Case Report

Psychotic Disorder after Left Posterior Cerebral Artery Stroke—An Atypical Event

Henrique Nascimento[1,*]
Bárbara Almeida[2]

[1]Neurology Department, Centro Hospitalar Universitário de Santo António (CHUdSA), 4099-001 Porto, Portugal
[2]Psychiatry Inpatient Unit, Hospital de Magalhães Lemos, 4149-003 Porto, Portugal

Abstract

Introduction: Stroke survivors usually present physical and neuropsychiatric complications. Post-stroke psychosis (PSPsy) is a particularly neglected sequel despite its disruptive nature.

Objectives: To present a case of early emerging neuropsychiatric symptoms following a left posterior cerebral artery (PCA) stroke. To review and discuss PSPsy clinical manifestations, pathophysiology, and clinical outcomes.

Clinical Case: A previously autonomous 68-year-old woman with vascular risk factors and depressive disorder presented to the emergency department with a 5-day history of disorientation, motor aphasia, and right hypoesthesia. Computer tomography revealed a left PCA stroke. She was started on acetylsalicylic acid and rosuvastatin and discharged the next day. Afterward, the patient developed a depressive mood, emotional lability, periods of confusion, delusions of persecution, guilt and unworthiness, auditory hallucinations, and suicide ideation. She was admitted to a psychiatric hospital and started on risperidone with a good response, being discharged after 15 days with the resolution of psychiatric symptoms.

Conclusions: PSPsy is more common after right hemisphere lesions and usually develops after some months. Nevertheless, our patient presented PSPsy following an ischemic event of the left PCA, with neuropsychiatric symptomatology dominating the clinic since the beginning. The involvement of the retrosplenial cortex or its connections was likely important for this atypical presentation. Due to the lack of guidelines on approaching PSPsy, most patients are treated with the same strategies used for non-stroke patients. A better comprehension of the anatomical basis underlining the symptomatology in these patients could deepen the understanding of psychosis and psychotic disorders.

Keywords

stroke; psychosis; left posterior cerebral artery; clinical-anatomical correlations

Introduction

Stroke is the second leading cause of death in people over 60 years [1]. Nevertheless, stroke is not always fatal, and patients can live for many years after a cerebrovascular accident, although usually with sequels. The physical sequels of stroke have been the focus of investigation and rehabilitation therapy for many years [1,2]. On the other hand, stroke is also associated with a spectrum of possible neuropsychiatric complications, both cognitive and non-cognitive [3,4]. The most common non-cognitive complications are depressive and anxiety disorders with a prevalence of up to 37% and 24%, respectively [5,6]. Psychotic disorders, on their turn, are a rare stroke sequel, occurring in around 5% of cases. However, considering the high incidence of stroke, there is a large number of people living with stroke-associated psychosis, many of whom are missing a formal diagnosis. In general, post-stroke psychiatric symptoms are underdiagnosed and undertreated, with a focus on the more frequent depressive and anxiety disorders. Post-stroke psychosis (PSPsy) is particularly neglected despite its disruptive effect on the quality of life [7].

There is no widely accepted diagnostic criterion for psychosis and psychotic disorders after stroke. According to the Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-V), post-stroke psychotic disorders would fall in the "psychotic disorders due to another medical condition" category. Like in other psychotic disorders,

*Corresponding author details: Henrique Nascimento, Neurology Department, Centro Hospitalar Universitário de Santo António (CHUdSA), Porto, Portugal. Email: hnascimento.neurologia@chporto.min-saude.pt

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the main symptoms of PSPsy are hallucinations or delusions, which may be accompanied by additional features such as disorganized thought or behaviour [8].

PSPsy can be a presenting symptom but usually appears to develop after an interregnum of some months. Furthermore, no perfect lesion-symptom relationship has been demonstrated to cause PSPsy; nonetheless, right hemisphere lesions are much more common in almost 80% of cases [7].

In this report, we presented an atypical case of early emerging neuropsychiatric symptoms following a left posterior cerebral artery (PCA) stroke. Afterwards, we reviewed and discussed the available literature on PSPsy, with a focus on presentation, lesion topography and clinical correlations, risk factors, treatment, and outcomes.

Clinical Case

At the end of May 2021, the patient, a 68-year-old previously autonomous woman, went to the emergency department (ED) with complaints of confusion and reduced strength and numbness in her right limbs.

Regarding relevant background information, she lived with her husband, daughter and grandchild, and was retired, having worked as a secretary with 12 years of schooling (high school). Her past medical history included diabetes mellitus type 2, hypertension, obesity, osteoporosis, and vertiginous syndrome. Regarding the patient's past psychiatric history, she had a mild depressive episode 25 years before (in reaction to life events), which was followed in primary care, medicated with sertraline (50 mg qd), and well-controlled until the events reported herein. Other usual medications included telmisartan + hydrochlorothiazide (80 + 25 mg qd), metformin (500 mg bid), beta-histine (24 mg bid), ibandronic acid (150 mg qw), and calcium carbonate + cholecalciferol (1500 mg + 400 mg qd). Family history was unremarkable from what could be assessed.

Five days before her admission, she started complaining about a strange feeling on her right limbs, "like something wasn't right", but couldn't explain it well. Despite these complaints, she continued her regular activities without apparent limitations. However, her husband observed that she was also presenting short periods of incoherent speech and had problems naming some objects. The patient confirmed that there was "something wrong in her head" in the previous days. She denied other symptoms, namely loss of conscience, traumatic injuries/falling, headaches, visual changes, and other motor or sensitive changes.

On admission to the ED, her general examination was unremarkable. Neurologic examination presented the following relevant findings: disoriented in time; verbose, although with tangential speech and some anomic pauses; difficulty in naming simple objects but no problem with repetition or following orders; no visual field defects on confrontation; no apparent motor deficit; inconsistent right hypoesthesia; and bizarre gait with no clear neurologic pattern (right leg approached the floor in a strange, seemingly antalgic, way).

A brain computed tomography (CT) scan with CT-angiography revealed a hypodensity of subcortical predominance in the internal part of the left occipital lobe, compatible with a subacute stroke of the ipsilateral PCA. The vascular study revealed an obstruction of the P2 segment of that artery. The images also presented a small old right striatocapsular ischemic lesion with extension to the respective corona radiata and subtle-moderate leukoencephalopathy. The remaining blood analysis and EKG were only relevant for a discrete increase of LDL-cholesterol.

She was diagnosed with a stroke of the posterior circulation of probable atherosclerotic etiology and started on acetylsalicylic acid 100 mg/day and rosuvastatin 20 mg/day. Due to clinical stability, she was discharged the next day.

Since the discharge, the patient presented a pattern of depressive mood with emotional lability and periods of confusion. She was evaluated in a neurology appointment five months after the events and, due to those complaints, was prescribed quetiapine 50 mg at night (that she did not take), sertraline was increased to 100 mg daily, and a psychiatry appointment was requested.

Approximately three months later, in the psychiatry appointment, the patient and her family described a history of a psychotic disorder, with persecutory delirium and sustained suicidal ideation, with apparent onset after the vascular event. The seriousness of the clinical picture warranted her admission to a psychiatric hospital.

At admission, the patient was distressed and restless, with a depressed mood. Her speech showed delusions of persecution, guilt, and unworthiness: she verbalized that she was being prosecuted because of wrong actions she took against her family; she said that all her family members had passed away and she deserved to be punished. The patient also presented auditory hallucinations with derogatory and threatening voices, suicide ideation, and initial insomnia.

Blood tests (which included a complete blood count, biochemistry with renal, hepatic, thyroid, ions, vitamin

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B12, and acid folic), urine analysis, and serological tests were drawn. The results were normal.

Given the clinical picture, sertraline was maintained, and risperidone (1 mg daily) for the psychotic symptoms and quetiapine (25 mg at bedtime) for her insomnia were added.

With the administration of the antipsychotic, the delusions and hallucinations were remitted, and her mood stabilized. However, it was notorious the presence of cognitive impairment in multiple domains, with dependence on her activities of daily living, such as personal hygiene and dressing. Cognitive evaluation was performed near the end of the hospitalization, after psychiatric stabilization. In Addenbrooke's Cognitive Examination-Revised, the patient scored 38/100 (with deficits in all of the six domains), and on the Frontal Assessment Battery, the score was 10/18.

After 15 days of hospitalization, the patient was discharged. At that time, the patient was calm, oriented in person, but disoriented in time and space; the mood was euthymic, without emotional lability; speech was spontaneous but poor in content; she didn't have delusions of any theme or presented auditory hallucinations; she slept well, and there were no ideas of death or suicide ideation. The patient was discharged with the following psychotropics: sertraline 100 mg (at breakfast), quetiapine 25 mg (at bedtime), donepezil 10 mg (breakfast), risperidone 0.25 mg (at breakfast and lunch), and risperidone 0.5 mg (at bedtime).

Discussion

Our patient had a presentation of PSPsy following an ischemic event of the left PCA. Neuropsychiatric symptomatology has dominated the clinic since the beginning, with the other changes in the neurologic exam, besides motor aphasia, being limited and inconsistent.

Posterior cerebral artery stroke accounts for around one-quarter to one-fifth of ischemic strokes. The most common sign is visual field defects (e.g., homonymous hemianopsia); other signs include aphasia, dysarthria, dysphagia, contralateral limb weakness and sensory deficits, as well as ataxia [9]. Considering the left PCA stroke, alexia without agraphia is its most characteristic sign [10].

Neuropsychiatric symptoms are common in stroke patients, being present in more than 30% of the cases. Depressive and anxiety disorders are particularly frequent [5,6]. One of the first studies that tried to find the prevalence of PSPsy was a cohort study published in 1991 and included 1191 stroke patients with a 9-year follow-up. Only five patients were identified as suffering from psychosis [11]. Stangeland *et al*. [7], in a 2018 systematic review, reported a prevalence of 4.67% and 5.05% for delusions and hallucinations, respectively, and a pooled prevalence rate of psychosis after the stroke of 4.86%. Patients with PSPsy had a mean age of 66.6 ± 16.6 years, a slightly higher percentage of men (53.5%), and a more significant proportion of ischemic events (around 80%). PSPsy usually followed right hemispheric stroke and was consequently accompanied more frequently by left hemiparesis (10.6%), headaches (4.2%), and dysarthria (3.8%), besides other less common symptoms such as left-sided neglect and left visual field defects. A relation with the type of stroke did not appear to be relevant as the proportion between ischemic and hemorrhagic strokes was similar in the general population and patients with PSPsy (80% of ischemic and 20% of hemorrhagic strokes) [7].

Psychosis was the only presenting symptom of stroke in 8% of PSPsy patients [7]. This "silent stroke" presentation can lead to wrongly diagnosing a pure psychiatric disease [5]. Usually, PSPsy accompanies other neurological symptoms in presentation or can develop after months [7]. In agreement, one study described delusions as usually appearing two days (range 1–3) after stroke and lasting a mean of 13 days [12], while an Australian retrospective study reported 6.1 months as the mean time for psychosis to develop [13]. In what long-term incidence is concerned, the same study reported a cumulative incidence of psychosis after stroke over a 12 years-period of 6.7% in patients without previous psychiatric disease [13]. The longer time suggested by the last study is more in line with the appearance of psychotic symptoms in traumatic brain injury (TBI) patients, which generally occur after two or more years following the trauma [7,14]. Since the presentation of the vascular event, our patient, on her turn, seemed to present psychiatric symptoms, such as emotional lability. The exact timing for the appearance of delusional and hallucinatory activity is more challenging to point out, but it also seemed to be present from the beginning.

In the review by Stangeland *et al*. [7], unspecified psychosis or psychotic features were the most common presentation, followed by delusional disorder, schizophrenia-like psychosis, and mood disorders with psychotic features. Delusion's themes were varied: persecutory delusions were the most frequent, while Othello syndrome/delusional jealousy, reduplicative paramnesia, and somatic delusions were present in a similar portion of patients. Regarding hallucinations, auditory was the most common modality, followed by visual hallucinations [7]. The overall higher prevalence of delusion over hallucinations and delusional theme distribution in stroke patients are similar to TBI cases. This fact,

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together with the already referred delayed onset, highlights that similar mechanisms are probably involved in psychosis development after stroke and TBI [7,14,15]. Our patient persecutory and delusions of ruin were, thus, in line with the most common psychotic manifestations after cerebrovascular accidents.

No perfect lesion-symptom relationship has been demonstrated to cause PSPsy. Nonetheless, right hemisphere lesions are much more common, being present in 79.1% of the patients, while left hemisphere and bilateral lesions were described in 14.2% and 6.7%, respectively [7]. Structural correlation studies reported that lesions related to psychosis are centered on the right frontal, temporal, and parietal lobes, right caudate nucleus, and white matter lesions with connectivity to those areas [2]. This agrees with right hemisphere pathology being associated with a range of perceptual anomalies and pathologies of belief [16,17]. Moreover, right hemisphere lesions can cause the loss of motivated withdrawal reactions to emotional stimuli, producing disinhibited behaviours, which can contribute to more noticeable psychiatric manifestations [6]. However, it is essential to consider that the limitations in speech that might accompany left hemisphere strokes could limit the capacity to perceive and diagnose psychotic disorders [7]. In fact, our patient presented with a mild form of motor aphasia, limiting the extent to which she could express herself. The presence of the old right striatocapsular lesion, reported in the CT scan, could also have contributed to the appearance of psychosis. Nevertheless, the temporal association of PSPsy onset and left PCA stroke makes it more likely that this was the precipitating factor. However, the contribution of a previous strategic lesion and cerebral small vessel disease to the overall clinical picture is more than possible.

Patients with little cognitive reserve are susceptible to the development of delirium, an acute disorder of attention and cognition, if subject to an insult (e.g., a metabolic imbalance or a urinary tract infection) [18]. It could be hypothesized that an underlying medical intercurrence could have been present at the time that our patient went to the medical appointment, contributing to the psychiatric symptoms. Yet, this did not appear to be the case as she had a more or less stable clinical course after the cerebrovascular accident, and the analysis done at hospital admission was normal.

In contrast, the probability of a vascular contribution to cognitive deficits in our patient is very likely. Cognitive assessment of inpatients should be done outside of disease exacerbation, and results should be interpreted with caution [19]. The evaluation of our patient was performed after the stabilization of psychiatric manifestations and demonstrated the presence of deficits in multiple domains. It has been reported that identifying cognitive impairment in hospitalized patients is associated with better outcomes as the care plan can be adjusted to the patient's needs [20]. In agreement with the results obtained, and besides psychiatry, our patient will maintain a follow-up in neurology, and the cognitive function should be reevaluated in ambulatory.

An interesting study using lesion network mapping to evaluate misidentification delusions reported that the left retrosplenial cortex was the area functionally connected to all lesion sites, potentially highlighting its role in the aetiology of PSPsy in PCA [21]. Although our patient did not present this particular type of delusion, her stroke could have also affected an area or neural networks globally involved in delusional activity. In agreement, a very similar case to ours was described by Castaño Ramírez *et al*. [5]. In that case, a woman in her sixties, without previous psychiatric history, had psychosis as the sole presenting feature, with no other findings in the neurologic examination. The clinical picture also included emotional lability, disorientation, complex visual hallucinations, and persecutory delusions. Magnetic resonance imaging (MRI) revealed an acute ischemic stroke in the territory of the left PCA with hemorrhagic reperfusion of the occipital cortex. Complete and spontaneous resolution of her clinical condition was achieved after approximately 15 days [5].

Risk factors for PSPsy are not well known. Previous psychiatric history, alcohol abuse, and depression are the most commonly reported comorbidities [11]. However, a history of psychiatric disease was only reported in 7.8% of the patients who developed psychosis. Our patient had a history of depression that, although well-controlled, probably contributed to PSPsy or made its symptoms more exuberant. Other risk factors reported include hypertension, hyperlipidemia, and diabetes, all well-recognized risk factors for stroke [7], and all present in the patient described in this case.

There is no consensus or guidelines on how to treat PSPsy. This is most likely related to the relatively lower percentage of that complication in comparison with depression or generalized anxiety disorders, but also due to the low number of reported cases and absence of clinical trials. Consequently, these patients are treated with the same strategies used on non-stroke patients with, usually, a good response. Approximately half of the patients who were treated with antipsychotics attained complete recovery, and partial recovery was reported in around one-quarter of the cases [7]. The most commonly used antipsychotic medications for PSPsy include haloperidol and second-generation

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antipsychotic drugs, such as risperidone, quetiapine, and olanzapine. On average, patients needed 3.5 months to achieve complete recovery, highlighting that patients respond well to the non-standardized treatment [7]. Likewise, our patient had an impressively fast response with a relatively low dose of medication (risperidone 1 mg/day and quetiapine 25 mg/day), and in less than two weeks, saw the resolution of psychotic symptoms and suicidal ideation.

Despite the generally good results obtained with traditional drugs, the use of antipsychotics for patients with stroke raises some questions. Some of these drugs have been associated with adverse metabolic effects on glucose and lipid homeostasis and could potentially contribute to a second cerebrovascular event. Olanzapine and clozapine presented the most unfavorable effects on metabolism in a meta-analysis [22]. In agreement, either first or second-generation antipsychotic drugs appear to increase the risk of stroke, especially for patients with vascular dementia [23,24]. This risk might be higher in patients treated with first-generation antipsychotic drugs [25]. The magnitude of increased stroke risk is likely not the same in all stroke patients with PSPsy. For example, a large case-control study found that neither first nor second-generation antipsychotic drugs increase the risk of stroke in non-elderly subjects with non-cognitive decline [26]. Patients with many cardiovascular risk factors, such as ours, would benefit from a regular evaluation of those factors. This would allow timely therapeutic adjustments, both by changing antipsychotics for others with a better metabolic profile and by introducing/substituting drugs directed at risk factors to improve their control (e.g., antihypertensives, antidyslipidemics, and antidiabetics).

The use of psychological/non-pharmaceutical approaches has presented interesting results in PSPsy. For instance, cognitive-behavioural techniques might help alleviate the distress caused by hallucinations or delusional beliefs [2,27].

Post-stroke psychiatric disorders, in general, are related to worsened outcomes, including lower quality of life and functional status, and increased risk of death and stroke recurrence [28,29]. Moreover, it appears that if not resolved in the initial period after stroke, psychosis tends to become a stable feature. Likewise, a study reported less than 5% variation in the prevalence of delusion or hallucinations over one year following a stroke [30]. Ten-year mortality was 51% higher in stroke patients with psychosis when compared with patients without psychiatric disorders. Part of this increased mortality and worse outcomes are caused by the same factors that affect patients with primary psychosis [13].

Conclusions

In conclusion, psychosis is a rare stroke complication when compared to other physical and neuropsychiatric sequelae. Nevertheless, it impacts the patient's functionality and quality of life, and might also weight on the family and caregiver. Also, the aging population will increase the incidence of strokes in the following decades, and consequently, even less common complications will amount to significant numbers.

Well-designed diagnostic criteria and assessment tools will be crucial to identifying and treating these patients earlier. This will also allow the study of treatments specifically directed to PSPsy patients.

Finally, a better comprehension of the anatomical basis underlining the diverse symptomatology present in vascular patients could deepen the understanding of psychosis and psychotic disorders in both stroke and non-stroke patients.

Availability of Data and Materials

Not applicable.

Author Contributions

HN and BA participated in the evaluation of the patient, review of clinical data, draft and final approval of the manuscript. The authors are accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This manuscript details a purely observational clinical study that does not require any intervention beyond standard medical care, and is therefore eligible for an ethical exemption. Written informed consent was obtained for the publication of this case report.

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Conflict of Interest

The authors declare no conflict of interest.

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