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# Post-Stroke Psychosis and Neuropsychiatric Disturbances in an Older Adult: A Diagnostic Challenge and Case Report

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**Abstract: Post-stroke neuropsychiatric disturbances may manifest as affective, cognitive, behavioral, or psychotic symptoms and may represent a diagnostic challenge, particularly when structural epilepsy coexists with brain lesions involving different vascular territories. We present the case of a 67-year-old man with a history of arterial hypertension, type 2 diabetes mellitus, and obstructive sleep apnea syndrome, who had experienced ischemic strokes in the territories of the right posterior cerebral artery and the left middle cerebral artery approximately one month before admission, with residual nonfluent aphasia and right upper-limb paresis. Fifteen days before admission, he experienced a late-onset focal seizure and was started on lacosamide. He subsequently developed progressive anxiety, depressed mood, psychomotor agitation, cognitive decline, behavioral changes, persecutory delusions, ideas of reference, and visual and auditory hallucinations. During the evaluation, he was conscious, without evident fluctuations in the level of consciousness or clinical findings consistent with delirium. Metabolic and infectious studies did not identify an alternative cause. Brain computed tomography showed right occipital and left frontoparietal encephalomalacia, as well as cerebral small-vessel disease, without evidence of intracranial hemorrhage or a new acute ischemic event. Video-EEG monitoring did not record epileptiform activity or electrographic seizures during the monitoring period. Brain magnetic resonance imaging was requested to rule out additional structural lesions; however, the study could neither be completed nor retrieved because the patient was transferred to another institution. During the observed hospital stay, he remained neurologically stable, without new focal deficits or recurrent seizures. He received symptomatic treatment with olanzapine, lacosamide was continued, and he underwent joint evaluation by the Neurology and Psychiatry services. The exact doses, subsequent pharmacological adjustments, and longitudinal course of the psychiatric symptoms could not be established because institutional follow-up was lost after the transfer. This case highlights the complexity of distinguishing post-stroke psychiatric manifestations from a new cerebrovascular event, delirium, nonconvulsive epileptic activity, postictal states, and primary psychiatric disorders. Integration of the clinical timeline, mental status examination, neuroimaging, and electrophysiological studies is essential to guide diagnosis and establish individualized treatment.**

***Keywords:*** *Stroke; Post-Stroke Psychosis; Neuropsychiatric Symptoms; Delusions; Hallucinations; Cognitive Decline; Post-Stroke Epilepsy.*

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## I. INTRODUCTION

Stroke is one of the leading causes of death and acquired disability in adults. Although its motor, sensory, and language consequences are widely recognized, patients may also develop cognitive, affective, behavioral, and psychiatric disturbances that significantly affect functional recovery, quality of life, and social reintegration.(1) These manifestations may occur during the acute phase, several weeks after the event, or following a latency period of several months.

Post-stroke neuropsychiatric complications include depression, anxiety disorders, apathy, emotional lability, cognitive impairment, delirium, and, less frequently, psychotic symptoms.(1,2) Post-stroke psychosis may

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manifest as delusions, hallucinations, perceptual disturbances, agitation, and marked behavioral changes.(2) Its recognition may be difficult, particularly in patients without a previous psychiatric history, because these manifestations may initially be interpreted as a primary psychiatric disorder, an emotional reaction to disability, or a confusional state associated with hospitalization.

The pathophysiology of post-stroke psychiatric disturbances is complex and likely multifactorial. Proposed mechanisms include direct injury to brain regions involved in emotional and behavioral regulation, disruption of cortico-subcortical networks, white matter involvement, cerebral small-vessel disease, and loss of connectivity among frontal, temporal, parietal, and limbic regions.(1,2) Although certain lesion locations have been more frequently associated with specific symptoms, no single anatomical region accounts for all psychiatric manifestations following a cerebrovascular event. Therefore, clinical interpretation should consider not only lesion location but also lesion extent, laterality, timing, and effects on functional brain networks.

The diagnosis of a psychiatric disturbance secondary to stroke requires the exclusion of other potentially treatable conditions, including a new cerebrovascular event, delirium, infection, metabolic disorders, adverse medication effects, substance use, and focal or nonconvulsive epileptic activity.(3–6) This distinction is particularly challenging in patients with post-stroke epilepsy because ictal states, postictal states, and psychiatric disorders secondary to structural brain injury may share cognitive, perceptual, and behavioral manifestations.

We present the case of a 67-year-old man with a history of ischemic strokes involving different vascular territories who subsequently developed progressive anxiety, cognitive decline, behavioral disturbances, persecutory delusions, and hallucinations. This case illustrates the diagnostic complexity of post-stroke psychiatric manifestations and the need to integrate the clinical timeline, mental status examination, neurological assessment, neuroimaging, and electrophysiological studies.

## II. CASE REPORT

A 67-year-old male teacher with a history of arterial hypertension, type 2 diabetes mellitus, and obstructive sleep apnea syndrome was evaluated. Approximately one month before admission, he had experienced an ischemic stroke involving the territories of the right posterior cerebral artery and the left middle cerebral artery, with residual nonfluent aphasia, distal paresis of the right upper limb, and post-stroke cognitive impairment. He subsequently developed affective symptoms for which he was receiving sertraline and quetiapine. He also experienced a late-onset focal seizure attributed to a post-stroke structural brain lesion, and treatment with lacosamide was initiated.

He presented to the emergency department with an approximately 15-day history of progressive anxiety, depressed mood, psychomotor agitation, behavioral changes, and persecutory delusions. During the days preceding admission, he had also experienced visual and auditory hallucinations, ideas of reference, and a significant decline in his usual level of functioning. The persecutory symptoms were mainly directed toward his wife, whom he believed had threatening intentions toward him. He had previously been evaluated at another institution, where anxiety and depressive disorders were initially considered and pharmacological treatment was initiated, without significant clinical improvement.

On admission, he was conscious and cooperative, with a blood pressure of 131/70 mmHg, heart rate of 120 beats per minute, respiratory rate of 19 breaths per minute, and temperature of 36°C. He had no hypotension or signs of hypoperfusion, although tachycardia was documented. On mental status examination, he was alert, without evident fluctuations in the level of consciousness, and exhibited an anxious affect and depressed mood. His thought process remained organized, but its content was characterized by paranoid and persecutory delusions accompanied by ideas of reference. He denied active suicidal ideation. No marked attentional impairment or fluctuations in mental status suggestive of delirium were identified.

Neurological examination revealed nonfluent aphasia and right-hand paresis, corresponding to the previously known sequelae of the stroke. No new focal neurological deficits were identified. Given the development of de novo psychotic symptoms in an older adult with recent cerebrovascular disease, a secondary organic etiology was initially considered, and diagnostic investigations were undertaken to exclude acute metabolic, infectious, pharmacological, epileptic, and structural causes.(4,5)

Laboratory studies, including a complete blood count, renal function tests, serum electrolytes, liver function tests, thyroid function tests, and vitamin B12 levels, showed no abnormalities that could explain the symptoms. Brain computed tomography revealed areas of chronic right occipital and left frontoparietal encephalomalacia, together with changes consistent with chronic cerebral small-vessel disease, classified as Fazekas grade III. No intracranial hemorrhage or computed tomography findings suggestive of a new acute ischemic event were observed.

Video-EEG monitoring did not record epileptiform activity or electrographic seizures during the monitoring period. This finding reduced the likelihood that ongoing epileptic activity accounted for the symptoms, although it did not completely exclude intermittent focal epilepsy or a possible postictal state.(3,16)

The patient was jointly evaluated by the Psychiatry and Neurology services. Based on the temporal relationship between the stroke and the onset of symptoms, the presence of structural brain lesions, and the absence of metabolic or infectious abnormalities that could account for the clinical presentation, the psychiatric manifestations were considered probably secondary to the previous cerebrovascular disease. Nevertheless, the differential diagnosis continued to include a new ischemic event, nonconvulsive epileptic activity, a

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postictal state, medication-related effects, and a late-onset primary psychiatric disorder.(3–6)

During hospitalization, he received symptomatic treatment with olanzapine, while anticonvulsant therapy with lacosamide was continued. Previous treatment with sertraline and quetiapine for affective symptoms was also documented; however, the exact doses and subsequent psychopharmacological adjustments could not be reliably reconstructed from the available information. The patient was jointly evaluated by the Psychiatry and Neurology services. Because of the persistence of psychiatric symptoms, brain magnetic resonance imaging was requested to exclude additional structural lesions or a new ischemic event not detected on computed tomography. During the observed hospital stay, he remained neurologically stable, without new focal deficits or recurrent seizures. He was subsequently transferred to another institution; therefore, the magnetic resonance imaging result was unavailable, the definitive discharge diagnosis could not be documented, and longitudinal follow-up of the delusions, hallucinations, and other psychiatric symptoms could not be performed. Based on the available information, the most likely diagnosis was considered to be a psychiatric disturbance secondary to cerebrovascular disease, while a small new ischemic event, intermittent epileptic activity, a postictal state, and a late-onset primary psychiatric disorder remained in the differential diagnosis.

## III. DISCUSSION

The present case highlights the diagnostic complexity of psychiatric disturbances following stroke, particularly when they manifest as anxiety, cognitive decline, delusions, hallucinations, and behavioral changes. These manifestations may occur during the acute phase, several weeks after the event, or after a latency period of several months, and they may be confused with a primary psychiatric disorder, delirium, recurrent cerebrovascular disease, adverse medication effects, or focal epileptic activity.(1,2) In the present case, the onset of de novo psychotic symptoms after stroke in a patient without a previously established primary psychotic disorder raised the possibility of a psychiatric disturbance secondary to cerebrovascular disease. The absence of evident fluctuations in the level of consciousness, preservation of an organized thought process, and lack of metabolic or infectious abnormalities that could explain the clinical presentation reduced the likelihood of delirium. However, the coexistence of a late-onset focal seizure and brain lesions involving different vascular territories required a broad differential diagnosis.

Post-stroke psychosis may manifest as persecutory delusions, ideas of reference, visual or auditory hallucinations, agitation, affective disturbances, and marked behavioral changes. Although it is considered less frequent than post-stroke depression or anxiety, its occurrence may cause functional decline, interfere with rehabilitation, generate family conflict, and increase the need for interdisciplinary care.(2) The temporal relationship between the cerebrovascular event and the subsequent onset of psychiatric symptoms is relevant when considering a possible secondary etiology; however, this association alone does not establish causality, and other neurological, metabolic, infectious, toxic, and pharmacological conditions must therefore be excluded.

Nascimento et al. (2024) described a 68-year-old woman with an ischemic stroke in the territory of the left posterior cerebral artery who subsequently developed emotional lability, episodes of confusion, persecutory delusions, auditory hallucinations, and suicidal ideation.(9) Although post-stroke psychosis has been more frequently associated with right-hemisphere lesions and with onset after a period of latency, this report showed that it may also occur early after a left-hemisphere lesion and constitute the predominant clinical manifestation. This pattern is comparable to that observed in our patient, whose psychiatric symptoms developed after the cerebrovascular event and required the exclusion of new structural lesions and epileptic activity before they could be attributed exclusively to a primary psychiatric disorder.(9)

Mota Freitas et al. (2023) reported the case of a 77-year-old woman without a psychiatric history who developed persecutory, grandiose, and mystical delusions accompanied by auditory and tactile hallucinations several months after a lacunar infarction of the right caudate nucleus. The absence of metabolic, toxic, or infectious abnormalities, together with the location of the lesion, supported post-stroke psychosis as the most likely diagnosis.(8) This pattern partially resembles our case and supports the need to exclude neurological and medical causes before establishing a primary psychiatric diagnosis in an older adult with newly developed psychotic symptoms.

Available evidence indicates that psychosis may develop either early or after a latency period of several months. The most frequently reported lesions involve the right hemisphere, particularly the frontal, temporal, and parietal regions, as well as connecting white matter pathways and subcortical structures such as the caudate nucleus.(2,8,9) Nevertheless, psychotic manifestations have also been documented in association with left-hemisphere and bilateral lesions, suggesting that these disturbances do not depend exclusively on a single anatomical location. Clinical interpretation should therefore consider lesion extent, laterality, timing, and potential effects on brain networks involved in the regulation of behavior, cognition, and perception.

Huang et al. (2022) described two patients hospitalized with COVID-19 in whom acute delirium preceded the development of hemiparesis. The symptoms were initially interpreted as being related to hospitalization, which prolonged the diagnostic process and prevented reperfusion therapy from being administered within the therapeutic window.(10) Although these patients had a clinical context different from that of our case, the reports demonstrate that newly developed cognitive or behavioral changes may represent the initial manifestation of a new cerebrovascular event and should not automatically be attributed to a psychiatric cause.

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In one of the patients reported by Huang et al., the initial computed tomography scan showed no acute lesions, and diffusion-weighted magnetic resonance imaging subsequently confirmed an infarction in the territory of the right middle cerebral artery.(10) This finding is relevant to our case, in which computed tomography revealed only chronic-appearing lesions and magnetic resonance imaging was therefore requested to exclude a recent ischemic lesion not visible on computed tomography. Accordingly, the absence of acute findings on computed tomography should not be interpreted in isolation when newly developed neuropsychiatric symptoms persist or when there is a significant change from the patient's baseline cognitive or behavioral status.(10)

In our patient, computed tomography showed left frontoparietal and right occipital encephalomalacia, together with chronic cerebral small-vessel disease classified as Fazekas grade III. These findings indicate the presence of structural cerebrovascular damage that may have contributed to the development of cognitive, perceptual, and behavioral manifestations through disruption of cortico-subcortical networks involved in the regulation of behavior, cognition, and perception.(1,11) Frontoparietal involvement may be associated with executive dysfunction, behavioral disturbances, impaired integration of information, and altered interpretation of environmental stimuli.(11) Occipital lesions, in turn, may be associated with complex perceptual phenomena, including visual hallucinations.(12) Cerebral small-vessel disease also represents an additional vulnerability factor for cognitive decline and post-stroke neuropsychiatric manifestations.(1,11) These associations should be interpreted as clinically plausible mechanisms rather than as definitive evidence of causality in an individual patient.

From a broader perspective, delirium and other cognitive or behavioral changes may constitute initial manifestations of stroke and may be confused with depression, psychosis, or encephalopathy.(10) Therefore, in patients with previous cerebrovascular disease and acute cognitive or behavioral changes, a broad diagnostic approach should be maintained, including recurrent ischemia, nonconvulsive seizures, metabolic abnormalities, delirium, medication-related effects, and secondary late-onset psychiatric disorders.(4–6,18)

Hassanin et al. (2022) described a 68-year-old man without a formal psychiatric history who presented with a one-week history of intermittent visual hallucinations, paranoia, sleep disturbances, depressed mood, and self-injurious behavior.(13) Magnetic resonance imaging demonstrated an acute ischemic stroke involving the left hemisphere associated with critical stenosis of the left internal carotid artery. Following treatment of the stroke and carotid endarterectomy, the psychotic symptoms resolved, and the patient was discharged without requiring neuroleptic therapy. This case demonstrates that the acute or subacute onset of psychiatric symptoms in an older adult with vascular risk factors should prompt investigation for an organic cerebral cause before a primary psychiatric diagnosis is established.(13)

Ahmed et al. (2025) described a 94-year-old woman with a history of arterial hypertension and diabetes mellitus who presented with visual and auditory hallucinations, macropsia, agitation, paranoia, and other psychotic symptoms. Brain computed tomography showed areas of encephalomalacia consistent with chronic infarctions in the right posterior parietal region and left occipital lobe, together with changes related to cerebral small-vessel disease. Although Alice in Wonderland syndrome was initially considered, the structural findings supported a possible cerebrovascular etiology for the perceptual and psychiatric disturbances.(14) The patient experienced improvement in hallucinations, paranoia, and sleep disturbances after treatment with quetiapine.

The report by Ahmed et al. (2025) is particularly comparable to our case because of the presence of parieto-occipital lesions, cerebral small-vessel disease, visual and auditory hallucinations, and paranoid symptoms. Both cases demonstrate that cerebrovascular lesions may manifest predominantly through perceptual, cognitive, and behavioral disturbances, even without the simultaneous development of a new focal motor deficit. This similarity also supports the importance of correlating symptoms with lesion location without assuming that such an anatomical relationship establishes definitive causality.(14)

The coexistence of a late-onset focal seizure in our patient required consideration of whether the behavioral and perceptual manifestations could represent focal epileptic activity, a postictal state, or postictal psychosis. These conditions may present with anxiety, agitation, affective disturbances, delusions, hallucinations, and behavioral changes, particularly in patients with cognitive impairment or structural brain lesions.(3,15)

Regala et al. (2023), in a case report accompanied by a literature review, noted that postictal psychosis may manifest through diverse affective, behavioral, and psychotic symptoms. Its recognition requires careful assessment of the temporal relationship between seizures, recovery of consciousness, and the subsequent onset of delusions, hallucinations, or behavioral changes, while also excluding ongoing epileptic activity and other neurological, metabolic, pharmacological, or psychiatric causes.(15) Likewise, the absence of epileptiform activity during an electroencephalographic monitoring period does not completely exclude intermittent focal epilepsy, because epileptiform discharges may not occur during the recording.(16)

In our patient, the history of a late-onset focal seizure required consideration of nonconvulsive epileptic activity, a postictal state, and postictal psychosis as part of the differential diagnosis. However, the absence of new seizures during hospitalization and the lack of epileptiform activity or electrographic seizures on video-EEG monitoring reduced the likelihood that ongoing epileptic activity explained the entire clinical presentation. Epilepsy was therefore interpreted as a factor that increased diagnostic complexity rather than as the definitive cause of the delusions, hallucinations, anxiety, and behavioral changes. The

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temporal relationship with the stroke, the presence of structural cerebrovascular lesions, and the initial exclusion of metabolic and infectious causes supported the consideration of a psychiatric disturbance probably secondary to cerebrovascular disease. Nevertheless, because the magnetic resonance imaging result and subsequent follow-up were unavailable, a definitive causal relationship could not be established.

Bell et al. (2023) analyzed nationally representative epidemiological data from the United States, the United Kingdom, Chile, and Colombia and found a prevalence of probable psychosis of 3.81% among individuals with a history of stroke, together with an adjusted association between the two conditions of OR 3.32.(17) The authors noted that the coexistence of cerebrovascular disease and psychotic symptoms identifies a population with substantial clinical needs that remains insufficiently studied. In this context, our report provides an individual description of a Colombian patient with cerebrovascular lesions involving different territories and subsequently developing psychotic symptoms, in whom a focal seizure broadened the differential diagnosis but did not adequately explain the full range of psychiatric manifestations.

The diagnosis of a psychiatric disturbance secondary to stroke requires the exclusion of potentially reversible causes, particularly when psychotic symptoms first appear in an older adult.(18) In this patient, no clinically relevant abnormalities were identified in the complete blood count, renal function, serum electrolytes, liver function, thyroid function, or vitamin B12 concentration that could explain the symptoms. No fluctuations in the level of consciousness, marked inattention, or clearly delirium-compatible clinical course were documented. The absence of new focal neurological deficits and acute findings on computed tomography reduced the likelihood of an evident recurrent cerebrovascular event, although it did not completely exclude a recent small ischemic infarction. Brain magnetic resonance imaging was therefore requested to complement the structural evaluation.(10)

Potential medication-related effects and the development of a late-onset primary psychiatric disorder were also considered.(5,18) Nevertheless, the onset of psychotic symptoms after the stroke, the absence of a clearly established previous psychotic disorder, and the presence of structural brain lesions supported a probable relationship with cerebrovascular disease. Accordingly, the diagnosis was based on integration of the clinical timeline, mental status examination, neurological assessment, laboratory studies, neuroimaging, and video-EEG monitoring.

This case reinforces the importance of avoiding diagnostic anchoring in patients with newly developed psychiatric symptoms after stroke. Prematurely attributing delusions, hallucinations, or behavioral changes to a primary psychiatric disorder may delay the identification of a new cerebrovascular event, a metabolic disturbance, delirium, or epileptic activity.(4–6,10,18) Conversely, assuming that all post-stroke behavioral manifestations are caused by epilepsy may lead to unnecessary escalation of anticonvulsant therapy.(3,6,16)

The present report has several limitations, including the absence of the brain magnetic resonance imaging result and the inability to document the medium-term psychiatric outcome because the patient was transferred to another institution and subsequently lost to follow-up. Furthermore, the precise doses and final adjustments of psychopharmacological treatment could not be recovered. These limitations prevent the establishment of a definitive causal relationship between the cerebrovascular lesions and the psychiatric symptoms and preclude an objective assessment of treatment response. Nevertheless, the temporal sequence, absence of a clearly established psychotic history, structural brain findings, and initial exclusion of metabolic, infectious, and ongoing epileptic causes supported the consideration of a psychiatric disturbance probably secondary to cerebrovascular disease.

## IV. CONCLUSIONS

Post-stroke psychiatric disturbances frequently include depression and anxiety.(1,19) They may also manifest as delusions, hallucinations, perceptual disturbances, cognitive decline, and behavioral changes.(1,2) Their diagnosis is complex because they must be differentiated from delirium, recurrent cerebrovascular disease, metabolic or infectious disorders, medication-related effects, epileptic activity, and late-onset primary psychiatric disorders.

In the present case, the temporal relationship between the stroke and the onset of psychotic symptoms, the absence of an identifiable metabolic or infectious cause, the presence of structural cerebrovascular lesions, and the persistence of symptoms without recurrent seizures supported the possibility of a psychiatric disturbance secondary to cerebrovascular disease. The late-onset focal seizure increased the complexity of the differential diagnosis but did not adequately explain the full range of clinical manifestations.

The evaluation of these patients should integrate the clinical course, mental status and neurological examinations, neuroimaging, and electrophysiological studies. Computed tomography without evident acute lesions or video-EEG monitoring without epileptiform activity should be interpreted within the clinical context and not regarded as isolated tests capable of completely excluding a new ischemic event or intermittent focal epilepsy.(10,16)

Early recognition of post-stroke psychiatric manifestations and interdisciplinary collaboration among emergency medicine, neurology, psychiatry, and rehabilitation specialists may reduce diagnostic anchoring, guide the rational use of complementary investigations, and facilitate individualized treatment.(7) The documentation of similar cases may contribute to a better characterization of their clinical presentation, anatomical relationships, timing, course, and therapeutic response.

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Table 1. Previously Reported Cases of Neuropsychiatric Symptoms after a Stroke

| Authors (Year) | Patient | Stroke location | Neuropsychiatric manifestations | Neuroimaging findings | Outcome / Clinical message |
|---|---|---|---|---|---|
| Nascimento and Almeida (2024).(9) | 68-year-old woman | Left posterior cerebral artery ischemic stroke | Emotional lability, depressive symptoms, episodes of confusion, persecutory delusions, delusions of guilt and worthlessness, auditory hallucinations, and suicidal ideation | CT: left posterior cerebral artery infarction | Psychiatric symptoms resolved after treatment with risperidone; the patient was discharged after 15 days. |
| Mota Freitas et al. (2023).(8) | 77-year-old woman | Right caudate lacunar infarction | Persecutory, grandiose and mystical delusions, auditory and tactile hallucinations | Right caudate infarct | Highlighted the importance of excluding metabolic and infectious causes before diagnosing post-stroke psychosis. |
| Huang et al. (2022).(10) | Two patients | Acute ischemic stroke in the right middle cerebral artery territory | Acute delirium and agitation preceding the appearance of focal neurological deficits | Initial CT without acute findings; diffusion-weighted MRI confirmed right middle cerebral artery infarctions | Demonstrates that acute behavioral symptoms may be the initial manifestation of stroke and may delay reperfusion treatment. |
| Hassanin et al. (2022).(13) | 68-year-old man | Acute left hemispheric ischemic stroke associated with high-grade left internal carotid artery stenosis | Intermittent visual hallucinations, paranoia, sleep disturbance, depressed mood, and self-injurious behavior | MRI: acute left hemispheric infarction; vascular imaging: high-grade left internal carotid artery stenosis | Psychiatric symptoms resolved after carotid endarterectomy, and neuroleptic treatment was discontinued. |
| Ahmed et al. (2025).(14) | 94-year-old woman | Chronic right posterior parietal and left occipital infarctions | Visual and auditory hallucinations, paranoia, agitation, sleep disturbance, and macropsia | CT: parieto-occipital encephalomalacia and cerebral small-vessel disease | Highlights the association between parieto-occipital vascular lesions and complex perceptual and psychiatric manifestations. |
| Regala et al. (2023).(15) | Case report and literature review | Long-standing focal epilepsy with right parieto-occipital encephalomalacia secondary to traumatic brain injury | Postictal psychosis with pleomorphic affective, behavioral, and psychotic manifestations | Right parieto-occipital encephalomalacia | Emphasizes the importance of the lucid interval and the temporal relationship between seizures and psychotic symptoms when differentiating postictal psychosis from other causes. |
| Present case | 67-year-old man | Previous right posterior cerebral artery and left middle cerebral artery ischemic strokes, followed by a late-onset focal seizure | Progressive anxiety, depressed mood, psychomotor agitation, persecutory delusions, ideas of reference, visual and auditory hallucinations, cognitive | CT: right occipital and left frontoparietal encephalomalacia, severe cerebral small-vessel disease classified as Fazekas III, and | The patient remained neurologically stable, without new focal deficits or recurrent seizures during the observed hospitalization. Brain MRI and longitudinal |

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|---|---|---|---|---|---|
| | | | decline, and behavioral changes | no evidence of an acute lesion | psychiatric follow-up could not be completed because the patient was transferred to another institution. The case illustrates the diagnostic overlap among post-stroke psychiatric manifestations, recurrent cerebrovascular disease, delirium, focal epileptic activity, and postictal phenomena. |

Table 2. Laboratory Findings at Presentation

| **Laboratory findings at admission** | | |
|---|---|---|
| **Parameter** | **Result** | **Units** |
| Leukocytes | 7770 | cells per µL |
| Neutrophils | 68.5 | % |
| Lymphocytes | 24 | % |
| Hemoglobin | 15.3 | g/dl |
| Hematocrit | 46.6 | % |
| Platelets | 377000 | cells per µL |
| BUN | 20 | mg/dl |
| Creatinine | 0.75 | mg/dl |
| Glucose | 131 | mg/dl |
| Sodium | 144 | mmol/L |
| Potassium | 3.68 | mmol/L |
| Calcium | 8,9 | mg/dl |
| Magnesium | 2.1 | mg/dl |
| TSH | 3.41 | uU/Ml |
| AST | 19 | U/L |
| ALT | 29 | U/L |
| Total bilirubin | 0.65 | mg/dl |
| Direct bilirubin | 0.34 | mg/dl |
| Indirect bilirubin | 0.31 | mg/dl |
| Albumin | 4.1 | g/dl |
| pH | 7.52 | |
| PaO2 | 56.5 | mmHg |
| $PaCO_2$ | 29.8 | mmHg |
| $HCO_3^-$ | 26.5 | mmol/L |
| Base excess | 1,6 | |
| $SaO_2$ | 92.70 | % |
| Lactate | 1 | mmol/L |
| $FiO_2$ | 21 | % |
| $PaO_2/FiO_2$ ratio | 269 | |

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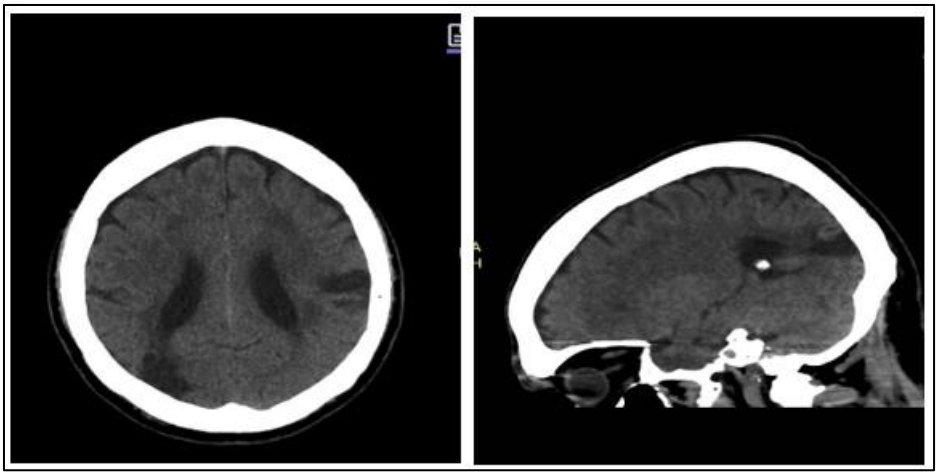

Fig 1 Computed Tomography Scan Showing Ischemic Sequale of Strok

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