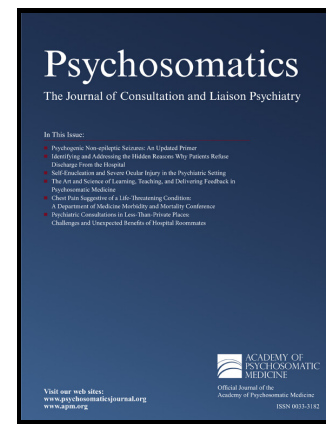


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Onset of psychosis following strokes to the cerebellum and thalamus

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Title: Onset of psychosis following strokes to the cerebellum and thalamus

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Clinical studies have suggested that the cerebello–thalamo–cortical circuit could be one of the neural networks contributing to the pathogenesis of psychosis.¹⁻⁵ However, current reports still remain limited regarding the onset of psychosis in association with structural lesions over both cerebellum and thalamus. Hereon we present the case who developed a new-onset psychosis after strokes to the left cerebellum and bilateral thalamus.

Ms. A was a 67-year-old woman without previous psychiatric or substance use history. She was physically healthy, except for the presence of untreated hypertension for approximately 10 years. When Ms. A was 67 years old, she was sent to the emergency department at another hospital because of acute consciousness change. She was subsequently admitted to the neurological ward after receiving a diagnosis of a hemorrhage over the left cerebellar hemisphere. Her consciousness gradually improved within the first two weeks of hospitalization.

Six weeks after the left cerebellar hemorrhage stroke, Ms. A was admitted to psychiatric ward at this hospital because of the persecutory delusions and irritable mood. Neurological examinations revealed dysarthria, scanning speech, and ataxia with a left-deviated gait. The Mini-Mental State Examination score was 26 of 30 (cutoff point: 24). A brain computed tomography scan revealed compatible findings with the history of hemorrhage over the left cerebellar hemisphere (Fig. 1A). In addition, the scan showed multiple small hypodense lesions suggestive of ischemic pathologies over the bilateral thalamus (Fig. 1B). Awake electroencephalogram and carotid and cardiac sonography were unremarkable. Blood tests revealed significantly elevated levels of triglyceride (250 mg per dL) and cholesterol (268 mg per dL). Her

medications for hypertension and hyperlipidemia are amlodipine 5 mg per day, valsartan 160 mg per day, and atorvastatin 20 mg per day.

After 2 weeks of treatment with haloperidol 2.5 mg per day, the persecutory delusions and irritable mood were gradually alleviated. Because of the occurrence of rigidity over the bilateral extremities, we administered aripiprazole 5 mg per day to replace haloperidol. Ms. A's psychotic symptoms were not exacerbated in the subsequent 2 months. Furthermore, she could independently take care of herself at home.

Emerging neuroimage literatures indicate that the cerebrocerebellar connectivity that accounts for psychosis may involve several brain regions including anterior cingulate, anterior hippocampus, superior temporal gyrus, and thalamus.¹⁻⁵ Therefore, cerebello–thalamo–cortical circuit is proposed as the possible neural network to explain the onset of psychosis. The present case is informative because it is possibly the first report demonstrating an onset of psychosis following strokes to both cerebellum and thalamus. The observations from this case provided further evidence supporting the hypothesis that lesions in cerebello–thalamo–cortical circuits could be associated with the onset of psychosis.

The patient in the present case was characterized by no previous psychiatric or family history. The prominent psychotic symptoms were noted for the first time in her life at age 67 following strokes to cerebellum and thalamus. In particular, her response to antipsychotics treatment was rapid, and recovery of function was favorable. These observations are therefore consistent with the findings of previous reports supporting the diagnosis of psychotic disorder due to stroke.⁵ However, we followed up this patient for only 2 months. Consequently, continuous monitoring of the psychotic symptoms and cognitive function was required before reaching the final diagnosis in this case.

DISCLOSURE STATEMENT

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

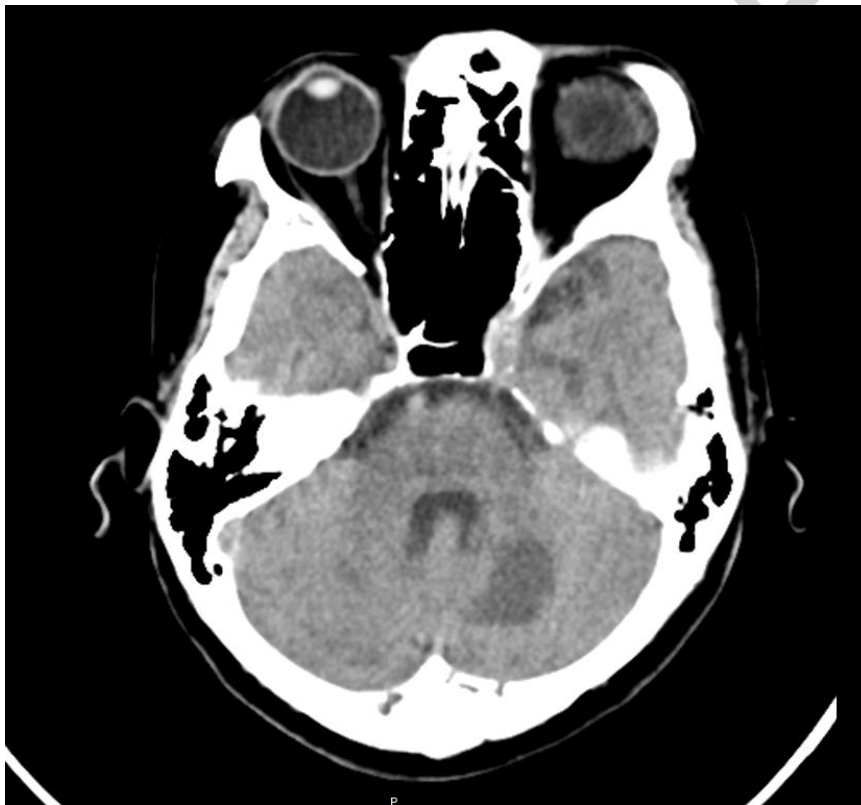
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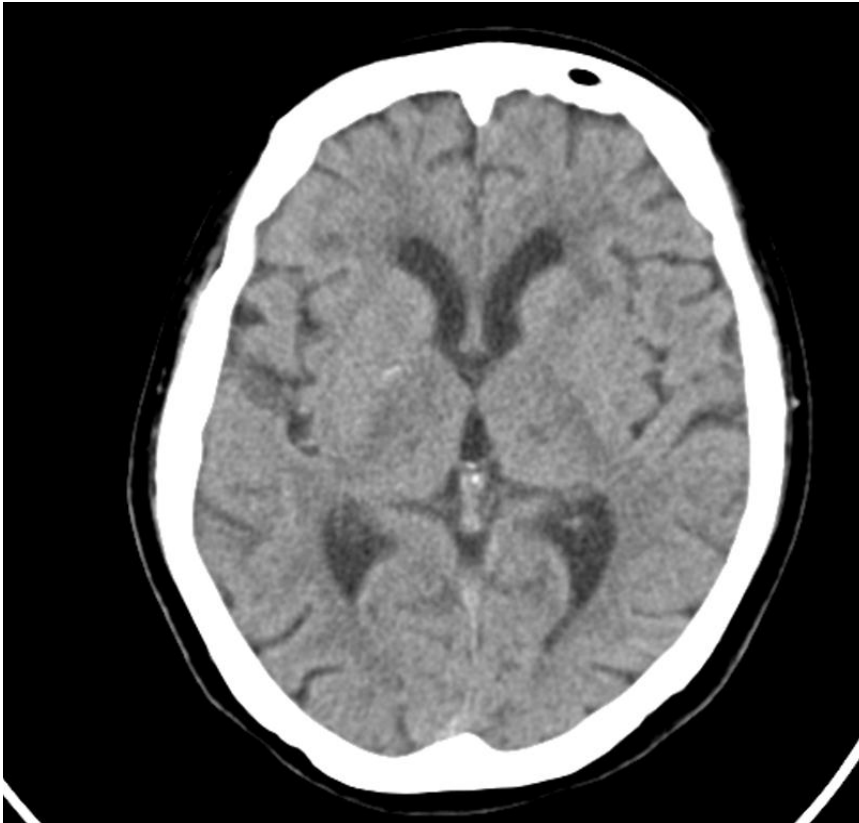
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FIGURE CAPTIONS

Figure 1. Brain computed tomography scan results.

The brain computed tomography scan revealed (A) one hypodensity lesion over left cerebellum hemisphere compatible with the history of left cerebellar stroke, and (B) multiple small hypodensity lesions suggestive of ischemic pathologies over bilateral thalamus.

FIGURES



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