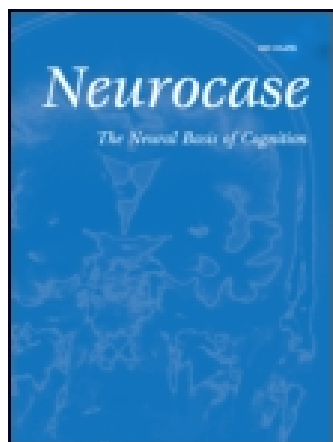


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# Schizophrenia-like psychosis associated with right lacunar thalamic infarct

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Thalamic dysfunction has been associated with schizophrenia and other psychotic disorders. We describe an adult patient with a lacunar infarct in the posterior region of the right thalamus exhibiting a paranoid schizophrenia-like psychosis as the only clinical manifestation. Neuropsychological assessment showed alterations in visuospatial memory and executive functions at follow up. This case highlights the role of information processing by the thalamus in the development of delusions. We suggest that dysfunction of the right mediodorsal and pulvinar thalamic nuclei disrupts both thalamic sensory processing and thalamo-prefrontal circuits mediating belief evaluation, leading to delusional beliefs.

**Keywords:** Thalamus; Schizophrenia; Psychosis; Cerebrovascular disease; Delusions.

Numerous studies have reported thalamic dysfunction and reduced thalamic volume relative to total brain size in patients with schizophrenia, a disorder with prominent psychotic features including delusions (Alelu-Paz & Giménez-Amaya, 2008; Andreasen et al., 1994; Byne et al., 2001; Byne, Hazlett, Buchsbaum, & Kemether, 2009; Csernansky, Schindler, & Splinter, 2004; Kemether et al., 2003; Konick & Friedman, 2001; Mitelman, Byne, Kemether, Hazlett, & Buchsbaum, 2006; Staal, Hulshoff-Pol, Schnack, Van der Schot, & Kahn, 1998). One of the primary functions of the human thalamus is the filtering or gating of sensory input. Abnormalities in thalamic circuits that mediate attention and sensory processing

may explain some of the diverse symptoms of schizophrenia (Andreasen et al., 1994), such as the development and expression of delusions. We present the case of a patient with a thalamic vascular lesion exhibiting a paranoid schizophrenia-like psychotic disorder in addition to visual memory deficits.

## CASE REPORT

A 37-year-old right-handed woman was admitted to the neuropsychiatric service with paranoid delusions and irritability, and given a provisional diagnosis of paranoid schizophrenia. She was separated

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from her husband, and until recently held a stressful administrative position in another country. The patient had been well until 4 years earlier, when she began to exhibit behavioral changes and paranoid delusions. She said that there was a government conspiracy against her and that they were watching her all the time through cables, electronic devices, and even through small ornamental figures. She also started to take baths in complete darkness for fear of being spied on. She became aggressive and mistrustful, and so was dismissed from work and left by her husband. She returned from the foreign country to her family and agreed to be checked by a neurologist because of mild headaches. The neurologist found her psychotic and she was referred to our psychiatry department.

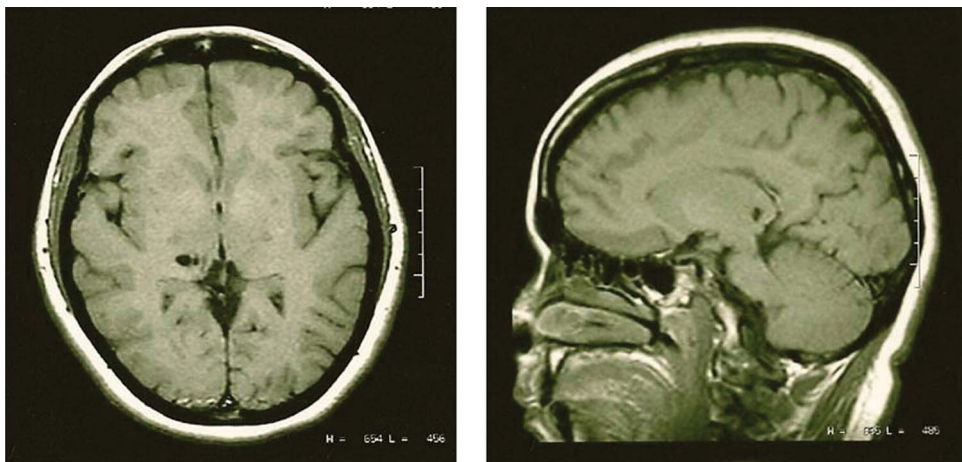
Her medical history was unremarkable. She had never been pregnant. She had smoked since age 18 but did not drink alcohol excessively. She had family antecedents on her mother's side with cerebral vascular strokes. Her mother had hypertension and dyslipidemia and her father had diabetes. She was polyglot and held a postgraduate degree; until the onset of this psychiatric disorder, she was considered to have excellent interpersonal skills.

On admission, she was suspicious and anxious, and expressed delusions of persecution, harm, and reference, including the belief that the government was following her, that there was a conspiracy preventing her from getting a job, and that the media was talking about her. Judgment and reality testing were impaired and she refused to be hospitalized. There was no evidence of auditory or visual hallucinations. She made corrections and gave recommendations to the forms that she was asked to

sign based on her knowledge of human rights and law. After reviewing the documents, she agreed to be hospitalized. Physical and neurological examinations were normal. Laboratory tests, including tests for renal and hepatic function, a complete blood cell count, coagulation tests, and blood levels of electrolytes, lipids, thyroid hormones, and cortisol were all normal. An electroencephalogram was normal, and no vascular dysfunction was found by intracranial and extracranial Doppler ultrasound.

A brain MRI showed a lacunar infarct on the posterior region of the right thalamus (Figure 1). The following causes of cerebrovascular disease were excluded: ischemic stroke resulting from large-artery atherosclerotic disease, small vessel or penetrating artery disease, or cardioembolism, and ischemic stroke resulting from hemodynamic mechanisms, non-atherosclerotic vasculopathy, or hypercoagulable disorders. The following risk factors were also excluded: hypertension, diabetes mellitus, hyperlipidemia, excessive alcohol use, obesity, and physical inactivity. The only documented risk factors were cigarette smoking and family history of stroke, hyperlipidemia, and diabetes mellitus. The final neurological diagnosis was ischemic stroke of undetermined etiology. Despite extensive evaluation, as many as one-third of ischemic strokes cannot be explained, especially in patients younger than 45 years old. Cardioembolic or hematological events that cannot be demonstrated at the time of evaluation may be the underlying cause (Billir, 2002).

She received risperidone at 2 mg/day and was discharged 1 week later with complete resolution of psychotic symptoms. On follow up, risperidone was



**Figure 1.** MRI study in a T-1 sequence, showing a lacunar infarct of the right posterior thalamus, in axial and sagittal projections.

reduced to 0.5 mg because of side effects (tremor and rigidity). Months later, the patient returned to work and has remained asymptomatic under treatment with risperidone.

At 1 year follow up, neuropsychological assessment revealed a total IQ of 91. Global memory was preserved (Wechsler Memory Scale). Deficits were found in visuospatial abilities, including visual memory (on the Rey-Osterrieth Complex Figure Test), and in planning and executive functions (as revealed by the Clox Executive Drawing Task and the Trail Making Test). The patient demonstrated partial functional recovery and began to work again, although she has not achieved her previous working status.

## DISCUSSION

Attention and information processing deficits stemming from thalamic dysfunction may lead to delusions in psychotic patients (Andreasen et al., 1994). Indeed, this hypothesis is supported by several studies showing reduced thalamic volume in patients with schizophrenia (Alelú-Paz & Giménez-Amaya, 2008; Andreasen et al., 1994; Byne et al., 2001, 2009; Csernansky et al., 2004; Kemether et al., 2003; Konick & Friedman, 2001; Mitelman et al., 2006; Staal et al., 1998). However, not all studies have documented gross thalamic abnormalities (Lesch & Bogerts, 1984); rather, the majority point to specific changes in signaling between mediodorsal, anterior, and pulvinar thalamic nuclei (Byne et al., 2001, 2002; Danos et al., 2005; Kemether et al., 2003; Mitelman et al., 2006; Qiu, Zhong, Graham, Chia, & Sim, 2009).

One of the most consistent findings in the thalamus of schizophrenic patients at autopsy is reduced volume and cell number in the mediodorsal nucleus (MDN), but these findings have not been replicated in all studies (Cullen et al., 2003; Dorph-Petersen, Pierrri, Sun, Sampson, & Lewis, 2004). In fact, the largest postmortem study (Cullen et al., 2003) found no difference in the MDN volume, the number of MDN neurons, or the size of individual neurons. Alternatively, a large scale MRI study by Kemether et al. (2003) did find significantly smaller MDNs in patients with schizophrenia. These conflicting results have been assessed by Danos et al. (2005), who also found a moderate volume reduction in the left MDN, but not in neuron density or the total number of neurons. The negative results reported by Cullen et al. (2003) may be due to

the age of the subjects (mean of 68 years), as age-related shrinkage may have obscured a disease-associated reduction in MDN volume. In addition to these changes in the MDN, reduced volume, disrupted regional cerebral blood flow, and fewer neurons have been reported in the pulvinar nucleus of schizophrenia patients (Byne et al., 2002; Danos et al., 2005; Heckers et al., 2000; Kemether et al., 2003).

The MDN is one of the largest association nuclei of the thalamus and is extensively connected to the dorsolateral prefrontal cortex and other regions implicated in schizophrenia (Byne et al., 2002); therefore, alterations in this thalamic nucleus could disrupt both cortico-subcortical and -cortical communication (Alelú-Paz & Giménez-Amaya, 2008). The pulvinar nucleus is the largest division of the human thalamus, and although more studies have examined changes in the MDN of schizophrenic patients, decreased size and reduced cell numbers in the pulvinar have been more consistently replicated. The pulvinar is important in visual and possibly auditory attention and it has prominent interconnections with the temporal lobe (Goldman-Rakic & Porrino, 1985; Grieve, Acuna, & Cudeiro, 2000; Kemether et al., 2003; Romanski, Giguere, Bates, & Goldman-Rakic, 1997). A study comparing 27 schizophrenia patients with 28 control subjects found that only the right pulvinar was smaller in the schizophrenia patients and had fewer neurons (Highley, Walker, Crow, Esiri, & Harrison, 2003). This deficit in the right pulvinar nucleus may lead to diminished neuropsychological performance and to changes in subjective experience since recognition of novel objects is associated with unilateral activation of the right pulvinar region (Heckers et al., 2000). In addition, an MRI study (Gilbert et al., 2001) localized the thalamic volume deficit in schizophrenia to the right posterior medial thalamic subdivision, a region including the right medial half of the pulvinar, and consistent with restricted loss of function to the right hemisphere, particularly to the temporal lobe regions associated with altered visual perception (Gilbert et al., 2001). Postmortem studies have also reported a reduction in the volume and neuron number of the right medial pulvinar nucleus (Byne et al., 2002, 2007).

Alterations in the MDN and pulvinar nuclei could affect the gating of sensory information, and thus the subjective experiences of the patient, in addition to information processing through thalamo-prefrontal circuits that constitute the belief evaluation system. Indeed, both altered

**TABLE 1**  
Case reports of psychosis associated with thalamic infarction

References	Cases	Localization	Symptoms	Comments
Guard et al. (1986)	68-year-old man	Right thalamic hemorrhage in pulvinar and DM	Anxiety-depression. Paranoid psychosis. Depersonalization	Frontal lobe (medial) syndrome developed
McGilchrist et al. (1993)	43-year-old man	Bilateral thalamic infarcts	Affective psychosis	SPECT: Bilateral frontal hypoperfusion
Yoshida et al. (2006)	73-year-old woman	Left DM	Frontal lobe syndrome Visual memory Auditory Hallucinations	Complete remission of psychosis (risperidone + Olanzapine) Frontal lobe symptoms developed
Arikan et al. (2009)	33-year-old man	Lacunar infarcts on posterior region left thalamus (DP, DL, and pulvinar)	Anterograde memory deficits Irritability, apathetic mood, persecutory delusions and hallucinations Visuospatial perception deficit	Symptoms resolution with risperidone and valproate

MD, mediodorsal; DP, dorsal posterior; DL, dorsal lateral.

sensory experience and aberrant belief evaluation are implicated in the two factor theory of delusions (Coltheart, Langdon, & McKay, 2007).

In the case presented, lacunar infarct was located in the posterior region of the right thalamus and mainly affected the pulvinar nucleus. Four other cases of psychosis associated with thalamic infarction have been reported (Arikan, Kütükcü, Karay, & Ozmen, 2009; Guard et al., 1986; McGilchrist, Goldstein, Jadresic, & Fenwick, 1993; Yoshida, Abe, & Yoshizawa, 2006) (see Table 1). Complete resolution of psychotic symptoms was achieved in three of these cases, but a frontal-like syndrome was observed at follow up.

The present case supports the role of thalamic dysfunction in altering subjective experience and disrupting the belief evaluation system of thalamo-prefrontal circuits. These two deficits could allow for the formation of delusions in the schizophrenic brain. Most cases, including ours, showed a favorable response to antipsychotics.

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