



Neuropsychiatric symptoms after stroke

Accurate identification is vital to optimizing recovery and quality of life

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Many patients experience neuropsychiatric symptoms following stroke. There is tremendous variation in the type, severity, and timeline of these symptoms, which have the potential to significantly impact patients' quality of life. Some symptoms occur as a direct result of ischemic injury to brain structures regulating behavior, executive function, perception, or affect. Other symptoms occur indirectly due to the patient's often-difficult experiences with the health care system, disrupted routines, or altered poststroke functional abilities. Psychiatric symptoms are not as easily recognized as classic stroke symptoms (such as hemiparesis) and are frequently overlooked, especially in the acute phase. However, these symptoms can negatively influence patients' interpersonal relationships, rehabilitation, and employment.

Patients and families may not realize certain symptoms are stroke-related and may not discuss them with their clinicians. It is important to ask about and recognize psychiatric symptoms in patients who have experienced a stroke so you can provide optimal education and treatment. In this article, we review the types of psychiatric symptoms associated with strokes in specific brain regions (*Table*,¹⁻¹⁰ *page 32*). We also describe symptoms that do not appear directly related to the anatomical structures affected by the infarct, including delirium, psychosis, depression, anxiety, and posttraumatic stress.

Symptoms associated with stroke in specific regions

Frontal lobe strokes

The frontal lobes are the largest lobes in the brain, and damage to areas within these lobes can cause behavioral and personality changes. Lesions



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A stroke that damages areas in the frontal lobe can cause multiple behavioral and personality changes



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Table

Psychiatric symptoms of stroke by anatomic location

Location		Symptoms
Frontal lobe	Lateral frontal lobe	Aprosodia, executive dysfunction, lack of empathy, poor self-regulation, trouble with attention
	Superior and inferior medial cortices	Apathy, lack of motivation, altered self-regulation, altered emotional processing, disinhibition
	Basal forebrain	Confabulation, reduced motivation, delusions (Capgras syndrome, reduplicative paramnesia)
	Orbital cortex	Personality changes, impulsivity, poor social judgment, reduced empathy, altered self-regulation, lack of goal-directed behavior
	Subcortical	Personality changes, reduced emotions, poor empathy, irritability
Parietal lobe	Dominant	Language deficits
	Nondominant	Neglect/inattention of the opposite side, denial of symptoms, anosodiaphoria, aprosodia, flat affect, personality changes
Occipital lobe		Visual hallucinations (colors, scotomas, metamorphopsia, palinoptic images, complex hallucinations, Charles Bonnet syndrome)
Other locations	Midbrain or thalamus	Peduncular hallucinosis
	Subthalamic nucleus	Hemiballismus, mood and behavioral disturbances, personality changes, hyperphagia
	Caudate	Dorsolateral caudate: abulia, psychic akinesia, decreased problem-solving ability, reduced abstract thinking, diminished spontaneity Ventromedial caudate: disinhibition, disorganization, impulsiveness, affective symptoms with or without psychosis

Source: References 1-10

in the lateral frontal cortex can cause aprosodia (difficulty expressing or comprehending variations in tone of voice), which can lead to communication errors. Lateral frontal cortex injury can cause executive dysfunction and a lack of empathy¹ as well as trouble with attention, planning, and self-regulation that may affect daily functioning. Strokes affecting the superior and inferior mesial cortices may result in apathy, lack of motivation, altered self-regulation, altered emotional processing, and disinhibition. Patients who experience a basal forebrain stroke may exhibit confabulation, reduced motivation, and delusions such as Capgras syndrome (the belief that a person or place has been replaced by an exact copy) and reduplicative paramnesia (the belief that a place has been either moved, duplicated, or exists in 2 places simultaneously). Strokes involving the

orbital cortex can be associated with personality changes, impulsivity, poor social judgment, reduced empathy, altered self-regulation, lack of goal-directed behavior, and environmental dependency.

Some strokes may occur primarily in the subcortical white matter within the frontal lobes. Symptoms may be due to a single stroke with sudden onset, or due to repeated ischemic events that accumulate over time, as seen with microvascular disease. In the case of microvascular disease, the onset of symptoms may be insidious and the course progressive. Infarcts in the subcortical area can also cause personality changes (though typically more subtle when compared to orbitofrontal strokes), reduced emotions, poor empathy, and irritability.¹ Patients may lack insight into some of or all these symptoms following a frontal lobe infarct, which makes it critical

to gather collateral information from the patient's friends or family.

Parietal lobe strokes

Symptomatology from parietal strokes depends on whether the stroke affects the dominant or nondominant hemisphere. Dominant parietal lesions cause language deficits, and psychiatric symptoms may be difficult to elucidate due to the patient's inability to communicate.² On the other hand, patients with nondominant parietal stroke may have neglect of, or inattention to, the opposite (typically left) side.³ This often manifests as a reluctance to use the affected limb or limbs, in some cases despite a lack of true weakness or motor dysfunction. In addition, patients may also have visual and/or tactile inattention towards the affected side, despite a lack of gross visual or sensory impairment.² In rare cases, a patient's stroke may be misdiagnosed as a functional disorder due to the perceived unwillingness to use a neurologically intact limb. In severe cases, patients may not recognize an affected extremity as their own. Patients are also frequently unaware of deficits affecting their nondominant side and may argue with those attempting to explain their deficit. Anosodiaphoria—an abnormal lack of concern regarding their deficits—may also be observed. Additionally, aprosodia, flat affect, and personality changes may result from strokes affecting the nondominant hemisphere, which can impact the patient's relationships and social functioning.³

Occipital lobe strokes

While negative or loss-of-function symptomatology is one of the hallmarks of stroke, occipital lobe infarcts can pose an exception. Although vision loss is the most common symptom with occipital lobe strokes, some patients experience visual hallucinations that may occur acutely or subacutely. In the acute phase, patients may report hallucinations of varied description,⁴ including poorly formed areas of color, scotomas, metamorphopsia (visual distortion in which straight lines appear curved), more complex and formed hallucinations and/or palinoptic images (images or brief scenes that continue to be perceived after

looking away). These hallucinations, often referred to as release phenomena or release hallucinations, are thought to result from disinhibition of the visual cortex, which then fires spontaneously.

Hallucinations are associated with either infarction or hemorrhage in the posterior cerebral artery territory. In some cases, the hallucinations may take on a formed, complex appearance, and Charles Bonnet syndrome (visual hallucinations in the setting of vision loss, with insight into the hallucinations) has been identified in a small portion of patients.⁵

The duration of these hallucinations varies. Some patients describe very short periods of the disturbance, lasting minutes to hours and corresponding with the onset of their stroke. Others experience prolonged hallucinations, which frequently evolve into formed, complex images, lasting from days to months.⁶ In the setting of cortical stroke, patients may be at risk for seizures, which could manifest as visual hallucinations. It is essential to ensure that epileptic causes of hallucinations have been ruled out, because seizures may require treatment and other precautions.

Other stroke locations

Strokes in other locations also can result in psychiatric or behavioral symptoms. Acute stroke in the subcortical midbrain or thalamus may result in peduncular hallucinosis, a syndrome of vivid visual hallucinations.⁷ The midbrain (most commonly the reticular formation) is usually affected; however, certain lesions of the thalamus may also cause peduncular hallucinosis. This phenomenon is theorized to be due to an increase in serotonin activity relative to acetylcholine and is often accompanied by drowsiness.

The subthalamic nucleus is most frequently associated with disordered movement such as hemiballismus, but also causes disturbances in mood and behavior, including hyperphagia and personality changes.⁸ Irritability, aggressiveness, disinhibition, anxiety, and obscene speech may also be seen with lesions of the subthalamic nucleus.

Finally, the caudate nucleus may cause alterations in executive functioning and behavior.⁹ A stroke in the dorsolateral

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Patients who have an occipital lobe infarct may develop a wide range of visual hallucinations



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Delusional disorder is the most common psychosis that develops in patients who have experienced a stroke

caudate may cause abulia and psychic akinesia, decreased problem-solving ability, reduced abstract thinking, and/or diminished spontaneity, whereas an infarct in the ventromedial region of the nucleus may cause disinhibition, disorganization, impulsiveness, and, in severe cases, affective symptoms with psychosis.¹⁰ Strokes in any of these areas are at risk for being misdiagnosed because patients may not have a hemiparesis, and isolated positive or psychiatric symptoms may not be recognized as stroke.

Symptoms not related to stroke location

Delirium and psychosis

Following a stroke, a patient may exhibit neuropsychiatric symptoms that do not appear to relate directly to the anatomical structures affected by the infarct. In the acute phase, factors such as older age and medical complications (including infection, metabolic derangement, and lack of sleep due to frequent neurologic checks) create a high risk of delirium.¹¹ Differentiating delirium from alterations in mental status due to seizure, cerebral edema, or other medical complications is essential, and delirium precautions should be exercised to the greatest extent possible. Other neuropsychiatric symptoms may manifest following hospitalization.

Poststroke psychosis often presents subacutely. Among these patients, the most common psychosis is delusion disorder, followed by schizophrenia-like psychosis and mood disorder with psychotic features.¹² Some evidence suggests antipsychotics may be highly effective for many of these patients.¹² Poststroke psychosis does appear to correlate somewhat with nondominant hemisphere lesions, including the frontal lobe, parietal lobe, temporal lobe, and/or caudate nucleus. Because high mortality and poor functional outcomes have been associated with poststroke psychosis, early intervention is essential.

Depression

Depression is a common problem following stroke, affecting approximately 35% of stroke patients.¹³ In addition to

impairing quality of life, depression negatively impacts rehabilitation and increases caregiver burden. There is significant variability regarding risk factors that increases the likelihood of poststroke depression; however, psychiatric history, dysphagia, and poor social support consistently correlate with a higher risk.^{14,15} Characteristics of a patient's stroke, such as lesion volume and the ability to perform activities of daily living, are also risk factors. Identifying depression among patients who recently had a stroke is sometimes difficult due to a plethora of confounding factors. Patients may not communicate well due to aphasia, while strokes in other locations may result in an altered affect. Depending on the stroke location, patients may also suffer anosognosia (a lack of awareness of their deficits), which may impair their ability to learn and use adaptive strategies and equipment. An additional confounder is the significant overlap between depressive symptoms and those seen in the setting of a major medical event or hospitalization (decreased appetite, fatigue, etc). The prevalence of depression peaks approximately 3 to 6 months after stroke, with symptoms lasting 9 to 12 months on average, although many patients experience symptoms significantly longer.¹⁴ Because symptoms can begin within hours to days following a stroke, it is essential that both hospital and outpatient clinicians assess for depression when indicated. Patients with poststroke depression should receive prompt treatment because appropriate treatment correlates with improved rehabilitation, and most patients respond well to antidepressants.¹⁶ Early treatment reduces mortality and improves compliance with secondary stroke prevention measures, including pharmacotherapy.¹⁷

Anxiety and posttraumatic stress

Anxiety and anxiety-related disorders are additional potential complications following stroke that significantly influence patient outcomes and well-being. The abrupt, unexpected onset of stroke is often frightening to patients and families. The potential for life-altering deficits as well as intense, often invasive, interactions with

the health care system does little to assuage patients' fear. Stroke patients must contend with a change in neurologic function while processing their difficult experiences, and may develop profound fear of a recurrent stroke. As many as 22% of patients have an anxiety disorder 3 months after they have a stroke.¹⁸ Phobic disorder is the most prevalent subtype, followed by generalized anxiety disorder. Younger age and previous anxiety or depression place patients at greater risk of developing poststroke anxiety. Patients suffering from poststroke anxiety have a reduced quality of life, are more dependent, and show restricted participation in rehabilitation, all of which culminate in poorer outcomes.

Many patients describe their experiences surrounding their stroke as traumatic, and posttraumatic stress disorder (PTSD) is increasingly acknowledged as a potential complication for patients with recent stroke.¹⁹ PTSD profoundly impacts patient quality of life. Interestingly, most patients who develop poststroke PTSD do not have a history of other psychiatric illness, and it is difficult to predict who may develop PTSD. Relatively little is known regarding optimal treatment strategies for poststroke PTSD, or the efficacy of pharmacotherapy and psychotherapeutic strategies to treat it.

Goals: Improve recovery and quality of life

Neuropsychiatric symptoms are common following a stroke and may manifest in a variety of ways. While some symptoms are a direct consequence of injury to a specific brain region, other symptoms may be a response to loss of independence,

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- Saha G, Chakraborty K, Patojoshi A. Management of psychiatric disorders in patients with stroke and traumatic brain injury. *Indian J Psychiatry.* 2022;64(Suppl 2): S344-S354.

disability, experience with the medical system, or fear of recurrent stroke. The onset of psychiatric symptoms can be acute, beginning during hospitalization, or delayed. Understanding the association of psychiatric symptoms with the anatomical location of stroke may assist clinicians in identifying such symptoms. This knowledge informs conversations with patients and their caregivers, who may benefit from understanding that such symptoms are common after stroke. Furthermore, identifying psychiatric complications following stroke may affect rehabilitation. Additional investigation is necessary to find more effective treatment modalities and improve early intervention.

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continued

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There is significant overlap between depressive symptoms and those seen after any major medical event or hospitalization

Bottom Line

Neuropsychiatric symptoms are frequently overlooked in patients with recent stroke. These symptoms include delirium, psychosis, depression, anxiety, and posttraumatic stress disorder, and can be the direct result of injury to neuroanatomical structures or a consequence of the patient's experience. Prompt treatment can maximize stroke recovery and quality of life.



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Most patients who develop poststroke PTSD do not have a history of other psychiatric illnesses

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