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# Aggressive Behavior and Posterior Cerebral Artery Stroke

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**Objective:** To describe the mechanisms leading to aggressive behavior among patients with acute posterior cerebral artery stroke.

**Design, Setting, and Patients:** We prospectively included all of the patients with posterior cerebral artery stroke and aggressive behavior admitted to our department from January 1, 2003, to December 31, 2004. Patients with history of stroke, cognitive impairment, or prior history of psychiatric disease were excluded.

**Results:** Aggressive behavior was found in 3 patients (7.3%) among 41 patients with posterior cerebral artery stroke. One patient had right occipitotemporal and ventrolateral thalamic stroke. The second patient had left occipitotemporal and lateral thalamic stroke. The third patient had right isolated occipital stroke. In addition to a contralateral homonymous hemianopsia, the patients, who were physically and emotionally balanced before the

stroke, suddenly manifested an acute, unusual, aggressive behavior. The patients became agitated and aggressive when they were stimulated by the environment, and they responded to solicitations by their relatives or medical personnel by shouting obscenities and hitting and biting others. In all of the 3 cases, temporary physical restraint was required and neuroleptics were administered. This unusual behavioral pattern resolved within 2 weeks after stroke.

**Conclusions:** Aggressive behavior is a rare presentation of acute posterior cerebral artery stroke, which may be difficult to diagnose in patients presenting with hemianopsia as the only concomitant neurological sign. The postulated mechanisms include dysfunction of the limbic or serotonergic system.

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**A**GGRESSIVE BEHAVIOR (FROM the Latin *aggressus*, past participle of *aggreddi*, to attack) is considered in patients whose primary purpose is to injure another person or organism, whether physically or psychologically.<sup>1</sup> It is regarded as the result of a dysfunction of the limbic system, implying the ventromedial hypothalamic area,<sup>2,3</sup> the amygdala, and the orbitofrontal cortex.<sup>4</sup> In stroke, aggression has been described after hemispheric infarction in the territories of the anterior cerebral arteries, middle cerebral arteries, and posterior cerebral arteries (PCAs).<sup>5-7</sup> However, these studies were based on large series of patients. Although both middle cerebral artery stroke<sup>8-10</sup> and PCA stroke<sup>11-14</sup> have been widely described to be associated with confusion, there is a lack of detailed descriptions of patients with stroke and aggressive behavior.

## METHODS

We included all of the patients admitted to our department with isolated PCA stroke and ag-

gressive behavior from January 1, 2003, to December 31, 2004, and prospectively entered in the Lausanne Stroke Registry. All of the patients were hospitalized in our stroke unit within 24 hours of stroke onset. All of the patients with hemorrhagic stroke, history of stroke, combined top-of-the-basilar stroke, or other brain lesion as well as those with history of psychiatric disease, cognitive impairment, or long-term alcohol consumption were excluded. Patients with lacunar strokes, middle cerebral artery strokes, anterior cerebral artery strokes, and deep-seated hemorrhagic strokes were excluded. All of the patients had full routine investigation for stroke, including neuropsychological bedside evaluation, standard blood tests, electrocardiography, chest radiography, and brain computed tomography.

## RESULTS

Among 728 strokes included in the Lausanne Stroke Registry from January 1, 2003, to December 31, 2004, 41 patients (5.6%) had PCA stroke, 276 had middle cerebral artery stroke, and 8 had anterior cerebral artery stroke. There were 403 pa-

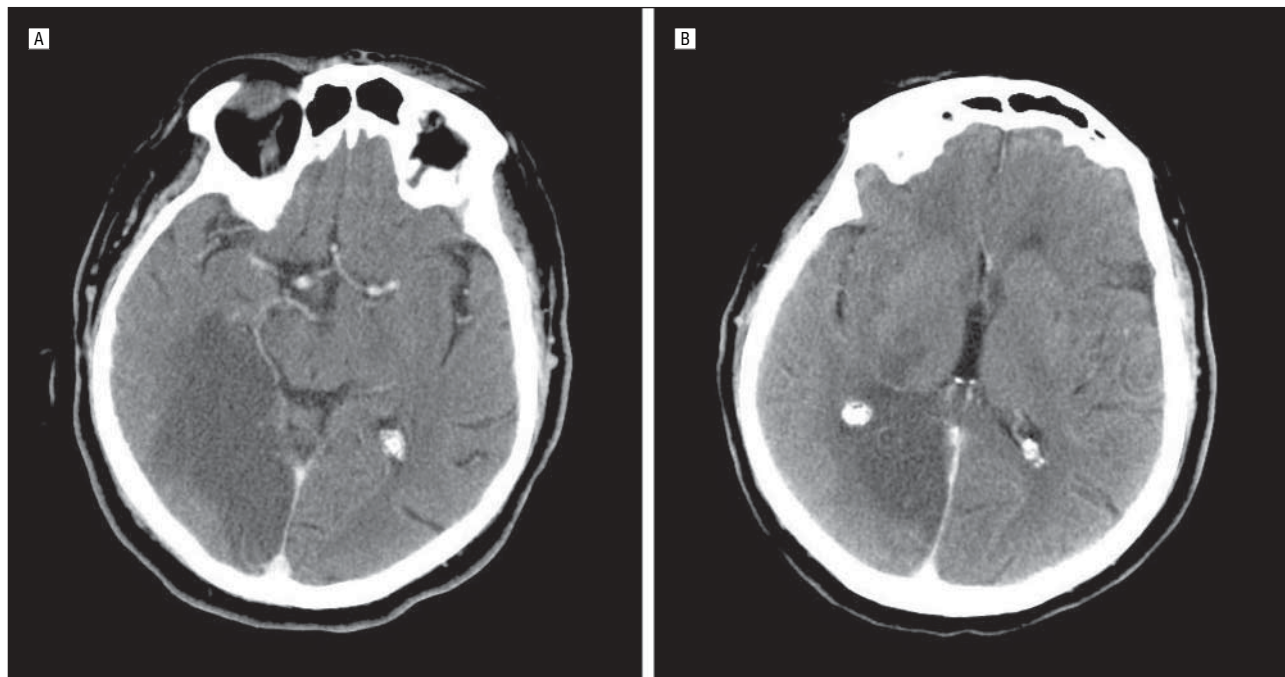
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**Table. Behavioral Changes After Isolated Posterior Cerebral Artery Stroke**

| Type of Behavior                           | Patients, No. | Thalamogeniculate Arteries <sup>a</sup> | Calcarine Artery <sup>a</sup> | Temporal Anterior Artery <sup>a</sup> | Temporal Posterior Artery <sup>a</sup> | Parieto-occipital Artery <sup>a</sup> | Choroidal Posterior Median Artery <sup>a</sup> | All Territories <sup>a</sup> | Partial Territories <sup>a</sup> |
|--|---------------|---|-------------------------------|---------------------------------------|--|---------------------------------------|--|------------------------------|----------------------------------|
| Aggressive                                 | 3             | 2                                       | 3                             | 2                                     | 2                                      | 2                                     | 2  | 2                            | 1                                |
| Confused                                   | 3             | 3                                       | 3                             | 3                                     | 3                                      | 3                                     | 3  | 3                            | 0                                |
| Agitated, not aggressive, not confused     | 1             | 1                                       | 1                             | 1                                     | 1                                      | 1                                     | 1  | 1                            | 0                                |
| Not agitated, not aggressive, not confused | 23            | 11                                      | 8                             | 13                                    | 16                                     | 12                                    | 7  | 2                            | 21                               |

<sup>a</sup>Values are expressed as the number of patients for each type of behavior who have a lesion in the respective arterial vascular territory.



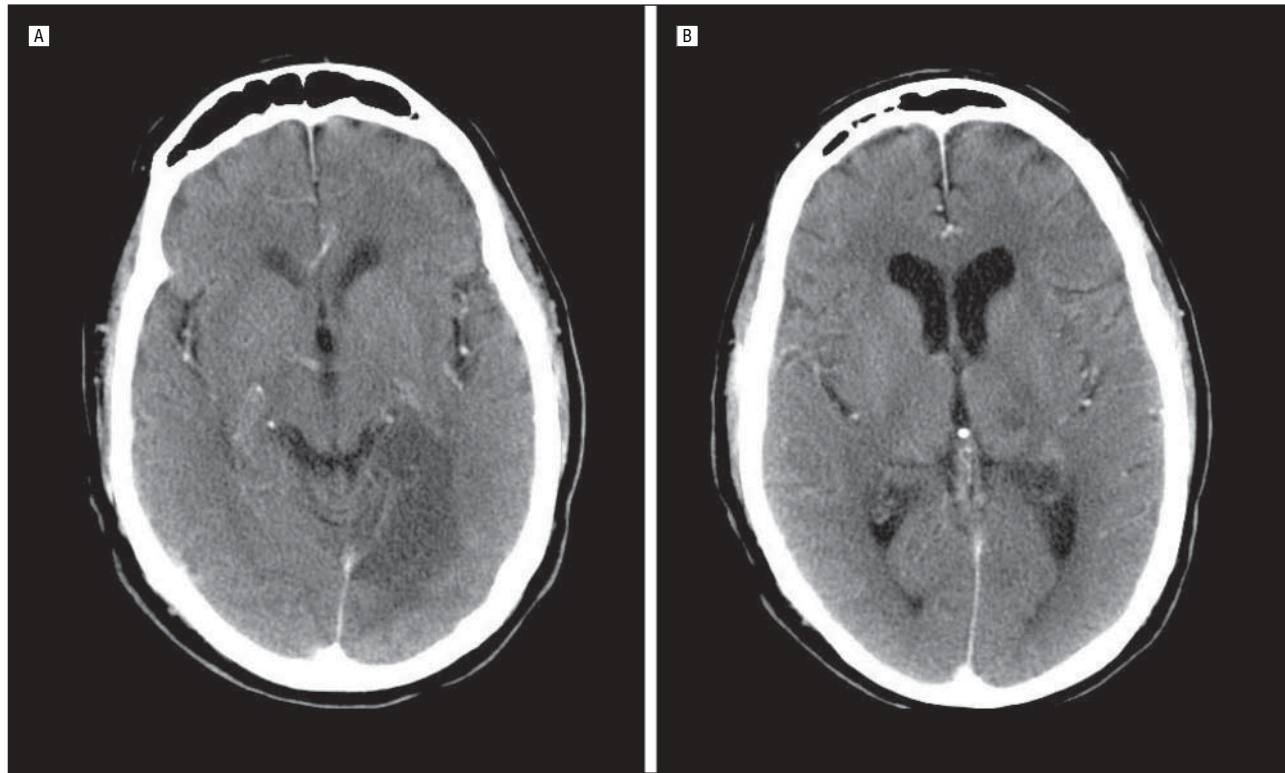
**Figure 1.** The patient had right occipitotemporal stroke (A and B) and ventrolateral thalamic stroke (B).

tients with lacunar stroke and deep hemorrhagic stroke. Three of the 41 patients with PCA stroke (7.3%) presented with aggressive behavior (3 men, aged 68, 74, and 83 years). One patient had right occipitotemporal and ventrolateral thalamic stroke. The second patient had left occipitotemporal and lateral thalamic stroke. The third patient had isolated right occipital stroke. All of the 3 patients displayed aggressive behavior that resolved within 2 weeks of the stroke, and all of the 3 patients had residual cognitive impairment after more than a year of follow-up. All of the 3 strokes had a cardioembolic origin. One patient with extensive PCA cortical stroke (sparing only the mesencephalus) was agitated but not aggressive. Three patients with full PCA cortical stroke were confused but not agitated or aggressive. Twenty-three patients with PCA stroke were neither agitated nor aggressive; of those 23 patients, 21 had partial cortical PCA stroke. The 2 patients who had extensive PCA cortical stroke and were neither agitated nor aggressive were young men (aged 32 and 40 years). The behavioral changes after PCA stroke are summarized in the **Table**. Eleven pa-

tients were excluded because they had concomitant infection, top-of-the-basilar associated stroke, or previous cognitive impairment.

### CASE 1

A 74-year-old man with a history of hypertension came to the hospital with left homonymous hemianopsia and left ataxic hemiparesia. At day 1, he was agitated, took off his oxygen mask and removed his intravenous lines, shouted obscenities, and tried to hit personnel whenever he was approached. One milligram of intravenous clonazepam was first administered, followed by intravenous neuroleptics. Physical restraint was finally necessary for the patient's own safety during the first 24 hours of hospitalization. Brain computed tomography demonstrated right occipital and temporal stroke with involvement of the ventrolateral thalamic nucleus (**Figure 1**). One milligram of haloperidol twice daily was continued during 10 days owing to recurrent outbursts of violence. The patient's behavioral disorder subsided, but mild



**Figure 2.** The patient had left occipitotemporal stroke (A) and lateral thalamic stroke (B).

cognitive memory impairment and left hemiparesia were still present after 18 months.

#### CASE 2

A 68-year-old man with a history of heart failure, diabetes, and hypertension was admitted for infectious endocarditis. At day 12 after admission to our hospital, the patient suddenly displayed aphasia with diminished verbal fluency and denomination ability, right homonymous hemianopsia, and right brachiofacial sensitive-motor deficits associated with ataxia. This man, who was known to be a quiet and reserved person, objected to daily care and responded angrily with familiar terms such as “I don’t give a damn!” He also flailed his arms and at some point tried to decisively hit the personnel. This behavior was apparent only when he was interacting with others. Brain computed tomography revealed left occipital and mesiotemporal stroke that included the lingual and fusiform gyri and the lateral thalamic nucleus (**Figure 2**). Intravenous neuroleptics were administered during 2 weeks before the patient could be discharged for rehabilitation. Neuropsychological examination 2 weeks later showed that the patient was disoriented in time and space and had deficits of attention but displayed no aggressiveness. Eighteen months later, the patient was apathic and had persistent anterograde memory deficits but did not display aggressive behavior.

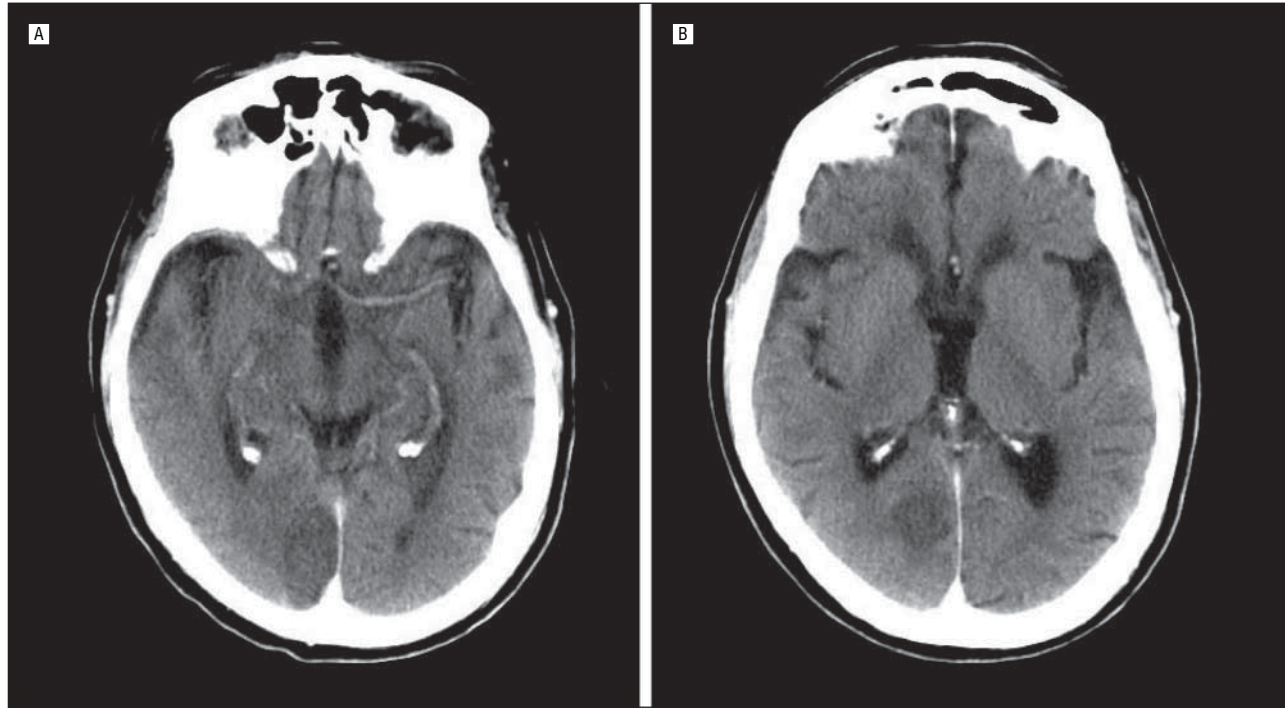
#### CASE 3

An 83-year-old hypertensive man was admitted to the hospital for diminished vision on the left side on awaken-

ing in the morning. Clinical examination revealed left homonymous hemianopsia and drift of the left arm. In the first day after admission, the patient objected to daily care and expressed paranoid delusions without hallucinations, as he was convinced that people wanted to capture and kill him. He wanted to get out of his room and out of the hospital. He became rapidly aggressive and tried to hit, bite, and scratch medical personnel. He only became agitated and aggressive when stimulated by contact with others. Physical restraint was not sufficient and intramuscular neuroleptic injections were required. Aggressiveness subsided and physical restraint was lifted the following day. On admission, brain computed tomography showed isolated ischemic right occipital stroke that included the lingual and fusiform gyri (**Figure 3**). Standard neuropsychological examination at day 5 showed partial orientation in time and space with anterograde memory disorder and mild executive dysfunction. However, no aggressive behavior or agitation was found. The patient left the hospital for rehabilitation 12 days after the stroke with persistent left homonymous hemianopsia. He returned home but had difficulties coping with stressful situations and became forgetful to a point where he was putting himself in danger. He was sent to a retirement home 1 year after the stroke. He did not, however, display violent behavior after the hospitalization.

#### COMMENT

These 3 patients, all with acute PCA stroke, presented with aggressive behavior and responded to trivial stimuli with purposely aggressive behavior. None of them had a



**Figure 3.** The patient had isolated ischemic right occipital stroke including the lingual (A and B) and pericalcarine (A and B) gyri.

history of alcohol consumption, metabolic disorder, or cognitive dysfunction and they were physically and emotionally balanced before the stroke. In addition to contralateral homonymous hemianopsia, the patients became agitated and aggressive when they were stimulated by their environment. They responded to solicitations by their relatives or the medical staff by shouting obscenities and hitting and biting others. In contrast, the patients never showed spontaneous violent verbal or physical signs without exposure to external stimuli. Temporary physical restraint and neuroleptics were required in the acute phase of stroke, but this pattern of behavior resolved within 2 weeks after stroke. Because the patients showed a selective and focused aggressive behavior toward their relatives and the medical staff, we suggest that their behavior should be distinguished from confusion or “agitated delirium,” which was reported after strokes in different PCA territories, including the paramedian thalamic territory<sup>15</sup> and unilateral involvement of the fusiform and lingual gyri,<sup>11,12</sup> or after more extensive lesions including bilateral PCA territories.<sup>13,14</sup>

The frequency of aggressive behavior in PCA stroke in our study is lower than in a previous study relying on oral self-reports given by the patients in which 17% of 254 patients with acute strokes in all territories displayed feelings of aggressiveness.<sup>7</sup> It is also lower than in another study where inability to control anger or aggression was reported in 33% of patients with occipital and thalamic stroke.<sup>5</sup> In our study, the strict inclusion of patients with restricted aggressive behavior and exclusion of patients with concomitant confusion or agitated delirium may explain in part the lower frequency of patients with aggressive behavior compared with these previous articles. Additionally, our study was prospectively conducted; it was based on direct clinic observa-

tion, which allows a better description than oral self-reports given retrospectively by the patients.<sup>5-7</sup> Our 3 cases had occipital lesions including lingual and fusiform gyri, and 2 of them had temporal and thalamic involvement. Our study shows that the more extensive the lesions are in the PCA territory, the greater the risk is of becoming confused, agitated, or aggressive. However, no particular region of interest, particularly not the thalamus, could specifically be pointed out as a predictor of aggressiveness. In the third case, an isolated occipital stroke restricted to the fusiform and lingual gyri triggered a paranoid delusion and short-lived aggressive behavior that resolved in less than 24 hours, suggesting not only that isolated occipital stroke may be sufficient to trigger aggressive behavior but also that other factors may contribute, such as old age and other environmental factors that remain to be identified. Traditional knowledge points to the limbic system as being the culprit, not only the orbitofrontal cortex but also the amygdala, which allows the evaluation and interpretation of emotions and represents the final pathway in the generation of aggressive outbursts.<sup>16</sup> Involvement of the amygdala or an interruption of the occipit limbic connections could be suggested in our 3 patients. The latter connections were illustrated in recent experiments in monkeys showing a link between the retrosplenial cortex and the associative visual area (V2)<sup>17</sup> and between the parahippocampal gyri and the prestriated visual area (V4).<sup>18</sup> The involvement of a more diffuse structure such as the serotonergic system<sup>19</sup> in the generation of aggressive outbursts could also explain why such diverse anatomical locations result in a similar behavioral disorder.

The description of our 3 patients further emphasizes the fact that aggressive behavior may occur after isolated PCA stroke with few associated clinical clues other

than hemianopsia. Also, the more extensive the lesion is, the greater the risk of developing an aggressive behavior seems to be. The recognition of this behavior pattern may be of great value in the diagnosis and management of acute PCA stroke.

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