

Letter to the Editor (Case report)

A case of psychosis associated with left thalamic lacunar infarcts

1. Introduction

Kraepelin et al. (1919) had assumed that manifestations of schizophrenia could be accounted for abnormalities in some of the brain areas. Latest studies have insisted on the thalamus being a substrate for defects in information processing in patients with schizophrenia (Sim et al., 2006).

The thalamus has a crucial anatomic position in the brain. It is comprised of multiple relay and diffuse projection nuclei (Kandel et al., 1991) receives input and output from cortical and brain stem regions. Conventionally, it has been considered to play a major role in “gating” or “filtering” information.

Patients with schizophrenia complain of being bombarded with stimuli, which they have difficulty screening out. This experience leads to an “input overload” leading difficulty in distinguishing between the “self” and the “not self.” Thus a problem with filtering stimuli and determining ego boundaries could lead to positive symptoms as well as negative symptoms (McGhie and Chapman, 1961).

To our knowledge, there has been only one reported case with schizophrenia like symptoms related with thalamic event (McGilchrist et al., 1993).

2. Case report

A 33 year-old right-handed man was referred to our department with the complaints of irritability and distress. Six years ago, he suddenly felt numbness on right side of his body, which gradually resolved in a few days. Afterwards he had difficulty in speaking, learning and concentrating. During the following months, he had progressively presented some auditory hallucinations and delusions of persecution. He had been prescribed some atypical antipsychotics for the last 4 years, but his drug compliance was poor.

On admission he was difficult to communicate, verbal content and fluency were decreased. His affect was apathetic, mood was irritable. He had auditory hallucinations and persecutory delusions. Judgment was poor, insight was partial. Medical/psychiatric personal and family history were unremarkable.

Routine investigations including full blood count, serum biochemical profile with fasting serum glucose, lipids and syphilis serology, were normal or negative. He was normotensive, nicotine and/or alcohol consumption have not been reported.

Physical and neurological examinations were normal. Echocardiogram and Carotid and Vertebral Doppler Ultrasoundography were normal. EEG revealed asymmetrical alpha activity in right posterior and temporo-posterior regions of the brain with no epileptic activity. On neuroimaging assessments which were performed after hospitalization, brain MR scan showed 2 mm lacunar infarcts on posterior region of left thalamus, while brain SPECT scan showed cerebral perfusion normal bilaterally (Fig. 1).

Neuropsychological assessment revealed a verbal IQ of 105, performance IQ of 69 and total IQ of 89 (Lezak, 1983a). Visiospatial perceptual priming defect was detected in Benton's Orientation Test (Benton et al., 1975). Frontal tests like Stroop Test (Jensen and Rohrer, 1966) were normal excluding Verbal Fluency Test (Green, 2000). Neuropsychological tests revealed deficits in IQ, linguistic performance and mostly in visiospatial perception skills.

Risperidone 4 mg/day was introduced. He presented with pressured disorganized speech, irritability, agitation, hallucinations and delusions on 2nd weeks of hospitalization which was assessed as a psychotic episode with affective symptoms. Na Valproate 1000 mg/day introduced, risperidone increased to 6 mg/day, his symptoms resolved in 2 weeks.

3. Discussion

Quantitative studies have revealed significant reductions in the total volume of the thalamic complex in subjects with schizophrenia (Sim et al., 2006). Some studies of thalamic nuclei suggest specific involvement of particularly the anterior nucleus, mediodorsal nucleus, and pulvinar (Danos et al., 2005). Evidence of quantitative involvement of thalamus in schizophrenics might clarify why schizophrenia like symptoms observed in the patients following a thalamic event.

A single case of affective psychosis was reported by McGilchrist and his colleagues (1993). However atypical psychiatric syndromes have also been reported following thalamic infarction. All of the patients reported had been survived but behavioural abnormalities had persisted for months after recovery of other neurological signs (Chung et al., 1996).

Case studies have described a higher prevalence of patent foramen ovale PFO in young stroke patients than in the controls (Jones et al., 1983). Cardiologic evaluations, Transthoracic Echocardiogram of our case were normal. Since Transcranial Doppler or Transesophageal Echocardiogram which were required methods for the definitive diagnosis, we couldn't comment on the possibility of presence of PFO.

In our case, lacunar infarcts were located in posterior region of thalamus, where dorsal lateral, dorsal posterior and pulvinar nuclei are found. Studies on individual thalamic nuclei have indicated reduced volume of the pulvinar in schizophrenia (Danos et al., 2005). Posner and his colleagues reported that pulvinar nucleus was involved in detailing targeted object (Sadock and Sadock, 2007), supporting visiospatial perception defect found in our case, especially in Benton's Judgment of Line Orientation Test (Benton et al., 1975).

In the largest series of thalamic lacunar infarcts reported in the literature, no patient presented a concurrent psychotic episode (Arboix et al., 2005). Therefore it should be added that psychotic disorders related to thalamic lacunar infarctions are exceptional, and it is difficult to establish a direct relationship between both conditions. However, since it's been well-known that clinically silent lacunar infarctions may occur (Vermeer et al., 2003) it might be hypothesized that there might

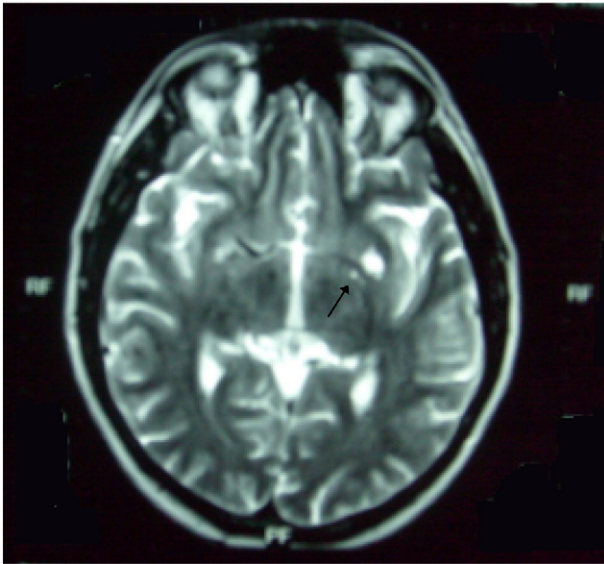


Fig. 1. a,b. A brain MR scans of the patient showed 2 mm hyperintense lacunar infarcts on posterior region of left thalamus, the hyperintensity superolateral to lacunar infarcts more prominent at the right one, were evaluated as a perivascular space. a. T2 weighted cranial MR scan in coronal section b. T2 weighted brain MR scan in transverse section showed 2 millimetric hyperintense lacunar infarcts on posterior region of left thalamus, the hyperintensity superolateral to lacunar infarcts were evaluated as a perivascular space.

be a subgroup of patients with psychotic disorders, where a relationship with a higher frequency of silent thalamic lacunes might exist.

4. Conclusion

There has been only one case report of affective psychosis associated with thalamic infarction. (McGilchrist et al., 1993). We presented a case whose verbal deficits, behavioral changes and psychotic symptoms emerged after transient right hemihypoesthesia, and responded well to an atypical antipsychotic and a mood stabilizer combination. His symptoms might have been correlated with the localization of lacunar infarcts, possibly to the pulvinar nucleus. An organic etiology should be suspected in patients with atypical symptoms, and the value of a careful history of the complaints should not be underestimated.

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27 January 2009