitofrontal damage. Akinetic mutism, amnesia, and congestion are observed in medial frontal lobe damage (Bonelli & Cummings, 2022). Cognitive and behavioral disorders have been commonly reported in cases of caudate nucleus infarctions (Wagner & Begaz, 2008). The caudate nucleus is the major relay center of the basal ganglia and thalamocortical circuits and connects the frontal, temporal, and parietal lobes via the cortico–pallido–nigral–thalamocortical circuits, which maintains the frontal lobe functions, fine motor coordination, and modulation of the associative functions (Kumral, Evyapan & Balkir, 1999). The rate of delirium in stroke cases is in the range of 6.7%–66% (Droś et al., 2020), and Rhee et al. reported in their meta-analysis that delirium is developed in the acute and subacute periods of anterior cerebral stroke (Rhee et al., 2022). Despite the finding of anterior cerebral infarction in this case, delirium was excluded based on the orientation and laboratory findings of the patient and the consistency of the psychiatric evaluation findings. We postulated that the movement disorders and behavioral changes were caused by the loss of frontal lobe functioning following caudate nucleus infarction. Vascular parkinsonism (VP) is differentiated from idiopathic Parkinson's disease (IPD) and is a separate clinical entity. Resting tremor in the upper extremity often begins unilaterally in IPD and responds well to levodopa (Adıgüzel, Öztürk & Altinayar, 2020). On the contrary, post-stroke parkinsonism often develops bilaterally and does not respond well to levodopa. The other hallmarks of VP include a history of hypertension, more prominent parkinsonism in the lower extremities, and the absence of tremor (Handley et al., 2009). This case was considered atypical because of the unilateral onset of symptoms in the upper extremity and the presence of resting tremor in the contralateral extremity. We hypothesized that the atypical symptoms were caused by the anatomical configuration of the cortico–pallido–nigral–thalamocortical circuits.

In previous studies, the onset of behavioral changes and temporary psychotic symptoms were reported 1 week after a right cerebral hemisphere stroke (especially in the posterior cerebral artery irrigation area), whereas the progressive atypical motor and psychiatric symptoms, in this case, occurred 2 weeks after the left basal ganglia infarction (in the middle cerebral and anterior arteries irrigation area) (Ferreira, Machado, Santos, & Machado, 2017). The development of cerebral hypoperfusion due to stroke increases the extent of damage in the infarction area. This damage causes structural and functional disorders associated with that area, which may lead to various psychiatric symptoms, such as hemiparesis and movement disorders, in addition to motor symptoms (Wagner & Begaz, 2008).

Delusions and hallucinations in the acute post-stroke period have been reported in approximately 4.86% of patients. In this case, uncommon psychiatric symptoms developed in the early post-stroke period. Additionally, contrary to the psychotic-like disorganized symptoms caused by the lower right frontal lobe and superior longitudinal fascial damage as reported in previous studies, the psychotic symptoms associated with left caudate nucleus lesions were observed in this case. This finding is suggestive of frontal lobe dysfunction.

CONCLUSION

In conclusion, only a few studies have so far reported the timeframe for parkinsonism and atypical psychiatric symptom development in the early post-stroke period. Herein, we described a case that presented with parkinsonism and atypical psychiatric symptoms, which continued and progressed in the acute–subacute post-stroke period. However, reassessment could not be performed as the patient died during the diagnosis and follow-up phase. The documentation of this rare case with remarkably atypical clinical features is expected to contribute to the literature.

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Author Contributions:

Concept: Sibel ÇIPLAK; **Design:** Sibel ÇIPLAK, Mustafa AKAN; **Data Collection or Processing:** Sibel ÇIPLAK, Mustafa AKAN, Melike ABA; **Analysis or Interpretation:** Sibel ÇIPLAK, Mustafa AKAN, Melike ABA; **Literature Search:** Sibel ÇIPLAK, Mustafa AKAN; **Writing:** Sibel ÇIPLAK, Mustafa AKAN.

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